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The Neuroendocrine Reactivity to Social Defeat: Threat/Challenge Appraisals and Socio-Economic Status

By

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Abstract

Extensive evidence demonstrates that lower socio-economic status (SES) is associated with poorer health and reduced opportunities to fully participate in society. Reasons for this exist within a latticework of socio-cultural, economic, political, and biological influences, in concert with psychological processes. The social gradient in health robustly illustrates how inequalities and social rank predict distribution of disease. Marmot (2004) argues that where people stand in relation to others in society is crucial for an individual's health and well-being. Similarly, Wilkinson and Pickett (2018) suggest the social gradient in health results from social rank and relative position on the social ladder, with subordination linked to limited resources and lack of control, rather than from health behaviours or access to medical care. However, neither of those theoretical positions empirically test specific biopsychosocial mechanisms through which status affects health. By exploring endocrine reactivity in response to an experimental social defeat task, cognitive moderators of this link, and the relationships of key psychosocial factors to endocrine reactivity and thereby health, this thesis advances research of health inequalities. It does so by providing insight into the concrete neuroendocrine mechanisms underpinning the social gradient in health. Although this study does not provide any firm, definitive conclusions about differences in endocrine reactivity and cognitive appraisals of the task between SES groups, it suggests that androgenic and glucocorticoid systems might indeed be involved in the social gradient of health. However, future research exploring those relationships on a larger scale is required. The results further demonstrate that the overall circulating T levels were higher in the high SES compared to low SES group in both competition conditions. Participants also display higher overall levels of circulating C levels on the day of the experiment compared to the baseline day. Moreover, the thesis suggests that testosterone (T) potentially plays an important role in the underpinning neuroendocrine reactivity that affects behavioural implications of social defeat/victory, before situating these within the broader contextual framework of socio-economic disadvantage (SED). Accordingly, whilst the relationship between T reactivity and motivational states was not found to be statistically significant, this thesis argues that public and health policy interventions should take cognisance of the behavioural and biological implications of social defeat

within lower SES groups. Doing so can aid in the minimisation of those consequences, and harvest positive health and behavioural outcomes which in turn respond to health inequality.

Chapter 1

Preamble

1. Introduction

This thesis is an interdisciplinary study of endocrine reactivity in response to an experimental social defeat task. Moreover, the thesis explores the role of cognitive moderators (threat/challenge appraisals) in the relationship between endocrine response to status competition outcome. The relationship of other psychosocial factors (i.e., sense of coherence, sense of control, personality, and mood/trait affect) to endocrine reactivity has also been explored. This approach has been taken due to the aforementioned psychosocial factors' purported links to physiological responses as damaging or protective factors (Steptoe & Marmot, 2002; Taylor & Seeman, 1999). In seeking to support previous literature which provides evidence of the existing links between psychosocial factors and physiological responses (Steptoe & Marmot, 2002), this thesis examines potential relationships between the above-mentioned psychological variables and neuroendocrine response to status outcome.

By comparing the long-term unemployed with those in high level employment the research advances health inequalities research by endeavouring to explain some of the concrete neuroendocrine mechanisms underpinning the social gradient in health. The implications of social defeat upon individuals having low status within society, and the extent to which these implications could transfer into behavioural and motivational drives for the maintenance of the transgenerational cycle of SED are also examined. This thesis brings insights of how re-thinking and re-addressing health inequalities research could contribute to higher succession of health and social public policies.

The chapter commences with an overview of the central literature topics of concern for this thesis, proceeds with the implications of health inequities research for policy

making, followed by the introduction of the study rational, aims and objectives, and research questions. Finally, the chapter finishes with an outline of the thesis structure.

1.1 Poverty

The WHO states that 'the world's most ruthless killer and the greatest cause of suffering on earth is extreme poverty' (1995), causing significant adversity to human health and well-being. As a multifaceted phenomenon, poverty has many dimensions: poor health, lack of control over resources and inability to satisfy basic needs. Extreme poverty contributes to enduring, sustained rates of infant and child mortality in various areas (UNICEF, 2020). Infants and children in poverty also suffer malnourishment and lack sanitary conditions, shelter, and education (UN, 2007). Deprived financial income also results in a large percentage of illiteracy (37% of the world's population), shorter life expectancies (e.g., 28 years less for the average African man compared to the European), and poorer quality of life (Klugman, 2009). The effects of poverty on one's mental and physical health, and well-being, then, appear detrimental.

The deleterious effects of poverty on human health are not only limited to developing nations but are a universal problem affecting even the largest, wealthiest and most powerful nations (Marmot et al., 2020). Despite the modernisation of contemporary societies, poverty-related diseases are still associated with economically disadvantaged groups in developed countries such as the US, UK and Australia (Forster et al., 2020, Marmot, 2004). The difference between those exposed to financial deprivation in modern or developing societies however, lies in the type of poverty experienced (i.e., relative versus absolute) and its implications for health. Thus, ill-health within Western industrialised societies has been predominantly associated with the experiences of relative deprivation and not absolute poverty. Absolute poverty does not fully account for ill-health experienced by the most impoverished groups within the most affluent countries (Wilkinson & Pickett, 2010; Marmot, 2004). The most deprived groups within developed nations no longer suffer the diseases commonly associated with absolute deprivation such as malaria, dysentery or starvation. Indeed,

they experience rather different socioeconomic causes of ill-health such as chronic exposure to stress, violence, lower educational quality, pollutants, and neighbourhood deprivation (Evans, 2004). These determinants are linked to higher risks of obesity, diabetes, cancer, cardiovascular diseases, dental problems, physical inactivity, asthma and mental disabilities (Miller & Chen, 2013). Consequently, albeit the implications of absolute deprivation upon health are immensely deleterious for those unable to meet basic needs, the consequences of relative deprivation appear to be no better, yet for different reasons.

Even with the absence of absolute poverty, people from lower socioeconomic groups still experience shorter life-expectancies, higher mortality rates and have poorer quality of life compared to those of higher SES (Kim et al., 2018). Western industrialised societies face a different problem - the issue of relative deprivation or relative poverty (Marmot, 2004; Wilkinson, 1997). Hence, as Marmot and colleagues (2020) suggest, what is embedded at the heart of unhealthy but no longer economically poor societies is not the issue of absolute poverty but the problem of social inequalities and relative deprivation.

Socioeconomic inequalities generate a range of health inequalities within developed countries. More specifically, they lead to a social gradient in health observed even in the most egalitarian and socioeconomically equal societies (Benach et al., 2003; Cavelaars et al., 1998a, 1998b). Furthermore, Marmot (1978, 1991) demonstrates in his research of the British Civil Service that the effects of socioeconomic circumstances are not confined to the most disadvantaged groups. Rather, a social gradient in health runs right through society, so that even among middle-class office workers, lower ranking staff suffer higher morbidity, earlier death, and poorer health than higher ranking staff (Marmot et al., 1978; Marmot & Smith, 1991; Marmot, 2004). Health gradients thus emerge not only in conditions of absolute poverty but in affluence and comfort.

1.2 Inequality

Egalitarians' motivation to achieve equality appears to be embedded in the foundations of human morality (Warburton, 1999). The desire for equality might then be deeply rooted in our religious beliefs, emerge from ethical theories such as Kantian's theoretical belief of equality of respect for all people, or could be based on the utilitarian idea that one should treat others equally in order to attain happiness. The debate around to what extent equality is achievable remains unsettled, however. This does not emanate from contra-egalitarian arguments suggesting complete uniformity is never attainable due to individual differences such as intelligence or genes. Rather, it is because certain facets of life such as equal distribution of money, employment opportunities and political power are not easily achievable in the context of modern stratified societies.

This further leads to discussions of whether the equal distribution of money is the most judicious method to minimise adversity and amplify happiness and wellbeing. However, this question is not settled and the concept of equal distribution of money is often seen as impractical and short-lived (Warburton, 1999). Importantly, when considering the model of equal distribution of money, one should also acknowledge that different individuals have different needs. Whilst proponents of the egalitarian view believe that moderate differences in income between individuals are only acceptable when those variances correspond to disparities in necessities, realistically, in order to survive one might need more financial resources than others. For example, individuals who require daily expensive medical or social care are highly unlikely to have long life expectancies and good quality of life in an egalitarian society, equally distributing the material shares across individuals. Even if it were possible to evenly distribute resources within societies, equality would remain short-lived and status hierarchies would emerge as a result of individual differences in spending strategies and needs. This, however, does not mean that as a society we should not strive for equality, but rather illustrates the complexity and multifaceted nature of disparities, and the need for multidimensional approaches and strategies to tackle those.

Nevertheless, the significant concerns here remain why attaining equality matters so much and the consequences of living in stratified groups. Living in a hierarchical society has resemblances to living in a pecking order system in the animal kingdom; some animals have more resources and power than others based on their social rank in the hierarchy. This will have inevitable effects on mortality rates, quality of life and morbidity (Sapolsky, 1999b; 2001; 2004). This is also observed within human structures, with lower socioeconomic groups experiencing higher mortality, morbidity rates and lower quality of life. Socioeconomic inequalities are unavoidably linked to health inequalities (Marmot, 2004), with the least egalitarian societies experiencing the highest levels of health disparities (Wilkinson and Pickett, 2010; 2018).

Marmot (2004) suggests that in order to understand how social status/position directly affects health, the implications of social rank should be considered through the lens of status syndrome. He further argues that the principal mechanism affecting biological effects of relative deprivation upon physical or mental health and well-being is the psychological experience of socioeconomic disadvantage (SED) rather than disadvantage per se. This suggest that it is not solely the lack of economic power that adversely impacts health, but rather the effect of the status gap between individuals that leads to this relative inequality.

1.3 Status

Status is associated with survival and reproductive success (Cluntton-Brock, 1998; Dewsburry, 1982; Ellis, 1995) and commonly defined as "a priority of access to resources in competitive situations" (Cummins, 2006, p.677). However, in environments where status is unstable reproductive success is directly linked to the duration of time during which the individual holds high rank (Altmann et al., 1995), demonstrating that the link between status and reproductive success is not one-directional. Inclusive fitness or "the reproductive success of individuals and their closely related kin" also robustly impacts social rank (Cummins, 2006, p.678). Although status affects every life domain and the outcome of competitive situations, additional factors such as inclusive fitness and settings also define social rank. Hence,

when exploring the implications of status rank upon the individual's survival and reproductive outcomes, one should not only consider the wide variety of factors influencing status rank, but also the inversed relationship where reproductive success determines social status.

Differences in status stem from levels of power, dominance, skills and influence result in the natural emergence of status hierarchies (Anderson et al., 2001; Berger et al., 1980; Cheng et al., 2013; Gould, 2002; Magee & Galinsky, 2008; Sidanius & Pratto, 1999). The effect of social hierarchies is responsible for allocation of limited resources such as food, territory and mates (Sapolsky, 2005), amplified individual status drive (Halevy et al., 2011) and impacts upon social learning (Henrich & Mcelreath, 2003). The tendency to organise into social hierarchies is not unique to humans and has been documented in almost every group-living species including apes, baboons, wolves, hens, ants and fish (Sapolsky, 2004, 2005, 2017).

Sexual selection theory (Darwin, 1859) posits that status-seeking behaviours are exhibited by most species, prompting organisms to covet increased numbers of surviving offspring (Koski et al., 2015). This in turn creates dominance hierarchies, within which status-related behaviours vary extensively due to evolutionary adaption to a great variety of environmental niches. Accordingly, some status-seeking behaviours enhance reproductive chances, whilst others aid individual's offspring survival rates (Von Rueden et al., 2011). In the animal kingdom, species of a greater physical size are usually deemed to hold higher rank than others (Ellis, 1995), however size just partially contributes to the attainment of a higher social position. More importantly, a collection of specific cognitive functions is required for the achievement of dominance, meaning that "selection favours those who have social and political intelligence" (Cummins, 2006; 682). In other words, in order to attain status within a hierarchy, individuals must; construct influential alliances founded on reciprocal commitments, predict and impact the behaviour of other individuals, and learn the implied principals of social groups that constrain behaviour and adhere to them. In human hierarchies relative social positioning depends on whether the individual is perceived as someone who corresponds to societal ideologies and goals.

Humans possess cognitive structures allowing them to promptly appraise status information and distinguish relative status roles in social groups (Cummins, 2006). Research on the underpinning mechanisms of status perception and sensitivity remains precarious and vague however, with no established results of the sensitivity of individual's perception of status. Besides, even without the abstract conceptualization of status, organisms still exhibit status-seeking behaviours and form social dominance hierarchies, driven by embedded neurochemical and androgenic mechanisms, including serotonin and testosterone responses (Cummins, 2006). This results in some individuals climbing higher on the social ladder than others. Individuals near the top are those who have greater access to – and control over - resources, more power and more influence compared to their lower-ranked contemporaries (Fragale et al., 2011; Mazur, 1985; Zitek & Tiedens, 2012).

This directly translates into an individual's morbidity, mortality, health outcomes and quality of life (Sapolsky, 2005). Dominant animals are less susceptible to immune, cardiovascular and reproductive dysfunction compared to their socially subordinate contemporaries for example (Cameron, 1997; Cohen et al.,1997; Kaplan & Manuck, 2004). In both the animal kingdom and within human hierarchies, status undoubtedly has implications for health and well-being of the individual, and will be explored later in this chapter.

1.3 Health Implications of Social Rank

The status gradient in health is observed in all cultures and nations as a result of naturally occurring status hierarchies and society's inability to achieve equality (Sapolsky, 2017). Some countries, however, have a significantly greater health gap than others; these often happen to be the most economic and socially unequal nations on earth (Marmot, 2004; 2015; Wilkinson & Pickett, 2010). Nevertheless, the effects of the health gradient are not only limited to the most unequal societies but are also found in countries with smaller degrees of economic and social inequalities such as the Nordic nations and Japan (Marmot, 2004; Popham et al., 2013). The presence of a health

gradient even in the world's most equitable nations points to the role of status syndrome and the importance of social rank or health.

This chapter has already outlined the prominence of primate research in better understanding social rank and its implications for individual's mortality and morbidity rates, susceptibility to disease, reproductive success, and survival. The effects, however, are not only confined to the species in the animal kingdom but are also observed in human social structures (Marmot, 2004; Sapolsky, 2017). Thus, when considering status hierarchies within the context of poverty, the experiences of lower socioeconomic groups often resemble those of subordinate species, and associated with multiple daily stressors, unpredictability of the environment, lack of control, and limited material and psychological resources to cope with stressors (Sapolsky, 2004, 2017). Indeed, socioeconomically disadvantaged individuals suffer the same health implications of low rank as subordinate animals, with biological consequences occurring across the whole lifespan. For example, childhood deprivation has been linked to higher risk of immediate and long-term hypothalamic-pituitary-adrenal (HPA) axis dysregulation, resulting in pathophysiology such as poor cognitive function, higher risk of psychiatric disorders and prevalence of chronic medical conditions later in life (Franz et al., 2011; Lupien et al., 2009; McEwen & Gianaros, 2010; Miller & Chen, 2013). In adulthood this is often associated with lower educational levels, higher morbidity rates, premature mortality, mental health diseases, higher rates of unemployment, marital difficulties and higher percentage of healthcare services used amongst these populations (Anda et al., 2010).

Early childhood deprivation is also linked to consistent production of inflammatory markers by the immune cells (Miller & Chen, 2013; Shonkoff et al., 2009), subsequently resulting in greater inflammatory body response. Whilst usually beneficial in the short term due to its function to promote faster injury recovery and provide larger defence against pathogens, in the long run it increases susceptibility to chronic inflammatory illnesses (Danese et al., 2009; Miller et al., 2011). This increased systemic inflammation often persists in adulthood and leads to increased levels of circulating inflammatory biomarkers such as interleukin-6 (IL-6) (Lockwood et al., 2018).

The predominant explanatory framework adopted to understand the implications of social rank for health is stress. The framework explores status disparities in health and the various protective factors and risk associated with these outcomes (Turner & Avison, 2003; Pepper & Nettle, 2017). For example, SED individuals often experience higher levels of stress due to unpredictability of life events, lack of control over material and psychological resources, higher exposure to risk environments, adverse neighbourhoods, negative life outcomes, and overall exposure to daily hassles and psychosocial stressors (Grzywacz et al., 2004; Pepper & Nettle, 2017; Turner & Lloyd, 1999; Senn et al., 2014). This leads to chronic activation of the stress response system, which whilst evolutionary advantageous in the short-term as provoking fight-or-flight survival mechanism, becomes deleterious when activated over the longer term. Prolonged activation results in inhibition of life-supporting systems such as the immune, metabolic and reproductive systems (Gustafsoon et al., 2010; Sapolsky, 2000). Moreover, chronic stress leads to persistently elevated levels of cortisol which detrimentally impact cardiovascular, reproductive, immune and metabolic systems; brain development, architecture and function (Lupien et al., 1998; Lupien et al., 2009; Scientific Council, 2014). Thereby, higher cortisol levels are often associated with higher risk of neurobiological dysfunction, cardiovascular diseases, autoimmune diseases, psychiatric conditions, impaired cognitive function, higher levels of mental illnesses, Type II diabetes, hypertension, gastrointestinal disorders and many other stress-related diseases (Blair & Raver 2012, Blair et al. 2011; Farah & Hackman 2012; Libby & Theroux, 2005; McEwen & McEwen, 2017; Ouellet-Morin et al., 2011a, 2011b; Sapolsky, 2000; Velupillai et al., 2008; Wright et al., 2005).

Subordinate animal species also experience a sluggish activation of the cardiovascular stress response after stress exposure, and deferred recovery after exposure (Sapolsky, 2004). This is closely related to the adverse impacts of SED on brain development, structure and neural function in humans. Thus, when exposed to early childhood disadvantage, individuals often experience toxic stress which subsequently disrupts the brain architecture and hormone systems which are evolutionary adapted to deal with stress in the short-term. However, when exposed to a significant amount of stress

over a prolonged period of time individuals develop stress system dysregulations. This can result in a lower stress threshold or overreactivity, where individuals may perceive events as stressful when in they are not to others. Alternatively, it may lead to a sluggish, close to shut down response to stress in the face of stressors (Loman & Gunnar, 2010; Zhang et al., 2004). Either outcome can potentially result in dysfunctional stress coping mechanisms such as impulsivity, high risk-taking or anxiety when exposed to stress.

Amongst humans, lower SES is not only linked to higher mortality and morbidity rates, but also related to a constellation of behaviours such as impulsivity, risk-taking, aggression, substance misuse, hostility, delinquency, and high rate of teen pregnancies (Haushofer & Fehr, 2014; Johns, 2010; Legleye et al., 2011; Peppers & Nettle, 2017). The prevalence of these issues, and many other health-related behaviours, is heightened amongst individuals from disadvantaged backgrounds who are more likely to engage in unhealthy diets, smoking, reduced physical activity, and alcohol and drug misuse than their more affluent peers (Melotti et al., 2011; Méjean et al., 2013; Mobley et al., 2006; Pecheya & Monsivais, 2016). However as Pepper & Nettle (2017) argue, this cannot solely be explained by Lazarus and Folkman's model of stress physiology (Lazarus & Folkman, 1984). It is true that some of these behaviours can be explained by the financial restrains experienced by people in disadvantage, for example limited financial resources leading to poor diets. However, the model cannot account for financially costly behaviours such as smoking and drug and alcohol consumption (Marmot, 2004, 2010; Pepper & Nettle, 2017). Hence, whilst partially explained by the theoretical frameworks of stress and financial restrain, these behaviours and emotional states appear to be more rationally associated with explanatory models encompassing status attainment and maintenance behaviours which has been linked to the androgen literature regarding circulating testosterone levels. Despite the great impact of testosterone upon behaviour (Mazur & Booth, 1998; Sharp, 2006), very little meaningful work has been conducted on the relationship between androgens and status disparities in health.

1.4 Mechanisms underpinning status disparity

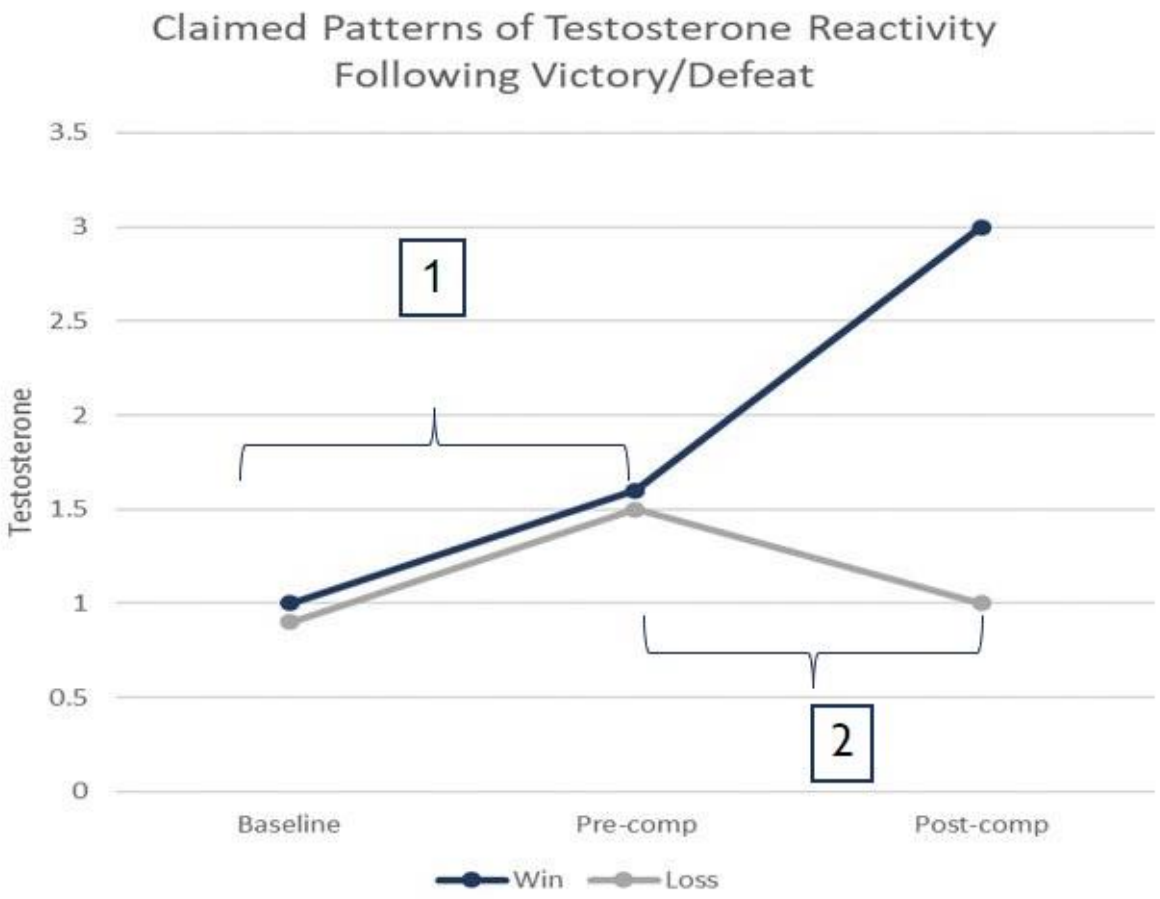
Despite the protracted emphasis on stress as a mediating factor between SES and health disparities, poverty per se does not cause ill health. Rather, aspects of poor health can derive from mechanisms related to stress physiology and associated immune system function. The stress model therefore does not account for the range of poor behaviours associated with SES such as hostility, aggression and risk-taking and which has been coined the "Behavioural Constellation of Deprivation" (Peppers & Nettle, 2017). Indeed, these behaviours and emotional states, predominantly observed in SED groups, are closely linked to the androgen hormone, testosterone. The central role of the hormone is to regulate sex drive, facilitate the adaptive fight-or-flight stress response and to regulate muscle development. It is also associated with status-seeking behaviours and dominance (Mazur & Booth, 1998; Knight & Mehta, 2014), subsequently contributing to the formation and maintenance of status hierarchies (Dugatkin & Druen, 2004; Murray, 2021) Thus, in order to understand the mechanisms underpinning status disparities in health, we first need to look at the context in which these disparities emerge, namely status hierarchies.

As outlined in the 'status' section of this chapter, status hierarchies can be explained though Darwin's sexual selection theory and are innately driven by an individual's status-seeking behaviours observed in all social group living species. More specifically, it is the challenge hypothesis, conceptually originating from Darwinian sexual selection theory that has been suggested to be the biological mechanism underpinning hierarchy formation and status disparity (Murray,2021). The hypothesis outlines the dynamic relationship between testosterone and status-related behaviours (Wingfield et al., 1990, 2000) and the implications for motivational, emotional state and behaviour (Mehta et al., 2008). The fundamental premise of the challenge hypothesis is that testosterone will rise preceding a status competition (Figure 1, phase 1). Following the encounter, the winner's testosterone levels will rise further and the loser's will fall (Figure 1, phase 2). Similarly, this prediction has been derived from Mazur's (1985) biosocial model of status, outlining the same relationship between testosterone and dominant behaviour in primates. These dynamic endocrine changes

have an effect on motivational states and behaviour (Knight & Mehta, 2014; Mehta et al., 2008).

Figure 1

The Challenge Hypothesis - Claimed Patterns of Testosterone Reactivity Following Victory/Defeat



As a means of examining status disparity, evidence for the challenge hypothesis in human males is drawn primarily from studies involving competition (e.g., Carré, 2009; Carré & Mehta, 2011; Knight & Mehta, 2014; Mehta & Josephs, 2006). In the light of these findings, it has been suggested that human males, in a variety of different situations, exhibit a characteristically predictable and stereotypical testosterone response, both prior to and following competition (Carré, 2009; Carré et al., 2013;

Wagels et al., 2018), directly analogous to the challenge hypothesis. Hence, if status-seeking behaviours are embedded in the human nature (Murray, 2021), whilst testosterone's central role appears to regulate these dominance-related behaviours (Sharp, 2006), then arguing that the social gradient in health could be partially explained by the "proximate causes" of it, i.e., androgen reactivity to status challenges, appears reasonable. A detailed explanation and discussion of the challenge hypothesis is provided in the literature review of this thesis (i.e., Chapter 2, p. 111).

In a broader evolutionary psychology context, behaviour is explained by ultimate and proximate causes (Mayr, 1961; Tinbergen, 1963), the purpose of which is to clarify why certain behaviours or traits are exhibited by a specified population in a particular environment, whilst accounting for the consequences of this behaviour or trait in the setting in question (Peppers & Nettle, 2017). Accordingly, the proposed ultimate cause of the social gradient in health is status, resulting in differences in access and control over material and psychological resources; ultimately creating status disparities and subsequent health inequalities (Marmot, 2004; Wilkinson & Pickett, 2018). However, in order to entirely understand the phenomenon "status syndrome", "proximate explanation" is also required. Proximate causes (i.e., specific psychological and biological mechanisms through which social rank impacts health) provide details about how ultimate causes are constructed, in this instance explanatory frameworks relating SES to health outcomes. Ultimate and proximate explanations cannot explain complex behaviours such as "status syndrome" solely; they are integral components and insufficient by itself (Scott-Phillips et al., 2011). Thus, whilst Marmot, Wilkinson and Pickett provide us with the ultimate causes of the phenomenon, the aim of this thesis is to elucidate the proximate causes. Hereafter, the suggested challenge hypothesis (the foundation of status hierarchy formation) and testosterone (the engine regulating status striving and dominance-related behaviours) are the proximate causes, or the biological mechanisms underpinning status disparities in health. These proposed mechanisms complement existing, and long established, biological models that have proven useful in the explanation of health inequalities, namely the theoretical framework of stress. The model alone, however, is insufficient to explain the "behavioural constellation of deprivation".

It is important to note that complex cognitive factors play a key role in moderating endocrine reactivity, particularly testosterone, to status challenges. Blascovich and Mendes (2000) report that individuals who regard events as either challenging or threatening experience differing physiological changes and alterations in general approach/avoidance behaviour. Furthermore, Senn and colleagues (2014) show that individuals from lower socioeconomic groups are often exposed to more daily stressors than those higher up the social ladder, and cope with them less efficiently than their counterparts (Mullainathan & Shafir, 2013). Thus, another factor that might be considered as a proximate cause of the status disparities in health is the cognitive appraisal of status encounter. In the light of these findings, this thesis asks whether low SES individuals appraise events and situations (in this case status challenges) as more threatening than challenging. Implications of those cognitions comprise subsequent impact on endocrine reactivity to stressors, and thereby health, but also consequences for future behaviour (i.e., motivational states) (Blascovich & Mendes, 2000; Mehta & Josephs, 2006).

Finally, proximate causes are rarely acknowledged by policy makers in the fields of public health and social policy. The aim of this thesis will be to endorse the application of proximate causes in policy research by supplementing Marmot, Wilkinson and Pickett's theoretical work on the social gradient of health. Moreover, it aims to encourage discussions on this matter not only amongst evolutionary psychologists and behavioural neuroendocrinologists, but also sociologists, epidemiologists, policymakers and other interdisciplinary partners.

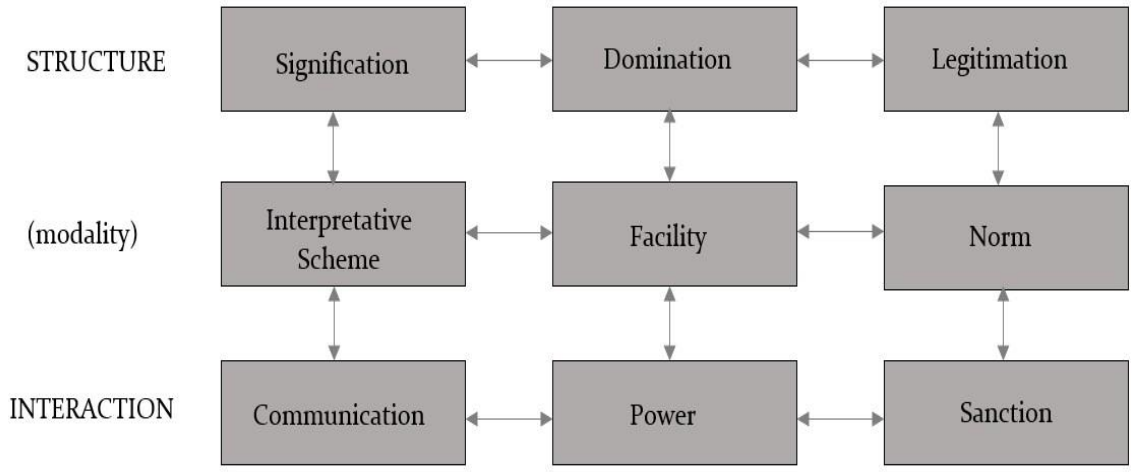
1.5 Social science and health

The relationship between human agency, social structure and health can be understood through multiple lenses or perspectives. A key debate within the existing body of literature concerns the extent to which social structure shapes an individual's behaviour and health. Giddens's (1984) structuration theory, for instance, argues that social life is predominantly agent controlled (Figure 2). In other words, individuals

continuously monitor and control their own behaviour in order to act in line with the behaviour of others and adhere to the norms of the societal structure (Giddens & Pierson, 1988). Similarly, critical realism theory claims that individuals have the critical capacity, creativity and reflexivity to construct social structure, whilst the inverse relationship is also conceivable where the social structure also possess the capacity to shape individuals, actors and agents (Archer, 1996). The commonalities between these two positions construct the foundations of the modern agency-oriented framework of structural inequalities, and their subsequent implications for health in British sociologists' and policymakers' work (Cockerman, 2013). As a result of this agency-oriented doctrine, a comprehensive body of health inequalities literature has been built upon the theoretical framework of structural inequalities (Cockerham, 2013).

Figure 2

Framework of the Structuration Theory



In contrast to this approach, a materialistic explanation of class disparities in health also emerged, most noticeably through the Black Report (Black et al., 1988). Known as the "alternative social causation approach", this model directly attributes health discrepancies to the unequal distribution of material resources (Bartley, 2016; Nettleton, 2006; Nettleton, 2020). Whilst this model emerged as the predominant

method of exploration of health inequalities in early health inequalities research, more recent literature focuses on alternative factors including psychological, behavioural and biomedical (Bartley, 2016). Although some sociological literature explores alternative explanations of health inequalities, the predominant behavioural research approach remains heavily agency-oriented, with a significant number of studies investigating the impact of structure on health in leading British publications (Horlick-Jones, 2011; Macintyre et al., 2002; Williams, 2003), resulting in little research from a bio-psycho-behavioural model.

A social constructionist view of health (Bury, 1986; Nettleton, 2006) argues that "health and illness are produced by subjectively, historically determined human interests and are subject to change and reinterpretation" (Cockerham, 2013, p. 50). In short, the model argues that all things are socially shaped and not identified or discovered (Turner & Wainwright, 2003) and in its most radical form would suggest that all social facts are socially constructed, including biologically objective issues like symptoms of illness (Gabe et al., 2004). Accordingly, adherents to this absolutist form of social constructionism might argue that the experiences of diseases are entirely – or at least largely - defined by cultural values and norms, public opinions and beliefs, and the interaction between individuals. In this sense diagnosis of an illness signifies the conversion of physical symptoms into socially accepted behaviour and results in change of social status (Gabe et al., 2004). The view that everything is socially constructed ultimately leads to a disregard for human agency and nature, and to hold the perception of individuals as blank slates or "tabula rasa" - entirely shaped by social structure (Pinker, 2002). Alternatively, if we reject the "tabula rasa" premise and accept that individual differences exist, we must also accept that those who find themselves in a position of disadvantage do not necessarily deserve the social position they have attained (Pinker, 2002).

The major criticism of the social constructionist model is therefore that it falls short in acknowledging biology and its implications upon our understanding of disease (Cockerham, 2013; Pinker, 2002). Similarly, policymakers struggle to integrate human

agency into health inequalities explanatory models, resulting in such 'straw-man' arguments that argue:

> "since the data in the social sciences are never perfect, and since a conclusion of inequality might be used to the worst ends by bigots or Social Darwinists, shouldn't we err on the side of caution and stick with the null hypothesis that people are identical? Some believe that even if we were *certain* that people differ genetically, we might still want to promulgate the fiction that they are the same, because it is less open to abuse" (Pinker, 2002, p. 151).

Sociologists commonly argue that utilising biological approaches to prove the link between social inequalities and health disparities is unethical, economically unprofitable and challenging, and thus not worth investigating (Bartley, 2016). In the absence of scholarship in this area, one might argue that sociology and social policy frequently struggle to account for the biological differences in humans, despite the significant body of research focused on the biological consequences and antecedents of SED (Bartley, 2016; Haslam et al., 2018). This study addresses this gap by exploring the role of neuroendocrine mechanisms in the multidimensional relationship between socioeconomic inequalities and health disparities, experienced by citizens of Scotland during the second decade of the 21st century.

1.6 Study Rationale

The robust association between social inequalities, and its implication for individual physical and mental health, mortality, and life expectancy is demonstrated by an extensive body of epidemiological literature (e.g., Marmot, 2004; Wilkinson & Pickett, 2010; 2018). Whilst the epidemiological research offers a theoretical explanation for why status disparities in health occur (i.e., the social gradient in health), it does not provide any experimental demonstration of any specific biopsychosocial explanatory mechanisms through which this phenomenon occurs. For this reason, this thesis aims to provide an understanding of some of the concrete neuroendocrine mechanisms underpinning status disparities in health. By applying a bio-cultural theoretical framework the study not only addresses the biological mechanisms underpinning major theoretical positions in research on low SES populations but also major

limitations in the SES/hormone literature. The study also aims to shed light on the argument as to why not all individuals who experience SED also experience the negative health connotations associated with it. For this reason, the study applies the methodological framework of threat/challenge cognitions in order to highlight the importance of moderating psychological factors for the health/status link. The study also seeks to provide further evidence of the proposed established links between psychosocial factors (i.e., sense of control, sense of coherence, personality, and mood/trait affect) and physiological responses, suggested by earlier epidemiological research (Steptoe & Marmot, 2002; Taylor & Seeman, 1999). Finally, the study explores the implications of social defeat upon individuals having low status within society, and the extent to which these implications could transfer into behavioural and motivational drives for the maintenance of the transgenerational cycle of SED. In doing so, the study seeks to promote further knowledge for the existing social and public policies tackling health inequalities.

1.7 Aims and Objectives

The aim of this research is to elucidate the neuro-endocrine mechanisms operating in response to perceptions of challenge and threat in different socioeconomic groups. Specifically, to investigate testosterone (T) and cortisol (C) reactivity over time. The proposed study will apply the challenge hypothesis/Mazur's biopsychosocial model of status (which essentially state there will be a rise in T following a successful status encounter and a fall following defeat in a status encounter) within the context of daily life and investigate hormonal reactivity to everyday stressors and its implication for social status, health, and behaviour. The study focuses on long-term unemployment as a subset of socioeconomic disadvantage. It can be a stressful life event with the potential to affect morbidity and mortality through unpredictability of events and scarcity of financial resources affecting psychological assets such as status, social support and time structure (Hughes et al., 2015; Linn et al., 1985).

The research also explores psychosocial factors (i.e., learned helplessness/sense of control – measure on the Attributional Style Questionnaire, personality – measured on the NEO Five Factor Inventory-3 instrument, sense of coherence – measured on Sense

of Coherence scale, and mood/trait affect – measured on Positive and Negative Affect Schedule-X), previously linked to neuroendocrine responses as damaging or protective factors (Steptoe & Marmot, 2002; Taylor & Seeman, 1999). This has been done in order to examine potential links/relationships between the aforementioned psychosocial variables and neuroendocrine response to status outcome, as previous literature provides evidence for those existing links (e.g., Steptoe & Marmot, 2002). Detailed justification for the consideration of each of the variables will be provided in Chapter 2 (sections: 2.3.4; 2.5.2; 2.5.5; 2.7.2). For this reason, threat/challenge cognitions are explored as potential moderators of the link between endocrine response and competition outcome, whilst the above-listed psychosocial factors are explored as potentially related to endocrine response, as the evidence appears to have reached a consensus that neuroendocrine response is associated with psychosocial factors that are unevenly distributed across social statuses (See Steptoe & Marmot, 2002 for a review of psychosocial factors).

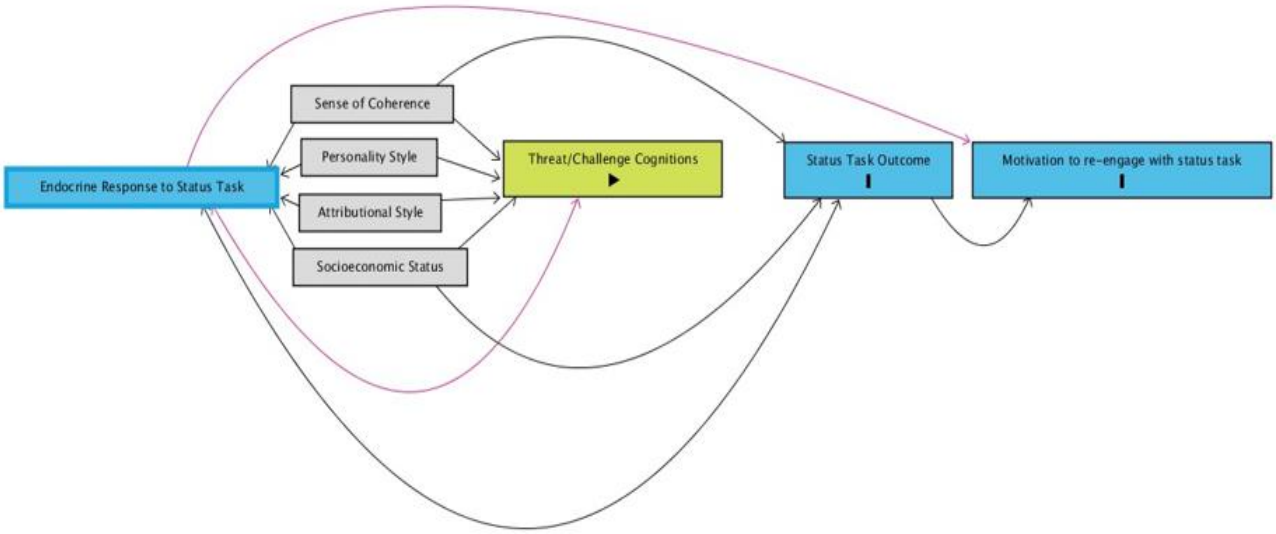
Moreover, acknowledging that endocrine changes to status outcome influence motivational states and future behaviour (Knight & Mehta, 2014; Mehta & Josephs, 2006), the study explores the implications of social defeat upon individuals who have low status within society. In doing so, the study seeks to investigate to what extent these implications could transfer into behavioural and motivational drives for the maintenance of the transgenerational cycle of SED.

Lastly, Scotland displays relatively higher morbidity and mortality rates compared to other European countries. These elevated rates drive much of the rise of health inequalities in the UK more broadly (McCartney et al., 2011). To investigate some of the mechanisms (i.e., neuroendocrine reactivity) and theories (e.g., sense of coherence, sense of control, personality) proposed to account for the disparities in health amongst the Glaswegian population (Marmot, 2004; Walsh et al., 2014), a Scotland-based population has been used.

Figure 3 presents a diagram of the research, incorporating all explored variables and their relationships.

Figure 3

Study diagram



By exploring the chronobiological endocrine reactivity to threat/challenge cognitions within a bio-cultural theoretical framework, several research questions are addressed:

1. Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?

2. Are psychosocial variables related to endocrine reactivity?

3. Does cognitive appraisal of threat/challenge moderate endocrine reactivity?

4. Does T reactivity correlate with a reduced motivation to engage socially?

In an endeavour to understand some of the neuroendocrine mechanisms underlying status disparities in health and to address the research questions and subsequently

emerging hypotheses most efficiently, this research draws on various quantitative methods. Moreover, to best capture the multifaceted nature of SED and its behavioural and biological consequences, both questionnaires and biological parameters are employed.

1.8 Thesis Structure

A central concern of this thesis relates to the implications of social defeat upon individuals attaining low status within human structures, and the extent to which these implications could translate into behavioural and motivational drives for the maintenance of the transgenerational cycle of SED. Consequently, this introductory chapter set out the links between SED, implications of status attainment and maintenance for the individual. It does so whilst acknowledging the different perspectives and theories through which health has been explored within the social sciences.

Chapter 2, the literature review, examines fundamental concerns, frameworks and theoretical models looking at the relationship between social and health inequalities. The chapter proceeds with the investigation of the importance of status rank for health, psychological, and behavioural outcomes. Meanwhile, evolutionary psychology frameworks of status foundations and attainment are highlighted. The literature review then examines the neuroendocrine mechanisms underpinning status through the proposed theoretical framework (i.e., challenge hypothesis/Mazur's biosocial model of status), whilst acknowledging the cognitive moderators of this relationship, and examining psychosocial factors previously recognized as protective factors by epidemiological research (Steptoe & Marmot, 2002).

The thesis continues with *Chapter 3*, discussing and justifying the chosen research methodology. The chapter engages with methodological concerns which questions the reliability of bio-behavioural research and its implications upon the established relationship between hormones and status. The chapter also explores the research philosophy whilst outlining the appropriate for this research epistemological and

ontological positions. Methods, materials, procedure and analysis are also described in this chapter.

Chapter 4 seeks to explore the impact of status competition for endocrine reactivity. The chapter commences with an analysis of the pre-competition data and continues with the post-competition analysis. The analysis provided in this chapter thus addresses the first research question and hypothesis.

Chapter 5 engages with analysis of psychological factors that have previously been suggested to impact endocrine reactivity to competition. Comparisons are drawn in relation to SES in an attempt to understand whether differences between SES groups occur and whether those variables could contribute to the broader question of why not all individuals experiencing SED would necessarily experience negative health outcomes (Marmot, 2004).

Chapter 6 combines the psychological and endocrine data in an attempt to evaluate potential existing links between psychosocial factors and endocrine response, thus addressing research question and hypothesis 2. The chapter then engages with analysis of cognitive appraisals (threat/challenge) as moderators of endocrine reactivity and thereby addresses research question and hypothesis 3. Further, the chapter proceeds with evaluation of the behavioural implications of competition victory and defeat. By doing so the chapter addresses research question and hypothesis 4.

Chapter 7 engages with a discussion of all three results chapters. The chapter then outlines the limitations of the present research and provides a summary of the findings. Future research directions are also discussed.

The final *Chapter 8* provides a summary of the discussion whilst suggesting future research which aims to facilitate our understanding of the relationship between micro and macro elements of SED. In particular, the chapter queries the future role of greater consideration of the implications of social defeat upon social policy.

Chapter 2

Literature Review

2 Introduction

This chapter provides a comprehensive review of the literature relating to the central themes touched on by this study, particularly drawing on a scholarship which explores the relationship between social and health inequalities. Prior to teasing out the complex links between social inequalities and health outcomes, this chapter commences with an examination of the concept of poverty and its relationship to social inequalities and health. Following this, the unsuitability of the accountability characteristics of the concept for the deleterious health outcomes across Western industrialised societies is highlighted. For this reason, the chapter then proceeds with the exploration of alternative and presumably more relevant concepts and measures which aim to explain and capture the link between ill-health outcomes and socio-economic factors in developed countries. The literature review then demonstrates that despite the wide range of concepts and measures adopted within research, a robust link between social inequalities and health outcomes exists. The importance of this link is then highlighted through examples of status disparities in health. More particularly, the chapter reviews the biological consequences of living in SED. The review also emphasises the bidirectional link between socioeconomic disadvantage and ill-health by illustrating examples of how the consequences of living and experiencing socioeconomic disadvantage may also become the antecedents of it. Subsequently, the mechanisms through which social inequalities impact health and wellbeing are investigated by drawing upon epidemiological literature which postulate the theoretical framework of status syndrome (status rank and status comparison) as model through which to explore and explain the phenomenon. To do so, the chapter shifts towards an examination of the evolutionary foundations and purposes of hierarchical structures, and importance of status rank across the animal and human kingdoms. The neuroendocrine mechanisms (androgenic and glucocorticoid systems) proposed to underpin status are then investigated through the lenses of the

methodical framework of the challenge hypothesis/Mazur's biosocial model of status whilst acknowledging the importance of cognitive moderators and relevance of psychosocial factors. Finally, the chapter demonstrates the importance of the neuroendocrine mechanisms underpinning status disparities in health, and considers the wider policy implications suggested by the literature which may be of relevance to strategies to address health inequalities.

2.1 Poverty

As a multidimensional phenomenon, the concept of poverty is often afforded complex and frequently inconsistent definitions amongst policymakers, social policy experts and anti-poverty campaigners (McKendrick, 2021; Ravallion et al., 2008). This arises from the fact that poverty has been associated with various forms of disadvantage and has been only broadly understood as "inadequate outcomes (such as not being adequately clothed); inadequate opportunities (such as not having access to an adequate education); or inadequate resources (such as not having enough disposable income to purchase what is necessary to maintain an adequate standard of living)." (McKendrick, 2021, p. 15). Similarly, extensive epidemiological and health research argues that the multifaceted nature of poverty affects health multidimensionally (Bartley, 2017; Marmot, 2004, 2005, 2010; Wilkinson & Marmot, 2003; Wilkinson & Pickett, 2010, 2018). This multidimensional, detrimental impact of poverty upon health and well-being has been illustrated through numerous studies associating poverty with material, educational, accommodational and nutritional deprivation (McKendrick, 2021); limited access to social services, transport, employment and social relations (McKendrick, 2021); and significantly higher risk of exposure to pollutants and poor environments (Bramley & Bailey, 2017; Haushofer, 2011). These various forms of disadvantage, albeit not in a straightforward manner, have negative implications for health and well-being frequently resulting in heightening the prevalence of mortality and morbidity rates, shorter life expectancies, health deterioration and amplified levels of crime across the most disadvantaged groups (Wilkinson, Marmot & WHO, 1998; Murali & Oyebode 2004; Wilkinson & Marmot, 2003; Wilkinson, 1997; Sapolsky, 2000, 2004).

Whilst logic may lead one to assume that developing countries disproportionately experience the adverse impacts of poverty on health and well-being due to their limited means to meeting trivial needs such as sanitary conditions, fresh water, food and shelter, the effects of poverty are not only confined to the 'poorest' countries (Deaton, 2013; Marmot, 2004; Wilkinson & Pickett, 2010; Wilkinson & Pickett, 2018). Evidently, considerable sections of the population within economically powerful nations such as the UK, USA and Australia live in poverty, are unable to escape poverty traps, consequence leading to a cycle of poverty, intergenerational poverty (Payne, 2005), and persistent poverty (Office for National Statistics, 2019). Rising childhood poverty, precariousness of employment contributing towards in-work poverty, and poverty amongst older people and vulnerable groups has also been documented in the UK (Brewer et al., 2021; Gibson, 2020; JRF, 2021). Indeed, in 2019/2020, 18% of the UK population were in relative poverty - before housing costs deduction - and 22% after (Francis-Devine, 2021). Importantly, even prior to the pandemic, in-work poverty had risen to an estimated 13% of households in 2018/2019 (Child Poverty, 2021; Fancis-Devine, 2021), whilst relative child poverty after housing costs reached 31%. These rates are further expected to rise post-pandemic (Brewer et al., 2021). Similar findings are observed in US population where 12.7% are classified as 'poor', with the burden of poverty disproportionately falling on women (Poverty USA, 2019). These figures remind us that whilst poverty prevails in the least developed countries many individuals living in the strongest economically performing countries also face the burden of unequal material distribution.

Western industrialised societies demonstrate heightened poverty levels and occurrences of individuals living below the federal poverty line, whereas empirical evidence suggests that the percentage of population unable to satisfy basic needs is significantly smaller than within developing countries (Ortiz-Ospina, 2017). The precise estimation of the levels of extreme deprivation in the richest countries is rather problematic due to the use of methodological approaches which adopt varying definitions of absolute poverty and thus fail to capture extreme levels of poverty (Ortiz-Ospina, 2017). Regardless of this lack of uniformity across methodology,

research reveals that even among the least well-off Americans (classified as living under the absolute poverty line, and constituting 12.6% of the entire population), some 80% of them possess material goods such as air-conditioning, a vehicle or second vehicle (75%/33% respectively), or some type of technology such as a computer (33%). None of these material goods are attributable to the 'poorest' individuals from developing countries (Wilkinson & Pickett, 2010). This shows that, in contrast to their counterparts in other parts of the world, disadvantaged individuals from rich societies acquire sufficient material resources to meet some basic needs and are likely to secure survival.

In a similar manner, some researchers argue that the presence of food banks in industrialised cities such as Glasgow signify western communities' inability to completely eradicate absolute poverty (MacLeod, 2016, p. 195); conversely, the presence of food banks may not merely result from a genuine lack of financial resources, but from cultural and societal factors which produce an expectation and pressure to attain particular standards of living even to the detriment of economic security. Indeed, considered within the analytical framework of life history theory, the prevailing 'poor' economic choices and life history strategies amongst the most disadvantaged are frequently explained by their short-term life aims and decisions related to proximal context rather than distal goals (Adams & White, 2009; Laran & Salerno, 2013; Sheehy-Skeffington, 2020; Watson et al., 2016). These short-term life goals could be considered a result of individuals' repeated exposure to higher levels of crime, violence, poor neighbourhoods, insecurity, unpredictability of events and general social and economic instability (Amir et al., 2018; Gennetian & Shafir, 2015; Sheehy-Skeffington, 2020).

Existing research suggests a variety of justifications for these present-oriented decisions (Pepper & Nettle, 2017). Some argue that impulsivity is a pathology resulting from deficient inhibitory processes (Bari & Robbins, 2013; Dalley et al., 2011), whilst others propose stress and negative affect as the cause of these short-term life outcomes strategies (Haushofer & Fehr, 2014). Nevertheless, there is a certain systemic necessity placed upon individuals to react to and deal with constant direct demands which result in a contextually appropriate response which is frequently associated

with the pursuit of short-term material goals (Pepper & Nettle, 2017). Lastly, individuals experiencing socioeconomic disadvantage could also strive towards the general societal aspirations of owning goods of status and 'luxury', leaving less resources to spend on the more basic, but necessary items (Wilkinson & Pickett, 2018). All these findings suggest that if disadvantaged individuals obtain enough material resources to meet basic needs, but not enough to fully participate in the acceptable daily life of the society, the 'poorest' individuals from richer societies face a different type of poverty from those living in extreme disadvantage, namely relative socioeconomic deprivation.

Epidemiological research demonstrates that ill-health within Western communities is rather weakly linked to the characteristics of absolute poverty diseases such as malaria, tuberculosis, malnourishment or dysentery, and supports the argument that within overall richer societies, it is relative poverty which these countries are suffering from, not absolute poverty (Deaton, 2013; Marmot & Wilkinson, 2001; Wilkinson & Pickett, 2010). This position emanates from findings of various studies of disadvantaged populations in Western communities indicating that individuals' ill-health is associated with different types of poverty-related diseases, the so-called 'diseases of affluence' often experienced by the least well-off in affluent communities, such as hypertension, Type 2 diabetes, cardiovascular diseases, cancers, and degenerative diseases (Marmot, 2004). These illnesses are frequently referred to as stress-related diseases and predominantly associated with 'relative poverty' or 'relative deprivation' (Sapolsky, 2017), whilst infectious diseases are robustly associated with a lack of sanitary conditions, shelter and food, conditions predominantly observed in the least developed countries. Additionally, whereas infectious diseases (common in the poorest countries) are particularly associated with early life and childhood, the diseases of affluence often affect later life or adulthood (Wilkinson & Pickett, 2010). Taking into account these differences in ill-health between the richest and the poorest countries, an alternative explanation of the presence of ill-health in Western industrialised societies has been pursued and suggested. Wilkinson (1997) suggests that higher mortality and morbidity rates are far more influenced by relative poverty or 'relative standard of living' than absolute poverty. His argument stems from the

aforementioned findings uncovering that it is not poverty *per se* that causes ill-health in rich societies. Rather, poor health emanates from different stress-related diseases associated with the economic, societal or environmental components of the multifaceted structural nature of poverty (Haushofer, 2011; Kim et al., 2018; Marmot, 2004; Wilkinson 1997; Wilkinson & Pickett, 2010; Wilkinson & Pickett, 2018).

As such, poverty appears to be contextual and in order for the concept to have utility in Western industrialised communities it must be viewed through the terms 'relative poverty' or 'relative deprivation', where an adequate standard of living is relative to the community within which the individual subsists, and not in the light of 'absolute poverty'. Before further discussing the differing sociological and epidemiological approaches and models which aim to elucidate the causes of ill-health in wealthy countries, it is important to examine some of the more relevant terminology.

2.1.1 Relative vs. Absolute Poverty

In the context of Western communities, it is particularly important to understand whether 'absolute' or 'relative poverty' has a more profound impact upon an individual's health and wellbeing. Absolute poverty refers to an inability to meet basic needs and sustain physical survival due to inadequate material resources (McKendrick, 2021), whilst its definition depends on a fixed financial threshold known as poverty line which varies across countries, household sizes and years (Arcaya et al., 2015). In contrast, relative poverty, also frequently described as 'relative deprivation', is characterized as a comparison between one's income and the societal standards of living in a specific time (McKendrick, 2021). More specifically, in the UK, relative poverty is defined as 60% of the median income (McKendrick, 2021) and in contrast to absolute poverty, the relative poverty line changes as economic growth rises. Nevertheless, both concepts of poverty place differing emphasis on either the objective or subjective dimension of income measures (McKendrick, 2021). As such, the subjective comparison of an individual's 'wealth' or 'poverty' in relation to the rest of the society appear to be better captured through relative poverty, whilst the objectivity of the criterion – income – through the dimension of absolute poverty.

The utility of 'absolute poverty' as an objective measure of wealth has been proven advantageous within the framework of the absolute income hypothesis (Kawachi et al., 2002), albeit not without its limitations. The fundamental premise of the absolute income hypothesis states that what impacts health most is individual's income per se rather than the income one acquires compared to the rest of the society, or relative income (Kawachi et al., 2002). Drawing from this principle, peoples' health should remain unaltered in situations when their income remains constant and the population gets wealthier. This, however, seems implausible due to the theory not accounting for economic dynamics such as alterations to the cost of material goods and other market forces which impact on an individual's ability to fully participate in society. More specifically, the hypothesis does not consider issues such as access to transport, communication devices and technology; resources which are fundamental in modern culture and lack of which might adversely impact psychological wellbeing (Kawachi et al., 2002). A lack of these resources is frequently associated with psychological discomfort, arising from subjective feelings of 'not being able to keep up with the average standards of living'; 'falling behind the rest of the society', stigma and shame (Arcaya et al., 2015).

Wilkinson and Pickett (2010) in their *"The Spirit Level"* also question the applicability of the hypothesis and absolute poverty as a measure of poverty. They suggest that absolute poverty is no longer a useful measure of poverty within rich societies as a result of its inability to elucidate the causes of ill-health amongst those demonstrating significantly better average material living standards and relatively low levels of extreme deprivation compared to their contemporaries in developing countries. Accordingly, the authors argue that the presence of ill-health in Western industrialised societies is better explained by relative income and the relative income hypothesis. This appears as a consequence of the fact that economic growth is not only no longer able to further facilitate the rise of wellbeing across rich societies, but also appears to further increase the levels of ill-health outcomes. Hence, even if a rich country gets wealthier, health outcomes would not necessarily be beneficially impacted by the rising standard of living (Wilkinson & Pickett, 2010). Evidently, their findings also illustrate that the transmission between 'developing countries' and

'modern societies' slowly decreases the beneficial effects of economic progress on the levels of health, wellbeing and happiness; subsequently arguing that individuals' absolute income in relation to health outcomes lacks utility in Western industrialised countries.

A strength of the relative income hypothesis is that it considers both the costs of social inclusion and the psychological pathways that link income to health (Arcaya et al., 2015; Garroway & De Laiglesia, 2012), the foundations of which focus on subjective measures of wealth rather than objective income as with the absolute income hypothesis. Consequently, social inclusion is viewed as one of the needs or 'capabilities' (Sen, 1983) that need to be acquired in order to achieve good quality of life (Garroway & De Laiglesia, 2012). This 'need' however, is difficult to explicitly measure or integrate within the absolute income hypothesis, making relative income once again a more effective instrument. Relative income approaches, as a criterion of poverty, consider how individuals compare themselves to others in society, and which factors play a significant role to the feelings of social exclusion. The approach thus questions whether low-income households only experience social exclusion when comparing to others of a similar socioeconomic status, such as relatively disadvantaged families, or also when compared to the prominence of more well-known public figures such as celebrities (Kawachi et al., 2002). Importantly, the relative income hypothesis serves additional purposes including facilitating our understanding of the distribution of wealth whilst affecting governmental and business investment decision making in serving those that are less well-off (Kawachi & Kennedy, 1997). Yet again, the concept appears to be a better facilitating measure due to its ability to conceptualise the complexity of the health-income relationship, doing so by recognising other stratification variables measuring the degree to which individuals fall behind others. This is something the absolute income measure fails to achieve.

The first two sections of this thesis have addressed the practical issues of how poverty, as a relevant operationalising term in the context of ill-health, can be measured and the utility of variations of this measure within developing and developed countries.

The next subsection proposes alternative concepts and measures frequently examined in research, in the endeavours to better determine the causes of ill-health and to prevent disease.

2.1.2 Alternative concepts and measures

Alternative stratification measures were reviewed in order to understand their utility when measuring access to resources and the implications for health and wellbeing in contemporary societies (Alkire & Santos, 2010; Arcaya et al., 2015; Cockerham, 2021; Redelmeier & Singh, 2001; Townsend, 1993; Wilkinson, 1997). Townsend (1993) for instance, utilized an alternative construct replacing the traditional measures of wealth, income and poverty, in order to evaluate the degree of socioeconomic deprivation in the UK. His instrument applied an index accounting for various factors such as clothing, diet, housing, recreation, work and education. This multidimensional poverty approach has also been used as an alternative concept in the context of developing countries (Alkire and Santos, 2010). Wilkinson (1997) for example, conceptualises the ill-health of modern countries as an issue of socioeconomic inequality rather than poverty or material/economic deprivation alone. Multilevel modelling methods distinguishing the impact of individual features from higher level structural influence, further support the shift in the research stream from poverty to social inequalities (Raudenbush, 2003). Resultantly, the interest in relative measures of SES has developed significantly alongside the ill-health research. Additionally, because the concept of poverty is frequently shaped and determined by features such as personal experiences, belief systems and value judgements, it is important to consider whether poverty per se or other conceptualisations of the causes of ill-health, capturing the experiential dimension of poverty, have better utility when determining the causes of ill-health.

Wilkinson and Pickett's argument, based on their extensive epidemiological research, is that ill-health and social problems appear more related to socioeconomic inequality than the average living standards and poverty per se (Wilkinson & Pickett, 2010). Wilkinson (1997) had earlier suggested that although income inequality and poverty are closely related, income inequality has greater pertinence when measuring health

due to its evident effects on psychosocial stress, subsequently affecting health and thereby increasing mortality and morbidity rates. Relatedly, Marmot and Wilkinson's latter work also suggests that health inequalities originate from the interdepended social and income inequalities (Wilkinson & Marmot, 1999; Wilkinson & Marmot, 2003). The Black Report (1980) was the first document profoundly investigating the relationship between social inequalities and health in Britain. A few models of explanation have been proposed in the report (i.e., selection, artefact, material and behavioural-cultural), with the early research predominantly concentrating on the economic aspect of the issue. Subsequently, between the 1980s and 2000s, research brought in and assessed three more additional factors in the exploration of the health inequalities field; psycho-social stress at work, life-course effects and social isolation (Bartley, 2017).

Prior to looking at the explanation of and the review of literature on the models endeavouring to elucidate health inequalities, this thesis will address the definition and importance of social inequalities as the origin of health inequalities. Moreover, the subsequent section will examine the various measures of social inequalities and specify the one that appears to have the highest utility for the purposes of this work.

2.1.3 Social Inequalities

Literature over the past 10 years demonstrates not only the lack of clarity around the understanding of social inequalities, but also the degree of conflict and confusion around the measures of it; all of which majorly impact health inequalities research (Bartley, 2017). This perplexity arises from a lack of clear definition and limited, vague descriptions of the measures of social inequalities (i.e., social class, social position) utilised in the research. For instance, terms such as 'high' and 'low' are briefly and unclearly used to accompany categories such as status, income, and social class, without any further clarification or differentiation between the descriptions of the concepts (Bartley, 2017). Krieger and colleagues (1997) not only emphasise the importance of distinguishing these 'dimensions of inequality' but also argue that terms should not be intermingled for the sake of research. This argument emanates from the fact that there is by no means a perfect concept encompassing all underlying ideas and

assets of terms (i.e., people with identical monthly income may experience entirely different socioeconomic positions based on their class, status or relative income). Because of the equivocality of the concepts used in literature and the lack of appropriate and comprehensive measure of social inequalities, Krieger and colleagues (1997) propose that every researcher should carefully choose the measure that seems most appropriate for the aims of their research. This should also be based on the specific health outcome of interest and the inequalities considered (by the researcher) to have the strongest impact on it. The measure considered to fit best for the purposes of this research will be made evident through the exploration of the next few sections of this thesis, however it will also be briefly outlined as a recruitment criterion variable at the end of this section. Prior to that, dissimilarities between the measurements of status, class, and income will be explored. Additionally, in order to shed some light on the origins of conflict in research, the ways these concepts have previously been evaluated will be noted. Taking into consideration the most up to date research within the social inequalities field, recent measures such as educational attainment will also be considered as a potential utilitarian measure.

Social class and social status are the two concepts most commonly used by research to discuss social position when deviating from single measures such as income and wealth (Bartley, 2017). Previously, measurements of both social class and social status had been used interchangeably, although this does not necessarily mean that they both correlate with the broad spectrum of health outcomes in an indistinguishable manner. However, by defining these two concepts we will be able to understand the exact relationship between economic rank outcomes, and the predictable implications for health. Whilst the measures of social class emanate from social structure theories (with Marx and Weber positing sociological theories centred around ownership of assets and property and occupational rank), the concept of social status encompasses the hierarchical idea of constructing society by rank "from top to bottom" (Bartley, 2017). Rank or pecking order exists within the context of social status and stratification of societies and it is frequently referred to as "prestige" or "social honour" by sociologist and anthropologists. In psychology, however, prestige refers to one of the two distinct pathways through which social rank is pursued and attained in human

societies; the second of which is dominance (Cheng et al., 2013). Another major distinction between social class and social status lies in the proposed definition of the terms with social status deriving from their ethnic, religious or tribal origin; entirely nonaligned to one's occupation, whilst social classes are classified on the basis of occupational group formation (Bartley, 2017). Because social status is often associated with non-progressive hierarchical systems, such as the Hindu caste system (Bartley, 2017), this raises questions around the utility of the measure in modern industrialised cultures. However, before completely discrediting the concept of social status as an outdated measure of social inequalities, with little contribution to the current understanding of health inequalities, one must acknowledge the distinct origins of the concept within the context of the American social epidemiology.

In America, Parsons's school of thought proposed an explanation of the concept of social inequality on the basis of 'structural-functionalism'. More specifically, societal structures comprise unequal occupational ranks accompanied by diverse employment conditions, prestige and income. Accordingly, Parson argues that individuals naturally inherit and acquire uneven abilities and skills, allowing some to achieve occupations within the fields of law, medicine, science which carry higher prestige and income compared to the rest of the social strata (Bartley, 2017; Marshall et al., 1988). 'High socioeconomic status' individuals were perceived to acquire higher occupational positions based on their 'skills and abilities', also frequently featuring in their education. Historically, the development of the measure of social position in the US was centred on occupational and educational ranks. However, upon a retrospective examination of the conducted studies it becomes apparent that those two dimensions were only used as a proxy for measuring social judgements of individual characteristics allowing one to acquire low or high prestige. Nevertheless, the method of measuring both education and occupation persisted and led to the establishment of a new measure of social inequalities; 'socioeconomic status' (Featherman & Hauser, 1976).

Currently socioeconomic status (SES) is used to determine one's status/rank within the economic and social hierarchy based on various indicators such as wealth, occupation, income and education, however subjective categories such as

neighbourhood-level crime rates, average house price, and despair are also commonly used in research (Braveman et al. 2005; Krieger et al. 1997; Lakshman et al. 2011; Pepper & Nettle, 2017). Resultantly, when outlining relationships between SES and health or behavioural outcomes, SES appears as a proxy measure rather than a strict definition referring to individual experiences of "affluence" or of "being less well-off", when categorising groups in society. Oakes & Rossi (2003) also argue that SES is a dormant concept, that is challenging to measure and define. This is due to the construct's frequent association with political ideologies, each of which adopt different normative vantage points on existing social structures and their desired transformation. Political ideologies, however, change over time, inevitably leading to a subsequent change of the understanding and definition of socioeconomic status (Oakes, 2016). The way the construct has been defined and applied by research therefore varies. Some have linked it to more assessable definitions such as annual income, whilst others have considered race and ethnicity as a core element of the measure (Oakes, 2016). Health status has also been recognised as an indisputable component of the measure by many, emanating from the close correlational and causational link between health and SES. Conversely, other scholars have discredited the link between SES and health, and SES and race, on the basis that "one should be able to improve their SES without changing their phenotype (including skin colour) or linguistic accent" (Oakes, 2016, p. 5); but also because if health is incorporated into the concept of SES, this inevitably impacts our abilities to evaluate health outcomes by SES (Kaufman et al., 1997; Oakes & Rossi, 2003). Importantly, although SES has been widely utilised in the research and falsely perceived as a universally understood concept, many struggle to correctly allocate individuals to particular SES groups, particularly when it comes to children, older people or those outwith the labour market. Regardless of the differing definitions and utilisation of the construct in research, the literature argues that SES could be broadly described as a concept which measures an individual or group's access to the culturally relevant resources required to succeed or obtain a higher rank on the social ladder. Reflection on the utility of the measurement across cultures, civilisations and times is necessary. This stems from the fact that whilst the antecedents of 'power' within societies might be similar, the degrees of social stratification and mobility vary profoundly, implying the necessity of

different SES measures in order to adequately correspond to the different nuances of each society's social strata (Henrich et al., 2005; Smith et al., 2011; Spilerman, 2000; Van Leeuwen & Maas, 2010).

The importance of the measure of SES within the research of health inequalities derives (as pointed out earlier on) from the evident link between SES and health and life outcomes (Oakes, 2016). SES serves as an indicator of individual's or group's access to valuable material and human capital resources. The access to these resources appears crucial due to their critical importance upon the prosperity of the individual or group. SES then reveals some of the theoretical underpinning issues, the magnitudes of which affect health differently. This appears to be a significant contribution of the multidimensional concept when it comes to considering the degree of its utility within the field. However, contemporary researchers also emphasise the importance of the construct as a measure of social systems. Thus, SES as a concept has been frequently used in opposition to more egalitarian and meritocratic views. Further, the concept has also been of interest to many concerned with the idea of SES entrenched impact on measures of health, wellbeing and life changes (Oakes, 2016). Nevertheless, despite competing approaches to defining and utilising the multidimensional construct of SES, its utility within research is indisputable.

Before exploring health inequalities in the next section of this review, I will briefly outline and consider the utility of other measures frequently adopted when investigating social and health inequalities. For example, European researchers and academics (e.g., frequently appear in favour of socioeconomic position (SEP) (Galobardes et al. 2006), a measure rather similar to SES. Krieger and colleagues (1997) define socioeconomic position as an "aggregate concept that includes both resource-based and prestige-based measures, as linked to both childhood and adult social class position" (p. 345). The degree of similarity between the concepts of SEP and SES is so high, however, that researchers often refer to the two terms as synonymous and interchangeable (Oakes, 2016). Some researchers (e.g., Lawlor et al., 2004; Lynch & Kaplan, 2000) appear to be in greater favour of SEP as a measure compared to SES, as a

result of the rather narrowed and limited interpretation of SES within the lines of occupational prestige and rank. This being said, the dissimilarities between "position" and "status" are genuinely insignificant. Whilst closely related to low SES, Kim and colleagues (2018) also suggest socioeconomic disadvantage (SED) as an alternative measure with better descriptive and explanatory utility; being more broadly conceptualised to include variables such as subjective perception of social position and contextual markers of disadvantage, like neighbourhood deprivation. And yet, even the construct of SES frequently utilises subjective dimensions such as neighbourhood-level deprivation. However, this is again being done idiosyncratically and on the basis of the specific interests and aims of the research. Researchers (e.g., Mirowsky & Ross, 2003) also demonstrate interest in the application and utility of educational attainment as a single measure of social position (individually considered outside the dimensional spectrum of SES) (Bartley, 2017). Regardless of the strong correlation between health and education, the utility of the measure could be easily discredited in the research on the basis of the concept not accounting for the complexity of the health inequalities field (Bartley, 2017). Expanding on this, another argument against the creditability of education as a single measure of health inequities is that the proportion of the population that access education and the level to which individuals get educated changes over time and it is not sufficiently robust when accounting for cross-cultural differences. The final two arguments against this approach relate to differences in access to education amongst sex and ethnic groups, and the easily adopted 'functionalist' frameworks which presume a direct link between intelligence and ill-health, whilst discrediting the complex health inequalities landscape (Gottfredson, 2004; Bartley, 2017).

In order to avoid the lack of clarity around the multidimensionality of SES as a construct, more direct and less ambiguously defined measures of inequalities such as the Theil Index and Gini Coefficient are commonly used within literature (Allison, 1978; Duro, 2008; Nolan et al., 2021; Richmond-Rakerd et al., 2020). These measures focus on one rather than many of the SES dimensions such as distribution of wealth and income and act as dispersion or variance instruments. The relative simplicity of these measures allows researchers to directly evaluate the degree to which

socioeconomic inequalities could be considered antecedents of health inequalities and what implications this has on populations (Adler & Ostrove 1999; Bowles & Gintis 2002). Considering this, one might argue or question why these measures are not consistently applied in order to avoid further equivocality and improve rigorousness within the literature. Thus, it is important to note that the high validity and reliability of the aforementioned measures of inequalities do not solely emanate from their computational effectiveness, but also of their ability to encompass the multifactorial nature of social inequalities and thus their relatedness to other more difficultly defined measures.

Differing degrees of utility amongst the wide range of measures raise the question as to which is the best measure of social inequalities. Unfortunately, literature remains in dispute over this issue. Considering the unsettled positions articulated here and following Bartley's (2017) and Oakes (2016) recommendations, one should utilise the measure that best allows the researcher to address their research questions and hypotheses. Hereafter, the chosen criterion for this thesis is SES. However, the construct is not simply based on measures of educational attainment and annual household income (as broadly utilised in research due to real world restrains and issues around disclosure), but encompasses household annual income, individual annual income, number of employed individuals within the household, educational attainment, marital status and employment status.

In summary, the measurement used to chart social inequalities is of importance. Researchers should be able to understand and differentiate between the concepts, whilst applying valid measures of those concepts for the sake of enquiry. Moreover, grasping definitions and utility of concepts and measures allow us to consider and profoundly investigate the specific correlational and causational relationships between SES and health. In our endeavours to reduce inequalities, we must ask ourselves what it is that makes people ill and what can we do to prevent it; is this a matter of income, working conditions or stress? There is little use in improving one's income when the root of the problem is located elsewhere, and thus our enquiries must endeavour to capture the most important and determining relationship.

2.1.4 Health Inequalities

As illustrated in the previous section, social epidemiology and health researchers often utilise various concepts and measures of social inequalities in order to better understand health inequalities. Thus, comparisons between groups on the basis of occupation, class, status or education are made in order to shed some light on the differences in health outcomes and the determinants of those. Both public policy and research were decisively influenced by the introduction of the topic in the first ever published document on health inequalities in 1980 - the Black Report (Bartley, 2017; Department of Health and Social Security, 1980; Townsend et al., 1986). The Black report documented the strong relationship between social inequalities, mortality, morbidity and class between the years of 1931 and 1971 (Bartley, 2017). Alas, the data from the 'most important single document on health' only tells us so much; limiting its attention to the health inequalities amongst males only. This stems from UK women's rather limited participation in the labour market during the 1920s and 1930s, causing failure by the study in allocation of women to particular social classes and inevitably resulting in their exclusion from the analysis. Nevertheless, the significant importance of these findings for health and policy making was echoed by the 1988 Acheson report on inequalities in health, which put forward a number of recommendations in the area of health, environment and social determinants (Acheson, 1988).

The findings of the Black Report saw the dawning of a new era for health inequalities research, with European and North American desire to produce the same outcomes and compare research results between countries (specifically between England and Wales), leading to data from as far back as the 1970s being tracked. European data appear much later than the report from England and Wales, putting the UK in an advanced position when it comes to longitudinal measurement of health inequalities, which significantly contributed to the identification and understanding of the general socioeconomic factors impacting health and wellbeing (Benach et al., 2003). What appears evident from the Black Report and the extensive analysis of European and US data since the early 90s is that morbidity, mortality, and life and health expectancies

nearly always follow the same trend, with individuals classified as of lower SES experiencing significantly higher mortality and morbidity and lower health and life expectancies (Cambois et al., 2020; Glymour et al., 2014; Haslam et al., 2018, Benach et al., 2003). However, whilst self-reported inequalities of morbidity are similar throughout Europe, this is not the case for the scale of mortality rates (Benach et al., 2003). Mortality inequalities between low and high SES groups appear to be smaller in more socioeconomically equal nations like the Nordic countries, whilst countries like the UK and US demonstrate higher - if not the highest - mortality inequalities. This being said, discussions around "relative" and "absolute" inequalities in health should be taken into consideration when drawing cross-cultural comparisons. This emanates from the fact that inequalities in the US and UK may be higher than those of Sweden when based on absolute differences (i.e., lower and upper social group rate differences). However, when measures such as "relative" health differences (i.e., mortality rate ratio for upper and lower social status groups) are taken into consideration, the evidence of smaller inequalities in the more egalitarian nations such as the Nordic countries vanishes (Benach et al., 2003; Mackenbach, 2020).

Importantly, mortality data also allows us to pinpoint the various causes of death across the countries. The dominant contributor to the socioeconomic gap in mortality in England, Wales and Ireland, and the Nordic countries is the higher risk of cardiovascular diseases predominantly observed within the groups experiencing lower SES (Harper et al., 2011; Kunst et al., 1999). On the contrary, the leading contributing factor in excess risk of premature mortality in France, Switzerland, Spain, Portugal and Italy is low SES, with the cardiovascular component playing a small role for this outcome (Kunst et al., 1999). Interestingly, the higher risk of mortality from cardiovascular diseases has not always been associated with lower SES groups, illustrating an example of the different stages of epidemiological development. Thus, higher cardiovascular disease mortality was once associated with higher SES groups (i.e., higher ischaemic heart disease mortality amongst those experiencing high SES in the 1950-1960s, with the trend changing course in the late 1960s-1970s) (Marmot et al., 1978; Marmot & McDowall, 1986; Stallones, 1980; Wing et al., 1986; Mackenbach et al., 1989). Regardless of the cause of mortality, trends of a widening gap in SES appear

evident and universal in the late 20th century (Lago et al., 2018; Mackenbach, 2012; Marmot, 2015; Marteau et al., 2021). Even within the earliest research of health inequalities in the UK, researchers examined potential frameworks to explore the mechanisms and factors by which socioeconomic inequalities impact upon health. In summary, four constructs have been adopted in the attempt to explain the social inequalities in health, namely: 'causal pathways and conditional health effects'; 'selection'; 'context versus composition'; and 'life course' perspectives (Arcaya et al., 2015; Benach et al., 2003).

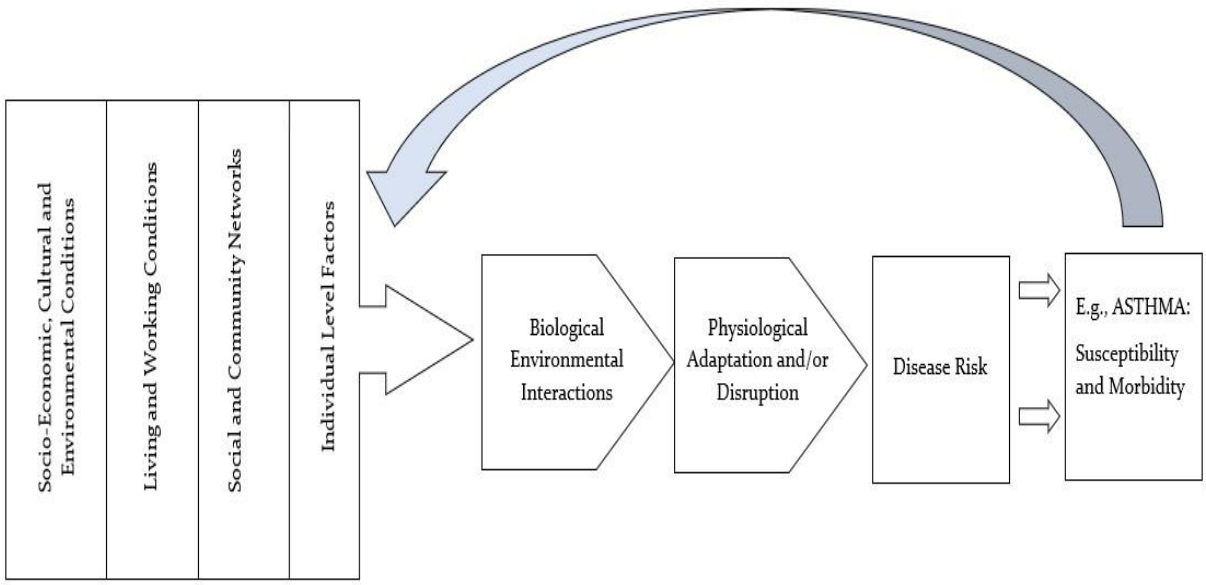
The 'causal pathways and conditional health effects' perspective (Figure 4) calls on those designing policies which aim to reduce health disparities to take into consideration the complex and indirect pathways through which one variable might affect health outcomes. For example, one should consider mediators and moderators of the link between factor and outcome variables (Bartley, 2017; Cockerham, 2021). Mediators are the variables lying between a predictor and outcome, thus frequently accounting for their causal relationship. For instance, we might be familiar with the link between occupation and blood pressure, and that income mediates this relationship. This results from the fact that occupation determines income, which subsequently might affect blood pressure by influencing whether one could afford healthy food, get adequate medical care, or experiences stress over financial matters (Arcaya et al., 2015). However, the relationship between occupation and blood pressure may not only be mediated income alone. It could be the case that even after increasing income, occupation continues to have an impact on blood pressure. If that is the case, this will mean that the occupation–blood pressure relationship is only partially mediated by income. Knowing that occupation has a direct (independent) impact on blood pressure might raise the question of what other variables might affect blood pressure (e.g., working conditions, job stress). In other cases, other variables (i.e., moderators) help explain the conditions under which an exposure and outcome are related. For example, evidence suggests that the relationship between self-rated memory function and depressive symptoms differs between those with low and high ratings of self-efficacy (O'Shea et al., 2016). For that reason, the variable self-efficacy may play a moderating role in the relationship between self-rated memory function

and depressive symptoms. Moderators can thus account for some of the variability in outcomes and address certain questions about who will be most responsive to a treatment/intervention.

In the light of those arguments, the thesis will go on to explore the relationship between endocrine reactivity to stress and performance outcomes could be moderated by cognitive perceptions of threat and challenge. Accounting for variables moderating or mediating a relationship allows social and health policy makers to more thoroughly consider the degree of complexity between those links.

Figure 4

Casual pathways and conditional health outcomes



The 'selective' perspective allows researcher to evaluate the degree to which an observed relationship is causal or self-selectional (reflecting one's personal characteristics and life choices). For instance, when analysing relationships between physical activity and neighbourhood walkability, it is important to address the question whether the link between physical activity and walkability is direct (i.e., walkability impacts physical activity) or more complex and linked to individual preferences and characteristics (i.e., more walkable areas are occupied by individuals

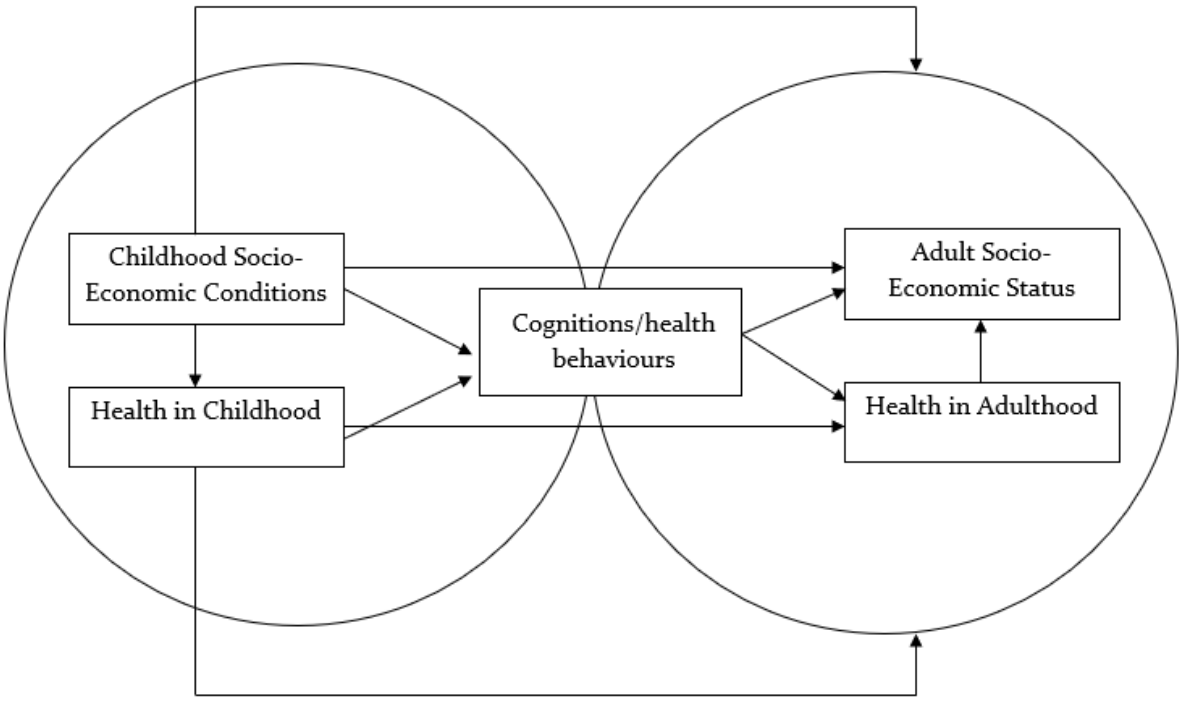
valuing physical activity). Another way that the selection framework has been utilised in the research of health inequalities is to draw links between socioeconomic disparities in health on the basis of educational, occupational status, and racial or ethnic backgrounds (Arcaya et al., 2015). Examples comprise, albeit rarely, researchers drawing links between health outcomes and race (i.e., some individuals having higher IQ, subsequently leading to higher education, occupation and generally better health) (Buchanan, 2003). Although these presumptions and links are generally rejected in health inequalities research, one cannot completely discredit the effects of neighbourhood deprivation, income, stigma and discrimination and occupation for health outcomes (Buchanan, 2003). The 'contextual versus compositional' framework, on the other hand, draws on differentiations between compositional (i.e., individuals' characteristics comprised by the environment) and contextual (i.e., impacts of neighbourhood or other higher-level unit) effects on health disparities. This allows inequality researchers to pinpoint whether the health disparities in a neighbourhood stem from the various health statuses of the individuals living in a setting (compositional) or from the effects of the higher-level unit itself, on the health status of the residents of this site (Subramanian et al., 2003).

Finally, the 'life course' concept adopts the idea that prolonged exposure to risk factors or higher levels of socioeconomic deprivation during the lifespan, and particularly at certain critical and more susceptible periods of foetal and early childhood development, carries risk of many health-compromising behaviours (e.g., smoking, alcohol misuse and substance abuse) and poor health (Arcaya et al., 2015; Benach et al., 2003; Ludwig et al., 2011; van de Mheen et al., 1998a). Impact of SES extends to other transitional and psychological factors such as locus of control and coping styles (Bosma et al., 1999). Health status in adulthood frequently reflects ill-health during childhood, demonstrating a feature of continuity. This suggests a potential accumulative effect of consequences of socioeconomic inequalities for health (van de Mheen et al., 1998b; Wadsworth, 1997). Importantly, this perspective allows researchers to consider the bidirectional nature of this relationship, i.e., neighbourhood deprivation might impact health outcomes, however, ill-health of individuals living in a certain area could also account for the classification of the area

as deprived (Arcaya et al., 2015; Ludwig et al., 2011). This perspective has been applied within the research framework; it being a model which accounts for the impacts of SES upon individual health and wellbeing, whilst considering moderating factors such as cognitive variables (Figure 5). Amongst these are threat/challenge cognitions which could conceivably moderate the causal relationship between SES and an individual's health and wellbeing. These moderators and other related psychological factors are sometimes referred to as 'resilience' factors. The concept, definition and use of the term resilience will be examined later in thesis.

Figure 5

Socio-Economic Inequlities in Health- A Life-Course Perspective



The following subsections will also explore the biological implications of SED across the lifespan, particularly in terms of stress physiology. Moreover, maintenance and causation of SED will be also considered within the postulates of this framework. However, before doing so, the last part of this section focuses on the various frameworks through which inequalities can be considered.

Four main models to explain health inequalities appear to be of central interest. These are as follows: (1) explanation on the basis of material factors; (2) psychological factors; (3) behavioural; and (4) biomedical explanations. Most of the earliest research on health inequalities (i.e., the Black Report) concentrates on explanations within the material factors, whilst most recent research expanded this view and brought in the idea of these additional explanatory panels (Bartley, 2017). The material explanation points to the link between access to material resources and physical risks (e.g., pollution) and the consequences for individual health (Arcaya et al., 2015). Without any doubt material deprivation and exposure to physical health risk factors appear to be a fundamental part of the understanding of health disparities. The absolute income hypothesis and measures of absolute resources are traditionally incorporated in the design framework of this model and yet, the model does not come without its' limitations. As illustrated earlier in the 'causal pathway' framework, there are many other factors that might impact upon the material deprivation/health interaction, or it might be the case that material resources and physical risk factors account to some degree for health inequalities, in line with many other factors.

Another explanatory category looks at psychological factors as a prism for elucidating disparities in health (Haslam et al., 2018). This area of exploration has been broadly considered when investigating the effects of psychological factors on health in this thesis (e.g., stress, social exclusion, low social support or social capital, and psychological responses to experiences). The research adopting this model commonly utilises relative measures of status position/rank, constructs evaluating stress or adverse childhood experiences (ACEs), or looks at subjective rather than objective variables (e.g., subjective perception of SES) (Kaplan, 1999; Marmot, 2004; McEwen & Gregerson, 2019; McEwen & McEwen, 2017; Rozanski; 2005). As this thesis proceeds all three measures will be further explored and discussed. The explanatory model proves useful in explaining the degree to which psychological factors impact health disparities, arguing that some groups are exposed to higher and continuous toxic stress, which supplement negative experiences. However, as with the material explanation, this approach cannot be adopted in its entirety without acknowledging the contribution of the other determinants of health in this rather complex

relationship. The third proposed model is based on behavioural differences. Accordingly, disparities in behaviours are the proposed mechanism for the explanation of health inequalities (Arcaya et al., 2015). These are health-related behaviours and habits such as smoking, alcohol misuse, drug abuse, or nutrition. Regardless of the variations of health-related behaviours across social groups (Krieger, 2001; Short & Mollborn, 2015) though, it is difficult to consider them as independent determinants of health (Arcaya et al., 2015). Finally, the biomedical explanation proposes that health disparities occur on the basis of distinctions between biological health factors across populations and settings (Koster et al., 2005; Skalická et al., 2009). Consequently, the model focuses on specific posterior biological risk factors without accounting for the greater contextual variations across groups. Utilising genetic or gene-by-environment explanations might appear useful in the case of inter-individual variabilities within a group, however, not when applying it to disparities between social status groups (Arcaya et al., 2015). Moreover, as with the behavioural explanation, this model imposes the same limitations and dangers of wrongly interpreting the multifaceted nature of health inequalities.

Acknowledging the wide variability of frameworks and explanatory models, scientific enquiry should not limit its perspective to only one framework or explanatory model which they are in favour of. On the contrary, one should apply complex, multidimensional, and multidirectional frameworks when exploring the wide variety of determinants involved in the convoluted relationship between health and socioeconomic inequalities.

2.2 Biological Consequences and Antecedents to Disadvantage

The following section will examine the reasons why relative deprivation and social inequalities are of importance by demonstrating in what ways individuals are affected by social structure. By focussing on the impact upon individuals, the chapter makes the point that it is not societies who are impacted by inequality, but rather individuals themselves. This is due to their susceptibility to social rank, the consequences of

which manifest in people's poor health outcomes, status anxiety, morbidity and mortality rates, in addition to life expectancies.

2.2.1 General Consequences

As previous sections have shown, living in a position of socioeconomic disadvantage (SED) or experiences of low SES have been associated with adverse effects upon both psychological and physical development (Blair & Raver, 2012; Adler & Rehkopf, 2008). Kim and colleagues (2018) inform us that SED is a contextualised term synonymous with SES, but which considers additional contextual factors such as neighbourhood deprivation and other factors such as subjective experiences of adversity and its impact on health. SES and SED, however, will not be used interchangeably.

SED correlates with poor language abilities, cognitive deficit and behavioural problems in early childhood (Blair & Raver, 2012). Whilst in adolescence, SED has been linked to higher levels of psychopathologies such as substance misuse, depression, anxiety and disruptive behaviours (Goodman et al., 2005; Sariaslan et al., 2014). In adulthood, SED impacts encompass increased levels of criminality, reduced productivity at work and higher rates of mental disabilities (Duncan et al., 2017). Additionally, there is an evident link between SED and physical health (Brooks-Gunn & Duncan, 1997). During infancy, it is associated with higher infant mortality and negative birth outcomes (Metcalfe et al., 2011), whilst in childhood and adolescence, the measure has been linked to higher risk of asthma, physical inactivity and dental problems (Miller & Chen, 2013). Importantly, research argues that regardless of adulthood exposure to SED, experiences of childhood adversity appear to have stronger and long-lasting psychological and physiological negative health effects, frequently featuring in impaired development (Cohen et al., 2006; Davidson & McEwen, 2012; Evans et al., 2004). The plausibility of this argument, however, will be further discussed in the next sections. Throughout adulthood, just as with SES, SED correlates with higher morbidity and mortality rates (Kim et al., 2018), higher risk of diabetes, cancer, and cardiovascular diseases (Seeman et al., 2001). And yet, regardless of the duration or specific life period of SED exposure, the severe negative implications of socioeconomic adversity upon health and wellbeing cannot be discredited.

As previously explored the relationship between the disparities in health and socioeconomic inequalities is rather complex and multifaceted, with competing frameworks and explanatory models offering diverse rationalisations. However, given that the primary interest of this thesis remains the neurobiological mechanisms through which SED impacts upon health, the next few sections will be considerably dedicated to the life course framework. They will aim to reveal how SES is linked to short and long-term consequences for health and wellbeing and life course development and the particular mechanisms through which SED "gets under the skin". Considering the predominant mechanism underpinning the relationship between socioeconomic inequalities and health, proposed by Kim and colleagues (2018), continuous exposure to toxic stress and the subsequently emerging elevated levels of cortisol will also be of central interest to this thesis. However, before engaging with the relationship between toxic stress exposure and health (in section 2.2.3 - Biological consequences of disadvantage across the lifespan), an overview of the complexity of stress and what stress is, will be provided.

2.2.2 Stress

The relationship between poor health and stress is well-established (Beasley et al., 2003; Clements & Turpin, 1996; McDonough & Walters, 2001). A range of health-related conditions such as stroke, memory loss, emotional dysregulation, heart failure and depression have been related to disruption of physiological systems which result from stress (Sapolsky, 1998). Indeed, four out of the ten leading causes of death (heart disease, musculoskeletal disorder or injury, suicide/murder, and stroke) have been directly linked to chronic stress exposure (Miller & O'Callaghan, 2002). However, literature argues that not all stress is destructive and noxious for vertebrates (Sapolsky, 2000). For instance, positive stress refers to short-lasting and moderate stress (acute stress) responses such as mild stress hormonal changes and increased heart rate in response to threat (National Scientific Council, 2014). Learning to adjust to these normal levels of positive (acute) stress is essential for healthy development. In order to better understand the ambiguous nature of stress and its impact on human life, a clear definition of the concept is necessary.

Arguably, Cannon first introduced the technical term 'stress' to the life science community in 1926, developing and empirically testing the fight/flight model. Subsequently, the concept of stress as applied to biological organisms rapidly developed through the work of Selye, who described the condition as 'the non-specific response of the body to any demand placed upon it' (Selye, p. 625-631, 1956). However, the term 'stress' is multi- dimensional, encompassing 'stressors', the 'stress response' and the concept of 'stress'. Thus, to identify the difference between stimulus and stress response, Selye had to create the word 'stressor' – or indeed anything perceived as challenging, threatening or demanding for the organism. Once the term 'stressor' was defined, the stress response was relatively straightforwardly characterized as a process which begins with the activation of the sympathetic nervous system, followed by an endocrine response that mobilizes and maximises muscular output and reaction speed (Viner, 1999).

The concept of homeostasis, defined as the body being in balance (Sapolsky, 2000), is crucial in understanding how organisms get sick from stress. Bernard (1865) coined the term Milieu Interieur which referred to the evolution of organisms to become more independent of the outside environment. For example, mammalian cells can exist only within certain ranges of temperature and acid-base balance (Sapolsky, 1993). The assumption of 'Milieu Interieur' is the ideal physical and psychological state for an animal is equilibrium. However, animals face many and varied stressors (e.g., environmental, social and psychological). If homeostasis is disrupted, energy has to be expended in restoring equilibrium and the goal of physiological systems is to buffer the internal environment (milieu) from environmental perturbations. Indeed, stress physiology is the study of the perturbations that upset physiological balance and an organism's attempts to re-establish that balance. This is reflected in some of the contemporary definitions of stress: "Stress is the recognition by the body of a stressor and therefore, the state of threatened homeostasis… adaptive responses are the body's attempts to counteract the stressor and re-establish homeostasis" Chrousos (1998, p. 311-335).

The stress response in humans has come to be principally associated with the fight/flight metaphor, first described by Cannon (1920) as a vertebrates' immediate reaction to threat. Following detection of a stressor, the Hypothalamic Pituitary Adrenal (HPA) Axis is activated. Through a cascade of endocrine and biochemical events, the body prepares to either run away or overcome the threat through fighting (Selye, 1956). Increased metabolic activity including release of epinephrine/nor-epinephrine from the adrenal medulla directly increases heart rate, triggering the release of glucose from energy stores, and increasing blood flow to skeletal muscle. Energy generated via the degradation of glycogen to glucose and the increased cardiovascular activity provide the body with the essential resources to manage the threat (Bartlett, 1998; Mizock, 1995). Thus, acute stress response or the fight/flight mechanism facilitates survival through an immediate physiological reaction to a perceived harmful event or a threat. Even being evolutionary adaptive however, the stress hormones that accompany stress response could also have devastating consequences to health (Sapolsky, 2000). This is because the evolutionary purpose of this mechanism is not to be chronically activated. Moreover, this could include non-life-threatening stressors such as constant worries about financial status and shelter (psychological stressors) continuously activating acute stress responses (Sapolsky, 2000). For this reason, prolonged exposure to continuous stress is associated with negative health outcomes through the repeated activation of the HPA axis and the subsequently chronically elevated glucocorticoid levels (Sapolsky, 2000; Zada et al., 2016). The following two sections (2.2.3 and 2.2.4) will explore in greater detail the relationship between SED and ill-health, and more precisely the function of chronic stress in this relationship.

Given the various types of stressors (e.g., environmental, psychological and social) faced by organisms, and the distinction between acute and chronic stress, a range of approaches to measuring stress have been developed, thus reflecting the different theoretical frameworks for conceptualizing the phenomenon (Hellhammer et al., 2010). Stress research thus broadly distinguishes between self-reported measures of stress and wellbeing (e.g., Perceived Stress Questionnaire) and physiological measures of stress (e.g., Trier Social Stress Test – TSST). The choice of stress measurement

approach should reflect the study research questions and the hypothesised mechanisms through which the stress type links to the outcome of interest (Crosswell & Lockwood, 2020).

2.2.3 Biological consequences of disadvantage across the lifespan

Moving from macro (lived experience) to micro (biological) within the context of SED research is not easily accomplished because the causal nature of SED, although multifaceted, is principally structural on economic, societal, or environmental levels. Nevertheless, and with full acknowledgement that biological makeup does not directly cause disadvantage, there are increasingly well-established neurobiological and ensuing behavioural consequences to SED. Other than environmental exposure to lead, perhaps, the principal mechanism underlying biological effects of SED on neural architecture and well-being is chronic exposure to stress. Evans (2004), for example, argues that SED during childhood is linked to higher stressor exposure such as separation from the parent, exposure to violence, lower educational quality, noise, crowding, pollutants, and harsh parenting styles. Moreover, lack of access to physical and cultural resources such as fresh fruit and vegetables, transportation and open space, as well as greater exposure to violence and crime are caused by neighbourhood deprivation (Meijer et al., 2012). This relationship carries through to adulthood with financial hardship, discrimination, stigma, lack of control and reward at work (Matthews & Gallo, 2011). And so, one of the distinct features of SED is that people are more likely to experience multiple stressors over a prolonged period (Kim et al., 2018). What follows next is an attempt at describing the neurobiological pathways of SED-stress exposure causing ill-health and well-being.

The hippocampus, amygdala, and medial prefrontal cortex constitute the three regions of the brain involved in the neurobiological mechanisms of stress. These areas, the hypothalamic pituitary axis (HPA axis) and the autonomic nervous system regulate physiological stress mechanisms (Ulrich-Lai & Herman, 2009). Chronic exposure to psychosocial stressors resulting from SED, can cause changes in the functions and microstructures of these regions (e.g., decreased hippocampal volume and inhibition of the processes of neurogenesis and synaptogenesis), subsequently leading to

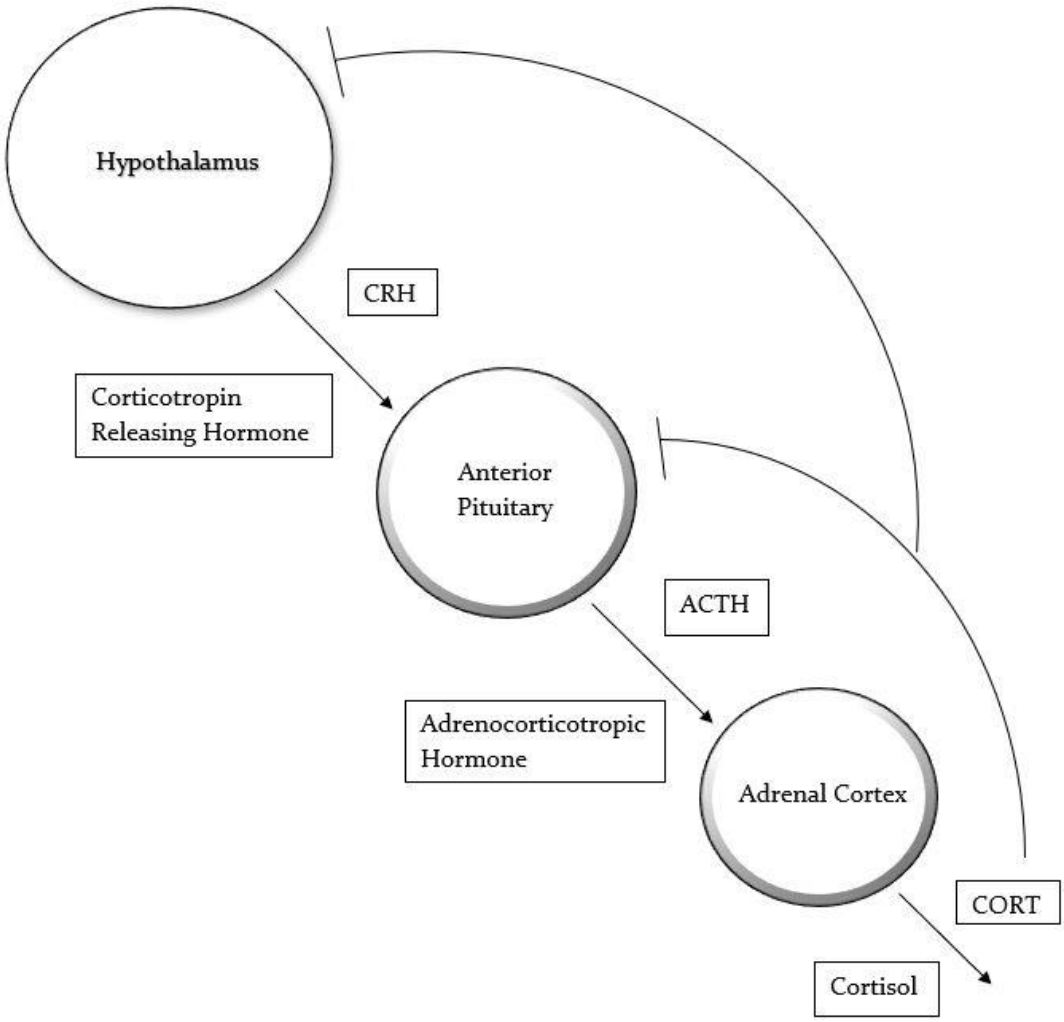
difficulties in executive functioning (Holmes & Wellman, 2009), and HPA axis dysregulation (Rodrigues et al., 2009). Additionally, chronically elevated cortisol levels also cause impairment of areas involved in language acquisition and social cognition, such as the insula, superior temporal gyrus, temporoparietal junction, and posterior cingulate cortex (Uddin et al., 2007; Sheehy-Skeffington, 2019). Two main models have been proposed to elucidate the mechanisms by which elevated stress levels influence the central nervous system (CNS) and physiological systems across the lifecycle. The first focuses on timing; the biological embedding model emphasises critical and sensitive periods, highlighting that early exposure to SED may lead to epigenetic modifications that alter CNS and physiological systems development which results in increased vulnerability to disease (Finch & Crimmins, 2004). The second model emphasizes the duration of SED, where prolonged exposure causes greater impact on neurobiological systems, subsequently impairing biological and behavioural resilience (Kim et al., 2018). Based on empirical evidence from animal and human research, the two mechanisms seem likely to be operating in tandem.

The initial process of stress evaluation occurs in the brain, where the primary physiological stress systems (i.e., the hypothalamic-pituitary-adrenal axis- HPA and the autonomic systems) are activated. Once stress-encoding signals from the amygdala are sent to the hypothalamus, starting the signalling cascade of the HPA-axis, corticotropin-releasing hormone and arginine vasopressin are released by the hypothalamus. Subsequently, this causes the secretion of adrenocorticotropic hormone by the pituitary gland, resulting in the release of glucocorticoids (the most important of which is cortisol), from the adrenal cortex (Figure 6). This whole process is of importance due to its impact upon other physiological systems such as the cardiovascular, immune, digestive, reproductive and many other systems in the human body. Moreover, the system has evolved to be an adaptive mechanism facilitating survival. This features through activation of the branches of the autonomic nervous system (i.e., sympathetic and parasympathetic nervous systems) provoking physiological responses to environmental and psychological stressors, such as the "fight-or-flight" response. Once the threat/challenge is overcome, the system regulates the excessive secretion of glucocorticoids (GCs) by automatically shutting off the

stress response. However, when exposed to continuous stress the system remains switched on for a prolonged period of time resulting in system dysregulation and constantly elevated levels of the neuroendocrine biomarkers of stress such as GCs and more specifically, cortisol. This in turn leads to dysregulation and suppression of the aforementioned systems (Danese et al., 2009; Lang et al., 2019). The biomarkers of these systems and the effects of their dysregulation on the overall physical health status will be further reviewed in the next section.

Figure 6

HPA Axis Regulation



2.2.4 Biomarkers of poor health

As aforementioned, dysregulation of the HPA-axis and continuously elevated cortisol levels frequently result in disruption and suppression, in addition to deterioration of the primary stress and other regulatory body systems (McEwen, 2001; McEwen & Stellar, 1993; Ryan, 2014). For instance, disruption of the HPA-axis as a result of continuous stress exposure has been associated with inflammatory system dysregulation, measured through biomarkers such as inerleukin-6 (IL-6), C-reactive protein (CRP), insulin-like growth factor 1 (IGF-1), interleukin- 12 (IL-12) and tumour necrosis factor alpha (Guan et al., 2021; Hintikka et al., 2009; Johnson et al., 2013; Levi et al., 2011; Lin et al., 2016). Accordingly, chronic stress appears to be associated with an increased production of proinflammatory cytokines resulting in a higher risk of inflammation, and neurogenerative, cardiovascular or autoimmune diseases (Kim & Maes, 2003). Oxidative stress has also been linked to metabolic suppression and disruption (Sapolsky, 2017). The manifestation of these disturbances is measured through the well-established biomarkers of metabolism function, i.e., high levels of low-density lipoprotein cholesterol, triglycerides, low levels of high lipoprotein cholesterol, glycosylated haemoglobin, insulin and glucose (Kim et al., 2018). Subsequently, suppression and dysregulation of the metabolic processes increase the risk of Type 2 diabetes, obesity and cardiovascular diseases (Ryan, 2014). Implications of disturbances in the metabolic processes and stress regulatory systems have also been related to poor health-related behaviours such as increased consumption of palatable foods, albeit structural and physiological brain changes partially account for these outcomes (Dallman et al., 2003; Lönn et al., 1994). An increased risk of cardiovascular diseases also arises from continued toxic stress and HPA-axis dysregulation, due to the associated prolonged exposure to increased blood pressure and elevated heart rate (both of which are biomarkers of heart diseases, i.e., systolic and diastolic blood pressure) (Krantz & Falconer, 1995). Finally, dysregulation of the primary stress response systems may result in severe architectural and physiological disturbances to the developing brain, poor developmental outcomes, inability of the organism to return back to homeostasis following stress exposure, delayed and sluggish response to stress, continuously elevated levels of GCs and the accompanying aforementioned disturbances and allostatic load (Kim et al., 2018; McEwen & Stellar,

1993; McEwen & McEwen, 2017). Exposures to deprivation, adversity and experiences of low SES and SED have been linked to exposure to repeated, severe and continuous stressors, frequently resulting in the ill-health outcomes and psychological diseases, some of which are outlined in this section.

Lastly, it is important to note that exposure to SED and ACEs has also been linked to "progeria", or accelerated ageing, and gene modification, measured by telomere length, histone modification, DNA methylation, and non-coding RNA. Consequently, epigenetic mechanisms appear to be exacerbated by chronic stress exposure, resulting in changes to or impairment of typical development and age-related physiological capacity (Lang et al., 2019). Epigenetic factors are also involved in the susceptibility of the organism to stress-related disorders, whilst the long-term effects of their modification have implication for later mental and physical outcomes (Griffiths & Hunter, 2014; Lang et al., 2019).

2.2.5 Maintenance and causation of socioeconomic disadvantage

Foetal origins theory (biological embedded model) proposed by Barker (1990), suggests that a woman's exposure to extreme psychological or physical stress during a critical prenatal period can cause long-lasting changes for child's neurodevelopment, resulting in at least partial placental dysregulation and exposure to elevated levels of circulating cortisol. Consequently, disturbances to neurobiological mechanisms lead to increased risk of physical diseases such as diabetes, cardiovascular diseases, hypertension or psychological illnesses like schizophrenia, anxiety, and depression in offspring (Eiland & Romeo, 2013), whilst indirectly influencing cognitive development in early stages of foetal programming (Fitzgerald et al., 2020). This frequently results in a higher risk of future negative life outcomes such as lower educational and occupational status, and potential subsequent danger of reoccurring SED exposure (Chase-Lansdale & Brooks-Gunn, 1997; Reiss et al., 2019). The negative long-term implications of SED for those individuals experiencing early year adversity or foetal programming and their associated high levels of stress, are often understood through the underlying neurobiological and structural disruptions of the brain (Kaiser et al., 2018). Research focused on neurobiological disruptions and micro and macro

structural changes of the brain reveal abnormalities in the previously outlined three critical brain area – the amygdala, the hippocampus and prefrontal cortex (Kaiser et al., 2018). The amygdala acts as a sensor of threats and involved into emotion regulation. Continuous exposure to stress and elevated cortisol levels has been associated with smaller amygdala size, resulting in physiological and behavioural stress response hyperactivity (McEwen & McEwen, 2017). Additionally, this might also result in behaviours such as aggression, hostility and anxiety, a manifestation of the emotional dysregulation.

The hippocampus is the second important area associated with learning, memory, and stress regulation (Jacobson & Sapolsky, 1991; McEwen & McEwen, 2017). However, its function in stress regulation is not rapid like the amygdala's but rather regulatory, expressed by glucocorticoid secretion regulation. Consequently, the brain region is crucially involved in the process of negative feedback (returning the activation of the HPA-axis to baseline) and is associated with high glucocorticoid receptors density (McEwen & McEwen, 2017). Excessive stress is therefore associated with hippocampal impairment (e.g., smaller hippocampal volume) resulting in the reduction of glucocorticoid receptors and impaired regulatory function. This in turn exposes the individual to higher risk of allostatic load, slower and sluggish responses to stress (slow activation when exposed to harm/risk), but also threatens to prevent the organism from returning to homeostasis and by that contribute to the accumulative nature of chronic stress. All these physiological implications are accompanied by behavioural and cognitive deficiencies such as linguistic or memory impairment, frequently resulting in the aforementioned long-term complications such as lower educational attainment.

Lastly, the prefrontal cortex is involved in higher executive functioning and behaviour regulation, and is also directly involved in the stress regulatory system by medial prefrontal cortex which has inhibiting function on the activation of the amygdala and hippocampus (Kalisch et al., 2006; Radley et al., 2006). Furthermore, the orbitofrontal cortex appears central to emotional regulation and appraisal by its direct effect on the amygdala, and subsequently the HPA-axis and autonomic stress responses (Milad &

Rauch, 2007). Chronic stress exposure has been associated with structural atrophy of the medial prefrontal cortex, neurogenesis suppression and increased orbitofrontal cortex activation, all of which are neural risk markers of a wide variety of mental disorders such as antisocial behaviours, depression and anxiety (Coccaro et al., 2007; McEwen, 2005; Mervaala et al., 2000; Shin et al., 2006). Importantly, brain morphometry abnormalities, measured by cortical thickness, white matter structure and grey matter volumes in these three brain regions, have been associated with severe psychiatric disorders (Phan et al., 2009; Price & Drevets, 2012).

All these biological and behavioural implications of chronic stress associated with SED appear to be the determinants of the intergenerational transmission of SED or poverty (Harper & Marcus, 2003). Crucially however, not all individuals exposed to elevated levels of stress develop neurobehavioral dysfunction (Romeo, 2015). The determinants that moderate the mechanisms of resilience are ambiguous but seem likely to include a complex interaction between environmental, biological and genetic factors (Rutter, 2006). The relationships between these "resilience" psychological factors and participants' endocrine response will be further explored in this thesis, whilst the definition of the term resilience within the context of this work will be also provided. Resilience factors such as social capital and social support have also been seen as crucial for the breakout of the intergenerational poverty cycles (Harper & Marcus, 2003). Nevertheless, the literature argues that escaping this cycle is not necessarily as easy as it has been earlier suggested by research (Binder, 1999; Corcoran, 1995; Solon, 1999).

It is important to note that the mechanisms through which intergenerational transmission of poverty and disadvantage may occur is not entirely and solely related to the private transmission of poverty (i.e., foetal and biological programming/exposure to stress; or from parents to offspring). Transgenerational and persistent poverty is also linked to public aspects such as transfer/lack of transfer of resources (i.e., tax income redistribution from previous generations towards educational resources for younger ones). This aspect is referred as "public" transmission of poverty or SED (Harper & Marcus, 2003). This review of literature,

however, will not focus on the macro level factors and the public transmission of disadvantage, but will rather concentrate on the biological consequence of living in SED and the mechanisms through which these could become the antecedents of it.

A key question therefore concerns the extent to which biological parameters might be modified through behavioural or cognitive interventions in a manner that is enduring and improves the quality of life. Furthermore, when, where and for whom should the intervention be developed are salient questions that policymakers must consider. Any attempt to develop a coherent strategy of intervention has to be set on a solid foundational understanding of the neurobiology of disadvantage. Despite extensive and sophisticated literature examining the deleterious effects of chronic cortisol exposure and subsequent effects on immune function, studies directly linking cortisol with poverty and inequality frequently employ basal measures. And yet, Sapolsky (2004) describes the shifting dynamics of cortisol reactivity to stress amongst dominant and subordinate primates, patterns of response which may be subdued, exaggerated, rapid or sluggish in returning to baseline. It seems entirely likely that cortisol reactivity is a better predictor of who is more susceptible to stress-reactive psychopathologies and responsive to intervention (Al- Dujaili & Sharp, 2012). Understanding whether atypical cortisol responses are encoded during foetal development and resist modification in the face of social experience is imperative if cognitive/behavioural interventions are to be designed effectively. There is some suggestion foetal programming of cortisol reactivity and negative effects of early childhood deprivation can be overridden during adolescence, but this remains controversial (Makes et al., 2020).

Whilst this section explored the biological implications of SED, the next aims to tease out some of the complex issues which affect those living in more affluent societies. Whilst most – if not all - people from developed countries no longer live in absolute material deprivation, many still experience poorer health outcomes, higher mortality and morbidity rates, are more susceptible to disease, and have shorter life expectancies than their fellow countrymen. The subsequent section therefore explains how susceptibility to status rank, control over life that comes with social status, and

the opportunities to engage and participate in society translate into health, wellbeing and longevity.

2.3 Status

The previous sections have explored the complexities and challenges around the definitions of various measures of social inequalities and the implications of their utility for the health inequalities research. Moreover, the diverse explanatory perspectives and frameworks applied to better understand the nature of health inequalities and the repercussions for social and health policy makers concerned with the health gap between socioeconomic status groups have been considered. This section now further examines the link between social and health inequalities through the prism of the social gradient in health or "status syndrome" (Marmot, 2004), with that being the framework used to explore the biological mechanisms underpinning the gradient in this research.

Whilst a wide range of social, psychosocial, biological and economic determinants of health have been explored within the field of health inequalities, the precise mechanisms through which SED 'gets under the skin' are still poorly understood and require further scientific clarification (Haushofer, 2011; Marmot, 2004; Peppers & Nettle, 2017; Sapolsky & Share, 1994). That being said, literature suggests stress as the major underlying mechanism in the link between social inequalities and health and thus particular attention has been paid to stress and the biological consequences of SED in earlier sections. Nevertheless, the transmissional nature between psychosocial determinants and the biological implications of living in disadvantage shall be expanded on. Whilst doing so, the thesis shall not merely consider stress as a framework but also the cognitive, psychological and biological aspects of 'status syndrome', as the core underpinning mechanism of status disparities and the resultant health inequalities.

Albeit people in Western industrialized societies no longer live in absolute material deprivation, they still experience the impact of a highly stratified society. Consistently,

social status within distinct structures is believed to have significant and widespread psychological and behavioural implications (Gordon & Townsend, 2000). As previously discovered, what is problematic with being poor in a materially rich society is not poverty per se, but the subordinate position of the disadvantaged in the social hierarchy. It is less about being poor and lacking economic means, and more about feeling poor and more precisely feeling more impoverished than those who surround you. Marmot (2004) argues that what is crucial for individuals' health and well-being is where they stand in relation to others in society. Wilkinson and Pickett (2018) also suggest that the mechanism underpinning the social gradient in health results from psychosocial factors such as subjective experience of social rank and relative position on the social ladder. Furthermore, even though Wilkinson (2000) and Wilkinson & Pickett (2010) argue that poorer health and greater inequalities are more apparent within more unequal countries such as the UK and US, the earlier discussion on the "relative health differences" amongst more egalitarian societies such as the Nordic countries should also be taken into consideration when making similar assumptions (Benach et al., 2003). The idea of relative health differences is thus consistent with Marmot's argument that the social gradient in health exists even within the most equal societies (Marmot, 2004, 2015).

Marmot (2004) further illustrates that whilst health disorders are concentrated in low SES populations, they are not confined to these groups; indeed, they occur throughout the various strata of society. This refers us back to the concepts of relative status/rank and relative deprivation/poverty as measures of social inequalities in health; whilst additionally clarifying that the effects of inequalities are not confined in any binary or categorical way to those living in poverty but affect everybody in the stratified societal structure (Marmot, 2004, 2010). Marmot refers to this as the social gradient in health emanating from 'status syndrome' (Marmot, 2004, 2010), or the psychological proposition that social comparison and status drive are deeply and evolutionary embedded in human nature. Correspondingly, Marmot implies that a health gradient runs right across society rather than only impacting upon those categorised as 'rich' or 'poor' determined by social differences in society such as social rank.

The social gradient in health (particularly observed in modern Western societies due to the widening gap between SES groups) is a robust illustration of how social inequalities and social rank predict patterns of disease in humans. Gradual deterioration in SES predicts a major rise in the prevalence of morbidity and mortality (Adler et al., 2000; Siegrist & Marmot, 2004). Among US states and cities, communities with greater income inequality have poorer health than the ones that are more equal, even when individuals from the same SES were compared (Wilkinson, 2000). This implies that income does not solely define health, as previously explored, but that the causal pathway between ill-health and social status/rank is rather complex. Further, the social gradient in health cannot be simply explained by factors such as limited access to health care resources (Sapolsky, 2005, 2017); indeed, only a small proportion of the SES-health relationship is reputedly explained by SES-related lifestyle disparities (Marmot, 2004).

Behavioural determinants such as higher rates of alcohol consumption, smoking, less healthy nutrition, fewer coping outlets such as holidays or gym memberships, crime-and-toxin afflicted communities and inactive lifestyles have also been proposed to explain elevated levels of mortality and morbidity across lower SES populations. However, even when considered collectively all these factors account only for a small portion of the variability in the socioeconomic-health gradient (Marmot, 2015; Siegrist & Marmot, 2004). Hereafter, the implications of stress in relation to the exposure to low SES and the frequently accompanying it factors - unpredictability of the events and the environment, the lack of control over life, social exclusion, and limited social support or social capital (as referred to by sociologists) should also be taken into consideration. The importance of the relationship between these concepts and SES, and their role as determinants of health (Haslam et al., 2018) will be further discussed as this review of literature proceeds. Correspondingly, Kunitz (1990, 1994) argues that a degree of "particularism" should always be applied in the attempt to understand the social determinants of population health. This also returns us to the idea of applying meta-theoretical frameworks when endeavouring to understand the disparities in health (Benach et al., 2003; Smith & Egger, 1996).

And yet, the field of health inequalities tends to heavily lean towards the psychosocial interpretation of the social disparities in health (Lynch et al, 2004.). This becomes apparent when looking at the work of well-established researchers such as Marmot, Wilkinson and Pickett. The framework derives from the perception-depended responses to status disparities, which ultimately translates into health disparities via various complex bio-psychosocial mechanisms. The explanatory model appears to be one that is highly accepted by the broader research community, and social and health policy makers due to its major contribution for the general rationalisation of health disparities within diverse contexts (Lynch et al., 2004). The framework has also been strongly supported by the primate literature on status and the stress-related diseases associated with hierarchy subordination (Sapolsky, 2004). Thus, links between natural social hierarchies among non-human primates and health inequalities research, articulated within Sapolsky's baboon studies and the Whitehall studies of British civil servants, have been drawn:

"The Whitehall and Serengeti studies are in a sense starting from opposite ends of a possible bridge. While the baboons show hierarchically associated variations in physiological responses to stress that are consistent with health effects, the civil servants show hierarchical variations in health outcomes that must emerge from some physiological pathway." (Evans, 2002, p.40)

As a consequence, the process of psychosocial relationship based on social rank perceptions has been argued to be an evolutionary, naturalistic and generalizable process which can be applied to human hierarchies (Adler & Ostrove, 1999; Clarkson et al., 1989; Marmot & Wilkinson, 2001; Sapolsky, 1999b, 2017; Wilkinson, 2000; Wilkinson, 1999; Wilkinson, 1997). This, however, does not come without criticism, including claims of cherry-picking selection processes within animal/primate literature (i.e., not acknowledging the diversity of hierarchical structures and the lack of universality of the implications of low rank for health between the different types of hierarchies), bias of positive findings, lack of systematic literature reviews on non-human primate studies and the complexity of human social structures (Lynch et al., 2004; Petticrew & Smith, 2012). Interestingly, similar critiques also have particular

limitations and more particularly their incomplete review of physiological biomarkers in relation to status-related differences in diseases, and lack of account for the wide variability of hierarchies and their diverse implications for status-rank.

The complexities around human social structures, the variability of animal hierarchies and their utility within societal groups will be further investigated in this thesis. The next sections will also discuss the animal and primate literature on the consequences of low rank in support of the social gradient in health. Moreover, psychological resilience factors frequently defining status rank within human hierarchies will also be reviewed and proposed as an alternative argument aiming to unpack the less straightforward link between subordination and stress-related diseases in humans. As much of the existing literature predominantly focuses on the negative association between status rank and health, it is worth noting that habituation to status rank may not only manifest in the form of negative outcomes but also in acclimatisation to the environment, where individuals are not psychologically disturbed by the limited resources the rank offers. This should be considered when unpacking the complex relationship between rank and health, and when posing the question as to why not all individuals living in SED encounter ill-health. This will be further explored within the 'resilient factors' and 'satisfied poor' sections, where forms of habituation to the environment are considered.

An important issue that has not been previously raised in the earlier sections, but which requires consideration is the saliency of "relative distance" comparisons (Wilkinson, 1997). This position queries whether individuals compare themselves to other groups of similar status, or is social comparison applied globally and generally to everyone regardless of SES and circumstances. It appears that status comparison is most powerful when based on local comparison, as suggested by Messner and Tardiff (1986) and Anderson and colleagues (2012) who argue that comparisons within local neighbourhoods are of greater importance for these conceivably health-damaging comparisons than those on national or state levels. Nevertheless, the authors also emphasise the significant role of variables such as gender, age and ethnicity when making relative social comparisons, meaning that what matters is the relatability

depending on identification, as well as on exposure to and availability of comparable information on those who one identifies with. All this being said, it is noteworthy that social comparisons implicitly measured on a global-level scale have higher validity and closer relationship to health, as suggested by income inequalities research (Lynch et al., 2004). For example, measurements of income inequalities in the US appear to have a closer link to health than those undertaken at local level (Subramanian et al., 2003). Similarly, US child mortality rates have been more closely associated with national rather than state income levels (Hillemeier et al., 2003). This does not necessarily mean that individuals do not apply multiple comparisons, or merely limit themselves to those in their close proximity (Lynch et al., 2004). The debate around relative distance comparisons therefore remains slightly inconclusive in literature and requires some thought when applying it to different contexts.

The results from the first Whitehall study of British civil servants (the origins of the idea of status syndrome) suggest that mortality and morbidity are dependently linked to levels of employment, whilst the second study concentrated on social determinants of health and diseases, applying a more rigorous recruitment process by including women in the research (Marmot et al., 1978; Marmot, 1991). Results from the second investigation shed some light on the link between employment and associated factors such as control/or the lack of it over the job and health status (Marmot, 1991). A wide variety of biomarkers have been utilised in the studies in order to evaluate individual health statuses, risk factors and causes of mortality in relation to the employment status. Of particular interest for this study is the presence of GCs as biomarkers of stress and the implications of social rank for the Whitehall participants' health. The emerging social rank-glucocorticoid relationship has been consistent with the primate literature on subordination and stress-related diseases (Marmot, 2004, 2015). Individuals of lower SES face higher risk of mortality and disease due to limited access to material and psychological resources, this in turn directly translating into psycho-neuroendocrine reactivity and/or stress-induced behaviours which indirectly lead to poorer health outcomes. This being said, the researchers did not account for an endocrine biomarker directly involved in hierarchy formation, such as testosterone and its implications for an individual's status, and behavioural outcomes. Thus, the

current research aims to address the gap in the literature whilst providing an additional biomarker and mechanism, within the context of threat/challenge cognitions, through which the implications of rank for health and behaviour could be better understood. Furthermore, implications of status upon endocrine response and health are not considered to be a one-way street (Knight & Mehta, 2014). For instance, circulating endocrine levels might be influenced by the social environment causing a rise or drop in status, which subsequently impacts health and behaviour. Fluctuations in endocrine levels may also result in subsequent effects where endocrine reactivity impacts status-seeking behaviours (Mazur & Booth, 1998). Consistently, Kemper argues that whilst other species are "overwhelmingly restricted to bio-social limitations", humans are "socio-bio-social species, to a significant extent freed from an immutable biology that lays down social arrangements by genetic fiat" (Kemper, 1990, p.2). Resultantly, endocrine mechanisms also influence future behaviours, such as status-seeking behaviours, and are thus rooted in the formation of status hierarchies. The discussion around the implications of endocrine responses for status-seeking behaviours and acquisition of status rank will be further expanded later in this work.

2.3.1 Status hierarchies

Smith and colleagues (2011) suggest that social stratification and hierarchies emerge universally and are recognised by everyone. Mazur (1973), for instance, portrays the status hierarchies and their multi-dimensional characteristics whilst arguing that "Kinship, courtship and consort linkages all provide additional structure, sometimes relating to status hierarchy and sometimes independent of it. Interaction…may be shaped by these…links more than by…status relationships" (Mazur, 1973, p.380). Albeit it cannot explain all social behaviours, dominance appears to be an important element of social structures in many different species, including humans (Cosmides & Tooby, 1987; Kokko et al., 2008, Murray, 2021; Trivers, 1972). This is evident in the universality of dominance hierarchy formation, emanating from status disparities (Dunbar, 1988; Mazur, 1973; Murray, 2021). This might be because animals living in groups do not only suffer the negative outcomes of the hierarchical structures (i.e., low status rank), but they also benefit from increased productivity, decreased predation harm, and availability of resources such as access to food and mates, all of which may be

facilitated, in part, by a hierarchical social structure (Camazine et al., 2020). Yet, where there is a hierarchical system, there is also a competition over pivotal resources, frequently resulting in conflict (Darwin, 1859).

Consequently, contests of dominance between animals commonly result when two unrelated animals are placed within the same setting, also referred to as the standard experimental resident-intruder paradigm. Diverse forms of interactions emerge subsequently: from violent physical combats to nonviolent recognition of higher and lower ranks between the individuals (van Vugt et al., 2015). These various interactions might proceed over the course of hours but also of weeks (Sharp, 2006). Determinants such as group size and animal class play a role in the frequency and the durations of the contests, eventually resulting in a stable hierarchical structure with dominants and subordinates (Chase, 1974; Wilson, 1975). Once this happens, the group structure is organised in a balanced way where competition does not outweigh the benefits of living within the pecking order (Richard & Schulman, 1982).

Such hierarchical group living translates directly into human structures, albeit not without its complexities (Gesquiere et al., 2011; Sapolsky, 2005). The differences between animal and human hierarchies will be further revealed in the human hierarchies' section. Nevertheless, the main argument against the transactional evolutionary nature of hierarchical structures from animal species to human beings emanates from the social constructionist approach which posits that culture content, rather than social structure, appears as an explanatory priority (Kemper, 1990). This culture content account proposes that power and status indirectly influence social structures through the establishment of ideologies and leveraging language (Gagnon, 1973; Tiefer, 1978).

Building on the established strong similarities between the basal hierarchical structures of animals and human beings, the next point of this literature review will address the variability of hierarchies and their level of sophistication.

2.3.2 Varieties of hierarchies

There is a wide variety of hierarchies that exist across species (Sapolsky, 2004). For instance, hierarchies can be gender-specific or involve both sexes, hereditary or labile, linear or contain circularities, or situational with rank fluctuating as a function of the resource contested or the presence of allies. Further, hierarchies might differentiate on the basis of the stressor type accompanying status rank (Sapolsky, 2004). Hereafter, one must consider the wide diversity of implications individuals might experience as a top or bottom rank within one or another type of hierarchical system. Instead, the implications of low rank (limited access to critical resources and disproportionate burden of stressors, and the supplementing physiological, psychological consequences) are not universally distributed across different hierarchical structures (e.g., marmosets and tamarins). Indeed, in some ranking systems it is not the subordinate animal which experiences the highest levels of stress, resulting from constant physical and psychological threats, and the accompanying physiological indices of stress (Sapolsky, 2004). This is because whilst in a traditional stable hierarchy, it is the dominant animal that might impose high levels of psychological stressors, in cooperative breeding species subordination is not associated with disproportionate share of stressors due to the fact that power is not forced from above (or from an animal of a higher rank) (Altmann et al., 1995; Blanchard et al., 1995; Sapolsky, 2004). Amongst marmosets for instance, there is an alpha individual who takes dominance over others, whilst all other relationships within the hierarchy system are equal with no gradations of rank (Sapolsky, 2017). Cooperative breeding and lack of gradations of rank has also been observed in animal species such as tamarins, white-browed sparrows, naked mole-rats, wild dogs, dwarf mongooses and Florida scrub jays (Abbott et al., 1998; Faulkes & Abbott, 1996; Creel et al., 1996; Creel, 2001). This variability is an example of how social context affects the "meaning of rank" (Sapolsky, 2017).

Another component of hierarchies which varies among species is the mechanism through which dominant animals acquire and maintain rank. For instance, amongst female baboons a kin selection occurs allowing rank to be inherited, whilst their male counterparts utilise physical conflicts and competition to attain rank. Whilst

hierarchies vary between sexes, of bigger interest is the question how rank is maintained once obtained; a challenge that is more complex than merely engaging in conflict and competitions to achieve rank and which involves the possession of skills such as social competence. Within classes like birds, primates, ungulates, and fission-fusion species, maintaining dominant rank requires social skills allowing the individual to balance between social complexities. This idea of primate social complexity and the brain development has been proposed by the British anthropologist Robin Dunbar and coined as the social brain hypothesis. The theory argues that these social complexities directly feature in the brain development of individuals, with dominant animals (living in rich ecosystems and variable group size) displaying larger brain in relation to the body ration but also larger neocortex in relation to the brain ratio. Further, in some of these species (e.g., fission-fusion), group size might vary drastically within the hours of the day, allowing rapid changes in ranks (Sapolsky, 2017).

Furthermore, the behaviours displayed by individual animals in order to acquire rank within a hierarchy also vary between species. This directly features in the psychological and physiological experiences of the rank within a hierarchy, not just for the dominant animals but also for the subordinates. For instance, the experiences of rank are highly dependent on factors such as how often the dominant animal is being challenged, the frequency of rank change and existing coping outlets for subordinate individuals such as kin relationships and grooming opportunities. Consequently, in subordinate species with limited coping outlets, subordination often resembles social defeat, lack of control and learned helplessness; all features demonstrated in individuals experiencing low SES within human hierarchies. Without doubt, this results in physiological indices such as elevated levels of GCs (Sapolsky, 2017). The concepts of learned helplessness, hopelessness, social defeat and lack of control will be further reviewed in the next few sections of the thesis. Accordingly, the link between physical and psychological indices of stress and status rank are profoundly impacted by the subjective perceptions of status rank individuals (either dominant or subordinate) experience within a hierarchical structure (Abbott et al., 2003; Sapolsky, 2004).

Lastly, instability of hierarchy could influence the experiences of status rank within structures. Events such as emigration, immigration or death of an individual with a central role amongst wild animal populations, or the creation or disintegration of coalitions can lead to dominant individuals no longer displaying the fewest stressors and thus lowest levels of basal GCs (Coe et al., 1979; Chamove & Bowman, 1976; Gust et al., 1991; Keverne et al., 1982; Mendoza et al., 1978; Sapolsky, 1993a). When such occurrences take place individuals of highest rank are the ones that begin to experience loss of control, less predictability, more physical stressors and the associated high levels of glucocorticoids rather than the ones obtaining lower rank. In contrast, when in captivity, animal hierarchy instability might occur within the first months of the group structure formation, subsequently affecting levels of glucocorticoids in dominant individuals (Sapolsky, 2004). Accordingly, these elevated basal glucocorticoid levels in dominant males during periods of hierarchical instability are associated with increased prevalence of coronary artery atherosclerosis, immunosuppression, highest risk of respiratory infections and highest testosterone levels (atypical for dominant individuals in a stable hierarchy) (Manuck et al., 1995; Masataka et al., 2010; Sachser & Prove, 1986; Rose et al., 1972; Eberhart & Keverne, 1979; Coe et al., 1979; Mendoza et al., 1979; Sapolsky, 1993a). The existing literature goes some way to articulate the complexities and variabilities of hierarchical structures and perhaps answer the inconsistent findings observed among various species regarding the link between basal GCs and status rank – something that has been discussed by Petticrew and Smith (2012) in their critique of the primate and epidemiological literature.

Nevertheless, when exploring social structures as complex as human hierarchies - which include consideration of social networks and culture and their impact on status for the individual - one should closely examine factors such as multiple hierarchies or the existence of more than one hierarchical structure of value for the individual. This further raises questions such as: If multiple hierarchies emerge within an individual's existence, does the implication of low rank within one hierarchy have a profound effect on the whole individual existence or do other ranks compensate for it? In the

latter case, inhabiting multiple hierarchical positions of varying valence may positively impact or act as a protective, resilience factor for physiological and psychological health and wellbeing of the individual. Moreover, if multiple ranks exist within human hierarchies, which one appears to be of the greatest value for the individual? Sapolsky (2004, 2017) argues that individuals value most the hierarchy within which they appear to be of highest rank. This will be further investigated in the sections of human hierarchies and multiple ranks later on in this literature review. However, before that, the implications of status rank in animal hierarchy models will be explored.

2.3.3 Biological consequences of social rank - animal models

Social rank has a robust impact on physiology and behaviour (Jimenez et al., 2017). It has been shown to have significant effects on individual susceptibility and resistance to diseases (e.g., Abbott et al., 2003; Kaplan, 2004; Morgan et al., 2002; Sapolsky, 2005; Virgin & Sapolsky, 1997) and can significantly influence the quality of life (Sapolsky, 2005). Nonhuman primate social groups have proven useful for studying status-related differences in disease vulnerability and resistance, with differences linked to predictable variation in physiological, neurobiological and behavioural characteristics. For example, socially subordinate monkeys are more susceptible to immune, cardiovascular, and reproductive dysfunction than their dominant counterparts (Cameron, 1997; Cohen et al.,1997; Kaplan & Manuck, 2004). Numerous nonhuman primate social groups are characterised by linear dominance hierarchies, where dyadic agonist interactions are formed and maintained through aggressive, submissive and affiliative behaviours (Kaplan et al., 1982). Dominant monkeys are typically the ones who obtain greater control over resources and maintain their superior status via physical aggression and/or intimidation; whilst subordinate individuals often experience a scarcity of resources, reproductive deterioration and fewer coping mechanisms (Kaplan & Manuck, 2004). It has been suggested that neurobiological and physiological differences observed between subordinate and dominant primates demonstrates that relatively greater amount of stress is experienced by subordinate monkeys (Henry & Stephens, 1977). However, recent research illustrated that the amount of stress experienced by different monkeys varies with allostatic load (Goymann & Wingfield, 2004; Haslam et al., 2018). In such hierarchies, stress

fluctuates extensively in accordance with the social structure, along with the hierarchies' stability, the availability of social support to subordinates and dominance style, namely whether dominance is maintained through aggression or non-physical intimidation (Abbott et al., 2003; Clarke et al., 1995; Sapolsky, 2005). Importantly, it has been demonstrated that social rank is the determinant of physiological outcome, rather than vice versa (Sapolsky, 2005, 2017). This is evident through various studies of individual captive animals where physiological profiles of singly housed organisms do not predict their ranks when they are subsequently placed within in a social group (Morgan et al., 2002). Frequently though the physiological and psychological implications of subordination determine future rank position, and thereby are no longer merely the consequences but also might become the antecedents of social rank.

Several stress-related physiological parameters have been found to be sensitive to social rank. GCs blood level is the most frequently studied endpoint. GCs are adrenal steroid hormones that are secreted during stress (i.e., cortisol or hydrocortisone in primates and corticosterone in many rodent species). They typify the double-edged nature of the stress response, as they facilitate adaptation to short-term physical stressors (i.e., fight/flight response), but nevertheless, are pathogenic when elevated secretion becomes chronic (Sapolsky, 2000). Consistently, animals who are more socially stressed within the dominance hierarchy show indices of hyperactivity of the GCs system. These indices encompass elevated baseline levels of GCs, enlarged adrenal glands that co-occur with the increased secretion, a sluggish GC stress reactivity in the face of a major homeostatic challenge and impaired system sensitivity to negative feedback regulation (Sapolsky, 2000, 2004).

In certain circumstances, it is the dominant animal that demonstrates this response. This involves species where dominant individuals have to repeatedly and physically reassert their rank, such as African wild dogs, male chimpanzees and female ring-tailed lemurs (Cavigelli, 1999; Creel et al., 1996; Masataka, 2010), those that are cooperative breeders (e.g. untamed wolves and captive marmosets and tamarins) (Abbott et al., 1998) and those with transient periods of major rank insecurity (untamed baboons and captive populations of rhesus monkeys, talapoin and squirrels)

(Sapolsky, 1993b). Controversially, the abovementioned is also observed among subordinate individuals in species where the hierarchy is stable and the high rank is maintained through nonphysical intimidation (e.g., feral male baboons, rhesus monkeys, rats and mice) (Eberhart et al., 1983; Barnett, 1955; Sapolsky, 1993b). Additionally, subordinates exposed to persistent social stressors with low availability of social support and insignificant presence of kin also experience the greatest physiological indices of stress (Cavigelli, 1999; Abbott et al., 2003).

Chronic stress exposure has a detrimental impact upon cardiovascular function, generating 1) hypertension and elevated heart rate; 2) platelet aggregation and increased circulating levels of cholesterol and lipids, jointly promoting atherosclerotic plaque formation in injured blood vessels, 3) vasoconstriction of damaged coronary arteries and 4) decreased levels of protective high-density lipoprotein (HDL) cholesterol and/or elevated levels of endangering low-density lipoprotein (Sapolsky, 2005). Additionally, organisms who are more socially stressed exhibit a sluggish activation of the cardiovascular stress response after stress exposure and also deferred recovery after exposure. Moreover, they demonstrate basal hypertension, a pathogenic cholesterol profile and increased vulnerability to the atherogenic effects of a high-fat diet (Sapolsky, 2005). Prolonged stress exposure also inhibits reproduction (Sapolsky, 2004, 2017). Amongst females it is associated with delayed puberty, decreased progesterone and oestrogen levels, increased incidence of anovulatory cycles, greater risk of miscarriage, impaired fertilization, prolonged interbirth intervals and increased reproductive senescence. Moreover, studies of feral baboons propose that higher rates of miscarriages are related to a subordinate position within a stable social structure. However, the literature remains ambiguous on this point (Altmann et al., 1995; Packer et al., 1995).

Lastly, chronic stress exposure (including social stressors) and elevated GCs levels are frequently associated with immunosuppression and the resultant increased risk of infectious diseases such as reactivate latent viruses (Ader et al. 2001; Cohen et al. 1991). Whilst some findings suggest a strong link between chronically elevated levels of GCs and high risk of susceptibility to infectious pathogens, the research on stress and

severe infectious diseases remains ambiguous (Capitano et al., 1998; Cole & Kemeny, 2001; Sapolsky, 2004). Nevertheless, a broad body of literature has demonstrated a clear relationship between stress and the suppression of the immune system basally (Dhabhar, 2014; Glaser & Kiecolt-Glaser, 2009; Padgett & Glaser, 2003; Schneiderman & Baum, 2018; Seiler et al., 2020). This demonstrates very evident and clear biological consequences of obtaining low rank within an animal hierarchical structure, frequently echoed in the human SES/health gradient. The following sections will consider psychological and behavioural implications of low status rank within animal and human hierarchies.

2.3.4 Learned helplessness, Learned hopelessness and Lack of control

Amongst socioeconomically disadvantaged populations, an inability to break the cycle of SED with the increased prospect of prolonged exposure to multiple physical and psychological stressors, may result in learned helplessness (Seligman, 1975), hopelessness (Minkoff et al., 1973; Wright & Beck, 1983) and lack of control. Learned helpless is a behaviour exhibited when individuals are repetitively exposed to aversive stimuli which is beyond their control to avoid (Seligman, 1975). Earlier research suggested that learned helplessness was acquired when individuals accepted their lack of control over aversive stimuli and no longer made attempts to avoid the stimuli, even in circumstances when they were unambiguously avoidable (Carlson et al., 2010; Nolen, 2014). Low SES populations are more frequently subjected to predominantly stressful, uncontrollable and unavoidable negative life events (Kim et al., 2018; Maier & Seligman, 2016), all of which are suggested to be determinants of learned helplessness (Seligman, 1975).

The theoretical model of learned helplessness proposes that people's inability to exhibit control over events or the environment frequently results in a learning process associated with the individual recognising that their responses do not affect situational outcomes (Abramson et al., 1978; Miller & Seligman, 1975). Repetitive exposure to uncontrollable events has been shown in a multitude of studies to result in consequent disruption of behaviour in both human and non-human primates (Fillippello et al., 2019; Hiroto & Seligman, 1975; Klein & Seligman, 1976; Landry et al.,

2018; Lennerlöf, 2020; Trindade et al., 2020) and comprises three narrowly linked deficits: cognitive, motivational and emotional (Seligman et al., 1979a,b). The cognitive deficit is associated with problem-solving skills and implies that the individual is unable to learn and utilise an appropriate response in a controllable situation. The motivational deficit often results in individual's lack of drive and effort, manifesting through the defeatist belief that regardless of skills and effort, one cannot change the outcome of a situation. The emotional deficit occurs in the form of depression. As these three deficits frequently occur as symptoms of depression, Abramson and colleagues (1978) and Seligman (1975) suggest that learned helplessness was also fundamental to the concept of reactive depression. It is important to note that more recent literature from the field of neuroscience suggests that the initial formulation of the theory of learned helplessness requires revision. Passive behaviour in response to aversive stimuli is not learnt but appears to be the default state of the brain, presuming control is not present (Maier & Seligman, 2016).

The authors thereby argue that this passivity can be overcome by individuals learning that aversive stimuli are controllable (Maier & Seligman, 2016). That is, individuals do not learn to be helpless following repetitive exposure to aversive stimuli, but this is rather a default state which nevertheless can result in a failure to learn to escape. Applied to the broader framework of socioeconomic disadvantage, this implies that due to the high degree of uncontrollability of events and the lack of control low SES groups often experience (Marmot, 2004, 2015), individuals might experience cognitive, emotional, and motivational deficits which render difficult any attempt to circumvent their circumstances which are frequently maintained by structural arrangements.

One of the criticisms of the model of learned helplessness is the lingering question over why every individual exposed to uncontrollable events doesn't develop depression and transfer the helplessness experience from one situation to another (Abramson et al., 1978). In response, Abramson and colleagues (1978) developed the 'learned theory of hopelessness depression', a subtype of depression, initially termed as the reformulation of the learned helplessness theory. The formulation of this theory not only elucidates the factors determining whether individual's learned helplessness

will be generalized across situations, but also lays the foundations for the common roots between learned helplessness, hopelessness depression and locus of control. As per these commonalities, attributional constructs appear to be central mechanisms for all the three concepts (Abramson et al., 1978; Furnham, 2009; Golin et al., 1981; Peterson et al., 1981; Schroder & Ollis, 2013; Seligman et al., 1979a,b). Resultantly, whether learned helplessness and/or hopelessness depression will be transferred cross-situationally, is highly dependent on the unpremeditated attributions the individual makes for those events (Peterson et al., 1982). Abramson and colleagues (1978) argue that a link between learned helplessness, hopelessness and depression can be drawn on the basis of attributional style. For instance, if an individual's failure to control events are attributed to themselves (internal attribution) rather than external factors/circumstances (external attribution), whilst holding the idea that these negative experiences will persist (stable rather than unstable attribution) and moreover, will be transmitted to and affect negatively other life domains (global rather than specific attribution), then learned helplessness, hopelessness and lack of control are more likely to occur (Abramson et al., 1978).

This amended model, however, is not immune to criticism. Indeed, the low correlations between the concepts of learned hopelessness and depression is the argument proposed to discredit the link between the models. Additionally, the utility of the factor - attributional style in this link has been questioned by researchers (Abramson et al., 1991; Anderson & Deuser, 1991; Barnett & Gotlib, 1988; DeVellis & Blalock, 1992; Peterson, 1991a, b; Peterson & Villanova, 1988). For instance, an attributional style such as internal-global-stable can, but may not, provide a sufficient trigger for hopelessness depression, suggesting that a broader range of factors (i.e., external factors such as social support), should be considered when establishing links between individual's vulnerability to depression and attributional style (Abramson et al., 1989; Rooke & Birchwood, 1998). This criticism overlaps to a great extent with the criticism of the initially formulated model of learned helplessness: arguing that not all individuals exposed to uncontrollable events and adversity or negative life events fell prey to depression and psychological defeat. This criticism points to the likelihood of "external factors" in development and susceptibility to depression. This will be further

recognised in the sections examining resilience or protective factors, status ranks and additional factors contributing to status perception (i.e., personality and subjective SES). Albeit the established correlation between hopelessness depression and individual's hopelessness has been weakened, this does not entirely refute the theory of learned helplessness per se (Schroder & Ollis, 2013). Indeed, a potential explanation for the low correlation between hopelessness and hopelessness depression might be the low utility of the attributional style diagnostic tool - the Attributional Style Questionnaire (ASQ) - as a predictor of depressogenic cognitive style (DeVellis & Blalock, 1992; Peterson, 1991a, b; Peterson et al., 1982; Peterson & Villanova, 1988). This stems from the fact that the three dimensions comprising the measure of attributional style are evaluated in isolation rather than synthesized and assessed as one undivided scale of depressive mode (Schroder & Ollis, 2013).

Beck and colleagues' work on hopelessness and its relationship to psychopathological conditions further point to a stronger relationship between risk of suicide and hopelessness can be established rather than between hopelessness and depression (Beck et al., 1985; Beck et al., 1974; Chochinov et al., 1998; Dyer & Kreitman, 1984; Ellis & Ratliff, 1986). Contrary to the instruments utilised to measure learned helplessness and hopelessness depression by Seligman (1975) and Abramson and colleagues (1978), the alternative Beck Hopelessness Scale (BHS) is not constructed on the basis of attributional theory and appears to be the most widely utilised measure of hopelessness nowadays (Velting, 1999). In fact, the BHS lays its foundations on three main aspects of hopelessness: feelings about the future, loss of motivation, and expectations. Nevertheless, the instrument was not initially designed to measure hopelessness, but rather individuals' levels of pessimism and negative attitude towards the future (Beck et al., 1974). BHS may therefore not be methodologically structured to identify differences and commonalities amongst disadvantaged populations prone to higher risk passivity towards uncontrollable events, lack of control and hopelessness.

This raises the fundamental concern regarding the measure of highest utility, allowing scholars to fully consider the complexities of the social inequalities in health. It is also worth mentioning that the variety of instruments utilised to measure hopelessness

across different population groups render different results, which might be another explanation for the reduced association between learned helplessness and hopelessness depression models (Beck et al., 1974; Glanz et al., 1995). For the purposes of this research, learned helplessness and hopelessness will be evaluated using the original Attributional Style Questionnaire – ASQ (Peterson et al., 1982). The questionnaire has been used across clinical and research populations, to measure both depressogenic attribution and academic performance respectively (Kleim et al., 2011). To my knowledge however, the questionnaire has not been utilised to measure differences in attributional style between SES groups. Nevertheless, the feelings of lack of control followed by exposure to multiple and continuous uncontrollable stressors predominantly observed across low SES groups implies that individuals experiencing disadvantage might exhibit higher passivity to escape aversive stimuli and score higher on specific attributional styles (e.g., internal-global-stable negative), further promoting passive failure to learn to escape due to limited control. Plausible differences in helplessness and hopefulness domains and attributional style between both status groups will be further revealed in the results section (section 5.2) of this thesis. Furthermore, the discussion section will also consider the implications of learned helplessness and hopelessness in relation to endocrine responses to stress.

The theory of learned helplessness directly feeds into the concept of lack of control and more precisely locus of control. Locus of control is defined as "the tendency of people to perceive that outcomes in a particular arena were either within or outwith their control" (McNabb 2003, p. 418). This stems from the fact that when exposed to uncontrollable and unpredictable negative life events associated with socioeconomic adversity, default cognitions may be assumed to presume that control over the situation and environment is lacking and thus a passive response to escape the circumstances is evident (Maier & Seligman, 2016). Importantly, whilst the concepts of hopelessness and helplessness have been used relatively consistently throughout research fields, the concept of control has been rarely recognised by sociological research and thus replaced by the term - powerlessness. Albeit defined differently, both terms refer to the same theoretical concept (Drew, 1990). The lack of uniformity and consistent terminology use across disciplines, alas, could cease the progression of

interdisciplinary research due to confusion and absence of clear communication of scientific results and conclusions.

Continuous exposure to stress and adverse environments has also been associated with a construct similar to learned helplessness, namely, social defeat. However, learned helplessness as a principle has been centred around the inability to escape negative events and absence of control, whilst social defeat focuses on social factors. Considering that social defeat has also been used as an animal model for depression in humans, and because of the close similarities between the model of learned helplessness and social defeat, such as their overlapping neurobiological implications, this thesis considers it of significant importance to review both models. This is drawn on the fact that as with learned helplessness, the concept of social defeat could be applied and considered within the framework of socioeconomic disadvantage. The next section thus aims to explore the construct of social defeat in animals and its relationship to the experiences of prolonged stress, adversity and socioeconomic disadvantage in humans (Hollis & Kabbaj, 2014).

2.3.5 Social Defeat

Social defeat is observed in humans and animals whilst considered a severe stressor finding its roots in the social dominance hierarchy. More precisely, social defeat encompasses the physiological and psychological implications resulting from repeated exposure to harmful and aggressive environments. The concept of social defeat originates from a series of experiments inducing the resident-intruder paradigm. When animals, typically rodents, are introduced to the cage of another dominant animal or group of animals of the same species a physical conflict occurs. Repetitive exposure to conflict and physical aggression is associated with chronic stress, threats and experience of defeat by the subordinate animal in the cage (Lehmann et al., 2020). The model has also been applied to acute stress response, where the animals are allowed to engage in conflict on a single occasion only. It is important to note that this is different from the continuous exposure to stress and more related to the ordinary stress response. The implications of social defeat for the subordinate individual as a result of repetitive exposure to threats from the dominant are therefore not simply

related to the physiological stress as discussed previously, but also behavioural implications (Bjorkqvist, 2001).

Consequently, severe implications such as social deficits, cognitive impairment, anxiety and depressive-like behaviours are related to long-term exposure to social defeat (Rohde, 2001; Selten & Cantor-Graae, 2005). Implications caused by social defeat differ from the ones triggered by other various stressors, however (Bjorkqvist, 2001; Meerlo et al., 1997). Whilst even a single exposure to social defeat has been associated with significant stress symptoms, long-term exposure has been linked to implications resembling those of depressogenic psychopathology (Koolhaas et al., 1997; Blanchard et al., 1995). Furthermore, Sgofio and colleagues (1999) suggest that in comparison to other threats, social stress triggers a shift in male rats from autonomic balance to sympathetic dominance, also associated with cardiac tachyarrhythmias. Additionally, the nature of social defeat has many implications for immunological and neurobiological and cardiovascular impairments (Alleva & Aloe, 1989; Abramchik et al., 1988; Biondi & Zannino, 1997; Cohen et al., 1996; Hyde, 1984; Kaplan & Manuch, 1997; Stefanski & Engler, 1999; Weiss & Sundar, 1992). It has also been related to health-related behaviours such as drug abuse, implying alterations in the serotonergic and dopaminergic (reward-stimulating) brain system, resulting in individuals being more prone to these health behaviours (Bjorkqvist, 2001; Huhman, 2006; Rygula et al., 2005; Laviola et al., 1999; Tidey & Mizcek, 1997). Most importantly for this thesis, however, are the implications of social defeat upon the neuroendocrine system, with social defeat being linked to steroid hormones and more specifically having effects on both corticosteroids and sex steroids.

Social defeat in subordinate animals has been related to impaired corticosterone response and fewer corticotrophin-releasing factor mRNA grains per cell compared to dominant individuals (Albeck et al., 1997). Furthermore, clear alterations in the hormonal and neurotransmitter responses have been observed as a result of social defeat. With continuous exposure to conflict in male rats resulting in winners displaying steady corticosteroid-binding globulin (CBG) concentrations and decreased total corticosterone, whilst losers have been associated with reduced CBG and

unchanged total corticosterone (Stefanski, 2000). These findings have been further supported by Otten and colleagues (2002), investigating the effects of winning and losing in pigs rather than rodents. Their results suggest that socially defeated pigs have experienced increased plasma epinephrine and norepinephrine, plasma catecholamines, adrenocorticotropic (ACTH) hormone, and heart rate compared to their counterparts. Moreover, losing conditions have been associated with decreased exploring behaviour and locomotor activity, suggesting social defeat implications are not confined to the physiological symptoms but also behavioural (e.g., fear and emotional distress) (Otten et al., 2002).

Studies of animals have been mirrored within human-focussed research, primarily examining dominance, defeat and testosterone (Knight & Mehta, 2014; Mehta & Josephs, 2006). Given the clear ethical implications of imposing social defeat upon humans, much of the current research relies on correlational and unclear links between human aggression, dominance, testosterone and social defeat. These studies often face methodological challenges and limitations including lack of baseline measurements, comprehensive sampling protocols and the use of biological material to determine hormone levels.

Some authors have argued that testosterone is related to dominance, status-related behaviours (Mazur & Booth, 1998) and aggression (Book et al. 2001). A particular emphasis has been placed on the link between aggression and testosterone levels in humans (Archer, 2006; Dabbs et al., 1987; Dabbs, 1997; Knight & Mehta, 2014) with higher levels of circulating testosterone generally being linked to higher rates of violence, social dominance among small group interactions and status related wins, or more generally wins in hormone status competitions (Ehrenkranz et al., 1974; Olweus et al., 1980; Mazur & Lamb, 1980). Nevertheless, the strength of this relationship remains uncertain with some studies supporting the links and others not (Björkqvist et al., 1994; Gadinger et al., 2011; Schaal et al., 1996; Tremblay, 1998; Vongas & Hajj, 2017). This inconclusiveness arises from the methodological issues and limitations of endocrine research (Sharp, 2006). For instance, the lack of comprehensive sampling protocols (i.e., single point sampling utilised), lack of established baseline and

evaluations of total rather than free testosterone. Methodological issues and the plausibility of the testosterone - status rank relationship will be profoundly discussed in the testosterone and challenge hypothesis sections of this thesis. Regardless of whether testosterone is reduced following social defeat or status loss, or it is the other way around, individuals with low basal testosterone levels are more easily socially defeated because they appear less threatening to others (Björkqvist, 2001). As such, the interaction between testosterone and dominance appears clear and further raises the question of which is the hen and which is the egg in the evolution and emergence of dominance hierarchies.

2.4 Evolutionary foundations of hierarchies

The previous section on status made links between status rank and health in order to provide further support for the social gradient in health. It did so by drawing on existing animal and primate literature which demonstrates that lower rank within hierarchical structures is frequently associated with negative health outcomes. The following section builds on this argument, emphasising that the relationship between status and health within human hierarchical structures is less straightforward due to the role of psychological factors in this relationship. Importantly, the section also demonstrates that although dominance hierarchy formation emanates from status disparities, social structures also have evolutionary purposes associated with positive outcomes for the individuals within those systems (i.e., access to food and mates). For this reason, this section explores the evolutionary foundations of hierarchical systems, their purposes and relations to status.

Status hierarchies emerge amongst non-human primates and other species. They appear to be omnipresent across modern and historical human societies respectively, with individual rank having effects upon access to resources in both animal and human ecospheres (van Vugt & Tybur, 2015). Resembling other species, evolved psychological mechanisms comprising of status strive and hierarchy navigation, seem to be embedded in the human nature. The function of these mechanisms is to motivate the individual to improve status position/rank in the hierarchical system, to

convert favourable status positions into fitness advantages (status capitalization), to monitor and appraise other members' position in the hierarchies (status assessment) and to manage and cope with both improved and reduced status (status management). Complex hormonal, cognitive, emotional, and behavioural systems' interactions coordinate those mechanisms (van Vugt & Tybur, 2015). Resultantly, natural selection therefore continues to have a role in the origins of hierarchies (van Vugt et al., 2008).

Broader evolutionary psychology frameworks suggest that the psychological systems underpinning status rank manifest in stimulus-response mechanisms that produce behaviours typically adaptive for the ancestral environment (Tooby & Cosmides, 2005). However, the arguments around which precise mechanisms construct this "modular status psychology" vary among evolutionary theories. For instance, some evolutionary theories propose individual competition as the foundation of status disparities, where an individual's interests act as a fundamental core of the status strive (Sapolsky, 2017). Williams (1966) foreshadows this, arguing that "the dominance-subordination hierarchy…is not a functional organization. It is the statistical consequence of a compromise made by each individual in its competition for food, mates and other resources. Each compromise is adaptive but not the statistical summation" (p. 218). This has also been supported by more recent evolutionary theorists highlighting the advantages of dominance ranking systems for both the individual and the group (Murray, 2021; van Vugt et al., 2008). Consequently, low status rank within a stable hierarchical structure might be more preferable than a high-status position in an unstable ranking system (Caporael, 1997; Ronay et al., 2012), and as a result one might argue that social hierarchies emerge as a product of selection operating not only at group but also at individual level (Wilson et al., 2008). Human social structures often show these same traits and organisational inclinations evident in non-human primate social groups.

2.4.1 Sexual selection theory

In Dobzhansky's famous dictum, nothing in biology makes sense except in the light of evolution (Dobzhansky, 1973). Thus, when we talk about biology, we need to have

some sense of how evolution has brought species to its current state. When considering evolution, one cannot neglect the important element of natural selection; that central mechanism of evolution which focuses on adaptation to facilitate survival. Presumably males and females face the same survival pressure – avoiding starvation, enhancing fitness and survival. However, none of that explains how individuals became sexually dimorphic. Males in general really are more violent than women, they really do instigate more wars, they tend to be more dominant and more likely to engage in financial corruption, be more assertive and risk-prone. Males are also more likely to fall prey to particular illnesses (Archer, 2004; Bauhr et al., 2019; Morrongiello & Rennie, 1998; Staniloiu & Markowitsch, 2012), whilst females cry more easily than men, are more sociable, display higher verbal abilities and are less openly confrontational (Buck, 1977, 1991; Chaplin, 2015). This raises important questions such as: do these differences emanate from our biology? If so, are the differences so significant as to think males and females are almost different species? (i.e., Men are from Mars). Alternatively, are any biological differences small, almost irrelevant and imperceptible but then subsequently amplified by socio-cultural and environmental expectations and effects?

Darwin acknowledged that natural selection couldn't answer the question of sex differences, subsequently proposing a second theory: sexual selection. As this theory attempts to explain why, broadly speaking, males are agentic and females passive, it was and remains controversial. Bateman (1948) thus drew on the reproductive differences between males and females, pointing to the number of offspring produced as a measure of male reproductive success, whilst females are considered reproductively successful when they mate with one partner only. Resultantly, this was considered a universal feature for all sexually reproducing species. Trivers (1972) constructed the theory of "parental investment" where Bateman's work appeared as a central subject. Subsequently, he suggested that female reproductive investment (i.e., metabolically expensive eggs) is so much greater than the "cheap" (low investment) sperm in males that sexes evolved to have different mating strategies (i.e., females mate monogamously, whilst males mate indiscriminately with multiple partners). This implies that different parental strategies and investment would also evolve. Parental

investment theory and the foundations of it (i.e., gamete size) will be reviewed in the forthcoming sections.

Given the intimate and direct relationship between status, survival and reproductive success, and risk to life within the animal kingdom, it is reasonable to hypothesise that a loss in status should send a warning signal directly to our physiology. Human and non-human animal studies suggest that hormones are one of the proximate mechanisms that facilitate the emergence, development and maintenance of status hierarchies in groups (Kemper, 1990; Sapolsky, 2004, 2017). Circulating levels of the androgen testosterone relate to individuals' relative status in both human and non-human samples, albeit in a rather non-straightforward manner (Archer, 1998; Ellis, 1995; Sapolsky, 1990). This has primarily been considered within the context of the challenge hypothesis (Wingfield et al., 1990).

2.5 Human hierarchies

With the disappearance of hunter-gatherer communities and the presence of equality as a fundamental tenet in human societies (maintained by what have been called 'counter dominance strategies'), human communities have started resembling the ranking systems of many other animal species (Wilkinson & Pickett, 2018; Sapolsky, 2017). Status hierarchies emerge early in the development of the human life and with little contribution from caregivers; the formation of status hierarchies is observed in preschool children as young as 2 years of age, for example (Schubert et al., 2008; Thomsen, 2020; Thomsen et al., 2011). Individuals from this age group differ amongst themselves on social dominance measures. Moreover, social dominance appears to be the earliest stable dimension of peer group social organization, but also the initial and most enduring observable personality trait (Cummins, 2005; Lemerise et al., 1998), with several studies demonstrating that toddlers prefer to associate with and imitate high-ranked individuals as opposed to subordinates (Boulton & Smith, 1990; Russon & Waite, 1991). Furthermore, infants demonstrate the ability to recognise status disparities even when subjects are used as a measure of status rather than human beings (Thomsen et al., 2011). This evidence suggests that human structures not only

resemble those of other species but also reflect the meaning of hierarchical systems and ranks.

Nevertheless, as this thesis has already established, hierarchical structures within human societies are not by any means as straightforward as those within the animal kingdom. This arises not only from the potential for human beings to belong to more than one ranking system, but also from contemporary research evidence suggesting that middle management is the group experiencing the greatest risk of stress-related diseases emanating from high work demands and limited autonomy (Sapolsky, 2017). Thus, the rather direct animal relationship low rank/ill-health appears somewhat convoluted amongst humans. Regardless of the multiple similarities to the animal species, human hierarchies display a degree of complexity not found amongst the animal domains. The following sections will review the concept of multiple ranks and examine additional determinants of the experiences of social rank.

2.5.1 Multiple ranks

Humans hold multiple ranks across a range of hierarchies. For example, an individual might be a subordinate at their workplace but hold the position of local councillor in the community, bringing them prestige and honour. Whilst Sapolsky (2004, 2017) suggests they are likely to value the one in which they are ranked highest above others, this is a point of contention. Other determinants also have an impact on the extent to which low status rank will translate into psychological stress and pathology. This includes the specialisation of some ranking systems, or the success of individuals in one ranking system but not others, or their subjective independent interpretation of events. Individual psychological interpretation of social rank is therefore fundamental for health and wellbeing.

Accordingly, what seems to be more important for health and wellbeing is an individual's perceived social status in the hierarchy that people value most, or their relative position to others. Adler and colleagues (2000) examined the relationship between poor health and 'subjective SES', testing healthy white women's thoughts and feelings of their SES by asking them a simple question; "in society, where on this

ladder would you rank yourself in terms of how well you're doing?". The findings demonstrated that subjective SES is as good, if not a better predictor of health measures as objectively ranked SES. Furthermore, Adler (2000) shows that subjective SES is built around education, income, and occupational position, plus satisfaction with standard of living and feeling of financial security about the future. Psychosocial variables associated with perceptions of status and stress may therefore relate to the impact of SES on health (Taylor & Seeman, 1999). For instance, stress, pessimism, and sense of control have been linked to both SES and poor health outcomes (Cohen et al., 1999; Kaplan, 1999; Kraus et al., 2009; Phillips & Klein, 2010). Additionally, passive coping strategies have also been linked to ill-health (Billings et al., 2000; Essex & Klein, 1989) but have not yet been linked to SES.

Thoits (1995) further argues that negative life events appear to be emotionally and psychologically distressing, and to have destructive physiological implications only if they are salient or influential for the individual's identity. More specifically, consequences of the event only arise if the individual "identifies with or is committed to the domains in which those events occur" (Thoits, 1995, p.72). Hereafter, the author argues that "identity-relevant" stressors might be the better predictors of psychological and emotional distress. This hypothesis though has not been confirmed by Thoits' findings, suggesting life events and changes are far more complex and should be interpreted within specific contexts (Brown et al., 1987; Brown & Harris, 1989). Nevertheless, both works stress the importance of individual's perception of rank for the implications on health and wellbeing. Experiences of rank in animals and human may vary on the basis of personality as a filter of the experiences of social rank. The next section explores this concept.

2.5.2 Personality as a filter of the experience of social rank

Research indicates that whilst large income inequalities reduce the availability of protective lifestyle factors for the disadvantaged in a community (Lynch et al., 2004), the ill-health outcomes of feeling 'poor' are often embedded in the psychosocial consequences of being made to feel poor by the settings (Wilkinson, 2000). Moreover, community's 'social capital' (e.g., levels of trust and social cohesion) is

characteristically decreased by increased income inequalities, whilst this decreased capital plays a significant role in the relationship between health outcomes and income inequality (Kawachi & Kennedy, 2006). Indeed, recent studies of the health gradient include consideration of psychosocial determinants of biological outcomes. Perception of deprivation therefore might be at the core of why disadvantage predicts poor health outcomes. Consequently, when trying to interpret the intricate associations between social rank and ill-health in humans, psychosocial factors and complexities such as multiple rank systems and perception of status should be taken into consideration in forming a more complete understanding of the mechanisms underpinning health outcomes and social inequality.

One of the psychological factors shaping the perceptions of rank is personality (Sapolsky, 2017). Within human hierarchies, personality also shapes the relationship between status rank and health to a great extent (Sapolsky, 2017). For example high-status individuals who are less open to novel experiences, less capable of positive reinterpretation of the events, do not use opportunities to take control over the events, cannot acknowledge victories and defeats as a result of one's inability to differentiate between those, and who have low coping outlets are less likely to be healthy regardless of their elevated position on the social ladder (Afshar et al., 2015; Pereira-Morales et al., 2018; Sapolsky, 2004; Sapolsky, 2017).

Alternatively, if an individual experiencing low SES has sufficient social support, is capable of positive reinterpretation of outcomes and of displaying status anxiety by recognising that there are others in potentially worse circumstances then they are more likely to enjoy improved health (Sapolsky, 2017). Consequently, maladaptive personality traits might prompt individuals to become more susceptible to stress, and thereby increase stress reactivity and subsequently the likelihood of ill health outcomes (Bolger & Schilling, 1991; Chapman et al., 2009), whilst adaptive traits (e.g., conscientiousness and extraversion) might act as a buffer of the experiences of stressors (Vollrath & Torgersen, 2000) whilst promoting more coping strategies and thereby relatively more positive health outcomes (Afshar et al., 2015). Additionally, particularly personality traits such as high consciousness, openness and extraversion

are found to be associated with growing up in a high SES home, whilst high neuroticism and low consciousness are related to early life experiences of low SES (Jonassaint, 2011).

In the light of this evidence and considering the prevalent risk of exposure to stressors within SED cohorts, one might argue that personality plays a very active and trivial role in the link between stress reactivity and health outcomes within lower SES populations (Hughes et al., 2021). This has also been echoed in the increased recognition of the importance of personality for socioeconomic implications and vice versa, in policy making and policy decisions (Bleidorn et al., 2019). Furthermore, personality traits have also been recognised as related to the relationship between status and steroid reactivity towards status encounter (Maner et al., 2008). Given the importance of those arguments throughout this thesis, potential links of personality to endocrine response to competition have also been explored. Lastly, understanding personality as a filter of the experiences of social rank allows us to shed light on phenomena within societal structures such as the "satisfaction paradox". The following section will focus on this.

2.5.3 The satisfied poor

The notion that social subordination in humans experiencing low SES is more stressful than inhabiting higher levels of social standing replete with relative affluence is a comparatively simplistic perspective which leads to an unrealistic dichotomy between these sections of the population. However, the relationship between social rank and its biological consequences is more complex than in animal models. For instance, well-being research demonstrates that some people living in objectively privileged conditions may experience dissatisfaction with their quality of life. In contrast, there are people living in an objectively disadvantaged conditions who express satisfaction with their quality of life, a phenomenon Zapf termed 'the dissatisfaction dilemma' (1984, p.24). The existence of the 'dissatisfaction dilemma' also known as the 'satisfaction-paradox' is recorded in several countries and adopts the lenses of adaption and resignation of poor individuals to explain their impoverished situation (Sardadvar et al., 2017, Zapf, 1984).

One way to better examine the satisfaction paradox is to consider it within the theoretical framework of learned helplessness (Seligman, 1975). Learned helplessness means the development of a mindset which believes that the factors impacting undesirable circumstances cannot be controlled by the distressed individual. The individual learns that whether or not they try to cope with the situation, the probability of changing the negative circumstances will remain the same. As a result, motivational, cognitive and emotional deficits emerge in the individual's personal temperament (Maier & Seligman, 2016). For instance, helpless individuals switch from asking for 'instrumental help', which supports individual's coping resources, to seeking 'in-kind-help' such as emergency services and in doing so disregard their own coping abilities and capacities. As a result, the individual's coping behaviour becomes ineffective or unrelated in terms of solving their general disadvantaged situation and they become more dependent on emergency services. Moreover, as a result of the learned helplessness, these disadvantaged individuals adapt to the underprivileged situation by justifying the cognitive disagreement that arises as a result of the stigma of poverty (Festinger, 1978; Sardadvar et al., 2017). This in turn allows people living with social disadvantage a positive level of satisfaction with life.

2.5.4 Resilience

Another important element in assisting individuals to face fewer health-related diseases or break the cycle of SED relates to the concept of resilience and protective factors. Before looking at the resilience or protective factors of interest for this research, I will review the various definitions and sources of resilience across the disciplines.

Resilience has been defined as "the ability to harness resources to sustain well-being." (Southwick et al., as cited by Wippold et al., 2021). However, the definition of the term evolved over time as more disciplines such as psychology, sociology, and biological disciplines endeavoured to understand the topic, resulting in a lack of consensus around the operational definition of the term. Despite this, the central interest

remains the question of why some individuals do not experience or experience negative outcomes in as acute a manner as others.

The first dispute arises over the question whether resilience should be coined as a personality trait or as a dynamic process (Herrman et al., 2011). Some authors describe the construct as an individual trait emerging as a consequence of a short-lasting trauma (Bonanno, 2004; Klohnen, 1996). Other scholars consider individual's assets and strengths, including social networks and support such as families, communities and services. The literature broadly focuses on the experiences of adversity as a core foundation of the concept and agrees on the significant negative implications for the individual experiencing it. The range of factors being explored as essential elements of resilience lead to a more comprehensive definition, namely "protective and vulnerability forces at multiple levels of influence - culture, community, family and the individual" (Cicchetti, 2010, p.151). Additional considerations include "the protective factor and processes or mechanisms that contribute to a good outcome, despite experiences with stressors shown to carry significant risk for developing psychopathology" (Hjemdal et al., 2006, p.94). It has also been defined as "an interactive concept that refers to relative resistance to environmental risks or overcoming stress or adversity" (Rutter, 2006, p.1) and a "multi-dimensional characteristic that varies with context, time, age, gender and cultural origin, as well as within an individual subject to different life circumstances" (Connor & Davidson, 2003, p. 76). Resilience might also be completely contextualised, temporal and impossible to generalize to other life domains. Consequently, the sources and pathways to resilience are multidimensional and frequently interrelated. These range from personal and biological factors, to environmental-systemic factors, and complex interactions between genetic, environmental and personal factors (Friborg et al., 2003; Luthar & Cicchetti, 2000; Masten, 2001).

Reflecting the diverse range pathways through which resilience might emerge, a wide variability of indicators and approaches to measure resilience at behavioural, emotional and developmental level are employed. To create greater transparency between research findings, to draw links between fields and to enhance the

understanding of resilience, researchers should provide clear description of the measurements or combination of measurements utilised in the study (Walsh et al., 2010). For those reasons, the following sections will shed some light on those factors considered protective or which are core contributors in the formation of resilience within this research. Next, the variables related to learned helplessness, hopelessness and lack of control as behavioural and psychological consequences of prolonged exposure to socioeconomic adversity will be reviewed. More specifically, the presence of control and the lack of learned helplessness and hopelessness will be considered protective.

2.5.5 Sense of coherence

Sense of coherence (SoC), a theory focusing on how people interpret, control and demonstrate resilience towards negative life events and stress, originates from Antonovsky's idea of "salutogenesis" (Antonovsky, 1987; Antonovsky, 1979). The term refers to the mechanisms endorsing and reinforcing good health, as opposed to pathogenesis which relates to the determinants of ill-health. Antonovsky's theory draws on his studies on female survivals of concentration camps, and argues that individuals might experience identical stressors but respond to the challenge posed by the stressor differently, based on the suitableness and effectiveness of the tension management system in place (Antonovsky, 1979). He argues that "what is important for the consequences of a life crisis is the subjective perception of the meaning of the event rather than its objective character" (Antonovsky, 1974, p.246). He adds that "despite the fact that the poor are screwed at every step of the way… they are not all sick and dying." (Antonovsky, 1979, p.7). This raises the question of which sources of strength allow some individuals to avoid the negative consequences of living in SED. Antonovsky refers to these sources as the generalized resistance resources (GRRs). There are three main GRRs: 1) adaptability (i.e., from physiological and psychological levels to social and cultural; 2) interpersonal relations and social support; and 3) Macrosociocultural GRRs encompassing the commitment and relationships between the individual and the community as a whole. The extent to which individuals acquire these GRRs dictates the extent to which they have a "generalised, pervasive"

orientation to life or a strong sense of coherence and thereby potentially more resilience towards negative life outcomes and poor health (Antonovsky, 1979).

Antonovsky created two versions of the same scale to measure an individual's sense of coherence. Both scales encompass three central themes: comprehensibility (understanding and predictability of individual's life events), manageability (resources allowing individuals to manage and control life events) and meaningfulness (the degree of meaningfulness and purpose one's life has) (Antonovsky, 1987). One of the versions comprise 13 (SOC-13) questions and the other 29 questions (SOC-29), with more modified versions occurring in recent research (Eriksson & Lindström, 2005; Walsh et al., 2014). Both versions are considered to have cross-cultural validity and reliability (Eriksson & Lindström, 2005), whilst strongly related to a wide variety of outcomes such as perceived health status, other measured of quality of life, mental health (including learned helplessness , hopelessness and depression), crime, psychopathology, health-related behaviours, anxiety and PTSD symptoms (Antonovsky, 1987; Eriksson & Lindström, 2007; Flensborg-Madsen et al., 2005; Gibson, 2003; Glanz & Maskarinec, 2005; Kouvonen et al., 2010; Nilsson et al., 2007; Ristkari et al., 2011; van Leeuwen et al., 2012). The relationship between SoC and physical health is not as straightforward; one camp within research argues that there is a direct link between the two variables, whilst another questions the relationship (Coward, 1996). Nevertheless, the link between sense of coherence and illnesses and health outcomes such as Type 2 diabetes, post-surgery recovery and mortality rates has been well-established (Agardh et al., 2003; Lundberg & Peck, 1994; Ray et al., 2003; Ristner et al., 2000), with strong SoC correlating with a 20% decreased risk of all-cause mortality (Wainwright et al., 2008).

Importantly, the concept has been directly linked to learned helplessness and hopelessness by the aforementioned GRRs, arguing that higher sources of GRRs imply lower risk of the "giving-up" syndrome. The author also draws links between control and the construct arguing that strong SoC does not necessarily imply that individuals are in control of their life, due to the active role of other factors such as identity in the experiences of life event (Antonovsky, 1979). Thus, in this study Antonovsky's sense of

coherence (SOC-29) is used as a measurement of resilience against negative life outcomes and as a factor related to endocrine response to status competition, but not particularly as a measure of control over life events. The measure of attributional style in this study focuses on the element of control.

Lastly, as a result of Antonovsky's argument that individuals and groups differ in their SoC based on socio-cultural and cultural-historical factors, Walsh and colleagues (2014) compared three UK cities with relatively similar social and health inequalities; Manchester, Liverpool and Glasgow. They found strikingly different results from what they hypothesised, with Glaswegians displaying distinctly greater SoC compared to Mancunians and Liverpudlians (Walsh et al., 2014). This subsequently discredited the previously suggested relationship between SoC and the higher prevalence of mortality in Scottish population (and particularly Glasgow) compared to the English cities. Nevertheless, the link between SES measures and SoC has been previously confirmed by multiple scholars (see Eriksson & Lindström, 2005; Ing & Reutter, 2003; Volanen et al., 2004) consolidating the credibility of SoC as an independent predictor of health disparities. This suggests that the concept of SoC is susceptible to cultural influences, similar to other self-reported health status instruments (Jürges, 2007; O'Reilly & Rosato, 2010; Walsh et al., 2014).

Furthermore, because Glaswegian culture has been associated with a strong sense of identity (Perchard, 2013; Richards, 2004) this evidence suggests a potential relationship between social identity (Tajfel, 1979) and SoC. These scholars suggest that that SoC might have a greater credibility as a measure of social identity rather than as a predictor of mortality rates and health outcomes. Responding to these suggestions, this study will provide analysis of the SoC scale in a Glaswegian sample who find themselves in low and high SES groups, thereby shedding light on this hypothesis and enabling a greater degree of confidence over the accuracy of such a measure.

2.6 Mechanism underpinning status

2.6.1 Testosterone

Despite the emphasis within literature on poverty's relationship to stress, cortisol, and inflammatory markers, poverty does not impact upon health directly through mechanisms related to stress physiology and immune system function alone. It is also associated with a constellation of behaviours and cognitions such as aggression, hostility, risk taking, delinquency, attention deficit, fear and impulsivity. Whilst these emotional states and behaviours have consistently been linked with testosterone, very few studies have been conducted which examine the relationship between androgens and poverty. Testosterone has a prominent role in influencing behaviour, including facilitating success in intra- sexual competition, mating effort and status striving (Buss, 2009). It is also involved in regulating sex drive, muscle development and the behavioural fight-or-flight response, and has been closely linked to dominance and status-related behaviours (Mazur & Booth, 1998), and aggression (Book et al. 2001). In particular, it appears to increase competition (Hegner & Wingfield, 1987), lead to confrontational responding in status and dominance challenges (Dabbs, 1997; Muller & Wrangham, 2004) and contribute to establishing and maintaining social hierarchies (Dugatkin & Druen, 2004). Thus, testosterone's role in behaviour appears to be one of signalling and regulating social status and the response to status challenges (Mazur & Booth, 1998), ideas which seem central to Marmot's work and the Whitehall studies.

Correlations between baseline levels of testosterone and SES have been reported in a few studies, the largest and most persuasive of which was conducted by Dabbs and Morris (1990), on 4,462 male U.S. military veterans. Self-reported measures indicated that those with levels of high testosterone participated in more antisocial and excessive behaviours such as higher likelihood of using drug and alcohol abuse, having numerous sexual partners and engaging in delinquency. When the individuals were divided into high and low socioeconomic groups (based on the US median of income and education), two findings were observed. Firstly, low SES group displayed higher testosterone levels compared to the high SES group, with the highest 10% testosterone range consisted of 14% low SES and only 6% high SES individuals. Secondly, results observed that the correlation between antisocial behaviours and testosterone levels

was shaped by SES; a significant relationship between antisocial behaviours and testosterone levels was observed amongst the low socioeconomic group, but not in the high SES group. These results suggest that there is an existing link between low SES and testosterone.

In a subsequent paper by Dabbs (1992), a relationship between occupational level and testosterone levels was established, where unemployed and blue-collar workers were related to higher levels of testosterone compared to white-collar workers. Accounting for confounding factors such as race and age did not amend the results. Based on the negative correlation between the Sevens & Cho's occupational status score (1985) and testosterone levels, observed among employed farmers, Dabbs argues that this correlation might be influenced by factors such as intelligence, education and antisocial behaviour. What remains unclear is the direction of the causality of this relationship. For example, Dabbs queries whether high testosterone during childhood due to its high heritability (Meikle et al., 1988) might affect levels of intelligence, antisocial behaviour and education later on in life. However, the opposite direction is also likely, where environmental factors related to low SES may directly impact testosterone level (Knight & Mehta, 2014).

Two subsequent studies from the same researcher also provide ambiguous evidence. The first study revealed that political ministers have lower levels of testosterone compared to football players and actors (Dabbs, 1992), whilst the second study, examining lawyers, suggested that trial lawyers (and thus classified as blue-collar workers amongst the high SES legal profession) have higher testosterone levels than patent lawyers and other highly esteemed roles (Dabbs, 1998). These range of contrasting perspectives point to the conclusion that the link between androgens and SES remains poorly understood. This thesis seeks to address that.

2.6.2 Cortisol

Cortisol's prominent role in stress is determined by two factors. Firstly, it is released by the organism as a response to external physical or psychological threat. Thus, in times of physical threat such as illness, physical exertion, body injuries or extreme

temperatures cortisol increases. Cortisol also elevates following psychological threats such as social stressors (Ferracuti et al., 1994). Secondly, as a stress hormone, cortisol affects the physiology by increasing blood sugar levels and supressing the immune system via suppression of pro-inflammatory cytokines such as IL-6 and IL-1, (Straub, 2004; Wilckens, 1995), which enables the organism to prepare to deal with external physiological or psychological threats. When chronically elevated, cortisol appears to have a toxic effect on the organisms. For instance, chronically increased cortisol levels cause mild but permanent elevations of the aforementioned cytokine levels (Kiecolt-Glaser et al., 2003) which then directly contribute to the development of disease such as cancer or atherosclerosis (Aggarwal et al., 2006; Coussens & Werb, 2002; Steptoe et al., 2001, 2002). Hence, transient cortisol facilitates survival via adaptive and protective mechanisms, whilst chronically elevated cortisol levels have detrimental effects on organisms.

An increasing number of studies identify the link between SED and cortisol (Haushofer, 2011; Knight & Mehta, 2014). Studies of children (Chen & colleagues, 2010; Evans & English, 2002) and adults (Cohen et al., 2006a, 2006b; Li et al., 2007) observe an association between SES and cortisol levels. In particular, higher levels of cortisol are reported amongst individuals within lower SES groups, where SES was measured by income, education and occupational level. Importantly, these increased cortisol levels, observed across disadvantaged groups, have behavioural and cognitive implications. Considering this link, the relationship between status and cortisol should be reviewed with a particular attention being paid to the variables impacting cortisol levels; health behaviours, sense of control, hostility and social support (Knight & Mehta, 2014). Indeed, the study presented in this thesis explores the relationship between these factors and hormonal response in low and high SES populations.

Health behaviours have been proposed as a potential mechanism to explain the relationship between cortisol levels and SES, with several studies identifying alcohol and tobacco as one of the main contributors (Cohen et al., 2006a, 2006b). Indeed, higher basal cortisol concentrations were observed in tobacco and alcohol users, whilst smokers displayed heightened cortisol secretion compared to non-smokers

(Knight & Mehta, 2014). The research appears to be less certain about the explanation of why low SES are associated with higher rates of smoking and alcohol-related mortality (Krueger & Chang 2008). Some suggest these could be used as dysfunctional coping mechanisms to deal with the high stress associated with low SES living (Hull & Slone, 2004; Muraven & Baumeister 2000; Pampel et al., 2010), whilst others link it to lower educational attainment, life-history strategies and cultural norms (Cutler & Lleras-Muney, 2008; Pampel et al., 2010).

SoC is another factor proposed to influence the link between SES and cortisol levels, with individuals from low SES groups self-reporting less control over life circumstances and outcomes (Cohen et al., 2006a, b; Knight & Mehta, 2014). Consequently, "shift and persist' intervention has been proposed as an effective mechanism to redirect attention towards maximizing the SoC and subsequently reducing the negative implications of the elevated cortisol levels (Chen & Miller, 2012). The intervention also aims to optimize resilience and optimism towards negative life events and stressors, and thus creating the traits associated with low basal cortisol levels in groups experiencing SED (Lindfors & Lundberg, 2002; Ryff et al., 2004).

Hostility has also been proposed to underpin the cyclical nature of SED where chronic exposure to stressors might facilitate an antisocial and hostile response to stressors which in turn further promotes interpersonal hostility (Gallo et al., 2006; Smith, 1994). Flattened cortisol slope and increased cortisol production were thus associated with high-trait hostility (Pope & Smith, 1991; Ranjit et al., 2009). Nevertheless, research has not established a clear link between cortisol levels and hostility, but rather between stress physiology, allostatic load and hostility trait (Hawkley et al., 2011; Kubzansky et al., 1999). Further research is thus necessary to address the question whether hostility has a direct impact on cortisol production and secretion, or whether the variable remains merely broadly associated with stress physiology.

Finally, social support has been proposed as a means to reduce the detrimental impacts of low status rank in animal species (Sapolsky, 2005). This is mirrored amongst humans experiencing SED, where the lack of social support and low SES has

been related to increased basal cortisol levels and higher acute response to laboratory stressors (Heinrichs et al., 2003; Pinquart & Sörensen, 2000; Taylor et al., 2006; Uchino, 2006). Limited social networks and perceptions of social isolation are stressors experienced by those encountering lower and less diverse social capital, resulting from the limited access to resources and status (Cohen et al., 2006a, b; Hawkley et al., 2012; Uchino, 2006). Several authors thus propose that the link between low SES and low social support, subsequently resulting in high cortisol levels, might occur as a result of individuals' mistrust of one another within deprived neighbourhoods, or due to the continuous exposure to stressors leading to inability to cope, seek and provide support (Cattell, 2001; Subramanian et al., 2003).

Considering the implications of status rank upon circulating basal and diurnal cortisol levels, one can argue that social rank alters cortisol functioning (Knight & Mehta, 2014). However, the question remains as to whether the reciprocal relationship proves similarly impactful; does cortisol levels impact upon status rank? Earlier research on rodents and rainbow trout suggests that GCs impact hierarchy formation (DiBattista et al., 2005; Timmer & Sandy, 2008). These findings are replicated, to some extent, in human research where cortisol does not directly impact social status per se, but has implications for status-related behaviours directly embedded in social rank acquisition (Anderson & Berdahl, 2002; Roelofs et al., 2005; van Peer et al., 2007). This study will develop greater understanding of the phenomenon by examining the extent to which behavioural, psychological and cognitive implications of cortisol reactivity reinforce social status.

2.7 Challenge hypothesis

The challenge hypothesis outlines the dynamic relationship between testosterone and aggression in mating contexts. It was originally proposed to account for testosterone-aggression associations in birds with a monogamous mating system (Wingfield et al., 1990). It holds that there are specific context-dependent increases in testosterone levels that are associated with aggression. Testosterone increases to moderate levels at the start of the breeding season, supporting reproductive physiology and behaviour.

During challenges relevant to reproduction, testosterone levels rise further amongst male birds. In turn, this facilitates aggression in the context of territory formation, dominance disputes and mate-guarding (Wingfield et al., 2000). The challenge hypothesis applies to a variety of monogamous bird species (Archer, 2006; Wingfield et al., 1990, 2000; Wingfield, 1985; Vleck & Brown, 1999), or rather to those that show paternal care (Wingfield et al., 2000), but not to all that have been studied (Moore et al., 2004; van Duyse et al., 2000). In polygynous birds without paternal care, males show high levels of testosterone throughout the breeding season and it is suggested that their lack of response to challenge occurs because testosterone levels are close to maximum (Wingfield et al., 1990, 2000). Similar biosocial model of status, associating testosterone with dominant behaviour and outlining the same endocrine patterns, has been formulated by Mazur (1985) for primates.

Most of what has been written on the challenge hypothesis concerns males. In different species of birds, there is marked variation in the testosterone levels of females, and it is clear from studies of mammals that androgens can play an important part in female aggression in some species (Clutton-Brock, 2009; Clutton-Brock & Huchard, 2013; French et al., 2013; Isaac, 2005). In an analysis of different species of socially monogamous birds, Wingfield and colleagues (2000) found that - when sexual dimorphism was less pronounced - testosterone levels were higher amongst females compared to their male counterparts. They suggest that in these cases testosterone may play a part in female competition for male parental investment.

The challenge hypothesis, as suggested by several studies, is seen to have a large-scale impact on a wide range of species including some breed of fish, two species of lizard, the ring-tailed lemur, the chimpanzee and the rhesus monkey (Hirschenhauser et al., 2004, Greenberg & Crews, 1990, Cavigelli & Pereira, 2000, Muller & Wrangham, 2004, Rose et al., 1972, 1974). These studies have documented that animals exhibit behaviour and hormone-related changes in a manner consistent with the challenge hypothesis and the biosocial model of status. Particular relevance to the human case has the study on wild chimpanzees (Muller & Wrangham, 2004). Due to dissimilarities in breeding and forms of relationship between bird species and chimpanzees, the

researchers argue that the challenge hypothesis may apply to mammals in a different manner. For example, male chimpanzees may maximise aggressive behaviour during female oestrus due to the rarity of access to receptive and fertile females, and in the presence of parous oestrous females, males exhibit more aggressive behaviour (Muller & Wrangham, 2004). Moreover, testosterone levels were also predicted to be higher amongst dominant males compared to subordinates since the dominant males are those who exhibit higher aggressiveness at all times. Results indicated that there was a significant increase in testosterone levels and aggressive behaviour among male chimpanzees during times of competition. Moreover, low-ranking males were generally less aggressive than dominant ones and produced lower levels of testosterone (Archer, 2006).

Archer (2006) argues that the predictions of the challenge hypothesis may apply to humans. Whilst the hypothesis has not been explicitly tested, the limited evidence in the human literature suggest that this position may be plausible. The evidence for the challenge hypothesis in non-human animals provides a foundation upon which to explore the relationship between testosterone and aggression in humans.

2.7.1 Animal species

Evidence from the animal species literature suggests that testosterone is involved in status processes and obtainment of social rank within the hierarchy, albeit through mechanisms which remains rather unclear. Bernstein and colleagues (1983) outline the importance of both testosterone and cortisol upon the formation, but not the maintenance, of pecking order systems. Numerous studies of various species such as rodents, non-human primates, hens and fish have been conducted not only in the pursuit of further clarification of the dynamic relationship between cortisol, testosterone and competition, but also in the endeavour to ratify the plausibility of this relationship in human species (Haslam et al., 2018; Kornienko et al., 2014; Sapolsky, 2004, 2017; Wu et al., 2017).

Animal studies demonstrate a link between corticosteroid response and competition (Bohák et al., 2018; Hudson et al., 2019; Sapolsky, 2004, 2017), with elevated levels of

cortisol being related to experiences of competition, with anxiety and stress occasionally accompanying it. Moreover, these studies indicate that cortisol remains high and increases further following defeat, whilst cortisol levels appear to gradually decrease in the aftermath of victory. In support of this finding, Mazur's biosocial theory of status suggests that endocrine responses to competition appear in an attempt to out-stress other competitors, whilst the implications of defeat are associated with the wider long-term implications of subordination (Mazur, 1985). Nevertheless, the literature also illustrates examples where victory is the outcome associated with higher corticosteroid production rather than defeat (Abbott et al., 1998; Chase, 1980). A potential explanation for this may be that individuals acquiring new status rank frequently become the target of others, subsequently leading to an exposure to multiple challenges. Sapolsky (2017) similarly suggests multiple additional factors such as stability of the ranking system, culture and the experiences of subordination within the hierarchical structure that appear to play a role in the determination of the corticosteroid system response. Human literature, however, builds a strong case for an association involving increase in cortisol levels following defeat and decrease following victory.

Given the increasing evidence of a dynamic relationship between testosterone, cortisol and male competition it is has become apparent, that as with the relationship between hormones and aggression, there is no simple, straightforward association. Amongst others, Elias (1981), Mazur and Lamb (1980), and Salvador and colleagues (2003) have suggested that psychological variables contribute to a framework whereby complex psychological mechanisms related to emotional and/or cognitive interpretation of the situation may be more important for hormonal responses than the outcome itself. In this regard, Gladue and colleagues (1989) state, 'the relationships between hormone and behaviour are complex, involving both hormonal effects on behaviour and experiential effects on endocrine function' (p.409).

2.7.2 Challenge hypothesis applied to humans

Human males also appear to exhibit a stereotypical and predictable testosterone response both before and after competition. This response appears to be parallel to the

challenge hypothesis/Mazur's biosocial model of status, with testosterone rising in anticipation to competition and levels amongst winners rising or appearing higher than those who experience defeat (Booth et al., 1989; Schultheiss et al., 1999; Mazur, 1985; Mazur et al.,1997). Importantly, the observed changes in winners' testosterone response may persist over the period ranging from minutes to days (Campbell et al., 1997; Elias, 1981; Mazur & Lamb, 1980).

Empirical research examining this typical endocrine pattern (i.e., the predicament of challenge hypothesis/Mazur's biosocial model of status) in humans has predominantly focused on physical competition such as wrestling or judo, albeit some studies validate this relationship in non-physical contests like chess matches (Gladue et al., 1989; Mazur et al., 1992). Bernhardt and colleagues (1998) suggest the relationship occurs even amongst spectators, where fans of a losing team experience decreased testosterone levels whilst levels amongst fans of the winning team elevate. Individuals who face a symbolic challenge also demonstrate rise in testosterone (Cohen et al., 1996). After graduation, medical students experiencing ecstatic mood display higher testosterone levels. Importantly, no change in the testosterone levels appear amongst students whose mood was not elated (Mazur & Lamb, 1980). The aforementioned studies lead to the conclusion that, as in animal species, human males also exhibit a stereotypical testosterone response in the face of a challenge in the period immediately afterward regardless of the nature of the contest.

However, this relationship remains disputed with some studies finding either an absence of, or a reversed interaction such as higher testosterone levels in losers than winners. A similar situation is found when examining steroid hormones and competitive behaviours (Gonzalez-Bono et al., 1999; Passelergue & Lac, 1999; Suay et al., 1999; Filaire et al., 2001; Mazur & Lamb, 1980). Uncertainty amongst scholars point to the importance of additional determinants of the relationship between the anticipatory and post-competition testosterone response and outcome. For instance, Mazur and Lamb (1980) suggest that elevated mood is absent if the event is perceived as insignificant, if there is a difference between the competitors' skills or if the outcome occurs as a result of luck (Gonzalez-Bono et al., 1999, Mazur et al., 1992;

Salvador et al., 1987). Resultantly, albeit the ostensible relationship between testosterone levels and competition is found in males across a wide variety of contexts, this 'is highly contingent on perceptions that gain, or loss of status is at stake' (Bateup et al., 2002, p.183).

Lastly, it has also been suggested that elevated testosterone following success in dyadic encounter encourages or increases the likelihood that competition will take place again (Carré, 2009; Carré & Mehta, 2011; Knight & Mehta, 2014; Mehta & Josephs, 2006). Conversely, subdued levels of testosterone following loss de-motivate and make less likely any desire to compete again (Bjorkqvist, 2001; Otten et al., 2002). Thus, there appears to be a corresponding theoretical concept here in learned helplessness, raising the question; does losing or in some other way being at the bottom of a hierarchy or low in status reduce testosterone to the extent that it makes even trying to change or succeed less likely? This research will aim to address this question and provide evidence for the behavioural implications of social defeat and victory. Furthermore, the study will examine a plausible dominating moderator of the testosterone/outcome relationship by adopting the biopsychosocial model of challenge and threat. The methodological design also accounts for the aforementioned factors.

2.7.3 Cognitive Appraisal Theory

Lazarus and colleagues (1964) first suggested that the perception of an external stimulus as either stressful or not is determined by an individual's cognitive appraisal of that stimulus and that this appraisal has later implications on hormonal and behavioural responses to the environmental challenge. Lazarus also argued that the experience of stress is dependent on the expectations an individual has of the significance and outcome of an event. Additionally, pattern of appraisals, determined by individual and contextual factors also proliferate and impact stress production. Thus, Lazarus' proposal (also known as cognitive appraisal theory) (Lazarus, 1966), and its several revisions (Lazarus, 1991; Lazarus & Folkman, 1984; Lazarus & Launier, 1978), explain individual differences in the quality, intensity and duration of stress in environments where external demands are constant across individuals.

In the latest iteration of Lazarus' theory (1991), stress is considered a relational concept by which stress refers to an association between an environment and an individual, moderated by primary and secondary appraisals. Primary appraisal is concerned with whether the stimulus is relevant to the individual's well-being and comprises three components: goal relevance, goal congruence and type of ego involvement. Goal relevance indicates the extent to which an encounter refers to issues about which the person cares, whilst goal congruence specifies the extent to which an event proceeds in accordance with goals. Type of ego involvement is concerned with self-esteem, moral values and ego-identity. Secondary appraisal is concerned with an individual's coping opportunities in particular circumstances and consists of three elements: blame or credit, coping potential, and future expectations. Blame or credit is the appraisal of who is responsible for a certain event, whereas coping potential refers to the evaluation of one's ability to undertake behavioural and cognitive operations that will be beneficial for a relevant encounter. Future expectations are related to the appraisal of the further course of an encounter with reference to goal congruence and incongruence.

Importantly, specific patterns of primary and secondary appraisal lead to different types of stress, namely challenge, threat and harm (Lazarus & Folkman, 1984). Harm is categorized as the psychological damage that has already occurred, whilst challenge and threat refer to future events relevant to the individual. Threat occurs in the anticipation of potentially imminent harm, whilst challenge occurs when an individual feels confident about mastering situational demands. For instance, Lazarus (1991) argued that for stress to be experienced, there must be some goal relevance to the encounter, goal incongruence must be high and ego-involvement must be concentrated on the protection of personal safety against threats. Therefore, threat is experienced when secondary appraisal indicates that an individual's coping potential is not sufficient, thus deeming harm potentially imminent. Challenge is experienced when secondary appraisal indicates that an individual's coping potential is sufficient, thus deeming harm less likely. The concept of adaptive and maladaptive responses to stressors is also evident within neuroendocrine research, where the employment of

physiological measurements of psychological stress such as Sympathetic Nervous System (SNS) activation, provides an insight of the mechanisms through which health consequences emerge and performance is impacted. Significant attention has been paid to the Sympathetic Adreno Medullary (SAM) and Pituitary Adreno Cortical (PAC) systems as a result of Cannon's (1920) and Selye's (1956) findings that these systems are involved in the stress response.

Endocrine reactivity and namely, the activation of the SAM and PAC systems when faced with psychological stressors and their impact on human functioning has been examined in many other studies through examination of urine and blood samples (Frankenhaeuser et al., 1968; Frankenhaeuser & Kareby, 1962). Frankenhaeuser and colleagues' research (1980) also investigated these two distinct stress responses, and differentiated between them based on the divergent substances excreted within urine. These researchers found that when faced with highly stressful situations, distressed individuals experience negative emotions and excrete cortisol, whilst less distressed persons experience positive emotions and excrete catecholamines. This suggests that there are two systems determining emotional arousal; SAM activity (associated with catecholamines) and PAC activity (associated with cortisol), and that the presence of these hormones within urine is dependent on the level of cognitive stress experienced by the individual. Dienstbier (1989,1992) further strengthens the idea of two separate stress response mechanisms (challenge and threat) and argues that these two stress responses are characterised by cognitive appraisal and associated neuroendocrine activity.

Furthermore, Dienstbier also suggests that an individual's ability to cope with an external stressor is associated with the system through which the arousal is provoked. Thus, PAC activity, accompanied by cortisol release, represents inadequate coping resources and a maladaptive arousal or threat response, whilst SAM activity accompanied by catecholamine release, represents a challenge response where positive secondary appraisal and positive emotions are experienced. Importantly, arousal is adaptive if coping resources adequately outpace environmental and contextual demands. To elaborate, when threat response occurs as a consequence of

acute stress exposure, it is not that cortisol directly impacts performance, but rather PAC activity affects the positive outcomes of SAM activity. Additionally, SAM activation is associated with successful performance, whilst PAC activation correlates with unsuccessful performance (Dienstbier, 1989, 1992).

Dienstbier's assertion that cortisol release may be a key determinant of inadequate coping mechanisms and subsequent performance disruption is also supported by numerous subsequent experimental studies. Harvey and colleagues (2010) found that elevated cortisol levels in the face of an external stressor is associated with performance impairment on tasks of memory, attention, decision making and clinical performance. Moreover, individuals with higher cortisol levels within urine when facing a threat, performed worse in a mental task than those with a lower cortisol response (Bohnen et al., 1990). Thus, literature suggests that cortisol is associated with performance disruption, particularly during cognitive tasks and cognitive appraisal of threat (Beilock & Gray, 2007; Kemeny, 2003; Mattarella-Micke et al., 2011).

2.7.4 The Biopsychosocial (BPS) Model of Challenge and Threat

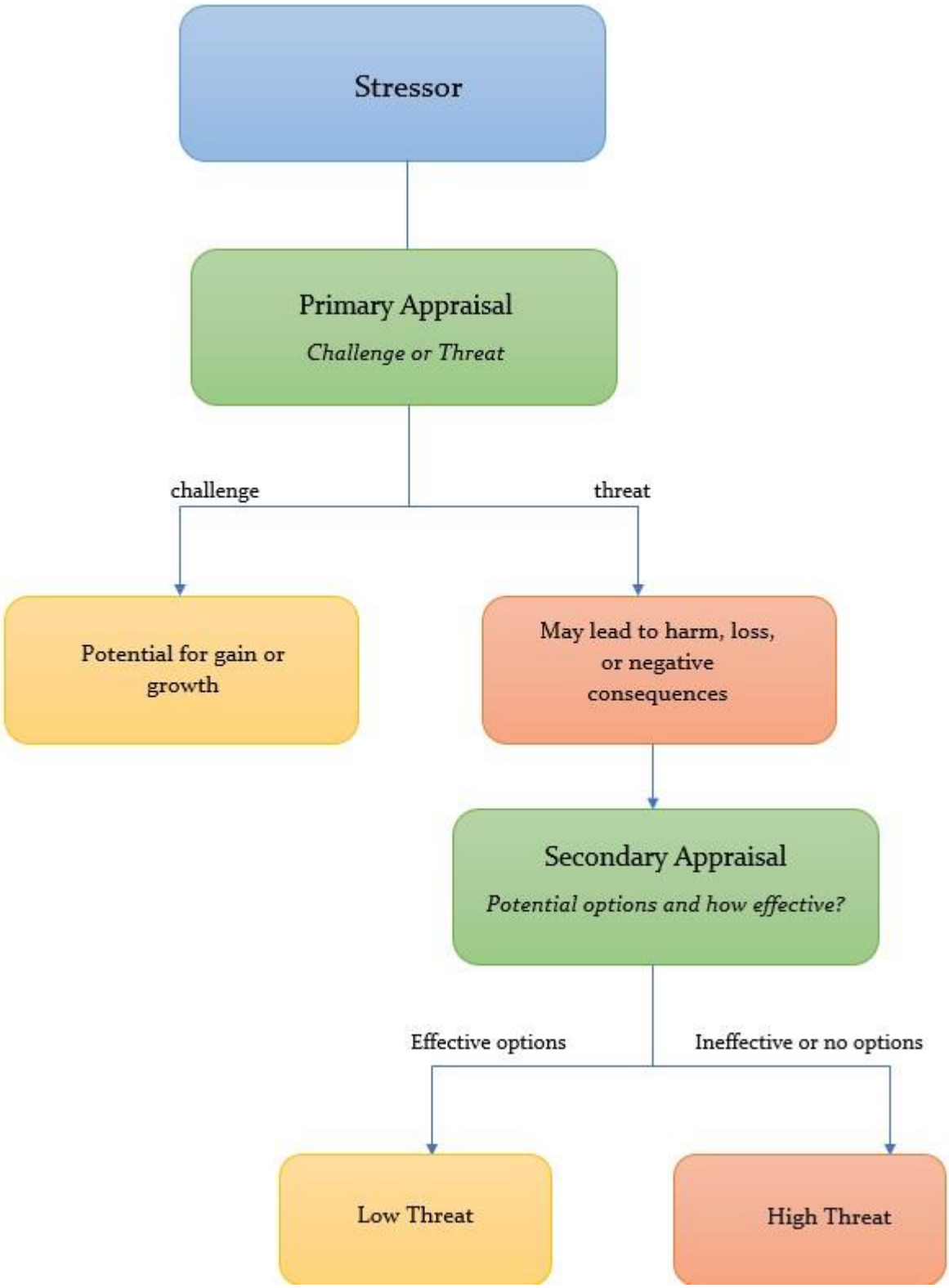
The most recent research on threat and challenges states is built on Lazarus and Folkman's (1984), Obrist (1981) and Dienstbier's (1989) work and frames the BioPsychoSocial (BPS) model of challenge and threat developed by Blascovich & Mendes (2000) and Blascovich & Tomaka (1996). The model (Figure 7) proposes a dichotomy in the way individuals respond to stress. The hypothesis suggests that when an individual perceives sufficiency or near sufficiency of resources to meet the demand of the situation, a challenge state occurs. However, if the individual appraises insufficiency of resources to meet the demand of the situation, a threat state is experienced. Resource appraisals relate to the perceived ability to cope with the demands of the situation and include skills, knowledge and external support. In comparison demand appraisal encompasses the perceptions of danger, uncertainty and required effort in a situation (Blascovich et al., 2003; Blascovich, 2008; Seery, 2011). The theory further argues that these cognitive evaluations anticipate the physiological responses to a stressful situation (Blascovich, 2008; Blascovich & Tomaka, 1996), an

argument supported by evidence from socially evaluative stress tasks (Tomaka et al., 1993; Tomaka et al., 1997).

Drawing on literature evaluating threat and challenge states (Blascovich & Mendes, 2000; Lazarus & Folkman, 1984) and the challenge hypothesis/Mazur's biosocial model of status, outlining the relationship between status and hormonal reactivity proposed by Wingfield and colleagues (1990) and Mazur (1985), it appears that cognitive appraisal of an event, social rank and resource appraisal may have fundamental implications on the hormonal reactivity to an environmental challenge. These in turn affect an individual's performance in the face of this challenge. The foundations of this theory may also provide an explanation of the contested nature of human hormones/status studies by casting light on the cognitive mechanisms which underpin hormonal reactivity to an external challenge.

Figure 7

The BioPsychoSocial (BPS) Model of Challenge and Threat


Stressor
Primary Appraisal
Challenge or Threat
challenge
threat
Potential for gain or growth
May lead to harm, loss, or negative consequences
Secondary Appraisal
Potential options and how effective?
Effective options
Ineffective or no options
Low Threat
High Threat

2.8 Study Rationale

An extensive body of literature demonstrates the robust link between social and health inequalities, and their implications upon an individual's physical and mental health, mortality, wellbeing, and life quality and opportunities. This link is particularly prominent within affluent societies, where a smaller proportion of individuals are affected by absolute deprivation compared to those in developing countries (Marmot, 2004; Wilkinson & Pickett, 2010, 2018). This poses the question as to why individuals within developed societies, whose basic needs are generally satisfied, still get ill. Marmot (2004) explains the disparities in health through the theoretical lens of status syndrome, arguing that an individual's relative social status plays a significant role in determining health and wellbeing. Individuals who obtain relatively lower social rank are more likely to experience more negative health outcomes, higher mortality rates and shorter life expectancies due to their limited resources not allowing them to fully participate in society and lack of control over life, leading to increased social anxiety and stress. Wilkinson & Pickett (2010; 2018) further support the postulated social gradient in health by emphasising that relative social position is indeed a more prominent factor for an individual's health compared to health behaviours and medical care access.

The epidemiological research from Marmot and Wilkinson & Pickett offers a theoretical explanation for the social gradient in health, however their work does not suggest any specific biopsychosocial explanatory mechanism through which this phenomenon occurs. For this reason, this thesis concentrates on one concrete process, namely the neuroendocrine mechanisms underpinning status disparities in health. The justification for this neuroendocrine focus stems from the prominent role of glucocorticoids in stress response and health more generally. Meanwhile, testosterone also influences and reflects social status. SED has been frequently associated with a constellation of behaviours and cognitions such as aggression, hostility, risk taking, delinquency, status-seeking, fear and impulsivity (Pepper & Nettle, 2017) which may be better explained though examining testosterone (Knight & Mehta, 2014). For this reason, this thesis seeks to explore testosterone's role in status disparities in health. In doing so, the study makes an empirical contribution to the theoretical paradigms

offered within of Marmot's *'Status Syndrome'* (2004) and Wilkinson and Pickett's *'The Inner Level'* (2018).

The study also aims to shed light on why not all individuals who experience SED also experience the negative health connotations associated with it. To achieve this, the study applies the methodological framework of threat/challenge cognitions in order to highlight the complexity around the relationship between status, endocrine reactivity, and health outcomes. It does so by outlining some of the important cognitive elements which moderate the links between these issues. In teasing out the complexities of this relationship, the impact of the previously reviewed resilience factors suggested to play a significant role in the link between status and health (namely, sense of coherence, personality, attributional style/sense of control), is also empirically tested. Moreover, the legitimacy of a focus on those factors stems from previous epidemiological research (e.g., Steptoe & Marmot, 2002) which not only points to a link between neuroendocrine response and psychosocial factors, but argues that those factors appear to be unequally distributed across the social gradient. This in turn has implications for health since fewer protective factors have been associated with an increased susceptibility to stress (Marmot, 2004).

By examining the neuro-endocrine mechanisms (specifically T and C reactivity) operating in response to perceptions of challenge and threat in different SES groups, the study seeks to address to what extent living in SED has implications for endocrine reactivity when engaging with a hormone competition task that resembles real life events such as financial form-filling. Moreover, by addressing how cognitive parameters (i.e., threat/challenge cognitions) moderate endocrine reactivity to social competition tasks, the study endeavours to explain why not all individuals who experience SED necessarily experience negative health outcomes (Marmot, 2004).

Finally, the study aims to distinguish between the role of basal and reactivity hormone levels upon behaviour and health, by addressing some of the methodological limitations of previous bio-behavioural research highlighted by Sharp (2006). The

methodological limitations of previous bio-behavioural research will be further outlined in the next chapter of this thesis.

In the light of the study rational, the following research questions have been formulated:

1. Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?

2. Are psychosocial variables related to endocrine reactivity?

3. Does cognitive appraisal of threat/challenge moderate endocrine reactivity?

4. Does T reactivity correlate with a reduced motivation to engage socially?

Chapter 3

Methodology and Methods

3 Methodology

As outlined in the previous chapter, there is wide-ranging evidence that lower socio-economic status is associated with poorer mental and physical health, higher mortality rates and reduced life opportunities. Reasons for this are multifaceted, existing within a latticework of socio-cultural, economic, political and biological influences. However one conceives of the antecedents of poverty, there are very definite biological consequences frequently related to chronic stress exposure. In this respect, a significant avenue of research has focused on the stress-hormone cortisol and its interaction with the immune system (Guan et al., 2021; Hintikka et al., 2009; Johnson et al., 2013; Lin et al., 2016). Despite the extensive and sophisticated literature on the deleterious effects of chronic elevated cortisol, studies directly linking it with socioeconomic disadvantage frequently employ basal measures. Previous literature describes the shifting dynamics of cortisol reactivity to stress amongst individuals experiencing low SES, where patterns of response which may be subdued, exaggerated, rapid or sluggish in returning to baseline (Fiocco et al., 2007; Rosmond & Björntorp, 2000a; Rosmond & Björntorp, 2000b; Sapolsky, 2017). Consequently, cortisol reactivity might be a better predictor of susceptibility to stress-reactive psychopathologies (Al-Dujaili & Sharp, 2012).

Low SES is associated with a constellation of behaviours and cognitions such as aggression, hostility, risk taking, delinquency, status-seeking, fear and impulsivity (Pepper & Nettle, 2017), which may be better explained by testosterone (Mazur & Booth, 1998; Knight & Mehta, 2014). However, very little meaningful work has been conducted on the relationship between testosterone and poverty, and that which has been conducted directly contradicts the cortisol literature (Haushofer, 2011; Mehta & Josephs, 2010). Since cortisol plays a critical role in stress responses and testosterone both influences and reflects social status, the current research investigates the possible

role of these hormones in responses to disparities in both social status and health. It is only by examining this complex endocrine/perception/behaviour relationship that an evidence-based case can be made for interventions which respond accordingly. Demonstrating that policies relating to state benefit application universal credit process represent continued exposure to social defeat, and has biological consequences which can lead to lack of control and learned helplessness, could result in important and much needed reform to public sector provision. Crucially, the converse is also true. If people are successful in undertaking tasks that are important to them, a biological effect increases the likelihood of engagement in future tasks. Thus, a case can be made for amendments to the system which encourage small wins in benefit claimants as a means of modulating behaviour. Lastly, the study will examine findings concerning the relationship between cortisol, testosterone, and SES.

The framework for the proposed methodology emanates from the challenge hypothesis/Mazur's biosocial model of status, which outlines the dynamic relationship between testosterone and status-related behaviours (Mazur, 1985; Wingfield et al., 1990, 2000). The challenge hypothesis conceptually originates from Darwinian sexual selection theory and it is the suggested biological mechanism underpinning hierarchy formation and status disparity (Kemper, 1990). The fundamental premise of the challenge hypothesis and Mazur's biosocial model is that T will rise preceding a status encounter, thus serving multiple physiological functions to prepare the organism to face an environmental challenge. Following the encounter, the winners' T levels will rise further and the losers' will fall, because the cost of loss can be significant for the organism. These dynamic endocrine changes have an effect on motivational states and behaviour (Knight & Mehta, 2014; Mehta et al., 2008; Mehta & Josephs, 2006). As a means of examining status disparity, evidence for the challenge hypothesis in human males is drawn primarily from studies involving competition (See: Booth et al., 1989; Schultheiss et al., 1999; Gladue et al., 1989; Gonzalez-Bono et al., 1999; Mazur et al., 1997; Mazur & Lamb, 1980; Salvador et al., 1987). In light of these findings, it has been suggested that human males in a variety of different situations, exhibit a characteristically predictable and stereotypical T response, both prior to and following competition (Booth et al., 1989; Elias, 1981; Gladue et al., 1989; Jiménez et al., 2012;

Mazur et al., 1997; Mazur & Lamb, 1980; Oliveira et al., 2009; Schultheiss et al., 1999), directly analogous to the challenge hypothesis/ Mazur's biosocial model of status.

The present research aims to apply this method to the broader contextual framework of socioeconomic disadvantage and examine potential differences in endocrine reactivity to hormone status competition between SES groups. Furthermore, acknowledging the methodological limitations of previous bio-behavioural and endocrine research; single-time point sampling, lack of meaningful baseline and use of invasive blood sampling procedures that determine not biologically meaningful testosterone, in the present study those challenges are raised and addressed. This has been accomplished by the utilisation of a comprehensive sampling protocol and a non-invasive salivary sample regime determining biologically free testosterone. Both methods are discussed in the following sections and considered to have a greater credibility and utility in bio-behavioural research (Sharp, 2006).

Complex cognitive factors play a key role in moderating endocrine reactivity, pre- and post-competition outcome (Kutlikova et al., 2021). For example, Blascovich and Mendes (2000) illustrate that individuals appraising events as either challenging or threatening results in contrasting physiological changes (i.e., blood pressure) and alterations in general approach/avoidance behaviour. Marmot (2004) argues that what is crucial for individual's health and well-being is where the person stands in relation to others in society. Wilkinson and Pickett also suggest that the mechanism underpinning the social gradient in health results from social rank and relative position on the social ladder (with subordination linked to limited resources and lack of control and therefore increased social anxiety and stress), rather than from health behaviours or medical care access (Wilkinson & Pickett, 2018).

Acknowledging this complexity and the presence of contextual and cognitive moderators of endocrine reactivity to status, the present study does not limit its findings to just endocrine parameters but also encompasses the importance of the cognitive element in the relationship between status, endocrine reactivity and health outcomes. Consequently, the study sets out to question whether endocrine reactivity

remains similar in the face of threat and challenge, irrespective of SES. Understanding to what extent threat and challenge cognitions moderate endocrine reactivity would also provide grounds for more nuanced debate around the question: why don't all individuals experiencing SED become ill? For the experiment to facilitate conditions in which both threat and challenge cognitions would occur, participants from both SES groups, affluent and SED, were exposed to competition which involved the individuals having to complete paperwork. Justification for the concrete experimental design is provided in the experimental task section. Additionally, other psychosocial factors such as attributional style, sense of coherence, personality, and trait affects - some of which are recognised as resilient factors by the literature and established to influence endocrine reactivity - were also examined in this research. This has been done in order to identify whether psychosocial variables associated with perceptions of status and stress (i.e., the aforementioned factors) may therefore relate to the complex relationship between endocrine response, SES and health (Taylor & Seeman, 1999). All psychosocial variables were examined via various validated and piloted psychological instruments.

Status-induced fluctuations in T levels however, are not only modifiable by cognitive and contextual factors, but they also have implications for subsequent social behaviours (Kutlikova et al., 2021; Mehta & Josephs, 2006) which may reinforce aspects of learned helplessness and hopelessness (Maier & Seligman, 2016). For example, whilst understanding patterns of chronobiological reactivity to perceptions of threat and challenge is important in its own right, it is also critically important to consider the implications of these patterns of low T following defeat and high T following victory. In animal and human research, it is suggested that low levels of T following defeat act to suppress any intention to compete further (Kutlikova et al., 2021; Losecaat Vermeer et al., 2020). Subsequently, it is not only the rise and drop in status that affects T levels, but the effect is reciprocal with T fluctuations further impacting subsequent status-seeking behaviours (Knight & Mehta, 2014). In the light of these arguments, the last RQ of this study seeks to determine whether lower levels of T following defeat/loss do indeed predict a reduction in motivation to re-engage in

subsequent daily activities. This is done by evaluating participants' motivation on a scale following exposure to a competitive task involving the completion of paperwork.

By examining the first research question, the study seeks to examine to what extent living in socioeconomic disadvantage has implications for the endocrine reactivity when engaging with a hormone competition task that resembles real life events such as financial form-filling. The second research question calls on the study to consider whether psychosocial variables, proposed to account for some of the disparities in health factors (Steptoe & Marmot, 2002; Taylor & Seeman, 1999), relate to endocrine reactivity to social competition tasks. Indeed, by addressing this question the research seeks to explore whether any of the psychosocial factors (i.e., attributional style, sense of coherence, personality, and trait affects) play the role of protective factors in the experiences of SED and their subsequent health implications. The third research question seeks to investigate whether cognitive perceptions of threat and challenge moderate endocrine reactivity to status competition. More broadly, by addressing this question the study endeavours to understand why not all individuals who experience SED necessarily experience negative health outcomes (Marmot, 2004). By addressing the last research question, the study aims to explore whether T fluctuations following status change (i.e., losing or winning a competition) have implications for future status-seeking behaviours and motivation. Prior research suggests that a drop in T following a social defeat, is associated with lower subsequent status-seeking behaviours, driven by individuals' motivation to avoid further loss of status (Knight & Mehta,2014). Locating these findings within the framework of SED, it could be argued that policies relating to state benefit application represent continued exposure to social defeat, and has biological consequences which can lead to lack of control, learned helplessness and lower motivation to engage with social task and the system. As such, this series of consequences may imply lower motivation to break the cycle of SED.

In light of the above, the current study investigates the following four key research questions:

1. Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?
2. Are psychosocial variables related to endocrine reactivity?
3. Does cognitive appraisal of threat/challenge moderate endocrine reactivity?
4. Does T reactivity correlate with a reduced motivation to engage socially?

3.1 Aims and Objectives

The study focuses on long-term unemployment as a subset of socioeconomic disadvantage. It can be a stressful life event with the potential to affect morbidity and mortality through unpredictability of events and scarcity of financial resources affecting psychological assets such as status, social support and time structure (Hughes et al., 2015; Linn et al., 1985). By exploring the chronobiological endocrine reactivity to threat/challenge cognitions within a bio-cultural theoretical framework, several major limitations in the existing SES/hormone literature (which render findings contradictory) are addressed.

First, quantitative determination of circulating T is possible from an assortment of biological material, i.e., plasma, serum, hair, saliva and urine. Principally though, there are two fluids which have been widely utilised for research purposes in the behavioural sciences: plasma and saliva. Early attempts at determining concentrations of circulating T, primarily for clinical purposes, traditionally utilised plasma or serum. Whilst this was advantageous in facilitating direct analysis of the circulating concentrations of total T in the blood, for the purposes of examining chronobiological changes there were and remain, several distinct limitations. As blood samples require time-consuming and often stressful venepuncture, obtaining invasive multiple samples over a period of hours can be painful and this procedure can be unattractive to participants (Dabbs, 1990). Moreover, measurement tends to be of the total rather than free, biologically active, fraction of T. The measurement of this free fraction is essential in bio-behavioural research as it represents in part, the fraction of total T that is available to exert a biological action at the cellular level (Smith et al., 1979). In the

proposed study, circulating T levels are obtained from salivary samples, thus allowing for determination of concentrations from the biologically meaningful 'free' fraction.

Second, establishing a baseline for T is not entirely straightforward. Whilst T production is under genetic control (Meikle et al., 1988) it is also responsive to a range of biological, environmental and psychosocial stimuli. In line with this, T concentrations have been shown to vary with time of day (Ahokoski et al., 1998; Baxendale et al., 1980; Dabbs, 1990; Turkes et al., 1980) and they fluctuate in a pulsatile fashion over minutes and hours (Al-Dujaili & Sharp, 2012; Veldhuis et al., 1987). Dabbs (1990) encapsulates some of the difficulties in designing suitable sampling regimens for bio-behavioural studies when he states, 'All this variability introduces error into behavioural studies, where stable measures are needed to characterise individual differences and changes over time. Without more information on these changes, one cannot know how many participants to run, how many measurements to take and when to take measurements' (p.83). Establishing a meaningful T baseline depends, at least in part, on what purpose the baseline is required to serve. In certain clinical practice for example, researchers have advocated collecting single samples each day over the course of a week and pooling the samples to provide a weekly average. However, when attempting to examine chronobiological changes in relation to social defeat this sampling protocol would be unsuitable. In an attempt to establish an appropriate baseline measure, researchers interested in hormonal responses to competition have, with few exceptions, utilised only one approach – to collect one-off salivary samples. Therefore, more recent guidance on salivary sampling protocols for bio-behavioural research are adopted (Al-Dujaili & Sharp, 2012) and employ a comprehensive salivary sampling protocol to address chronobiological reactivity in T and C.

Third. In animal and human models, it is suggested that given the potential costs to the life of the organism, decreased levels of T following defeat act to suppress intention to compete further (Knight & Mehta, 2014; Mehta & Josephs, 2006). The

current study seeks to determine whether potentially lower levels of T following defeat/loss do indeed predict a reduction in motivation to re-engage in subsequent status-related activities. This is accomplished by use of the motivational questionnaire which has been used to measure motivation following competition defeat/victory.

Fourth. Because endocrine parameters are modifiable not simply by objective competition outcome but complex perceptions of relative status, the research seeks to address issues related to threat/challenge cognitions. The study further explores specific psychosocial variables (i.e., sense of control, coherence, personality and trait affect) as they have been suggested to shape the subjective perceptions of status rank (Sapolsky, 2017; Walsh et al., 2014), and thereby relate to the complex relationship between endocrine response, status rank and health (section 2.5.2 for a review of the variable personality; section 2.5.5 for a review of sense of coherence; section 2.7.4 for threat/challenge). Moreover, it has been suggested that a number of those variables are closely related to protective factors (i.e., sense of control and sense of coherence) contributing to the construction of threat/challenge cognitions (Antonovsky, 1987; Denton et al., 2004). Considering the importance of all these factors (some of which have been identified as resilient characteristics within extant the literature) upon hormonal response to status and formation of threat/challenge cognitions, the present research seeks to address the relationship of each of those psychosocial determinants to endocrine response. Furthermore, drawing on the BioPsychoSocial model of challenge and threat, which suggests that cognitive appraisal of a stimulus has later implications for hormonal and behavioural responses to the environmental challenge, the study seeks to investigate the role of threat/challenge cognitions as moderators of the link between endocrine response and status competition outcome.

Furthermore, because motivation to re-engage with consequent homologous tasks has been associated with post-competition status-induced fluctuations in T (Knight & Mehta, 2014), the present study aims to further shed some light on this relationship within the context of socioeconomic disadvantage and social defeat.

3.2 Testosterone

As previously explored, social, political and economic disadvantages and inequalities disproportionately affect certain populations, with the worst health outcomes being observed amongst female, ethnic minority and migrant groups (Varela-Silva & Santinho, 2016). Consequently, a sophisticated body of literature focuses on the deleterious effects of social inequalities upon health, with particular attention being paid to the implications of continuous stress and the subsequent elevated levels of corticoids for a variety of fundamental body functions such as the immune, reproductive and digestive systems. Considerably less attention has been paid to the relationship between SED and testosterone, however.

The limited research which has been undertaken suffers from four major impediments. Firstly, studies employ biomarkers for their own sake with no significant development of biocultural theory. Secondly, studies utilize plasma samples and testosterone concentration is subsequently determined using radioimmunoassay, which almost invariably measures total rather than the biologically active 'free' fraction of testosterone. This renders interpretation of simplistic correlational studies highly problematic. Thirdly, even though androgens impact upon female cognitions, emotion and behaviour every bit as much as males, women are overlooked in these studies. As the burden of poverty falls disproportionally on women this oversight is unfortunate. Fourthly, studies consider putative basal testosterone levels which are then correlated to low SES or behaviours claimed to be concomitant with poverty. However, androgens are highly labile and levels are modified in response to a wide range of social stimuli, especially those related to status. Whilst being socially subordinate through facing SED is not inevitably detrimental to health and psychological well-being, in the theoretical context of the challenge hypothesis/Mazur's biosocial model of status, where a subjective experience of an objective outcome (win or loss) impacts upon circulating testosterone levels, the resultant disparity has been demonstrated to have emotional and behavioural consequences that may underpin the maintenance of poverty. Several physiological

consequences of this 'winner effect' are likely moderated by cognitive interpretation of events as being either challenging or threatening.

Consequently, this study addresses the four major limitations inherent in poverty/androgen research by adopting a range of measures that will contribute to a greater understanding of the phenomenon. Firstly, the use of salivary androgenic biomarkers is positioned within a well-developed conceptual and theoretical framework, drawing upon ethological and evolutionary traditions. Secondly, circulating testosterone levels will be determined from salivary samples, thus allowing determination of concentrations from the biologically meaningful 'free' fraction. Thirdly, this study employs a highly sensitive in-house enzyme-linked immunosorbent assay (ELISA), optimised specifically for the levels of testosterone typically found in women and young children, thereby allowing inclusion of cohorts experiencing lower testosterone levels in analysis. Finally, using a comprehensive salivary sampling protocol, this study will investigate chronobiological changes in testosterone reactivity rather than static basal levels, allowing exploration of the role that modification of testosterone levels when encountering threat and challenge has upon cognitions and behaviours reinforcing SED.

3.2.1 Blood vs Saliva samples – total vs. free testosterone

The first concern that will be addressed in this thesis is related to the procedures used to determine circulating testosterone levels in research and the plausibility of the measurements. A wide variety of biological material has been used in research to determine circulating testosterone levels including serum, hair, plasma and urine. However, saliva and plasma are the two most commonly examined within behavioural endocrine research (Sharp, 2006). Whilst in clinical research, studying traditional serum or plasma serves the purposes for direct analysis of total circulating testosterone levels in the blood, chronobiological changes - focus of bio-behavioural research, require slightly different strategies. Thus, measurements of total rather than biologically free testosterone imply limitations for chronobiological changes. This

occurs as free testosterone is just a fraction of the total circulating testosterone levels, however only this fraction of testosterone acts as a biological activator at a cellular level and not total testosterone (Laurent et al., 2016). Another advantage of measuring free testosterone is that it does not require the adoption of an invasive collection procedure utilised in the collection of total testosterone, such as venepuncture. Subsequently, the benefits of collection of free testosterone in the saliva compared to total testosterone in blood are several; it is non-invasive, it encompasses chronobiological changes (a central focus of bio-behavioural research) and it does not create difficulties for participants to collect the required amount of saliva (3-5ml) from a passive drool into a disposable tube (Al-Dujaili & Sharp, 2012; Dabbs, 1991; Riad-Fahmy et al., 1983). Salivary sampling protocol thus appears to be the preferable technique for a non-invasive, chronobiologically changing observant and allowing vulnerable groups participation in the research method.

3.2.2 Basal vs Reactivity

When looking at chronobiological changes of hormones in bio-behavioural research, one should consider the importance of hormonal inflections such as circadian rhythm. Failing to do so might result in serious misinterpretation of results (Sharp, 2006). Testosterone levels fluctuations are observed not only as a result of circadian rhythm but also from the effects of the production and release of gonadotropic releasing hormone (GnRH) and luteinizing hormone (LH), resulting in rapid testosterone level changes frequently observed within short periods of time (Filaire et al. 2009; James & Baxendale, 1994; Kivlighan et al. 2005). The accuracy of previous hormone-competition studies where researchers fail to include a baseline into their study design (see Elias, 1981), but rather assess the relative alteration between pre- and post-competition endocrine levels, are therefore questionable. Furthermore, studies of this nature frequently only include a single salivary or blood sample (Sharp, 2006). Nevertheless, in instances where researchers do not discredit the importance of the establishment of baseline and circadian rhythm fluctuations, their measures again tend to be limited due to their inability to account for episodic hormonal fluctuations by only collecting samples at roughly the same time of the day. For example, if a pre-

competition sample is arranged for 12pm, the researchers establish a baseline by collecting a one sample the day before at around 12pm. Resultantly, researchers interpret any hormonal variance as a result of competition outcome or individuals' perception of it (Sharp, 2006). In order to avoid these misinterpretations, this study draws on a meaningful baseline. Further details of the comprehensive sample protocol adopted in this study will be provided in the following section.

3.2.3 Sampling

Due to the impact of various environmental, biological and psychosocial factors on testosterone levels, establishing a baseline is not an easy task (Sharp, 2006), with physical and sexual activity, alcohol consumption, cognitive states, nutrition, mood, status-related behaviours, immune function, stress and aggressive behaviour (Morris et al., 1987; Swift, 1989) all capable of affecting testosterone levels. These levels can also fluctuate through the day and across different seasons (Demir, 2016; Stanton et al., 2010; Stern et al., 2020; Lee, 2020). Whilst testosterone levels in males exhibit a pulsating pattern over short periods of time (Veldhuis et al., 1987), in women, testosterone levels fluctuate across the menstrual cycle with individuals experiencing higher concentrations around the middle of the cycle (Vermeulen & Verdonck, 1976). Nevertheless, literature findings on testosterone levels and menstrual cycle are ambiguous, with the implications for free testosterone also remaining vague (Dabbs, 1990). Indeed, Dabbs stresses the importance of these determinants by arguing that 'all this variability introduces error into behavioural studies, where stable measures are needed to characterise individual differences and changes over time. Without more information on these changes, one cannot know how many participants to run, how many measurements to take and when to take measurements.' (p.83).

Subsequently a meaningful baseline is required when designing studies and scholars should reflect on what purposes of the established baseline is, to calibrate it accordingly. For clinical studies a sample protocol utilising single-point day samples over the course of a week, establishing a weekly average, that allows researchers to

determine a clinical condition or the lack of it based on testosterone levels analysis has often been regarded as the most appropriate approach. With bio-behavioural research however, the situation is slightly different because the main interest here are the chronobiological changes. In light of this, a sample protocol will appear to have a low utility for the purposes of this research. Nevertheless, as described in the previous section, bio-behavioural research also appears to predominantly lean towards a single-point sample protocol when establishing baselines. Table 1 (adapted from Sharp, 2006) provides a comprehensive summary of the various sampling strategies used by researchers in their endeavour to determine baseline testosterone levels.

Table 1

Baseline Levels of Testosterone: Bio-Behavioural Research and Sampling Times

Authors (year)	Sex	Baseline	Pre-Competition
Bateup et al. (2002)	♀	24hrs prior	15 mins prior
Mazur et al. (1997)	♂ ♀	10 mins prior	3 mins prior
Filaire et al. (2001)	♂	3 weeks prior	5 mins
Gonzalez-Bono et al. (1999)	♂	None established*	45 mins prior
Passelergue and Lac (1999)	♂	15 mins	immediately
Suay et al. (1999)	♂	45 mins prior	immediately
Eubank et al. (1997)	♂	None established*	10 mins prior
McCaul et al. (1992)	♂	None established*	immediately
Booth et al. (1989)	♂	Approx. 24hrs prior	15 mins prior
Gladue et al. (1989)	♂	10 mins prior	5 mins prior
Salvador et al. (1987)	♂	None established*	10 mins prior
Elias (1981)	♂	None established*	10 mins prior

Note. In the studies where baseline has not been established, authors have adopted the strategy to use pre-competition sample as a baseline (Sharp, 2006).

The lack of consistency around sampling protocols and establishment of a meaningful baseline gives a potential explanation of the debate amongst existing literature on misinterpretation of results (Salvador et al., 1987). Sharp's work (2006) on female

competition provides a comprehensive guide on the utilisation of a multiple point sampling protocol, whilst establishing a meaningful baseline and sheds some light on the uncertainties around hormonal responses to competition. Considering all these issues, one should interpret with caution the reliability and validity of early assay technology studies due to the significant limitations of their methodological designs. Nevertheless, background literature proves to be important not only for the exploration of previous design limitations, but also allows us to discredit the argument supporting the credibility and utility of single time-point measures in current bio-behavioural research.

This study adopts a comprehensive sampling protocol where multiple samples are collected across two non-consecutive days, allowing the research to establish a meaningful baseline and control for circadian activity. Furthermore, the study methodologically accounts for additional factors impacting circulating testosterone levels.

3.3 Cortisol

The previous literature review chapter has examined the impact of low SES or experience of SED upon basal cortisol levels, circadian activity throughout the day and cortisol reactivity, whilst the ways in which cortisol affects status rank has also been discussed. Compared to testosterone, the relationship between cortisol and status-related behaviours seems to be better understood. Nevertheless, research examining cortisol reactivity and status-related behaviours should also be interpreted with caution due the overlapping methodological limitations in steroid hormone research. In light of these limitations, this study will proceed in the following manner: (1) circulating cortisol levels will be determined from salivary samples, thus measuring biologically free, rather than total cortisol concentrations; (2) employing a highly sensitive in-house enzyme-linked immunosorbent assay (ELISA); (3) adopting a comprehensive salivary sampling protocol and meaningful baseline will allow the investigation of chronobiological changes in cortisol reactivity rather than diurnal and

basal cortisol levels, and thereby investigating how cortisol levels modifications in relation to threat and challenge appraisals and subsequent behavioural and cognitive implications affect the cycle of SED.

3.3.1 Basal vs Reactivity

Whilst much of the existing literature is concerned with the effects of SES on basal cortisol levels, this study will focus on chronobiological changes and thus examine acute cortisol fluctuations. In doing so, it will expand on the known link between SES and cortisol response to psychosocial and physiological stressors.

A small number of studies demonstrate that individuals experiencing lower SES appear to have higher cortisol response to laboratory induced psychosocial stressors (Adler et al., 2000; Fiocco et al., 2007; Kristenson et al., 1998). Moreover, following exposure to pharmacological challenge of the HPA-axis function, low SES has been associated with a cortisol hyperactivity but not high SES (Rosmond & Björntorp, 2000a). Another study from Rosmond and Björntorp (2000b) demonstrates less efficient cortisol concentration reduction, following a dexamethasone suppression test (i.e., testing HPA-axis' ability to supress cortisol production in response to a cortisol agonist – dexamethasone), exhibited by individuals from low SES compared to individuals from higher SES backgrounds. These findings suggest that individuals from lower SES might exhibit a stronger activation of the HPA-axis and slower return of the elevated levels back to baseline, thus rendering that the effects might be insignificant if they happen once or twice a week or month. However, in repeated exposure, an accumulative effect occurs resulting in chronically elevated cortisol levels, inability of the individual to return to homeostasis, slow and sluggish recovery and augmented cortisol reactivity to stressors. Cortisol reactivity therefore, might be a better predictor of the susceptibility to stress-reactive psychopathologies and receptivity of intervention (Al-Dujaili & Sharp, 2012).

Studies to capture the dynamic model of status has been undertaken by numerous scholars examining the relationship between cortisol reactivity and competition outcome. Findings however, remain inconsistent. Whilst some studies report a well-established relationship between lower cortisol levels following competition victory and higher cortisol levels following defeat, as is found in animal species (Jiménez et al., 2012; Sapolsky, 1999a,b; Stanton & Edelstein, 2009), others either find a lack of, or a reversed relationship where cortisol levels are lower after victory (Hasegawa-Ohira et al., 2011; Oliveira et al., 2009; Suay et al., 1999). This suggests that findings should be interpreted with caution due to research failing to utilise a meaningful baseline whilst interpreting any hormonal deviation as a result of contest outcome. Furthermore, as with testosterone, research frequently fails to utilise comprehensive sampling protocols. Lastly, inconsistencies in research findings where some research finds null relationship between testosterone and status-related behaviours (Archer, 1998), whilst others find no relationship between cortisol and status (Gadinger et al., 2011), might stem from the fact that the two hormones may interact in their implications for status related behaviours rather than act independently (Knight & Mehta, 2014). This will be further reviewed in the cortisol/ testosterone contradiction section of this literature review.

3.3.2 Sampling

Research of cortisol lack a meaningful baseline and comprehensive sampling protocol when it comes to examining the relationship between reactivity and status related behaviours in competition studies (Bateup et al., 2002; Hankin et al., 2015; Stanton & Edelstein, 2009). This study will endeavour to create not only a meaningful baseline but also a comprehensive sampling protocol that allows the researcher to redress any methodological limitations and shed some light on the inconsistent social endocrinology research findings.

3.4 Cortisol and testosterone contradiction

Mehta & Josephs (2010) argue that testosterone and cortisol should act in tandem when it comes to status-related behaviours, with testosterone positively only affecting status-seeking behaviours and the obtainment of higher rank when GC levels are low. Conversely, high cortisol levels should result in minimal impact of testosterone upon status-related behaviours. This argument has been coined the dual-hormone hypothesis (Mehta & Josephs, 2010). Consistent with Mehta & Josephs' hypothesis, several other studies examining testosterone/cortisol interactions within the context of sport, decision-making, leadership, social network popularity, dominance-related traits and management report the same findings (Casto et al., 2019; Edwards & Casto, 2013; Mehta & Josephs, 2010; Mehta & Prasad, 2015; Pfattheicher, 2017; Ponzi et al., 2016; Sherman et al., 2016). The consistency of these findings lends weight to the argument for the prevailing role of cortisol as a moderator of the relationship between testosterone and social dominance. Thus, an explanation for the inability to find a relationship between testosterone and social rank in previous research on the context of the challenge hypothesis might stem from the fact that researchers fail to account for the inhibiting effects of cortisol (Knight et al., 2020).

Furthermore, the authors suggest that the interaction between testosterone and cortisol might feature on a psychological level due to the relationship between testosterone and status-seeking motivation, and cortisol and social-approach inhibition (Mehta & Josephs, 2010). Resultantly, social approach (associated with low cortisol levels) and high-status drive (associated with high testosterone) might lead to higher social rank and social dominance, whilst the reversed relationship (social inhibition associated with high cortisol, or high status-seeking motivation associated with high testosterone) might result in lower rank and compliant behaviours. This argument sits well within a broader evolutionary framework of modulating complex social behaviour in relation to stress (HPA axis) and reproductive axis (HPG) (Carré & Mehta, 2011). Reproductive related behaviours could possibly be inhibited by high environmental stress due to their metabolic cost and threat for survival mechanisms.

Logically then, status-seeking behaviours are exhibited when they are not metabolically costly within a low stress environment (Knight & Mehta, 2014).

The proposed argument however, lacks empirical justification and it remains highly speculative. Further research is required to clarify the precise relationship through which testosterone and cortisol interact. This study, by adopting a comprehensive sampling protocol, therefore aims to reconcile the cortisol and testosterone anomalies in relation to status-seeking behaviours amongst groups differing in SES.

3.5 Research Philosophy

An underpinning philosophy of research has been highlighted as important (Bahari, 2010) and is relevant to both natural and social sciences. Moreover, it has been suggested that when researchers do not consider epistemological and ontological philosophical positions, it can compromise the quality of their research (Easterby-Smith et al., 2002). Indeed, research philosophy has been associated with the development of knowledge and its application in the social world. It encompasses one's subjective experiences and views of the world. Thus, directly reflecting on researcher's notion of research design and methods and affecting the general understanding and thinking of the research process (Bahari, 2010; Cohen et al., 2018). Easterby-Smith and colleagues (2002), emphasise the following three main reasons why an underpinning philosophy should be considered, understood and applied in research. Firstly, it promotes clarity regarding research designs; secondly, enhances the understanding of which design would best suit the purposes of the research and lastly, enables researchers to identify, create and design studies even outside the spectrum of their background experiences and knowledge.

Considering the use of combined quantitative objective measures and observations, questionnaires and biomarkers, the application of specific research questions and variables, and the test of theories in this research, the most appropriate epistemological position for the current study appears to be positivism, whilst the

ontological is objectivism. The next section will further expand on these two ontological and epistemological orientations, outline their benefits for research and identify limitations.

3.5.1 Positivism as an Epistemological Orientation

Positivism is a widely accepted, recognized and utilized epistemological position in quantitative research (Bahari, 2010). It posits that the social world exists externally, whilst there are social facts that comprise of an objective reality. Easterby-Smith and colleagues (2002) report that positivisms lead one to the position that the only knowledge of significance is considered to be the one that stems from independent, objective observations of the external reality, meaning that the ways through which researchers discover and acquire knowledge are neutral and technical processes (Lee, 1992). Researchers must therefore apply independent objective experimental methods that allow them to test theories and hypotheses which in turn will "gradually develop and refine universal laws of nature" (Bahari, 2010, p.23). Staiton-Rogers (2006) further argue that positivist dogmas regard definite relationships between events and things in the external world and one's knowledge of those. The logic of experimental designs derived from natural sciences are those that drive and directly impact upon the positivistically oriented research methodologies. Thus, positivist research frequently involves formal questionnaires and large-scale surveys to explore a wide variety of research topics. Another significant characteristic of this approach is the use of statistical analysis, measures of associations and measurement models (Bahari, 2010).

Since the present research utilises objective endocrine parameters to explain regularities in human social behaviour (Easterby-Smith et al., 2002), and seeks to examine the causes of changes in social facts through objective instruments and quantitative analyses (Firestone, 1987), a positivist epistemological position seems apposite. Another aspect that supports the adoption of the outlined epistemological orientation is the fact that this research utilises generalized theoretical statements and measurements that are universally accepted and applicable. For example, the NEO-FFI questionnaire used to measure personality style in the current study has frequently been used to assess personality style in other studies in this area (Bahari, 2010).

Positivist orientation of course, does not come without its limitation, including critique of the stance that all knowledge and concepts are based on experience. However, not all concepts are based on experience, for instance, time, space and cause. These ideas are better understood as social, contrasted by the society and circumstances. The present research aims to address this issue by acknowledging the importance of cognitive factors (threat/challenge) and individual's perceived experiences of SES for endocrine reactivity to status encounter. Hence, although capturing this aspect numerically, through questionnaire and statistical analyses and thereby in a strictly positivistic manner, the study endeavours to address the importance of individual beliefs and subjectivism, to some degree, by using objective measures. Consequently, the current study hypothesises that endocrine responses to competition only tell us so much and thus, what appears to be of fundamental importance for the research are individual's subjective experiences (threat/challenge) of an objective outcome (competition outcome). This prediction is derived in part from the previous findings that not all individuals who experience SED necessarily experience negative health outcomes (Marmot, 2004) and that subjective perceptions of SES appear to be better predictors of health than objective SES (Adler et al., 2000).

3.5.2 Objectivism as an Ontological Orientation

Considering the utilisation of objective research methods, statistical analyses and independent observations in this research, objectivism as an ontological orientation seems an appropriate position to adopt. According to objectivist dogmas, "social phenomena and their meanings have an existence that is independent of social actors" (Bryman, 2004, p. 16). By adopting this ontological orientation then, research aims to predict reality in the most objective manner (Davies et at., 1993). Moreover, objectivism is also associated with methods extracted from the natural sciences, in particular, the numerical objective measurement of relationships between 'things' (David & Suton, 2004). This allows the results to be verified against the most possible "theory- neutral objective data", thereby resulting in the best achievable "genuine empirical knowledge" (Bahari, 2010, p. 25).

Capturing endocrine parameters and psychological variables by numerical measurements in this research, thus arguably allows to apply the objectivists view of the social world as real, hard and concrete as the natural, affecting the individuals in various ways (Bahari, 2010; Morgan & Smircich, 1980). This has been echoed in the present research as endocrine parameters and psychological data are explored in two objectively classified SES groups. However, it is also hypothesised that amongst individuals of the same SES group, a universal response attuned to objective status would not be demonstrated due to various ways in which this concrete reality affects individuals and based on their subjective experiences of this social world. Considering that individuals' subjective experiences have been captured numerically and assessed on self-reported measures, or indeed by adopting a positivistic approach, rather than via qualitative measures (e.g., interviews) and the suitable for those - interpretivism, it could be argued that this applies a degree of limitation to the research in relation to how well self-reported instruments capture subjectivism.

3.6 Methods

In an endeavour to understand some of the neuroendocrine mechanisms underlying status disparities in health and to address the research questions and subsequently emerging hypotheses most efficiently, this research draws on various quantitative methods. Moreover, to best capture the multifaceted nature of SED and its behavioural and biological consequences, both questionnaires and biological parameters are employed.

Hypotheses:

1. Socio-economically disadvantaged population will demonstrate a dissimilar pattern of endocrine response to a social defeat stimulus compared to a higher SES population.
2. Psychosocial variables will relate to endocrine reactivity.
3. Cognitive appraisals of threat/challenge would moderate endocrine reactivity in both SES populations.
4. Reduced motivation to engage with subsequent social tasks would positively correlate with a decrease of T reactivity.

The lack of direction in the patterns of endocrine reactivity in hypothesis 1 and direction of moderation for hypothesis 3 stems from the fact that the endocrine literature is extremely equivocal and results remain inconsistent (as discussed in section 3.3.1, p. 139). Considering the contradictory results from the past literature and methodological limitations of studies, providing a concrete direction of how endocrine response will differ (hypothesis 1) and cognitive appraisals will moderate endocrine reactivity could be misleading. For that reason, H1 and H3 were tested as two-tailed hypotheses.

In pursuit of answers to the first research question, and to examine the above hypothesis, this section discusses the methodological approach through which hormones are extracted, determined and analysed. In the interest of the second and third research question and hypothesis, validated psychological instruments utilised to measure relationships to and moderators of endocrine reactivity are discussed. This section also focuses on the nature of the chosen experimental task and its' justification, experimental procedure, study design, sampling, and the applied statistical and hormonal analytical strategies.

3.6.1 Study Design

The study employs a factorial repeated measures design to determine testosterone and cortisol reactivity in response to a non-physical, dyadic, status-related, experimental task. The independent variables are: personality (NEO-FFI-3), employment status, SES, attribution style, perceptions of threat/challenge, importance of the task for the individual (prior exposure), sense of coherence and competition outcome (win/loss). The dependent variables are: circulating levels of salivary testosterone and cortisol, affect state (PANAS-X) and task difficulty (NASA-TLX workload).

3.6.2 Materials

Various materials were utilised for the different components of this research. For screening purposes of the exclusion criteria and in order to recruit the targeted populations, the study utilises a demographics questionnaire. To address whether socio-economically disadvantaged population demonstrate a dissimilar pattern of

endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status, the study utilises endocrine parameters. In order to create a competition condition, the study uses a financial form-filling task; detailed justification for the chosen task is provided later on in the text. Because endocrine parameters are modifiable not simply by objective competition outcome but by complex perceptions of relative status, the research seeks to address issues related to threat/challenge perceptions. Furthermore, as discussed in the literature review, resilient/protective psychosocial factors (i.e., sense of control, sense of coherence, personality, and trait affect) also appear to be closely related to the experiences of SED and link to physiological response (i.e., endocrine reactivity). For these purposes, the questionnaires: Attributional style, Sense of Coherence, Threat/Challenge Questionnaire, NEO-FFI- 3 and PANAS-X were used to measure the abovementioned concepts respectively. Considering the importance of all these factors, some of which identified as resilient characteristics by the literature, the present research seeks to address the relationship of the aforementioned psychosocial factors to endocrine response and moderating role of threat/challenge cognitions in endocrine reactivity to competition outcome. Lastly, because motivation to re-engage with consequent homologous tasks has been associated with post-competition status-induced fluctuations in T (Knight & Mehta, 2014), the present study aims to further shed some light on this relationship within the context of socioeconomic disadvantage and social defeat. This is accomplished by use of the Motivational Questionnaire, capturing motivational scores following exposure to competition defeat/victory.

Firstly, in order to obtain information about volunteers' background and classify them into two separate objective SES groups, a demographics questionnaire has been used at the first stage of the study. The *demographics questionnaire* comprised age, educational and occupational levels, number of individuals working within the family and marital status (Appendix I). Example questions: "Please choose your highest educational qualification"; "Please indicate your annual income". The demographics questionnaire allowed the researcher to monitor for some of the exclusion criteria (e.g., age), to be able classify individuals into two stratified samples; low and high SES, and to report demographic characteristics of the sample.

Endocrine parameters – were examined in 12 salivary samples across two non-consecutive days. The first day served the purposes of a meaningful baseline to which endocrine reactivity on the second day (day of competition) were compared. On the first day, 48hrs prior to the competition, 4 time-matched salivary samples were collected. On the competition day, the collected salivary samples were 8; 4 time-matching samples with the baseline and 4 used to examine endocrine reactivity following the competition exposure. Following Sharp (2006), the utilisation of multiple sampling protocol and meaningful baseline allows the study to address methodological limitations of previous research, outlined in the last sections of the literature review. Detailed study procedure is provided later in this chapter.

To identify whether there are differences in endocrine reactivity in response to threat/ challenge task appraisals and between SES groups, the study creates an environment where physiological stress response will be demonstrated by participants from both SES groups (due to exposure to hormone status competition). This is done by the utilisation of an experimental task – financial form-filling competition - that aims to create a competition setting in which stress response will be triggered and endocrine response will be observed. The *Financial Questionnaire used for the form-filling competition* (Appendix II) comprised questions related to the participants financial ecosystem: "Does the person you live with share financial responsibility with you or make a contribution to your financial situation?"; "How many properties do you own, or partly own, in the UK and/or overseas?"; "Do you (and your partner) have finances in savings accounts, ISAs, bonds, premium bonds, invested in shares, funds etc.?". The estimated time for completion of the task was 40 minutes (approximate time based on performed pilot study comprising 5 individuals from both SES groups; individuals were not included in the sample size); however, time and outcome were manipulated by the confederate's response (i.e., the researcher manipulated whether confederate or opponent will win the competition prior to exposure to it). Additional paperwork was potentially required for the completion of the questionnaire (e.g., credit history, mortgage paperwork). Whilst this may have resulted in incomplete or false answers provided by the participants, what was important was not the answers provided by the

participants on the finance questionnaire (all data were immediately destroyed after task completion), but the endocrine response elicited by the task.

There were several methodological and theoretical considerations in choosing the experimental task. First, it was necessary to devise an experimental task which would produce threat cognitions (distinguishable from challenge cognitions). Consequently, a financially oriented task was used in order to facilitate a framing effect. This draws upon the theoretical position that disadvantaged individuals who lack financial resources might realistically be expected to experience bandwidth overload when engaging with a finance-related task (Mullainathan & Shafir, 2013). Second, because the study sought to ascertain whether differences in perceptions of a task (threat/challenge) produce a dissimilar endocrine response, both groups (low and high SES) were exposed to the same objective experimental conditions; what differed was subjective interpretation. Hence, based on Mullainathan and Shafir's work on the mental bandwidth taxation hypothesis (2013), it was anticipated that threat cognitions would occur predominantly in the disadvantaged population, although this was by no means a certainty or necessity (Marmot, 2004). This results from the fact that although higher levels of stress and the subsequent negative psychological and physiological implications have been found to be more concentrated in low SES populations, not all individuals from disadvantaged groups experience elevated levels of stress and threat cognitions (Kim et al., 2018; Marmot, 2004; Sapolsky, 2017).

Amongst lower SES populations there will be individuals who will exhibit resilience towards perceptions of disadvantage and the multiple stressors frequently associated with living in socioeconomic disadvantage. And so, in order to successfully facilitate disparity in the IV of task cognitions (with two levels: threat and challenge) between the two groups, the chosen financial form has been utilised. If the task were to be changed to something which is neutral (and it is impossible to know a priori whether it would be neutral because we are dealing with individual perceptions of the task) (Tomaka et al. 1993; Tomaka et al., 1997), there is the distinct likelihood that both groups, low and high status, would perceive the task as a challenge; thus, undermining the purpose of the experiment. As such, this would not allow the investigators to

address the research question of whether there are differences in endocrine reactivity in response to threat or challenge task appraisals.

Personality style, sense of coherence and attributional style (capturing sense of control) are amongst the factors established as shaping the perceptions of SES and thereby the implications of SED for health and wellbeing, whilst threat/challenge appraisals even more vividly illustrate psychological and physiological differences in responses to stress. This directly translates into the fact that endocrine reactivity to social defeat is modifiable by cognitive parameters and thereby, more broadly, could account for why experiences of SED are associated with ill-health in some individuals but not in others (e.g., the satisfied poor) (Marmot, 2004). In the light of these arguments and drawing on bio-behavioural research which highlights the significant cognitive moderators of endocrine reactivity in status encounter (Blascovich & Mendes, 2000; Lazarus & Folkman, 1984), and suggests links between psychosocial variables and physiological responses (Steptoe & Marmot, 2002; Taylor & Seeman, 1999), this study accounts for those factors by measuring them on the following validated, widely utilised and well-established amongst psychological studies instruments:

Threat/Challenge Questionnaire - In conjunction with collection of salivary samples, participants were asked to respond to a number of written questions concerning how threatening or challenging the task was for them and how important they believed it to be for themselves (Appendix III). Threatening/challenging perceptions were scored on a 9-Likert point scale, where -4='Threatened', 0='Neither' and +4='Challenged', where threat "may be associated with lack of emotional, psychological or physical resources to cope with the task", whilst challenge "may be associated with slight anxiety and fear of the task, however sufficient emotional, physical or psychological resources to cope with the task". The importance of the task was scored on a 6-Likert point scale, where 0= 'not at all' and 5= 'very much so'. As with the Financial Questionnaire, a pilot study (n=5, low and high SES) was conducted prior to the experiment, to test the clarity, reliability and validity of the threat/challenge cognitions and importance of task (individuals were not included in the experimental

sample size). The two questions (how threatening/challenging the task was for the participants, and the importance of the task for the participants) were scored and analyzed independently. For the 'threat/challenge' question a high score indicates challenge appraisal, whilst a low score indicates threat appraisal of the competition task. Similarly for the 'importance of the task' question a low score reflects little importance, whilst a high score reflects significant importance of the task for the individual.

NEO–Five-Factor Inventory-3 (NEO- FFI-3) - the instrument comprises 60 items (statements) and it is the shortened version of the Revised NEO Personality Inventory (NEO-PI-R) produced by Costa and McCrae. The tool is designed to measure personality based on five personality domains: Neuroticism, Extraversion, Openness to Experience, Agreeableness and Conscientiousness. Questionnaire statement examples include: "I am not a worrier."; "I like to have a lot of people around me."; "I laugh easily.". All statement responses are recorded on a scale from 'strongly disagree' to 'strongly agree'. Once all statements are recorded, a raw total score for each of the aforementioned personality domains is produced. For instance, for the Neuroticism domain, a sum of all the values of the marked responses (12 items) is calculated. The same procedure is applied to the remaining domain scores. Following that, the raw scores from each subdomain are used to produce profiling scores (T-scores). This is done by plotting them on a scale (from ≤25 to ≥75) indicating whether the scores are very low, low, average, high, or very high. A high raw score (e.g., 36) on a subdomain (e.g., Neuroticism) reflects a high profiling T-score (i.e., very high neuroticism trait). The original tool (NEO-FFI) has demonstrated to be a reliable and valid measure of personality, with excellent internal consistency (0.68-0.86) across cultures (Aluja et al., 2005). However, the revised version demonstrates improved psychometric properties and readability, which is why the present research utilises the latest version of the NEO-FFI instrument (NEO-FFI-3). NEO-FFI-3 is considered to have stronger structure and increased reliability. The instrument is also more suitable for younger individuals (Costa & McCrae, 2010).

PANAS- X - Emotional states were evaluated by the Positive and Negative Affect Schedule Expanded Form (PANAS-X, developed by Watson) prior to and following experimental exposure. The instrument demonstrates good reliability and validity, whilst also considered a responsive measure of emotional experience, regardless of subject population or time frame (Watson & Clark, 1994). The self-report 60-item instrument measures 11 specific affects (Fear, Sadness, Guilt, Hostility, Shyness, Fatigue, Surprise, Joviality, Self-Assurance, Attentiveness and Serenity) and two general Positive and Negative affects. The scale consists of a number of words and phrases (e.g., cheerful, disgusted, attentive) that describe different feelings and emotions related to the abovementioned 11 specific affects. The individuals are required to provide an answer next to each of the words, based on the extent to which participants have felt that way during the past few weeks. A scale from 1 to 5 is used, where 1= 'very slightly or not at all' and 5= 'extremely', to record the answers. A total score for each scale is produced by summing all the responses to the items belonging to that scale. For instance, for General Negative Affect a sum of the responses to the items; afraid, scared, nervous, jittery, guilty, ashamed, irritable, hostile, upset, and distressed is required. The higher the score on the General Negative Affects scale, the higher the negative affect. The instrument has been validated in several languages (including Spanish and French) and demonstrates high internal consistency - alpha = .89 and .91 for Positive Affect and Negative Affect in women, respectively; and alpha = .87 and .89 for Positive Affect and Negative Affect in men, respectively (Díaz-García et al., 2020). The tool also demonstrated a very strong overall internal consistency (alpha= 0.86) in this study.

NASA Task Load Index (NASA-TLX) - Upon completion of the form-filling competition, participants' workload was assessed by a modified NASA-TLX, comprising six numerical scales (namely; Mental Demand, Physical Demand, Temporal Demand, Performance, Effort, and Frustration), rated on a scale from -5 to 5 (Hart & Staveland, 1988). The study utilises this instrument in order to measure individuals' subjective experience of the task, combining the indicators previously outlined. Thus, a high score of 5 on the subscale 'Mental Demand' would reflect a very high mental demand. The original NASA-TLX tool has been developed by the Human

Performance Group at NASA's Ames Research Centre and used to measure task workload. However, the instrument has been modified to suit the purposes of the study by eliminating the weighting process (i.e., weighting the subscales) resulting in utilisation of the commonly named Raw TLX (RTLX). There are several methods by which the results from the measure could be analysed, including evaluating each of the subscales separately (particularly in cases where the subdomains appear irrelevant for the research purposes), or by adding or averaging the scores on the subscales and generating a total workload. In this study, the scores of the NASA-TLX subdomains have been added to create one overall score prior to the inferential analysis (Table 10). The modified version of the instrument has been utilised by Hart (2006) and proven to be a sensitive and reliable measure of task workload. The tool demonstrates high internal consistency (>0.80) (Xiao et al., 2005). For this study we found an adequate Cronbach's alpha (0.50). However, this did not replicate the high internal consistency found by other studies.

Attributional style questionnaire - The study utilised (ASQ) in order to measure participants' explanatory style for bad and good events. The instrument is comprised of three causal dimensions: internal versus external, stable versus unstable and global versus specific causes (Peterson et al., 1982). The questionnaire includes 12 hypothetical situations, consisting of 6 'good' and 6 'bad' events (e.g., 'You meet a friend who compliments you on your appearance'; 'You have been looking for a job unsuccessfully for some time'). Each of these situations is followed by a series of 4 questions. The first question following each situation asks for the major cause of the situation and serves as an aid to better answer the following three questions (the first question is not added to the total score). The next three questions evaluate whether individual's responses are: 1) internal versus external; 2) stable versus unstable; and 3) global versus specific causes. Example questions: for internal versus external – "Is the cause of your friend's compliment due to something about you or something about the other person or circumstances?"; for stable versus unstable – "In the future when you are with your friends, will this cause again be present?"; for global versus specific causes –"Is the cause something that just affects interacting with friends or does it also influence other areas of your life?". Each response is recorded on a scale from 1 to 7.

For good events, a score of 1 represents the lowest score, whilst a score of 7 represents the highest. In comparison bad events, a score of 1 is the highest, whilst a score of 7 is the lowest. Due to the reversed order of scoring, when analysed, scores for good events should be separated from the scores for bad events (Peterson et al., 1982).

The instrument demonstrates respectable overall internal consistency (range 0.75-0.72) and stability (range 0.65- 0.69). Cronbach's coefficient alpha for each individual subscale demonstrates an internal consistency of (range .44 to .69) and a mean reliability of .54. For the composite bad and good events reliabilities were .72 and .75, respectively. The authors (Peterson, Semmel, Von Baeyer, Abramson, Metalsky & Seligman) however, suggest the utilisation of the composite scores (i.e., Composite Negative Attributional Style (CoNeg), Composite Positive Attributional Style (CoPos), and Composite Positive minus Composite Negative (CPCN)) rather than the individuals (i.e., Internal Negative, Stable Negative, Global Negative, Internal Positive, Stable Positive, Global Positive, Hopelessness, Hopefulness) as they obtain higher reliability and internal consistency (Peterson et al., 1982). Meta-analysis by Sweeney and colleagues (1986) consolidates this argument. Cronbach's alpha for the composite items in this study was high (0.70). Composite negative (CoNeg) and composite positive (CoPos) scores are obtained by summing up the individuals' scores on all three dimensions for the bad event and for the good event, respectively. CPCN (i.e., the full-scale score) is obtained by subtracting CoNeg from CoPos. CPCN, arguably, has the highest reliability and validity for predicting outcomes (Peterson, et. al, 1982). The scores can range from minus 18 to positive 18. For this study, the composite score will be utilized to classify those with a more negative style (internal, stable and global for bad events) and those with a more positive style (internal, stable and global for positive events). Those scoring on the negative end of the axis will be classified as having a more negative attribution style while those scoring on the positive end will be classified as having a more positive attribution style.

Sense of Coherence Scale - Orientation to Life Questionnaire (SoC) was developed by Antonovsky (1987). The 29-item questionnaire concerns three main components: 1) comprehensibility/ perceived understanding of your existence; 2) manageability/perceived ability to handle, control events; 3)

meaningfulness/perceived meaningfulness of life. A total of 11 of those items measure comprehensibility, 10 items measure manageability, and 8 items measure meaningfulness (e.g., "When you talk to people, do you have the feeling that they don't understand you?"; "In the past, when you had to do something which depended upon cooperation with others, did you have the feeling that it:"). The responses are measured on a semantic scale that ranges from 1 to 7 points. A high score in the meaningfulness subdomain reflects high perceived meaningfulness of life. The instrument also produces a total 'sense of coherence' score (ranging from 29 to 203 points) by summing up the total points from all three subscales (Eriksson & Mittelmark, 2016). Points between 160 and 190 on the total 'sense of coherence' represent a strong sense of coherence, whilst points under 70 are considered as low sense of coherence. Individuals with low sense of coherence are more likely to experience hardships and challenges (Antonovsky, 1987).

The tool has excellent internal consistency ranging from 0.70 to 0.95 across 32 countries (Eriksson & Lindström, 2005). Stability of the instrument ranges from 0.69 to 0.78 (1 year), 0.64 (3 years), 0.42 to 0.45 (4 years), 0.59 to 0.67 (5 years) to 0.54 (10 years), demonstrating decent stability over the years (Eriksson & Lindström, 2005). Cronbach's alpha for the tool in this study was very high - 0.83. The study did not follow strict protocol for the time completion of the questionnaire due to the fact that sense of coherence is a trait rather than state (Antonovsky, 1987) and participation in a competition is a situational factor that would not affect or amend individual's responses on the questionnaire. The instrument has been used in various experimental designs in the past, none of which follow a strict timing procedure (Walsh et al., 2014). However, the scale has been used predominantly in SES context (e.g., Gibson, 2003; Eklund et al., 2001; Nilsson et al., 2010), and to my knowledge, has not been utilised in competition studies. The *Sense of Coherence Scale* has also been utilised as a measure of protective/resilient factors contributing towards the formation of threat/challenge cognition (Antonovsky, 1979).

In order to address the last research question: 'Does T reactivity correlate with a reduced motivation to engage socially?', a *Motivational Questionnaire* was utilised following the form-filling competition. Participants were asked to complete the

questionnaire in which their motivation to re-engage with subsequent social activities (e.g., apply for a job, complete another financial form, seek help for funding matters, join a finance wellbeing course, in order to acquire knowledge of how to improve your financial situation, apply for mortgage/credit/loan) following victory/defeat at the contest was assessed (Appendix IV). Items in the motivational questionnaire were scored on a 6-Likert point scale, where 0='not at all' and 5='very much so'. Pilot study has also been conducted for the following questionnaire (n=5, low and high SES). The tool demonstrated a good overall internal consistency (alpha= .76) in this study.

3.6.3 Participants

The aim of the study was to recruit an equal number of individuals classified as long-term unemployed and from low SES, and as long-term employed and from high SES, in order to be able to examine potential endocrine dissimilarities in reactivity to a social defeat stimulus between the two populations. For these purposes, a stratified sampling strategy was applied. This allowed the researcher to divide the sample into two strata, low unemployed and high employed SES groups, by utilising poverty indicators such as SIMD, educational level, employment status, household income which allowed individuals to be classified in those two groups. The study did not meet the targeted population size (n = 188) due to low compliance with the study regulations across the low SES population and the implications of the Covid-19 pandemic (i.e., lockdown periods, social distancing rules, lab closures and the risks associated with collecting saliva).

Accessing low SES populations appeared more challenging than the high SES group. This stems from the fact that most of the high SES individuals were recruited on university campus allowing more flexibly and generally easier participation, whilst establishing links with low SES groups required the involvement of gatekeepers, recruitment off-campus, challenges around convenient venues and time for the participants. Albeit participants' travel expenses to the university were covered, the complexity of the research design and its time-consuming nature appeared to impact decisions about participation. Resultantly, thirty-one healthy males aged between 21 and 45 (M = 31.2, SEM = 1.3) were recruited using a stratified random sampling

approach from two discrete populations. Eleven participants were long-term unemployed: defined by the Organisation for Economic Co-operation and Development as a state of joblessness for 12 months or more. Based on several poverty indicators (SIMD, educational level, household income level, and marital status) they were classified as socio-economically disadvantaged. Twenty participants were employed (for at least 2 yrs.), with an income per annum of at least £30,000 (poverty indicators were also applied).

Within these stratified samples, unemployed participants were recruited from local community organisations and centres, third sector organisations, associations and programmes (i.e. Glasgow Life Clydebank Gateway programme network meetings/sessions; Violence Reduction Unit – Street and Arrow Project; North West Glasgow Recovery Communities workshops/meetings within recovery centres, locations - Whiteinch Recovery Café, Drumchapel Saint Mark's Church; Safe as Houses Project; thread on Trust Deed Scotland forum; job centres; job clubs, locations - Possilpoint Community Centre, Maryhill Housing Association, Maryhill Community Central Hall, Queens Cross Housing Association). Recruitment posters (Appendix V) were also placed in community centres. The interested candidates were able to contact the researcher via email. The researcher has also visited the centres on a regular basis in order to be able to meet and recruit volunteers. Employed males were recruited through social media platforms (e.g., Facebook, Twitter), via all-staff email in the departments of Social Work and Social Policy, Psychological Sciences and Health, and Pharmacy and Biomedical Sciences at the universities of Strathclyde and Glasgow. Snowball sampling within the university, companies and organisations situated near Glasgow City centre was also used as an additional recruitment approach where required. Where snowball sampling has been employed, participants were asked to send the recruitment advert to other prospective participants (via emails, social media platforms or physical copies of the recruitment poster) without disclosing any additional information about the study (apart from the one in the advert and the PIS). Future prospective participants were asked to follow the same recruitment procedure, without disclosing any additional information. The risk of disclosing information is conceivable even between participants who do not know each other. However, this is a

degree of risk that exists in research (May, 1993). Researcher, thus attempted to generally recruit prospective participants from different groups. Initial contact with potential volunteers was established by social media platforms, recruitment advert (Appendix VI).

As circulating T has been shown to fall with normal aging in males (Al-Dujaili & Sharp, 2012; Harman et al., 2001; Feldman et al., 2002), the sample was restricted to a cohort between the age of 20 and 45yrs. Although there is some suggestion the fall in T is not precipitous and can be mitigated by physical activity (D'Andrea et al., 2020), focusing on this age group reduces the potential for extreme inter-individual endocrine differences in not only basal levels but potentially reactivity. Because T and the carrier protein sex-hormone binding globulin (SHBG) are susceptible to modification by a variety of environmental and psychosocial factors it is necessary for endocrine research to have a number of exclusion criteria that may ordinarily seem overly limiting. Exclusion criteria were presence of obesity, acne, diabetes, hypertension, being on a strict diet or seriously restricting calorific intake, previous history of kidney or liver disease, having consumed illicit drugs during the previous month. For criteria that could not be visually inspected, volunteers were asked to verbally confirm that they do not obtain any of the aforementioned conditions in order to further proceed with their participation in the study.

Participants were not offered any straightforward financial inducement to participate. However, in order to elicit a status-facilitated endocrine response, the competition had to be engaging. This was accomplished by offering a financial incentive for 'winning'. Mazur and Booth (1998) point out, '…in the reciprocal model, as exemplified by the competition studies, T will not rise in response to a challenge when the outcome is a certainty or there is little by way of status or resources at stake' (p.388). This theme is echoed by Bateup et al. (2002) who note that the T-competition relationship is, 'highly contingent on perceptions that gain or loss is at stake' (p.183). Consequently, in order to amplify feelings of gain/loss a financial incentive of £25 cash was offered for participants in the winning condition. Participants in the losing category were informed they would receive nothing and this, in line with the

literature, was expected to trigger a fall in circulating T. Since the participants in the low SES group invariably had low financial status, the reward was deemed likely to be a significant inducement to engage seriously. Both high and low SES groups were equally rewarded once the study was completed, so as not to disadvantage any participants. Travel expenses (public transport) comprising travelling to the university on the day of experimental exposure, was also covered for the participants.

3.6.4 Procedure

Once individuals had emailed the researcher to indicate their interest in this research, they were sent details of the exclusion criteria and a plain language statement which described the study via email (Appendix VI). If criteria for inclusion in the study was met, the experimenter confirmed eligibility to participate. At this stage participants had the procedure explained verbally, had the opportunity to ask questions and were provided with detailed written instructions. Written informed consent was obtained (Appendix VII) and participants were handed the remaining materials (i.e., psychological and behavioural instruments; salivary collection tubes; chewing gum; instruction and information sheets (Appendix VIII and IX). Physical data were collected from the participants at a time and place convenient for them such as community centres, recovery cafes or the university campus. Once informed consent was obtained, the study proceeded as follows:

The experimental procedure comprised two major study phases (baseline and experimental) and was executed over a total of 4 days. The baseline phase of the study comprised the collection of 4 baseline salivary samples, time-matched to the samples collected at the experimental phase, which took place 48 hrs after the baseline. So, if the financial form - filling competition (experimental phase) took place at 4pm on a Wednesday, pre-experimental salivary samples were collected at 9am, 3pm, 3.30pm, 4pm on a Monday. This time-matching allows the experimenter to account for circadian dynamics and episodic fluctuation of salivary T which would otherwise render the salivary data un-interpretable (Al-Dujaili & Sharp, 2012).

In the second (experimental) phase of the study, participants were exposed to a form-filling competition task and were asked to collect 8 salivary samples, pre and post competition. Salivary samples were used for testosterone and cortisol hormone determination, thus allowing to address the first research question: 'Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?'. Utilising a meaningful baseline (48hrs prior to the experimental phase) allowed control over circadian activity and avoid potential misinterpretation of results (Salvador, 1987, as discussed in section 3.2.3), whilst the utilisation of a multiple time point sampling, 4 for baseline and 8 for the experimental procedure, compared to single - point day sampling appears more suitable approach when investigating chronobiological changes of endocrine levels in bio-behavioural research (please refer to sections 3.2.2 and 3.2.3).

During the baseline phase, individuals were also asked to complete two psychometric tools; the personality (NEO-FFI-3) and sense of coherence (Orientation to Life) questionnaires and provide demographic information. Participants were also asked to respond to a number of written questions about their daily activities during baseline saliva collection, enabling the researcher to monitor for confounding factors (Appendix X). The sense of coherence and personality measures were used to capture potential links to endocrine reactivity, whilst the demographics data was used to objectively classify individuals into two separate SES groups. The purpose of the sense of coherence scale was also as a tool measuring factors that might contribute towards the construction of threat/challenge cognitions.

On the day of the experimental competition, participants were asked to complete an affect questionnaire (PANAS-X - 30 mins prior to competition exposure). This allowing to evaluate individual's affect state prior to task engagement. Participants were unfamiliar with the competitive element of the experimental task up until 10min before the actual exposure, this serving the purpose to create an endocrine response in the volunteers. Knowledge of the true nature of the experimental task prior to the competition would have jeopardised any endocrine reactivity observations.

Threat/challenge cognitions and participants' level of engagement with the task were measured on a questionnaire immediately before the competition exposure. This addresses the second research question: 'Does cognitive appraisal of threat/challenge moderate endocrine reactivity?'.

Prior to engaging in the form-filling competition task, participants were given verbal instructions regarding their opponent (the confederate), the nature of the task, and the requirements to win the competition. The confederate was presented as a person of a similar status (e.g., unemployed for the unemployed group of participants) to the participants. Participants were instructed that in order to win the competition, they will have to complete the financial form as quickly and accurately as possible. For that reason, it has been clarified to participants that they could not win the competition solely due to swift completion of the tasks, and that they will be asked to spend extra time filling in empty or inaccurately completed fields. In order to further enhance commitment to succeed in the competition the cash reward (£25) was placed on the table during the task and the participants reminded that if they were unsuccessful, they would forfeit the reward.

An equal number of disadvantaged and affluent participants were allocated to one of two different competition conditions: loss or win. Block randomisation was used for the allocation to loss and win conditions. Outcome was manipulated via a male confederate who following researcher's instructions would either purposely win or lose the competition task. The confederate was instructed to either purposely win or lose the competition (through successful completion of the form-filling task) prior to the arrival of the participant. Only one participant, who was randomly assigned to lose, ended up winning the competition. To avoid compromising the random assignment of participants to winning and losing conditions, the participant was excluded from the sample size. To further enhance deception, the confederate was asked to bring supplementary financial paperwork (e.g., credit history, mortgage paperwork) that was present on the table next to him during the entire competition. The theoretical and methodological justification for the choice of a financial form-filling task is presented on page 148/149 (section 3.6.2).

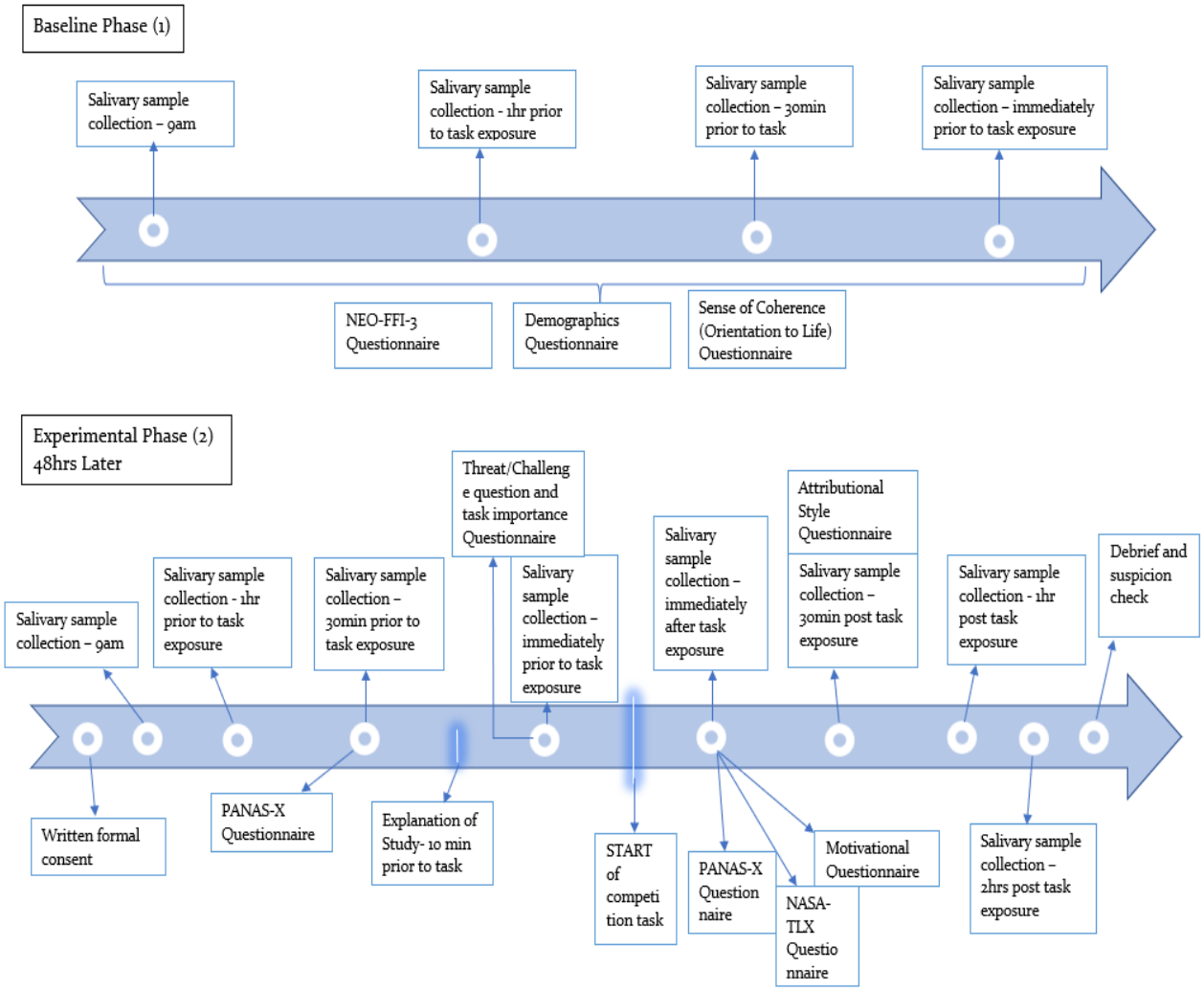
Mean competition length was 20.6mins (range: 10-38mins). Upon completion of the task (outcome was manipulated so that participants were either successful or unsuccessful) participants in the winning condition were handed the cash reward to increase perception of status and participants (in both conditions) began collecting post-task salivary samples; immediately post, 30mins, 1hr and 2hrs post task. Additionally, participants were asked to complete another four psychometric tools: the affect (PANAS-X) form that they have completed prior to the competition, workload (NASA-TLX) form, attributional style and motivational questionnaire. NASA-TLX, PANAS-X and attributional style questionnaires were used to measure previously suggested links between endocrine response to status competition and psychosocial variables (Steptoe & Marmot, 2002). The motivational questionnaire was used to measure post-competition motivation to engage with subsequent social tasks, thereby allowing to address the last research question: 'Does T reactivity correlate with a reduced motivation to engage socially?'.

Finally, participants were debriefed (Appendix XI), final informed consent obtained and those in the unsuccessful condition given the cash reward. In order to control for deception, participants were asked if they genuinely believed the experiment and whether they have won or lost the competition task; but this only took place after the debriefing had taken place. All respondents informed the researcher that they fully believed the legitimacy of the experiment (including the veracity of the competition outcome regardless of whether they have won or lost, and the confederate being a genuine opponent of the same status).

Figure 8 below illustrates the stages of the study procedure i.e., when and how salivary samples were collected, and the application of psychometric tools. A sample collection timeline is also provided in the PIS (Appendix XII).

Figure 8

Timeline of Study Procedure



3.6.5 Procedure for salivary samples collection

In order to establish a comprehensive baseline data for the hormones T and C, participants were asked to collect four time-matched salivary samples 48hrs prior to task exposure. This has been done in order to address previous methodological limitations in bio-behavioural research, namely the lack of meaningful baseline (Sharp, 2006). Failure to establish a meaningful baseline would have resulted in a lack of control over circadian activity and potential misinterpretation of results (Salvador, 1987).

When collecting salivary samples, participants were required to rinse their mouths thoroughly three times with water in order to minimise the risk of blood contamination in the saliva. The oral environment was allowed to normalise for two mins so that samples were not diluted. Participants then chewed on a quarter stick of sugar-free gum, discarding the first mouthful of saliva which contained cellular debris from the gum. They continued to chew on the gum and deposit saliva into a 10ml universal collection container up to the 5mL mark. Once sufficient saliva had been collected the cap was replaced tightly and samples refrigerated prior to return to the experimenter (which happened within 24-48hrs) and subsequently placed in a -80°C freezer, at the University of Strathclyde Institute of Pharmacy and Biomedical Sciences (SIPBS), until ready for assay.

It is important to note that although gum-chewing is no longer recommended for saliva collection (van Anders, 2010) due to its large effects on salivary testosterone, estradiol, and secretory immunoglobulin, the use of indirect assays during this study allowed the for assay to be clean and free from these contaminations. The gum thus did not interact with the assay as may happen with commercially prepared kits from Salimetrics using direct assays. Commercially available ELISA kits, employ the direct method, where salivary samples are not treated prior to assay. However, several limitations occur when utilising this approach (Sharp, 2006). Despite the benefits of the non-invasive approach, saliva as a substance contains various contaminants: i.e., bacteria, leukocytes, mucins, and extremely importantly for enzyme assays, endogenous enzymes. All these agents can interfere with ELISA assays, and salivary samples are particularly sensitive to interfering factors (e.g., pH imbalance) which produces results that are unpredictable - either too high or low (alkaline samples, for instance, tend to generate low results). Indeed, Uribe-Alvarez and colleagues (2021) report that low saliva pH can yield false positive results and comprise assay performance, more generally. Sharp (2006) further cites Aldercreutz who, referring to a range of studies, noted '...testosterone assays do not work well in non-extracted plasma' (1990, p.387). Finally, Jones and colleagues (2004) argue that '...some samples from female subjects give falsely high results when measured with direct immunoassay' (p.51). To alleviate some of these fundamental limitations, the method

utilized throughout the study is the indirect method, where salivary samples were treated with an extraction step that removes interfering agents.

3.6.6 Hormone determination

Salivary testosterone and cortisol were estimated by highly sensitive and specific ELISAs by modification of previously tested and published methods (Al-Dujaili et al. 2011; Al-Dujaili et al. 2012; Welling et al., 2007).

Initially, all samples were placed in a -80°C freezer. When required for assay the frozen salivary samples were removed from the freezer and allowed to thaw at fridge temperature for 24hrs. Once thawed they were centrifuged at 3000rpm for 10 mins in order to break down muco-polysaccharides (Sharp, 2006). Samples were then aliquoted into a series of smaller Eppendorf tubes (1mL each), and either re-frozen or transported in Medical cool carriers from SIPBS, Strathclyde to Queen's Medical Research Institute laboratories (QMRI), Edinburgh, where hormonal determination was performed.

Testosterone Assay:

A 96-well ELISA plate was coated overnight at 4 °C with Donkey Anti Rabbit Serum IgG prepared in house from the Scottish Antibody Production Unit, UK in sodium bicarbonate coating buffer (pH 9.6)). Plates were washed twice with wash buffer and dried before blocking with 0.5% BSA in PBS, pH 7.4 buffer for 1hr at room temperature. After 2 washes, standard, QC's and samples (50mL) were added in duplicate to wells with 50mL of Testo-HRP (ASTRA Biotech) and 50mL of Testo-Ab (R3S07-259, Meridian Life Science) (both HRP and Ab diluted in assay buffer). Plates were incubated for 2hrs at 28°C with shaking. Plates were washed 4 times before the addition of 120mL of TMB for 15mins in the dark with shaking. The reaction was stopped with 80mL of 1N Sulphuric Acid. The plate was then read at 450nm for analysis using SoftMax Pro (Version 7.1, Molecular Devices) ELISA reader. ***Cross reactivity:*** 100% Testosterone and 13.8% DHT. All other steroids tested gave <0.3%

cross reactivity. ***% CV;*** Intra-Assay %CV = 8.4% and Inter-Assay %CV = 16.4%. ***Sensitivity:*** Limit of detection = 10 pg/mL and Limit of quantification = 28 pg/mL.

Cortisol Assay:

A 96-well ELISA plate was coated overnight at 4 °C with Goat Anti Mouse IgG; Arbor Assays in sodium bicarbonate coating buffer (pH 9.6)). Plates were washed twice with wash buffer (300mL) and dried before blocking with 0.5% BSA in PBS, pH 7.4 buffer for 1hr at room temperature. After 2 washes, standard, QC's and samples (20mL) were added in duplicate to wells with 80mL of Cortisol-HRP (ASTRA Biotech) and 50mL of Cortisol-Ab (ASTRA Biotech) (both HRP and Ab diluted in assay buffer = 0.1% BSA PBS). Plates were incubated for 2hrs at 28°C with shaking. Plates were washed 4 times before the addition of 120mL of TMB for 15mins in the dark with shaking. The reaction was stopped with 80mL of 1N Sulphuric Acid. The plate was then read and analysed as for testosterone ELISA above. ***Cross reactivity:*** 299.1% Cortisol, 4.7% Corticosterone, 3.6% 11-Deoxycortisol, 1.7% Cortisone. All other steroids tested gave <1% cross reactivity. ***% CV:*** Intra-Assay %CV = 3.6%and Inter-Assay %CV = 10.5%. ***Sensitivity:*** Limit of detection = 0.05ng/mL and Limit of quantification = 0.2ng/mL.

To view the full protocol on assay determination, please refer to Appendix XIII.

3.6.7 Hormonal data analysis

In order to reduce inter-individual variability, raw endocrine data were normalised by dividing a participant's recorded scores by their mean daily levels (performed separately for both T and C) and thus anchoring all values to an absolute value, i.e., a maxima of 1 - an approach adopted from previous endocrine studies (see; Bateup et al., 2002; Sharp, 2006; and Mazur et al., 1997).

The inter-individual variation has the additional effect of masking individual patterns of reactivity and reduces the likelihood of finding significant differences in group mean data. In order to circumvent this difficulty, the magnitude of any changes in levels of T and C in relation to competition phase are illustrated with percentage

change from the mean (Elias, 1981). This was facilitated by determining the mean of each individual's levels across all time points and calculating the percentage change for each data point from that mean figure.

3.6.8 Statistical analyses

The main strategy for analysis of the endocrine data was repeated measures and mixed two-way measures ANOVAs. Utilising ANOVAs allowed the investigation of chronobiological changes of T and C, where the within-subject factor was time and between-subject factor (SES). Findings from these analyses assisted to address RQ1 and H1 by drawing endocrine comparisons between the two SES groups. Mixed two-way and repeated two-way measures ANOVAs have been performed since this is the most common method used to test for interactions.

Psychological data analysis was also performed in relation to SES, in order to draw potential differences between the affluent and disadvantaged groups, thus further allowing to evaluate the most significant differences and use them to build models for the analysis of potential relationships between endocrine reactivity and the psychosocial factors (i.e., sense of coherence, sense of control, personality and trait affect). Findings from the analyses addressed RQ2 and H2. One of the psychosocial variables, sense of coherence, was also analysed in relation to another cognitive factor, threat/challenge perceptions, in order to address the question whether SoC scores contribute towards the formation of threat/challenge appraisals, as suggested by Antonovsky (1987) and Denton and colleagues (2004). This has been achieved via an additional MANOVA. For this purpose, parametric tests were performed: one-way MANOVAs, separate one-way ANOVAs (in cases of multicollinearity). For data that did not follow a normal distribution or did not meet the requirements for these parametric tests, non-parametric equivalents were used: Mann-U-Whitney and Multiple Kruskal-Wallis. These analyses have been carried out to ensure SES groups differ in psychosocial factors in the predicted way (e.g., differences in personality traits, sense of coherence and attributional style). Moreover, unpacking potential differences in psychosocial factors between SES groups assists in shedding some light on the complex relationship between experiences of SED, resilient/protective

psychosocial factors and physiological responses. For this reason, the analyses serve as a building block to addressing RQ2 and H2.

In seeking to provide evidence of the links between psychosocial factors and physiological responses proposed by Steptoe & Marmot (2002), the study conducts a series of analysis (i.e., Pearson's r Correlations for normally distributed and ratio data, Spearman's Rank Correlations for ordinal data, and multiple linear regressions) where relationships between sense of coherence, sense of control, personality, trait affect and endocrine response are explored. In doing so, the study directly addresses RQ2 and H2.

The research proceeds with analyses of individuals' subjective experiences of an objective outcome, namely the importance of cognitive factors (threat/challenge) for endocrine reactivity to status encounter. Hence, the study endeavours to address the importance of individual beliefs and subjectivism in objective parameters – competition outcome. Justification for this approach has been drawn on Marmot's (2004) argument that not all individuals experiencing socioeconomic disadvantage would necessarily experience negative health outcomes. Furthermore, subjective perceptions of SES appear to be better predictors of health compared to objective SES (Adler et al., 2000). Thereby, by evaluating the extent to which subjective parameters play a role in physiological responses to objective outcomes/circumstances, this research offers an arena for more nuanced debate around the question: 'Why do not all individuals experiencing socioeconomic disadvantage get ill?'. Cognitive moderators of endocrine reactivity to status competition were captured via custom model ANCOVAs. Moderation rather than mediation analysis was performed as the current research is interested in the interactions between cognitive factors (threat/challenge appraisals), endocrine reactivity and competition outcome. In other words, the study seeks to explore whether the moderator cognitive appraisals affect the strength and direction of the relationship between endocrine response and competition outcome.

To test whether a decrease in T reactivity positively correlates with a reduced motivation to engage socially, the study performed a linear regression model analysis. The performed analysis allowed to address RQ4 and H4.

Finally, power analysis for endocrine response to competition outcome was also performed (Appendix XIV). Sample size was calculated based on the effect size found in previous work on the relationship between competition outcome and testosterone (in studies conducted outside laboratory) which was 0.19 (Geniole et al., 2017). Considering an alpha= 0.05 and a power to detect the effect of 0.80, sample size was 188. Considering the small sample size of 31 participants, the current findings should be explored with caution, whilst the study regarded as an explanatory/feasibility, aiming to underpin larger scale research in the future (Button et al., 2013). Additionally, future research should do a pre-planned power simulation according to the expected effect size and, if possible, report design and analyses alongside estimated test power.

All analyses were performed using International Business Machines Statistical package for social sciences (IBM SPSS), software version 25.

3.6.9 Ethical Approval

Ethical issues were respected in accordance with Strathclyde University Ethics Committee (UEC) and the Code of Practice on Investigations Involving Human Beings (RKES). The study was approved by the Strathclyde University Ethics Committee (Appendix XV).

Chapter 4

Salivary testosterone and cortisol reactivity to threat/challenge cognitions in socioeconomically disadvantaged and affluent males

- The analyses reported in this chapter address RQ1 and H1:

"Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?"

"Socio-economically disadvantaged population will demonstrate a dissimilar pattern of endocrine response to a social defeat stimulus compared to a higher SES population."

- In this and the following two results chapters (i.e., Chapter 5 and 6), p-value of .05 will be used as a threshold of significance. Therefore, results above this threshold will be discussed as non-significant, whilst the one under .05 considered significant.

4 Introduction to Endocrine Results

To address the first research question and hypothesis, in this section endocrine data are analysed in relation to socioeconomic status and competition outcome. Firstly, normalcy checks, skewness, kurtosis and sphericity are performed for all endocrine data. If skewed or not normally distributed, all hormonal data are log-transformed and anchored to a maxima of 1 in order to reduce inter-individual variability and address issues of normality. The chapter proceeds with data analysis of the baseline and pre-competition hormonal states in order to be able to, firstly, validate the experiment, and secondly to be able to identify whether anticipatory hormonal response occurs regardless of SES, as suggested by the challenge hypothesis/Mazur's biosocial model of status (Archer, 2006; Mazur, 1985; Wingfield et al., 1990). Subsequently, analysis of the post-competition endocrine data is performed. The data is analysed in relation to the objective competition outcome (win/loss) and SES. This allows to test the challenge hypothesis' credibility in its second phase (post hormonal competition exposure) and

sheds some light on the findings of the previous hormonal competition studies. Endocrine reactivity to hormone status competitions though is complex due to the multiple contextual, cognitive and psychosocial factors involved, and shifting patterns of hormonal response (Sharp, 2006). In addition, Scheepers and Ellemers (2005) also report that low and high-status groups exhibit different and complex physiological responses to status encounters. Therefore, in order to tease out some of the complexity of endocrine pattering and to address the first research question: 'Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?', endocrine data analysis also considers social groupings (high/low SES). The chapter starts with analysis of testosterone and proceeds with cortisol data.

4.1 Testosterone Data Characteristics

Skewness and kurtosis tests were performed in order to ascertain if T data met requirements for normalcy. The DV was levels of circulating T. The IV was task phase (baseline and competition). Table 2 Illustrates z scores for both skewness and kurtosis. Time-points for raw T data were not normally distributed. Additionally, a Shapiro-Wilk test was performed and was significant for most baseline time points and some experimental (p <.05), indicating data were not normally distributed. Finally, Q-Q plots and stem-leaf plots were examined visually. These showed the same pattern revealed by the Shapiro-Wilk test. The results from these tests and visual examinations indicate that the data was not normally distributed, and outliers were present in the dataset due to high inter-individual variability in hormone levels. Inter-individual variability and skewness were reduced by data log-transformation and anchoring to a maxima of 1. This is a standard procedure in hormone studies for addressing the data issue.

Table 2

Z Scores for Skewness and Kurtosis of Testosterone Data by Phase

Time	Baseline	Day of Task

	Skewness	Kurtosis	Skewness	Kurtosis
9am	3.02*	1.35	1.5	-0.4
1hr	1.38	-0.23	-0.3	-0.1
30 mins	0.37	-0.64	-0.4	0.6
Immediately Prior	1.13	-0.82	3.53*	5.81*

Note. N=31

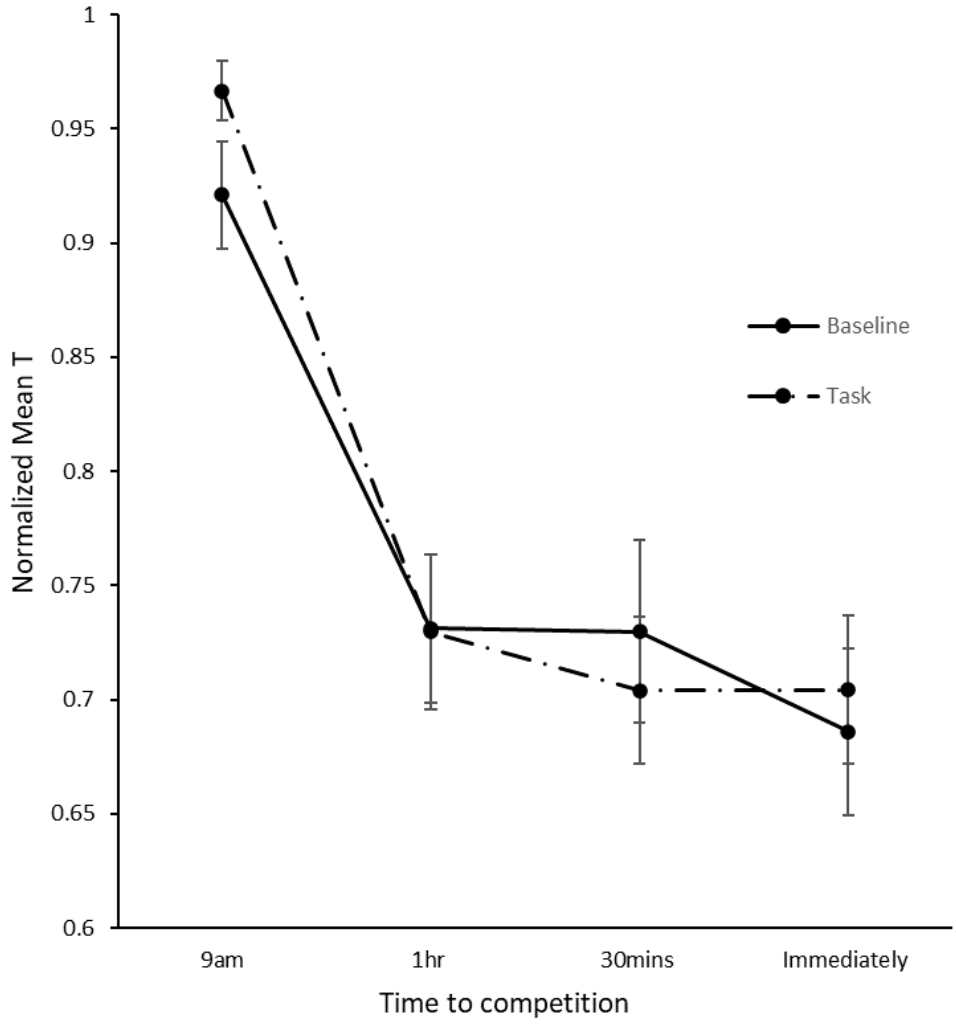
**Z-scores for Skewness and Kurtosis significant at (> 1.96)*

4.1.1 Baseline and Task Phase Testosterone Data for all Participants (Mean ± SEM)

Figure 9 illustrates that between 9am and 1hr prior to contest (time matched) baseline and pre-competition mean T levels fell perceptibly, following clear circadian activity, where T levels are higher in the morning and gradually declining towards the evening. The observed hormonal patterns replicate previous research findings (Sharp, 2006).

Figure 9

Normalized Mean Testosterone Under Baseline and Task Phases (n = 28) (Mean and ± SEM)



In order to test the relationship for time under the baseline and pre-competition conditions, a two-way repeated measures 2 (day) X 4 (time of day) ANOVA was performed on normalised mean data. Three participants were excluded from the analysis as outliers. Employing the Greenhouse-Geisser correction procedure (adjusting for lack of sphericity) revealed the main effect for time reached significance F (2.22, 60) = 19.99, $p < .001$, $\eta^2 = .43$. Pairwise comparison using Sidak revealed that testosterone concentrations were significantly higher at 9am compared to 1hr, 30min and immediately before the competition. The main effect for day was non-significant,

F (1, 27) = 0.03, p = .86, η^2= .001. The interaction between day and time (sphericity assumed) was also non-significant, F (3, 81) = 1.74, p = .16, η^2 = .06. These results indicate that the change of T levels over time was not significant between days (baseline and competition). Meaning that there is a lack of anticipatory rise in T in response to a non-physical competition task. The importance of these results will be further discussed in Chapter 7, section 7.1.1. SPSS output for this analysis provided in Appendix XVI.

4.1.2 Post-Competition Testosterone Data by Outcome and SES

In the light of previous evidence suggesting that low and high-status groups exhibit different and complex physiological responses to status encounters (Scheepers & Ellemers, 2005) whilst victory/defeat impact upon endocrine reactivity (Mazur, 1985), results in the present study were analysed through the prism of social groupings (high/low SES) and competition outcome (win/loss). Table 3 and 4 Illustrate z scores for both skewness and kurtosis. Immediately After and 30 mins post competition raw T levels were significantly skewed whilst 1hr and 2hrs after levels appear normally distributed. This means that a higher inter-individual variability in hormone levels is observed in the collection points immediately after and 30min post competition compared to 1hr and 2hrs post. Higher inter-individual variability at these points does not come as a surprise considering the argument that T levels fluctuations appear shortly after the competition (Vermeer et al., 2016). Additionally, a Shapiro-Wilk test was performed and was significant for immediately after and 30 min post time points (p<.05), indicating data were not normally distributed. Q-Q plots and stem-leaf plots were also examined visually. In order to minimise inter-individual variability and reduce skewness, all data were log-transformed and normalised to a maxima of 1.

Table 3

Z Scores for Skewness and Kurtosis of Testosterone Data by Post-Competition Outcome

Time	**Win**		**Loss**	
	Skewness	Kurtosis	Skewness	Kurtosis
Immediately After	2.61*	3.47*	1.94	3.00*

30min Post	0.7	-0.8	5.12*	9.2*
1hr Post	-0.6	-0.4	0.3	0.4
2hrs Post	-0.04	-0.52	0.1	-1.2

Note. n=31

**Z-scores for Skewness and Kurtosis significant at > 1.96*

Table 4

Z Scores for Skewness and Kurtosis of Post-Competition Testosterone Data by SES (Low/High)

Time	**High SES**		**Low SES**	
	Skewness	Kurtosis	Skewness	Kurtosis
Immediately After	1.19	2.1*	2.30*	1.91
30min Post	0.85	0.13	3.61*	5.28*
1hr Post	-0.12	0.14	0.78	0.15
2hrs Post	-0.34	-0.06	0.06	-1.44

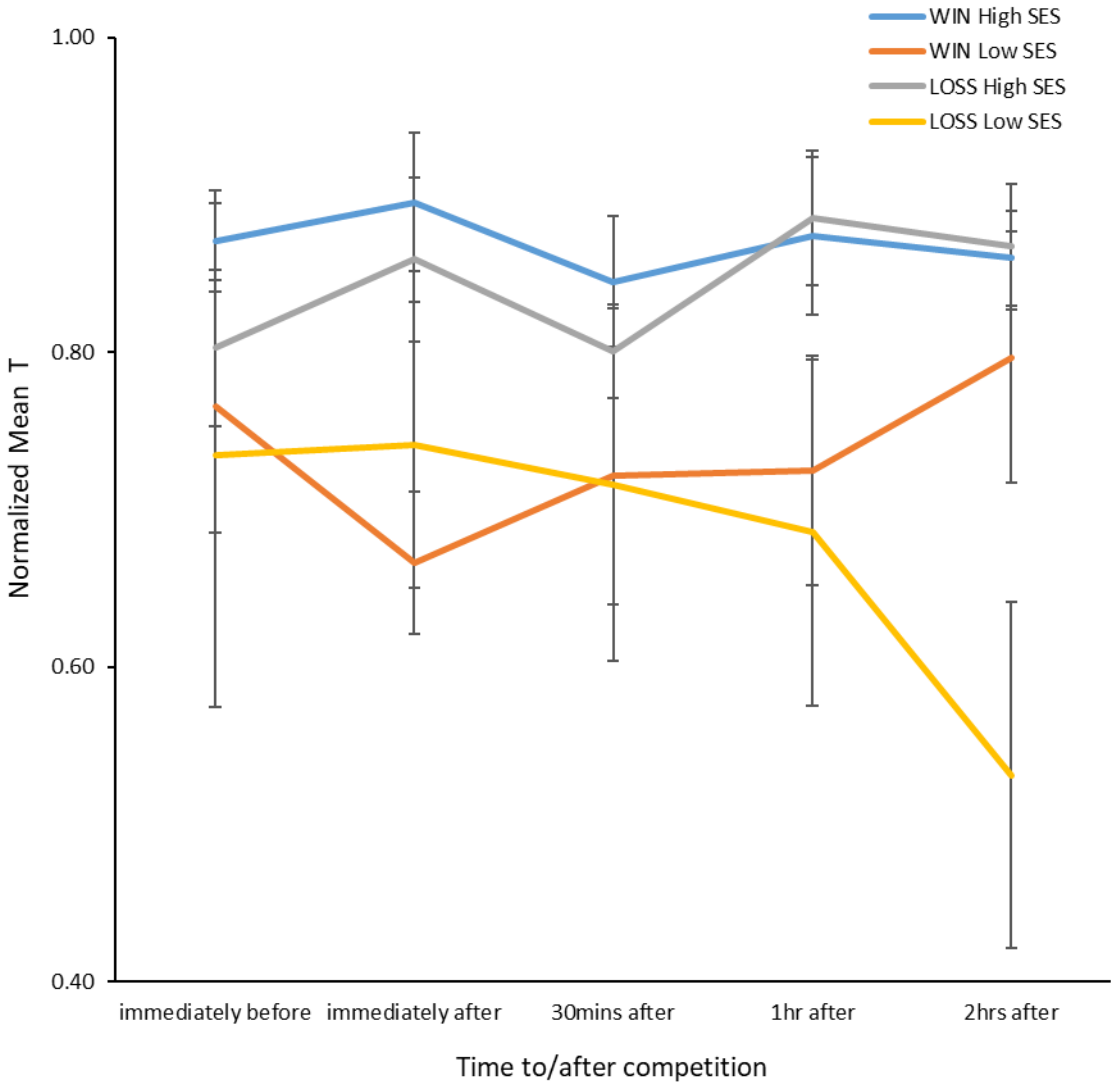
Note. n=31

**Z-scores for Skewness and Kurtosis significant at > 1.96*

Figure 10 illustrates the difference between T levels in the winning and losing conditions for both socioeconomic status groups.

Figure 10

Normalized Mean Testosterone By Competition Outcome and SES (Mean and ± SEM) (n = 31)



To test the relationship between time, competition outcome and socioeconomic status, a 2 (outcome) x 2 (SES) x 5 (time) ANOVA was performed on normalised T data. The within-subject factor was time whilst the between-subject factors were outcome (win/loss) and SES (low/high). The DV was circulating T levels. Main effects and all possible two-way and three-way interactions were reported. Results revealed a non-significant main effect (sphericity assumed) of the within-subject factor time on mean T levels F (4, 108) = .22, p = .90, η^2 = .01. Moreover, there was no significant main effect of the between subject variable competition outcome F (1, 27) = 1.51, p = .23, η^2 =

.05 on mean T levels. The main effect of SES on mean T levels, however, was significant F (1, 27) = 21.03, $p < 0.001$, $\eta^2 = .44$. Pairwise comparison using Sidak revealed that circulating T levels were significantly higher in the high SES group compared to the low SES population. The two-way interactions between time and outcome F (4, 108) = .87, $p = .48$, $\eta^2 = .03$, time and status F (4, 108) = .68, $p = .61$, $\eta^2 = .03$, and status and outcome F (1, 27) = .18, $p = .70$, $\eta^2 = .01$, were not significant. The three-way interaction between time, outcome and status, F (4, 108) = 1.6, $p = .20$, $\eta^2 = .05$ was not significant. These results indicate that in this analysis we did not detect a statistically significant effect of competition outcome on overall T levels. Moreover, the analysis revealed a non-significant association between T levels and time. However, circulating T levels were higher in the high SES compared to the low SES group. Lastly, the change of T levels over time was not significantly different between competition outcomes (win/loss) or status groups (low/high), whilst the relationship between circulating T levels across SES groups and competition outcome was not statistically significant. The relevance of these findings to the core RQ1 and H1 will be discussed in section 7.1.2 of Chapter 7. SPSS output provided in Appendix XVII.

4.2 Cortisol Data Characteristics

Table 5 Illustrates z scores for both skewness and kurtosis. The table demonstrates that the raw cortisol data is not normally distributed. Additionally, a Shapiro-Wilk test was performed and was significant for all time points ($p < 0.05$), indicating data were not normally distributed. Finally, Q-Q plots and stem-leaf plots were examined visually, revealing the same results as from the Shapiro- Wilk test. These results suggest the presence of outliers and lack of normality due to high inter-individual variability in hormone levels. In order to address this issue, all data were log-transformed and anchored to a maxima of 1.

Table 5

Z Scores for Skewness and Kurtosis of Cortisol Data by Phase (n=15)

Time	**Baseline**		**Day of Task**	
	Skewness	Kurtosis	Skewness	Kurtosis
9am	3.64*	4.79*	10.85*	27.93*
1hr	5.80*	8.09*	8.3*	21.98*
30 mins	4.9*	5.7*	7.73*	12.81*
Immediately Prior	5.1*	8.2*	8.24*	16.7*

Note. n=15

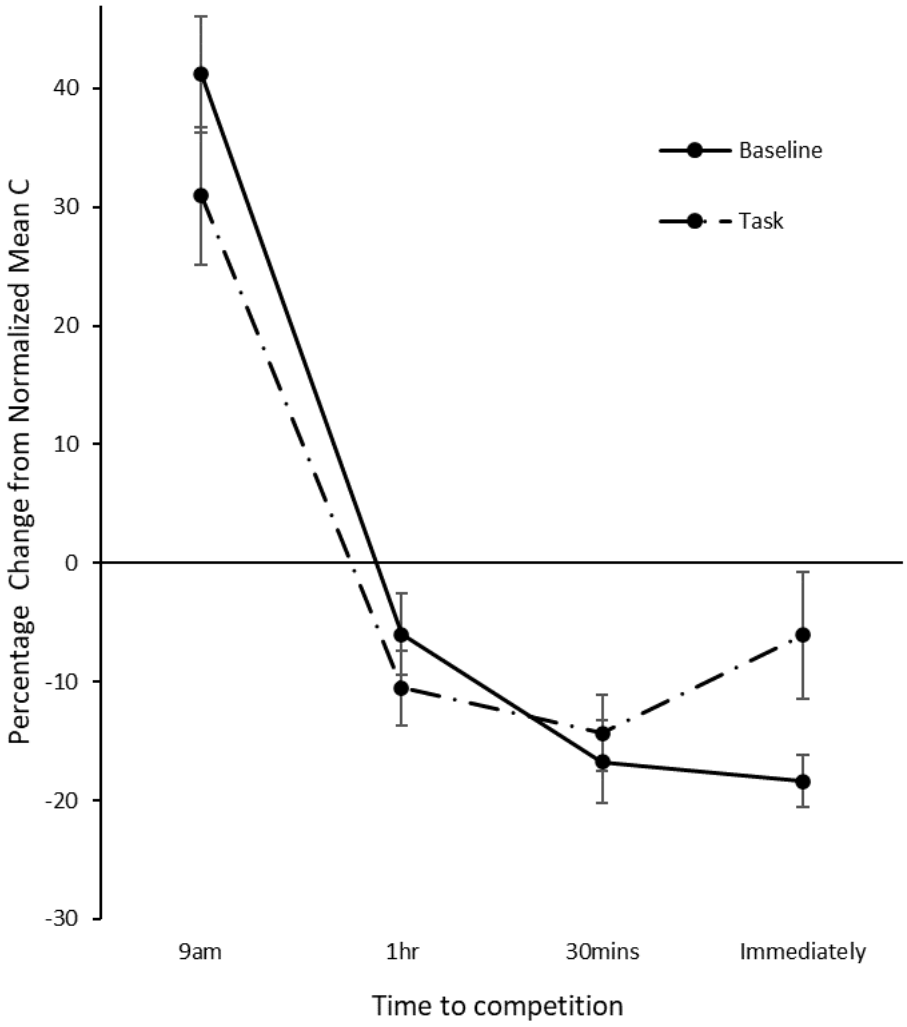
**Z-scores for Skewness and Kurtosis significant at (> 1.96)*

4.2.1 Baseline and Task Phase Cortisol Data for All Participants (Mean and ±SEM)

Figure 11 illustrates that between 9am and 1hr prior to competition (time matched) baseline and pre-competition percentage change C from the mean. As demonstrated on the graph, C levels drop significantly following a typical daily circadian rhythm.

Figure 11

Percentage Change in Normalized Cortisol from Mean Under Baseline and Task Phases (n=28) (Mean and ± SEM)



A two-way repeated measures 2 (day) X 4 (time of day) ANOVA on the percentage change from the mean cortisol (n = 28, due to missing data) was performed. The dependent variable was circulating C levels, whilst the independent variable was task phase (i.e., baseline and day of competition). The ANOVA demonstrated significant main effect of day on C levels (sphericity assumed), F (1, 27) = 6.5, p = .02. With individuals exhibiting higher levels of cortisol on the day of the competition exposure compared to baseline. The main effect for time was also significant (Greeenhouse-Geisser correction procedure applied to adjust for lack of sphericity), F (1.93, 52.1) =

43.1, $p < 0.001$. Pairwise comparison using Sidak calculation revealed that the difference between time lies between the 9am and 1hr, 9am and 30min, and finally 9am and immediately prior. The interaction between day and time (sphericity assumed) was non-significant, F (3, 81) = 2.6, p = .059. These results reveal that not only C levels differed between days, with participants demonstrating higher levels on the competition day, but also that C levels changed across the different collection points. For a discussion of these findings please refer to section 7.1.3 of Chapter 7. SPSS output provided in Appendix XVIII.

4.2.2 Post-Competition Cortisol Data Comparison by Outcome and SES

As with T, post-competition cortisol data was analysed by competition outcome and SES. Tables 6 and 7 illustrate z scores for both skewness and kurtosis. The tables demonstrate the raw cortisol data were notably skewed and lacks normality. Again, a Shapiro-Wilk test was performed and results were found to be significant for all time points (p <.05), indicating data were not normally distributed. Q-Q plots and stem-leaf plots were also examined visually. Following these result outcomes, all data were log-transformed and normalised to a maxima of 1.

Table 6

Z Scores for Skewness and Kurtosis of Cortisol Data by Post-Competition by Outcome

Time	**Win**		**Loss**	
	Skewness	Kurtosis	Skewness	Kurtosis
Immediately After	4.32*	6.25*	3.64*	4.6*
30min Post	5.79*	10.68*	6.4*	12.5*
1hr Post	6.47*	12.8*	6.45*	12.72*
2hrs Post	5.92*	11.14*	6.25*	12.11*

Note. n=30

**Z-scores for Skewness and Kurtosis significant at (> 1.96)*

Table 7

Z Scores for Skewness and Kurtosis of Post-Competition Cortisol Data by SES (Low/High)

Time	High SES		Low SES	
	Skewness	Kurtosis	Skewness	Kurtosis
Immediately After	4.01*	3.91*	2.84*	3.04*
30min Post	7.1*	13.88*	1.06	-0.95
1hr Post	6.52*	11.34*	2.86*	3.09*
2hrs Post	5.61*	7.71*	1.56	0.22

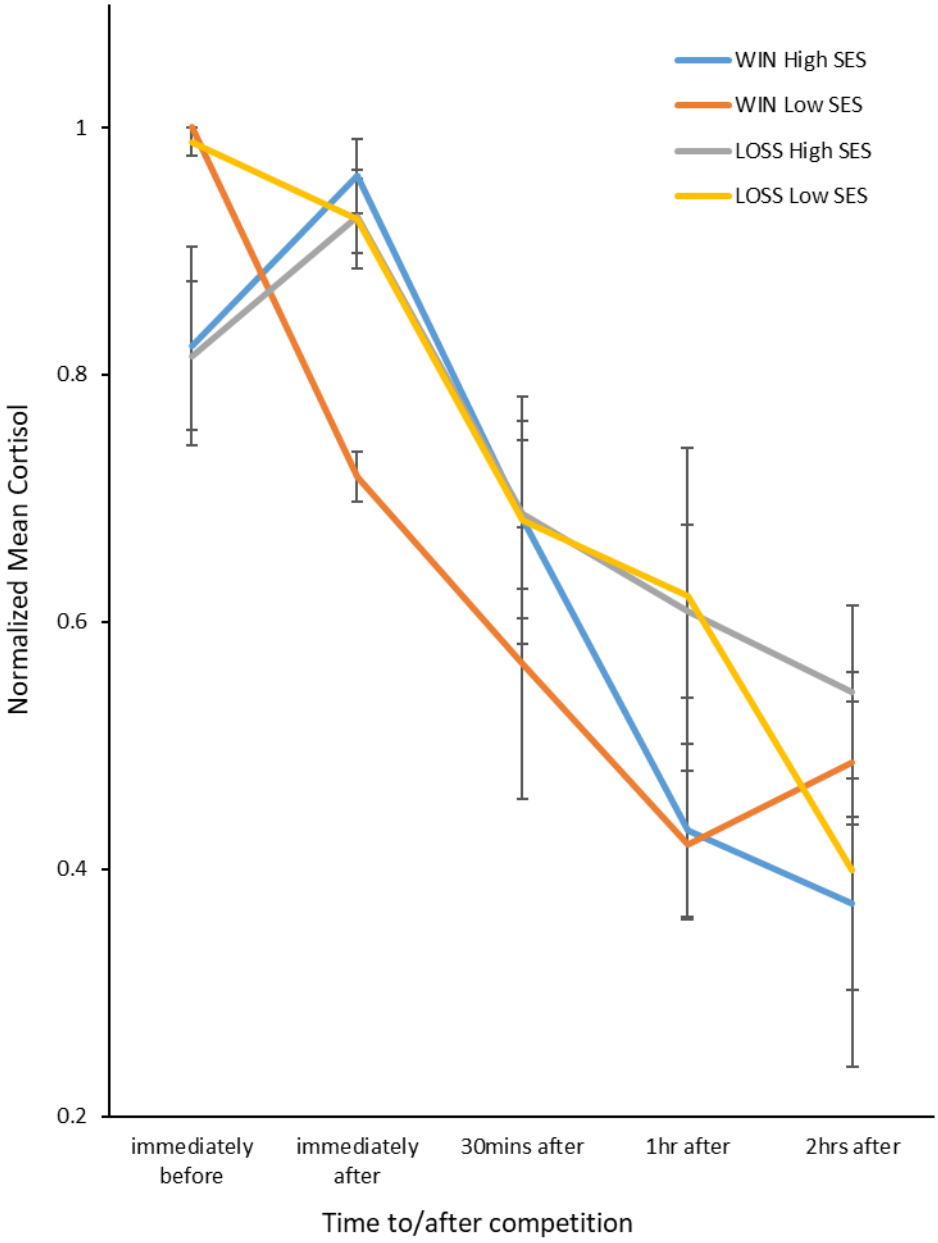
Note. n=30

**Z-scores for Skewness and Kurtosis significant at (> 1.96)*

Figure 12 illustrates the difference between C levels in winning and losing condition for both socioeconomic status groups.

Figure 12

Normalized Mean Cortisol By Competition Outcome and SES (Mean and ± SEM) (n=26)



To test the relationship between time, competition outcome and socioeconomic status, a 2 (outcome) x 2 (SES) x 5 (time) ANOVA was performed on normalised C data. Prior to inferential statistics the dataset was examined and four participants were excluded as outliers, whilst one participant's data was incomplete, resulting in n = 26. The within-subject factor was time whilst the between-subject factors were outcome (win/loss) and SES (low/high). The DV was circulating C levels. Main effects and all possible two-way and three-way interactions were reported. Results revealed a significant main effect (Greenhouse-Geisser correction procedure adapted) of the

within-subject factor time on mean C levels F (3.1, 67.9) = 30.6, $p < 0.001$, η^2 = .60. Pairwise comparison using Sidak revealed that cortisol concentrations were significantly higher at time 1 (immediately before task exposure) and 2 (immediately after task exposure) when compared to times 3 (30min after task exposure), 4 (1hr post task exposure) and 5 (2hrs post task exposure) (please refer to Appendix XIX for pairwise comparison table). The main effects of the between subject variables competition outcome F (1, 22) = 2.7, p = .11, η^2 = .11 and SES F (1, 22) = .01, p = .92, η^2 = .00, on mean C levels, were not significant. The two-way interactions between time and outcome F (3.1, 67.9) = .97, p = .41, η^2 = .04, time and status F (3.1, 67.9) = 2.2, p = .09, η^2 = .09, and status and outcome F (1, 22) = .07, p = .80, η^2 = .003, were not significant. The three-way interaction between time, outcome and status, F (3.1, 67.9) = 1.5, p = .23, η^2= .06 was also not significant. These results indicate that we did not detect a statistically significant effect of competition outcome on overall C levels, in this analysis. Moreover, circulating C levels did not significantly differ between the two SES groups (low and high). The analysis, however, revealed a significant association between cortisol levels and time. The change of C levels over time was not significantly different between competition outcomes (win/loss) or status groups (low/high). Furthermore, the relationship between circulating C levels across SES groups and competition outcomes was not statistically significant. The relevance of these findings to the core RQ1 and H1 will be discussed in section 7.1.4 of Chapter 7.

Chapter 5

Reactivity of salivary testosterone and cortisol to threat/challenge cognitions in socioeconomically disadvantaged and affluent males

- The analyses reported in this chapter contribute towards the analyses performed in Chapter 6, allowing to address RQ2, H2, RQ3, H3 and RQ4 and H4.

5 Introduction to Psychosocial Data

This chapter engages with the analysis of psychosocial data, by drawing comparisons between the two SES groups. In doing so, it aims to explore on which psychosocial measures SES groups differ most. This would subsequently feed into the analyses of the relationships of psychosocial variables to endocrine data and the cognitive moderators (threat/challenge) of endocrine reactivity to hormone status competition (explored in chapter 6).

Thus, the chapter, starts with analysis of the sense of coherence scale (SoC). Comparisons between low and high SES groups on the four-dimensional scale are drawn. The data are also analysed by grouping the individuals into threat and challenge conditions, in order to identify whether SoC scores contribute towards the formation of threat/challenge appraisals, as suggested by Antonovsky (1987) and Denton and colleagues (2004). The chapter then proceeds with the analysis of the psychological variables – attributional style and personality - by SES groups. Allowing exploration of whether SES groups differ significantly on the attributional and personality style measures.

Moving towards the thesis central concerns, threat and challenge appraisals are analysed by SES. The section thus aims to identify whether there are any statistically significant differences in the appraisals of the hormonal status competition between SES groups. More specifically, the section is concerned with whether low SES groups perceive events as more threatening compared to high SES groups. As low SES

populations are more frequently subjected to predominantly stressful, uncontrollable and unavoidable negative life events (Kim et al., 2018; Maier & Seligman, 1967), this thereby proposed to generally affect their appraisals of events.

Finally, the chapter engages with analysis of the motivational questionnaire. Differences in this analysis are drawn on the basis of the objective competition outcome (win/loss) rather than SES. This is done in order to evaluate whether based on competition outcome, groups will differ in their motivation to engage with subsequent relevant activities. In Chapter 6 (section 6.2), this analysis is explored in relation to post status competition T reactivity, in order to fully address the last research question, hypothesis, but also to generally test the credibility of the argument that "status-induced fluctuations in T influence future status-seeking behaviours" (Knight & Mehta, 2014, p.5).

> RQ4: "Does T reactivity correlate with a reduced motivation to engage socially?"
>
> H4: "Reduced motivation to engage with subsequent social tasks will positively correlate with a decrease of T reactivity."

5.1 Sense of Coherence

SES group differences in the sub-domains of sense of coherence were tested using a one-way MANOVA. Assumptions for running the MANOVA were met. The Box's M value of 16.2 was associated with a *p* value of .21 (equal covariance matrices between the groups assumed), interpreted as non-significant based on guidelines by Huberty & Petoskey (2000). However, with values being close to 0.9 (see Table 8), it could be argued that multicollinearity between the subdomains exists. Consequently, separate one-way ANOVAs were performed to examine any significant difference between the two status groups on each sense of coherence sub-domain (Meyer et al., 2006). The DVs are subdomains of the sense of coherence scale, whilst the IV is SES.

Table 8

Correlations between sense of coherence sub-domains

		1.	2	3.	4.
1.	Meaningful	-	.257	.628**	.776**
2.	Perceived Understanding	.257	-	.514**	.733**
3.	Perceived Ability to Control Events	.628**	.514**	-	.895**
4.	Global	.776**	.733**	.895**	-

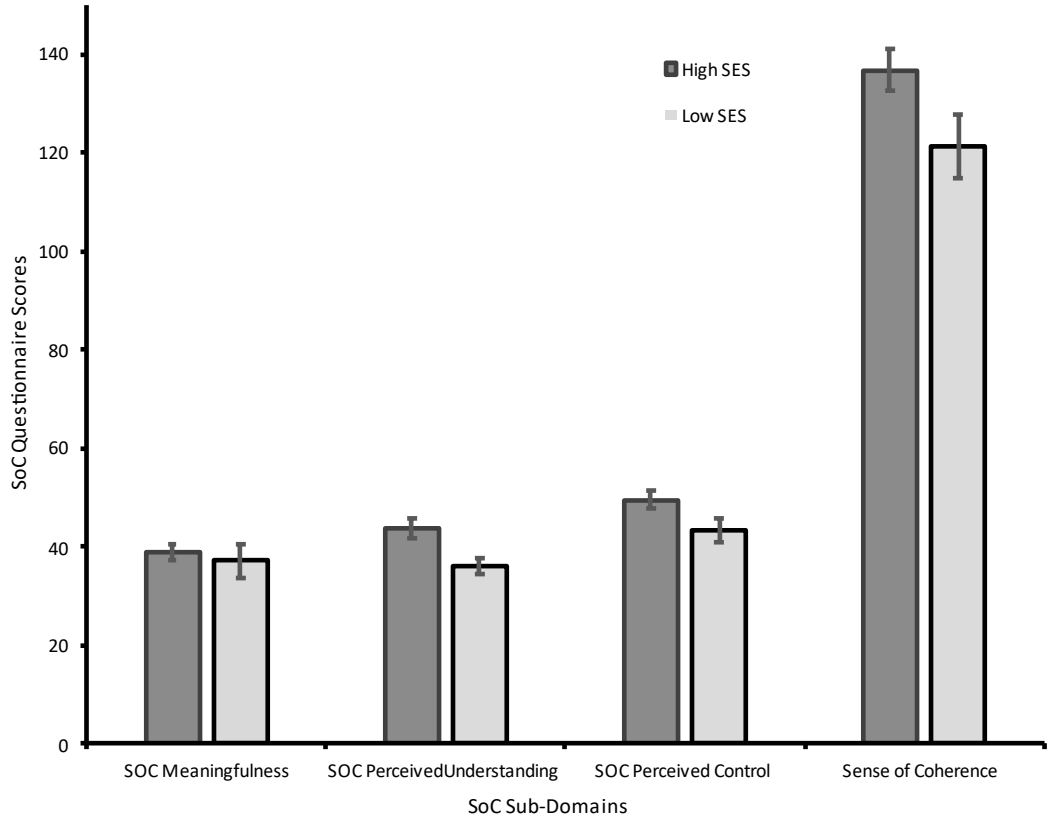
Note. n=31

*Correlation is significant at the 0.001 level (2-tailed)***

As illustrated in Figure 13, the low status group (n = 11) had a perceived understanding sense of coherence (SoC Perceived Understanding) M = 36.00 (SEM = 1.48). By comparison the high SES (n = 20) were marginally higher M = 43.80 (SEM = 2.00). The graph also reveals a meaningful sense of coherence (SoC Meaningfulness) of M = 37.09 (SEM = 3.34) for the low status group. Whilst the high SES group were very marginally higher M = 38.8 (SEM = 1.48). The low status group had a perceived ability to control life events sense of coherence (SoC Perceived control) M = 43.27 (SEM = 2.47), whilst the high SES were marginally higher M = 49.50 (SEM = 1.88). Finally, the low status group had a global sense of coherence M = 121.09 (SEM = 6.53) compared to higher M = 136.70 (SEM = 4.16) for the high SES group.

Figure 13

Sense of Coherence Scores by Socioeconomic Status (n=31) (Mean ± SEM)



The MANOVA results revealed a non-statistically significant difference across status groups on a linear combination of the sense of coherence's sub-domains, F (4, 26) = 2.1, p = .11; Wilks' Λ = .76 (unequal samples), partial η^2 = .244. The follow-up univariate ANOVAs revealed a statistically significant difference between the groups in scores of perceived understanding of existence F (1, 29) = 7.13, p = .01, and global sense of coherence F (1, 29) =4.45, p =.04. The groups did not differ significantly in meaningful sense of coherence, F (1, 29) =.29, p =.60; and perceived ability to control life events, F (1, 29) = 3.95, p = .56. SPSS output provided in Appendix XX.

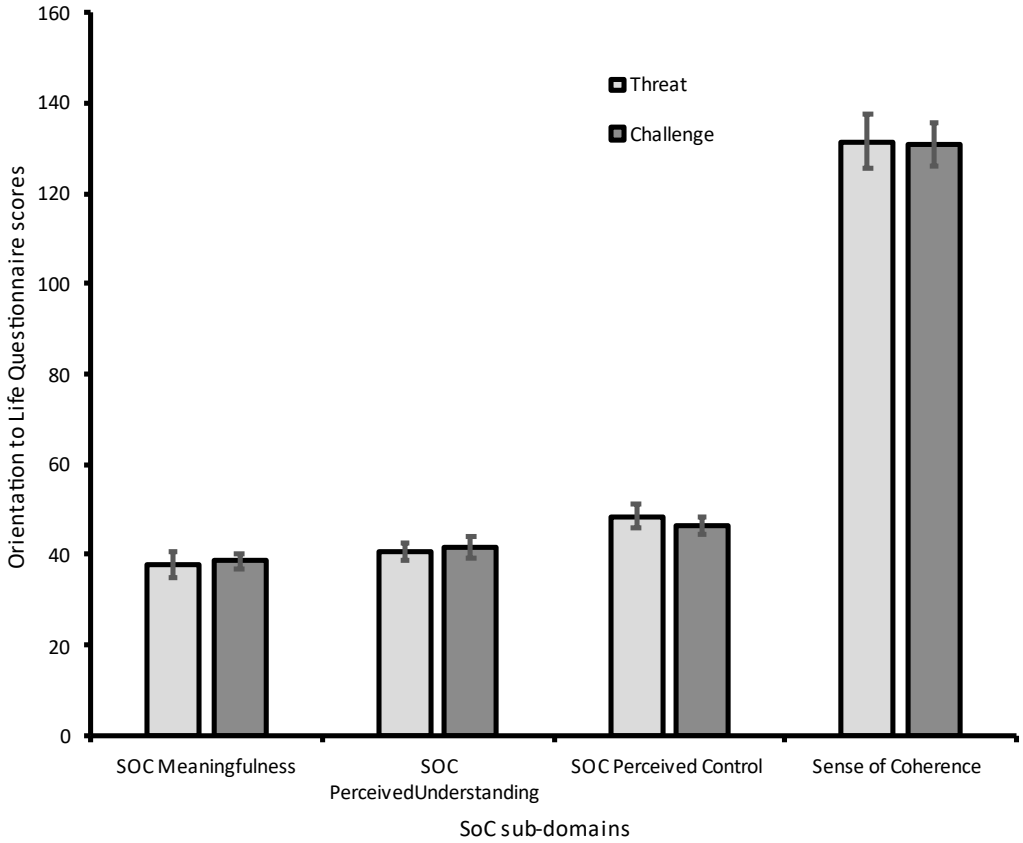
The additionally performed separate one-way ANOVAs (for robustness checks) on each of the sub-domain of sense of coherence scale (i.e., meaningful sense of coherence, perceived understanding of existence, perceived ability to control life events, global sense of coherence) yielded the same results (Appendix XXI).

Following this analysis and considering the relationship of sense of coherence to protective/resilience factors, the scale has been also analysed in the light of threat/challenge appraisals of the task competition. As with SES, assumptions for running a MANOVA have been tested and met prior to the analysis (checks for multicollinearity appear the same of those in the previous analysis, as the DVs overlap). In this analysis, however, the IV is threat/challenge cognitions.

Figure 14 reveals that the threat group (n = 14) had a perceived understanding sense of coherence (SoC Perceived Understanding) M = 40.5 (SEM = 2.0). By comparison the challenge group (n = 17) were marginally higher M = 41.5 (SEM = 2.3). Meaningful sense of coherence (SoC Meaningfulness) for the threat group was M = 37.8 (SEM = 2.7). Whilst for the challenge group it was M = 38.5 (SEM = 1.7). Surprisingly, the threat group had a higher score for perceived ability to control life events, sense of coherence (SoC Perceived control), M = 48.4 (SEM = 2.7), compared to the challenge group - M = 46.4 (SEM = 1.9). Finally, the threat group had a global sense of coherence M = 131.5 (SEM = 6.1) which again appeared higher to the challenge group, M = 130.8 (SEM = 4.7).

Figure 14

Sense of Coherence Scores by Threat/Challenge (n = 31) (Mean ± SEM)



The MANOVA results here also yielded a non-statistically significant difference between threat and challenge groups (i.e., groups perceiving the task competition as either threatening or challenging) on a linear combination of the sense of coherence's sub-domains, F (4, 26) = .51, p = .11; Wilks' Λ = .73 (unequal samples), η^2 = .07. The follow-up univariate ANOVAs did not reveal any statistically significant differences between the groups in scores of each of the sense of coherence subscales (all $p > .05$).

5.2 Attributional Style

A one-way MANOVA was performed in order to compare the effect of status on attributional style. Following Peterson and colleagues' recommendation (1982), only the composite scores were examined (i.e., *Composite Negative Attributional Style*; *Composite Positive Attributional Style*; *Composite Positive minus Composite Negative*, *Hopelessness*, and *Hopefulness*) due to the lower reliability and validity of the

individual dimension scores (i.e., *Internal Negative*, *Stable Negative*, *Global Negative*, *Internal Positive*, *Stable Positive*, and *Global Positive*). Assumptions for running a MANOVA were checked and met prior to analysis. The Box's M value of 1.27 was associated with a *p* value of .22, non-significant. However, as with the sense of coherence measure, it could be argued that multicollinearity between the subdomains exist (Table 9). Consequently, separate ANOVAs have also been run for robustness purposes.

Table 9

Pearson Product Moment Correlations between Attributional Style Questionnaire sub-domains

	1	2	3	4	5
1. Composite Negative	-	.103	-.707**	.862**	.078
2. Composite Positive	.103	-	.630**	.124	.854**
3. Composite Pos minus Composite Neg	.707**	.630**	-	-.585**	.546**
4. Hopelessness	.862**	.124	-.585**	-	.126
5. Hopefulness	.078	.854**	.546**	.126	-

Note. n=31 for all variables.

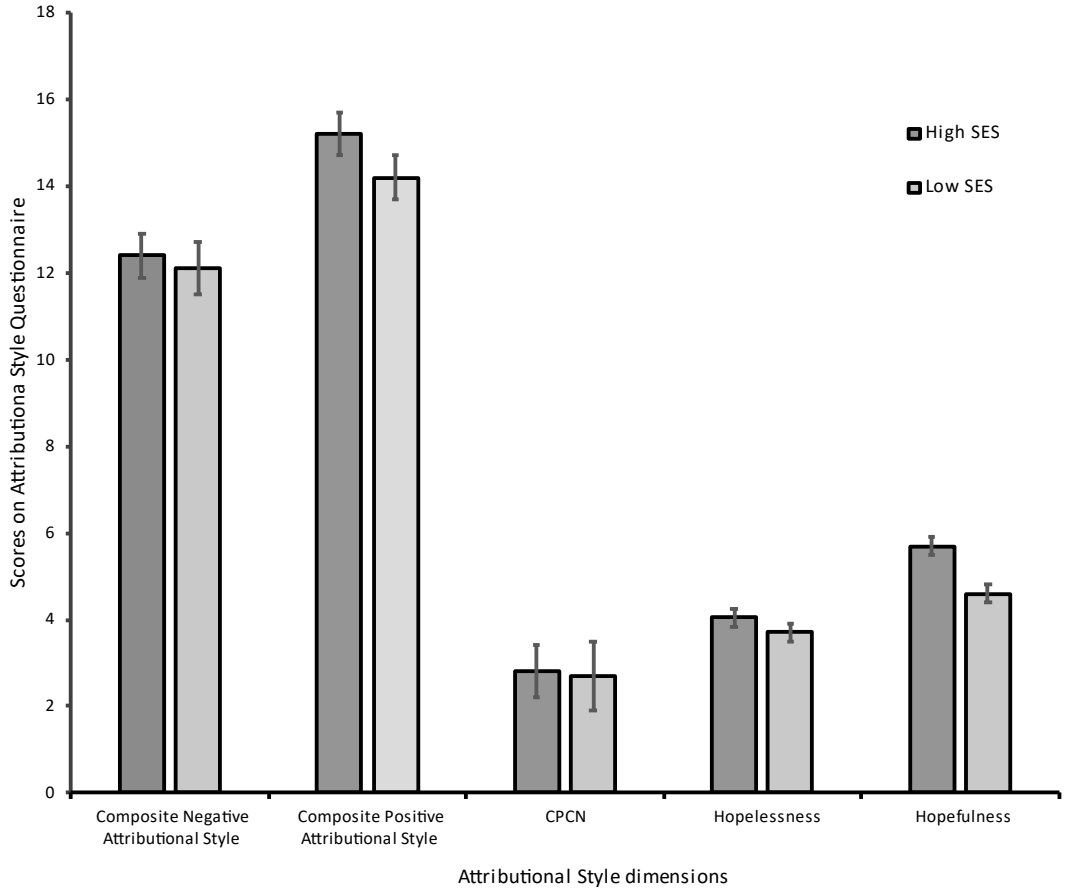
***Significant at the 0.001 level (2-tailed).*

As demonstrated on Figure 15, the low status group (n = 11) had a lower score for composite negative attributional style M = 12.07 (SEM = 0.63) compared to the employed group (n = 20) who had marginally higher scores – M = 12.4 (SEM = 0.51). As illustrated, lower SES groups also exhibit lowers scores for composite positive attributional style M = 14.2 (SEM = 0.54) compared to higher SES groups M = 15.2 (SEM = 0.45). For CPCN (Composite Positive minus Composite Negative), lower SES group had once again lower M =2.16 (SEM = 0.82) scores compared to the high SES group: M = 2.81 (SEM = 0.65). Moreover, lower status group demonstrate marginally lower hopelessness score (M = 2.2; SEM = .83) compared to the higher status group (M = 2.81; SEM = .65). And finally, low socioeconomic group demonstrating lower scores on the

hopefulness (M = 4.6; SEM = .22) compared to higher socioeconomic groups (M = 5.3; SEM = .75).

Figure 15

Attributional Style By Socioeconomic Status (n=31) (Mean and ±SEM)



MANOVA results demonstrated that there is not a statistically significant difference across statuses on a linear combination of the ASQ sub-domains, F (5, 25) = 1.93, p = .13; Wilks' Λ = .72, η^2 = .28. The follow-up univariate ANOVAs revealed that the only statistically significant difference between the groups lies in the mean scores of Hopefulness, F (1, 29) = 5.7, p = .02, η^2= .17. The difference between the group mean scores for the rest of the ASQ dimensions were not significant: composite negative attributional style, F (1, 29) = .155, p = .70, η^2= .01; composite positive attributional style, F (1, 29) = 1.73, p = .20, η^2= .06; CPCN (Composite Positive minus Composite

Negative), F (1, 29) = .37, p = .055, η^2= .01; and finally hopelessness, F (1, 29) = 1.1, p = .31, η^2= .04. SPSS output for this analysis provided in Appendix XXII.

The additionally performed separate one-way ANOVAs (for robustness checks) on each separate sub-domain yielded the same results (Appendix XXIII).

5.3 NEO-FFI-3 (Personality Style)

A one-way MANOVA was performed in order to compare the effect of status on personality style. Most of the assumptions for running a MANOVA were met prior to analysis. However, there were violations of the covariances matrices of the dependent variables across groups and the data lacked normalcy. As aforementioned MANOVA has been considered a robust test against normality assumptions arguing that the investigator would be allowed to proceed with the analysis. In order to address the lack equality of multiple variance-covariance matrices (the Box's M value of 38.4 was associated with a p value of .013- significant), Pillai's Trace correction has been adapted when reporting the results.

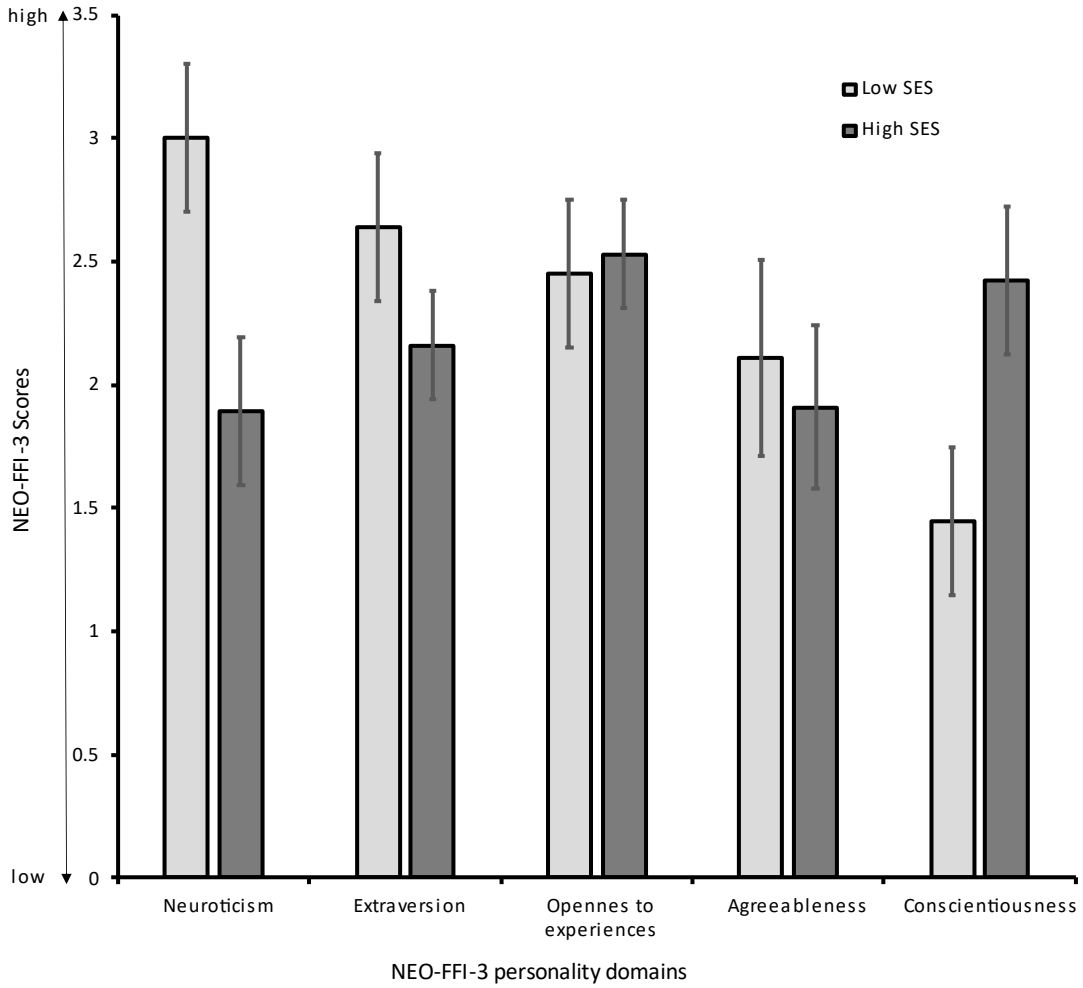
One might argue that the violations of these assumptions and the small sample size do not allow proceeding with the chosen analysis. For this reason and for robustness checks, separate one-way ANOVAs have also been performed to examine any significant difference between the two status groups on the separate NEO-FFI-3 domains.

As demonstrated on Figure 16, the low status group (n= 11) demonstrated considerably higher score for neuroticism M= 3.00 (SEM=.27) compared to the employed group (n= 19) who scored lower - M= 1.89 (SEM=.30). The lower SES group also exhibited higher scores for extraversion M= 2.64 (SEM= .30) compared to higher SES groups M= 2.16 (SEM= .22). For Openness to Experiences, lower SES group scored lower M=2.45 (SEM= .30) compared to the high SES group: M= 2.53 (SEM= .22). Moreover, lower status group displayed lower score on agreeableness (M= 1.91; SEM= .40) compared to the higher status group (M= 2.11; SEM= .33). And finally, low socioeconomic group

demonstrated marginally lower scores on conscientiousness (M= 1.45; SEM= .34) compared to higher socioeconomic group (M= 2.42; SEM= .30).

Figure 16

Personality Style Across Socioeconomic Groups (n= 30) (Mean ranks and ±SEM)



MANOVA results illustrated a statistically significant difference across status on a linear combination of the NEO-FFI-3 personality domains, F (5, 24) = 3.6, p = .02; Pillai's Trace = .43, η^2 = .43. Subsequent follow-up univariate ANOVAs revealed that the groups displayed statistically significant differences in mean scores of neuroticism, F (1, 28) = 6.7, p = .02, η^2= .19 and conscientiousness, F (1, 28) = 5.2, p = .03, η^2= .16. Further to this, individuals from low SES scored significantly higher on neuroticism

and significantly lower on conscientiousness compared to those from the higher SES group. SPSS output for this analysis provided in Appendix XXIV.

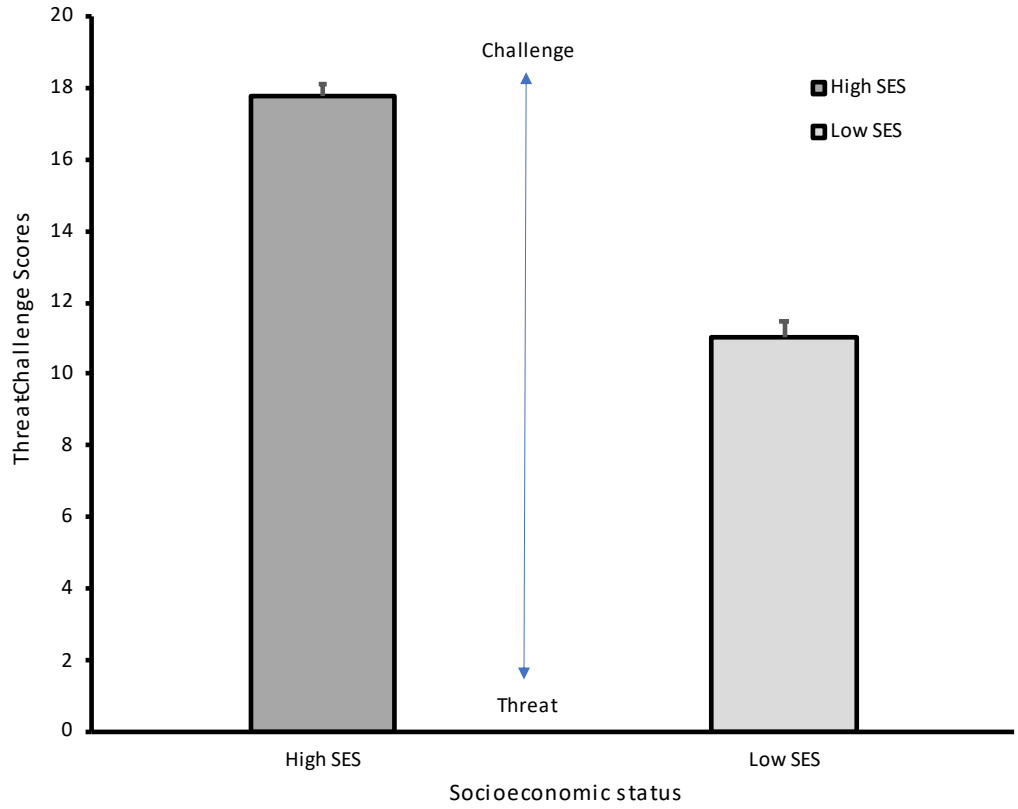
Additional one-way ANOVAs (for robustness checks) on each separate sub-domain yielded the same results (Appendix XXV).

5.4 Threat/Challenge Appraisals by SES

Figure 17 demonstrates that higher SES group cognitively appraises the competition event as more challenging (Mean rank= 17.8) than threatening compared to the lower SES (Mean rank= 11.0). A Mann-U-Whitney test was performed in order to test whether the difference between mean ranks was statistically significant. Data was not normally distributed ($p < .001$), and the dependent variable was measured on an ordinal scale, suggesting the utilisation of a non-parametric test. The dependent variable was perceptions of threat/challenge, measured on a Likert scale from -4 to 4, where -4 was most threatening, 0 was neutral, and +4 was most challenging. The independent variable was socioeconomic status. One participant from the low SES cohort was excluded from the analysis due to missing data. Resultantly, the samples size was 30 (n=30) with 20 individuals from the high SES group and 10 from the low.

Figure 17

Appraisals of Threat-Challenge Across Socioeconomic Groups (n=30) (Mean ranks and ±SEM)



The results yielded statistically significant difference between the groups (U= 55.0, p = .04, r= -0.4), with higher SES groups perceiving the event as more challenging than threatening compared to the lower SES group. SPSS output provided in Appendix XXVI.

5.5 Task Workload and Threat/Challenge Appraisals

As previously outlined, psychosocial variables will be taken into consideration in the analysis of factors related to endocrine reactivity in the face of threat and challenge (Chapter 6, sections 6.1.3 and 6.1.5). In this section, however, task workload will be explored as psychological variable that does not in any straightforward manner relate or impact endocrine reactivity, but rather contributes to the formation of threat and challenge cognitions, similar to SoC. Hereafter, interactions between task workload

and threat/challenge cognitions were the primary focus of this analysis. An overall score for the NASA-TLX has been created by adding the scores of the instrument's subdomains (Table 10). Subsequently, assumptions for running Spearman's rank correlation have been tested and met.

Table 10

NASA-TLX (Mean and ±SEM)

Variable	*Mean*	*SEM*
Mental	-.97	.47
Physical	-3.9	.32
Temporal	.12	.56
Performance	1.9	.57
Effort	-.38	.50
Frustration	-1.95	.52
Total Raw TLX Score	-5.3	1.6

Note. n=30

Table 11

Spearman's Rank Correlation Matrix between Task Workload and Threat/Challenge Cognitions

		Total Raw TLX Score	Threat/Challenge Cognitions
Total Raw NASA TLX Score	Correlation Coefficient	-	.289
Threat/Challenge Cognitions	Correlation Coefficient	.289	-

Note. N=30

All correlations non-significant.

The results revealed no statistically significant correlation between workload and threat/challenge cognitions, r_s (28) = .30, p = .12, Table 11. SPSS output provided in Appendix XXVII.

5.6 Motivational questionnaire

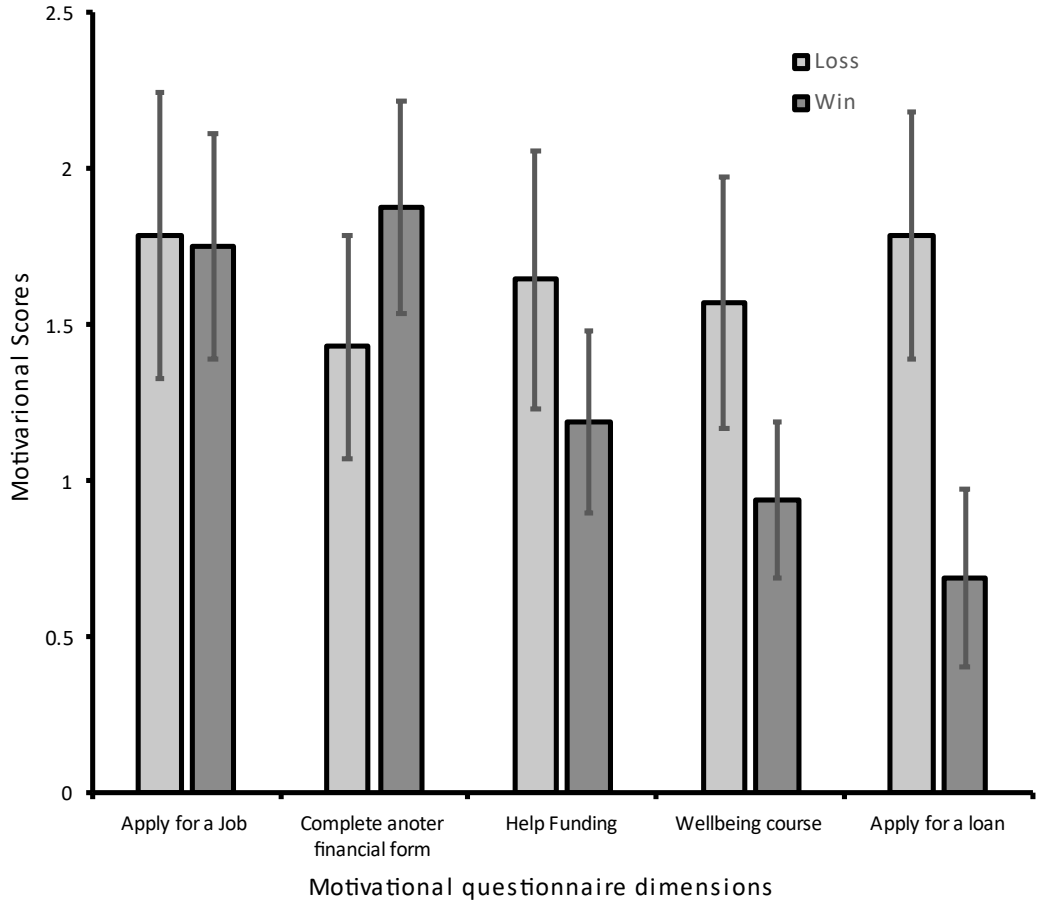
In order to test whether there are differences between the mean scores on motivational questionnaire by outcome group (win/loss) multivariate Kruskal-Wallis analysis was performed. The independent variable was outcome (win/loss), whilst the dependent variables were scores on the motivational questionnaire (i.e., how motivated individuals were to complete any of the following tasks immediately after experimental exposure: Apply for a job; Complete a financial form; Seek help for funding matters; Join a finance wellbeing course, in order to acquire knowledge of how to improve your financial situation; Apply for a mortgage/credit/loan). Normality checks indicated that the data was not normally distributed across outcome groups. The dependent variables were also ordinal data, measured on a Likert scale (from 0 to 4). Hereafter, non-parametric tests were utilized. However, one might argue that the dependent variable could be treated as a scale rather than ordinal data, whilst MANOVA is considered a robust test against the normality assumption (Real Statistics, 2020). Resultantly, for robustness purposes, one-way MANOVA was performed complementing to the non-parametric analyses.

Figure 18 represents differences between motivational scores on five of the dimensions of the motivational questionnaire across outcome groups (win/loss) immediately after the competition. Winners (M = 1.8; SEM = .40) and losers (M = 1.8; SEM = .50) displaying almost similar motivational response when it comes to their incentive to apply for a job immediately following victory/defeat at the contest. Regarding completing another questionnaire immediately after the form filling competition, winners at the contest displayed marginally higher desire (M = 1.9; SEM = .34) to compete again compared to the losing group (M = 1.4; SEM = .40). For seeking help for funding matters, join a finance wellbeing course and apply for a mortgage/credit/loan losing groups displayed higher mean motivational scores (M = 1.6 SEM = .41: M = 1.6

SEM = .40; M = 1.8 SEM = .40) compared to the winners (M = 1.2 SEM = .30: M = .94 SEM = .30; M = .70 SEM = .30).

Figure 18

Motivational Questionnaire Scores Across Outcome Groups (n= 30) (Mean and ±SEM)



Multivariate Kruskal-Wallis results yielded non statistically significant differences between the two outcome groups (win and loss) on: motivation to apply for a job, motivation to complete another financial questionnaire, motivation to seek help for funding matters, and motivation to join a financial well-being course, following competition exposure. However, the two groups differed significantly in their motivation to apply for a loan, X2 (1) = 4.67, p = .031, with the winning group expressing lower motivation to apply (mean rank = 12.44) compared to the losing group (mean rank = 19). Interestingly, following defeat, the losing group expressed a lower motivation to complete another financial form (mean rank = 13.96) compared to

the winning group (mean rank = 16.84). However, this difference did not reach a significant level. SPSS output provided in Appendix XXVIII.

Closer examination of the data (i.e., differences in motivational scores between losing and winning within lower SES and higher SES populations) yielded non-statistically significant results between any of the motivational questionnaire domains and within any of the cohorts (low/high SES) (Appendix XXIX).

MANOVA results demonstrated that there is not a statistically significant difference across outcome on a linear combination of the motivational questionnaire sub-domains, F (5, 24) = 1.91, p = *.13;* Wilks' Λ = .72, η^2 = .30. The follow-up univariate ANOVAs, alike the multivariate Kruskal- Wallis, revealed that the only statistically significant difference between the groups lies in the mean motivation scores to apply for a loan F (1, 28) = 5.3, p = .03, η^2= .16.

Chapter 6

Salivary testosterone and cortisol reactivity to threat/challenge cognitions in socioeconomically disadvantaged and affluent males

- The analyses reported in this chapter address RQ2, H2, RQ3, H3, and RQ4, H4:

RQ2: "Are psychosocial variables related to endocrine reactivity?"

H2: "Psychosocial variables will be related to endocrine reactivity."

RQ3: "Does cognitive appraisal of threat/challenge moderate endocrine reactivity?"

H3: "Cognitive appraisals of threat/ challenge will moderate endocrine reactivity in both SES populations."

RQ4: "Does T reactivity correlate with a reduced motivation to engage socially?"

H4: "Reduced motivation to engage with subsequent social tasks will positively correlate with a decrease of T reactivity."

6 Introduction

This chapter aims to draw links and relationships between the endocrine and psychosocial analysis performed in chapter 4 and 5, respectively. Hereafter, the section explores whether the explored psychosocial variables appear to be related to endocrine reactivity, and whether testosterone and cortisol reactivity to status competition are moderated by cognitive appraisals, thereby addressing RQ2 and H2, and RQ3 and H3:

RQ2: "Are psychosocial variables related to endocrine reactivity?"

H2: "Psychosocial variables will be related to endocrine reactivity."

RQ3: "Does cognitive appraisal of threat/challenge moderate endocrine reactivity?"

H3: "Cognitive appraisals of threat/challenge will moderate endocrine reactivity in both SES populations."

The chapter starts with a general evaluation of the relationships between the psychosocial variables that appear to yield significant differences amongst SES groups (drawn on analysis in Chapter 5), SES (as an objective measure), and endocrine

reactivity to status competition. The analyses performed for this purpose are Pearson's and Spearman's correlations followed by a multiple regression model. Based on the outcomes of the analyses, and because cognitive appraisals in this thesis were hypothesised to be moderators of the relationship between endocrine reactivity and competition outcome rather than independent predictors or simply correlated to endocrine response, the chapter then proceeds with an evaluation of the threat/challenge appraisals as moderators of endocrine reactivity to competition outcome. This analysis is performed with the aid of custom ANCOVAs.

Drawing on the findings from Sharp (2006), which indicate the importance of mood on endocrine reactivity, the chapter proceeds with analysis of the relationship between trait affect and post status competition endocrine reactivity. All analyses are performed on T data initially, followed by C data.

Finally, the chapter explores the relationship between endocrine reactivity and post status competition motivation. This is built on the motivational data provided in chapter 5, in this instance however, the data is not analysed in relation to competition outcome only but endeavours to tease out the complexity of the relationship between status changes, post status competition T fluctuations, and future motivation/status - seeking behaviours. In doing so, the chapter addresses the last research question and hypothesis:

> RQ4: "Does T reactivity correlate with a reduced motivation to engage socially?"
>
> H4: "Reduced motivation to engage with subsequent social tasks will positively correlate with a decrease of T reactivity."

6.1 Psychosocial Variables, Cognitive Moderators and Endocrine Response

6.1.1 Psychosocial Variables, SES and T reactivity

To test the relationships between those psychosocial variables which appear to yield significant differences amongst SES groups (drawing on an analysis within Chapter 5), SES (as an objective measure), and endocrine reactivity to status competition, a series

of analyses were performed. Specifically, these analyses were Pearson's and Spearman's correlations followed by a multiple regression. In doing so, the research addresses address RQ2 and H2. Firstly, data were entered in a Pearson's r and Spearman's rank correlations in order to identify the strongest correlations between post-competition T reactivity and the psychosocial data. A multiple linear regression was thereafter conducted to measure the impact of the predictor variables socioeconomic status and threat/challenge cognitions on the criterion variable post-competition T reactivity.

Spearman's and Pearson's correlations results revealed that the only significant correlation between the psychosocial and endocrine data were between threat/challenge and T reactivity (Table 12 and 13).

As illustrated on Table 12, none of the correlations between the psychosocial variables: Perceived understanding of existence (sense of coherence subscale), Perceived ability to handle/control events (sense of coherence subscale), Global sense of coherence, CPCN (ASQ subscale), Hopefulness (ASQ subscale) and post-competition Testosterone reactivity were significant ($p > .05$) (Appendix XXX).

Table 12

Pearson's R Matrix Correlation between Psychological data and Post-competition T Reactivity

			1.	2.	3.	4.	5.	6.
1.	T reactivity	r	-	-.201	-.213	-.227	-.044	-.119
2.	SoC Perceived Understanding	r	-.201	-	.514**	.733**	-.103	-.097
3.	SoC Perceived Control	r	-.213	.514**	-	.895**	.314	.299
4.	Global sense of coherence	r	-.227	.733**	.895**	-	.214	.160
5.	CPCN-Attributional Style	r	-.044	-.103	.314	.214	-	.546**
6.	Hopefulness	r	-.119	-.097	.299	.160	.546**	-

Note. n=31

*Correlation is significant at the 0.01 level (2-tailed)***

*Correlation is significant at the 0.05 level (2-tailed)**

Table 13 illustrates that there is only one close to statistically significant negative correlation between the post-competition T reactivity and psychosocial variables (Appendix XXXI). Namely, the relationship between threat/challenge cognitions and T reactivity (r_s (29) = -.351, p = .053). Because of the central importance of SES and its' relationship to T reactivity for this research, status has also been included in the further regression model.

Table 13

Spearman's Rank Matrix Correlation between Psychological data and Post-competition T Reactivity

		1.	2.	3.	4.	5.	6.	7.
1. Help with Funding Matters	r	-	.671**	-.203	-.562	-.031	.576	.220
	n	30	30	29	29	30	30	30
2. Wellbeing Course	r	.671**	-	-.203	.416*	-.168	-.413*	.042
	n	30	30	29	29	30	30	30
3. Conscientiousness	r	-.203	-.203	-	-.348	.302	-.388*	-.056
	n	29	29	30	30	30	30	30
4. Neuroticism	r	.562**	.416*	-.348	-	.124	.433*	.304
	n	29	29	30	30	30	30	30
5. Threat/Challenge Cognitions	r	-.031	-.168	.302	.124	-	-.433*	-.351
	n	30	30	30	30	31	31	31
6. Socioeconomic status	r	.576**	.413*	-.388*	.433*	-.433*	-	.309
	n	30	30	30	30	31	31	31
7. T reactivity	r	.220	.042	-.056	.304	-.351	.309	-
	n	30	30	30	30	31	31	31

Note. n=30

*Correlation is significant at the 0.01 level (2-tailed) ***

*Correlation is significant at the 0.05 level (2-tailed) **

To test for multicollinearity between predictor variables, Pearson Product Moment correlations were calculated among the two predictors. None of the correlations reached the threshold of 0.80, demonstrating lack of closely related variables (Table 14).

Table 14

Correlation Coefficients Between Predictor (status and threat/challenge cognitions) and Criterion Variables (testosterone reactivity)

	1.	2.	3.
1. Reactivity	-	.277	-.255
2. Status	.277	-	-.452*
3. Threat/Challenge	-.225	-.425*	-

Note. N=31

**Pearson Product Moment Correlations significant at ≤0.01 (1-tailed)*

Using the enter method it was found that socioeconomic status and threat/ challenge perceptions did not significantly predict T reactivity: F (2, 28) = 1.4, p = .270, R^2 = .09, $R^2Adjusted$ = .024. The R^2 of .09 indicates that 9% of the variance in T reactivity is explained by the predictor variables. Closer examination of the relation coefficients reveals that the predictor variable – socioeconomic status had a higher impact on the criterion variable, β = .221, t = 1.09, p = .28) compared to threat/challenge cognitions (β = -.13, t = -.62, p = .54) (Appendix XXXII).

6.1.2 Threat/Challenge cognitions as moderators of T reactivity

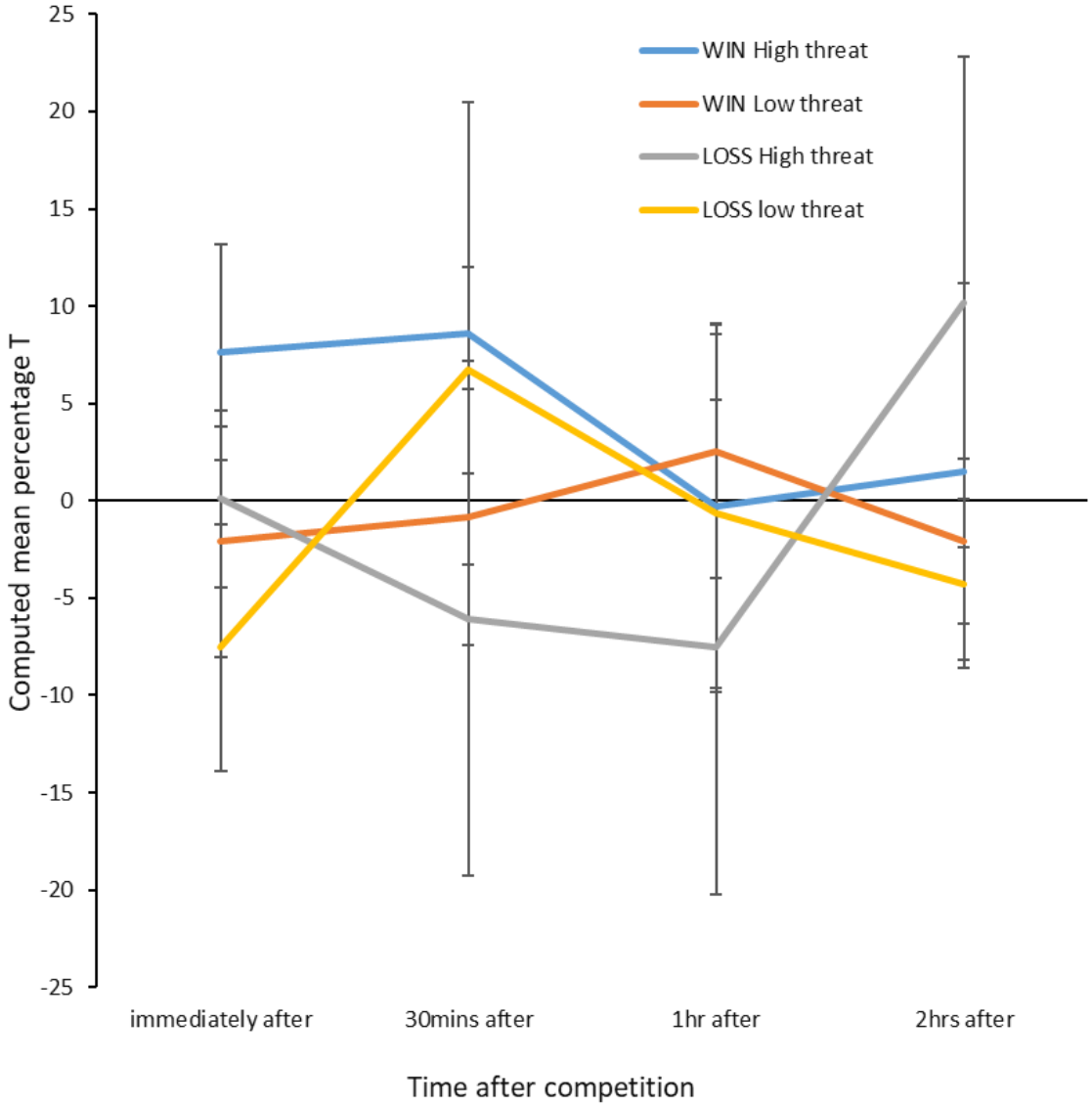
In order to address how cognitive appraisal of threat/challenge moderate endocrine reactivity, a custom ANCOVA has been performed on Testosterone post-competition reactivity data. Prior to any inferential analysis the Threat/Challenge scores have been recoded from the original 9-Likert point scale (where -4= 'most threatening'; 0=

'Neither'; and +4= 'Most Challenging') to High and Low Threat (0= 'low threat' and 1= 'high treat'). The variable was recorded as a dichotomous following a graph distribution illustrating that the moderator could be split into the abovementioned two main categories (Threat and Challenge) without losing meaningful data. Analysing the variable as initially recorded (from -4 to +4) would have distributed the variable across all recorded points, therefore capturing very little meaningful information and proving very little within the study due to its' limited sample size. Computed variables for the Testosterone reactivity have also been created based on the subtraction between the mean T levels immediately before the competition and levels immediately after the competition. All assumptions for running the custom ANCOVA were tested and met prior to analysis.

Figure 19 illustrates the difference between T levels in winning and losing condition for low and high threat groups.

Figure 19

Computed Mean Percentage T Change by Post-Competition Outcome and Threat/Challenge Cognitions (Mean and ±SEM) (n=31)



To tests whether threat/challenge cognitions moderate T reactivity following victory/defeat a custom model ANCOVA analysis has been performed (n = 31). The model has been customised in order to include the interaction between the covariate and the categorical predictor (Field, 2018). The independent variables were time (withing-subject factor with 4 levels - time points) and outcome (between-subject factor with 2 levels- win/loss). The dependent variable was computed mean

percentage T levels. Covariate was threat/challenge cognitions. Descriptive statistics are presented at the table below (Table 15):

Table 15

Descriptive Statistics for Custom ANCOVA - Post-competition computed testosterone difference by outcome and status (Mean, SEM)

	Win		Loss	
	High Threat	Low Threat	High Threat	Low Threat
Immediately post	7.65(5.54)	-2.1(5.94)	0.102(4.55)	-7.54(6.32)
30mins post	8.6(11.9)	-0.85(6.6)	-6.1(13.2)	6.72(5.3)
1hr post	-0.31(9.33)	2.56(6.53)	-7.54(12.7)	-0.65(9.21)
2hr post	1.5(9.7)	-2.1(4.23)	10.2(12.6)	-4.26(4.32)

Main effects of each of the residual and all possible two-way and three-way interactions were reported. The results yielded non-significant main effects of time, F (3, 81) = .70, p = .60, η^2 = .03 or outcome, F (1, 27) = .64, p = .43, η^2 = .023, after controlling for the effect of threat/challenge cognitions. The covariate, threat/challenge cognitions, was also not significantly related to the T reactivity, F (1, 27) = .86, p = .36. The two-way interactions between time and outcome F (3, 81) = 1.80, p = .20, η^2 = .06, time and threat F (3, 81) = .60, p = .64, η^2 = .02, and outcome and threat, F (1, 27) = .30, p = .60, η^2 = .01, were not significant.

However, there was a significant three-way linear interaction between time, outcome and threat, F (3, 81) = 3.5, p = .02, η^2 = .11. Table of contrasts examining polynominal trends revealed that the three-way interaction time*outcome*threat appears significant at a linear level (p = .02), meaning that the group mean T levels increase proportionally over the collection time. Closer examination of the table of contrasts also revealed a significant two-way linear interaction between time*outcome (only

when adjusted for threat/challenge cognitions, $p = .03$) The profile plot revealed some different patterns of the adjusted means between winning and losing group at immediately after to 30mins post competition, and from 1hr to 2hrs post competition (Appendix XXXIII).

Two separate follow-up ANOVAs (one on the threat condition and one on the challenge condition) were performed in order to unpack the observed significant three-way interaction (i.e., time*outcome*threat). The within-subject factor for both ANCOVAs was time, whilst the between-subject factor was outcome. The results revealed non-significant main effects of time in both threat and challenge conditions ($p = .50$; $p = .35$, respectively). The main effects of outcome in both groups (threat and challenge) were also non-significant ($p = .70$; $p = .90$, respectively). Moreover, the two-way interactions between time and outcome for both groups (threat and challenge) were also not significant ($p = .36$ - for threat condition; $p = .54$ – for challenge condition) (Appendix XXXIV). Although the follow-up ANOVAs were not statistically significant and the study was underpowered, there are detectible patterns. For instance, those in the loss high threat condition demonstrate a moderate response immediately after, followed by a steep drop, and end up with the highest cortisol 2 hours after. Whilst the loss low threat condition climes steeply after 30 minutes, but then drops steeply thereafter. Further, participants in the win low threat condition display consistent response throughout. Finally, the win high threat starts high until after 30 minutes and then drops after an hour and stays low (table 15 and figure 19 for reference). A more detailed interpretation of these relationships is provided in the discussion section (p.253/254).

6.1.3 Impact of Trait Affect and Competition Outcome on T Reactivity

Drawing on the findings from Sharp (2006), indicating the importance of mood on endocrine reactivity, a multivariable linear regression has been performed on testosterone post-competition reactivity data in order to address whether trait affect, and competition outcome predict endocrine reactivity. For the purposes of this analysis the general negative and positive affect PANAS-X scores (sum of the ten items on both positive and negative emotions) were used. This is because the study was not

interested in individual sub-domains of the PANAS-X scale but in the total negative and positive affect scores exhibited prior and post competition exposure. Prior to any inferential analysis computed variables for the testosterone reactivity have been created based on the change between the mean T levels, immediately before the competition, and levels immediately after the competition (T percentage difference). Computed variables for the residuals - general negative and general positive affect have also been created, based on the change between affect prior to the competition and after it (general negative affect difference and general positive affect difference). All assumptions for running the regression models were met prior to analysis.

Multiple linear regression was conducted to measure the impact of the predictor variables competition outcome and trait affect on the outcome variable T reactivity in percentage. Table 16 presents the descriptive statistics for PANAS-X data (subscales and total) at pre- and post-competition (n = 30).

Table 16

PANAS-X for Pre- and Post-Competition (Mean and ±SEM)

Variable	*Pre-Competition*	*Post-Competition*
General negative emotion	14.5 (1.2)	14.5 (.97)
General positive emotion	32.3 (1.4)	30.3 (1.5)
Fear	9.0 (.70)	9.0 (.55)
Sadness	8.1 (.80)	7.4 (.60)
Guilt	8.9 (.94)	8.4 (.81)
Hostility	8.7 (.71)	9.6 (.98)
Shyness	7.9 (.55)	6.8 (.41)
Fatigue	8.1 (.62)	7.0 (.48)
Joviality	25.1 (1.2)	23.7 (1.41)
Self-assurance	19.4 (.80)	17.2 (.90)
Attentiveness	13.6 (.55)	12.7 (.61)
Serenity	10.2 (.50)	8.8 (.50)
Surprise	5.6 (.53)	6.9 (.60)
Basic Positive Affect	19.5 (.80)	17.9 (.90)

Basic Negative Affect	8.7 (.72)	8.6 (.60)

Note. N=30

Descriptive stats for all variables entered in the regression model outlined in the table below (Table 17):

Table 17

Descriptive Statistics for Testosterone Percentage Change, General Positive and Negative Affect PANAS-X Scores, and Competition Outcome (Mean and SD)

Testosterone Percentage Change	-.26(16.13)
General Negative PANAS-X Score Change	-.03(4.48)
General Positive PANAS-X Score Change	2(6.64)
Competition Outcome	.53(.51)

Note. N=30

The results revealed that positive and negative trait affects, and competition outcome do not significantly predict the T change in reactivity: F (3, 26) = 1.6, p = .22, R^2 = .16, R^2Adjusted = .06. The R^2 of .16 indicates that 16% of the variance in T reactivity is explained by the predictor variables (Appendix XXXV). Closer examination of the relation coefficients reveals that the predictor variable negative affect had a higher impact on the outcome variable, (β = .32, t = 1.8, p = .09) compared to competition outcome (β = .23, t = 1.2, p = .24). The correlations table revealed a weak positive correlation (.164) between the residual competition outcome and the criterion variable T reactivity. One might argue that this requires the variable to be extracted from the model. Thus, the analysis has been repeated without the residual competition outcome. The model results revealed a non-significant relationship between the predictors – negative and positive affects and the criterion variable T reactivity, F (2, 27) = 1.5, p = .21, R^2 = .11, R^2Adjusted = .04 (Appendix XXXVI).

6.1.4 Psychosocial Variables, SES and C reactivity

As with T, to test the relationships between the psychosocial variables that appear to yield significant differences amongst SES groups, SES, and endocrine reactivity to status competition, correlation and regression analyses were performed. In doing so, the research addresses RQ2 and H2. Similarly to T, data were entered in a Pearson's r and Spearman's rank correlations in order to identify the strongest correlations between post-competition C reactivity and the psychosocial data. Subsequently, a multiple linear regression was conducted to measure the impact of the predictor variables socioeconomic status and threat/challenge cognitions on the criterion variable post-competition C reactivity. All assumptions (e.g., rejecting multicollinearity, Table 20) were met prior to the analysis. Results revealed that the only significant interactions between the psychosocial and endocrine data were between status and C reactivity, and threat/challenge and C reactivity (Table 18 and 19).

Table 18 reveals that none of the correlations between the psychological variables: Perceived understanding of existence (sense of coherence subscale), Perceived ability to handle/control events (sense of coherence subscale), Global sense of coherence, CPCN (ASQ subscale), Hopefulness (ASQ subscale) and post-competition Cortisol reactivity were significant ($p > .05$) (Appendix XXXVII).

Table 18

Pearson's R Matrix Correlation between Psychological data and Post-Competition C Reactivity

			1.	2.	3.	4.	5.	6.
1.	C reactivity	r	-	.006	-.136	-.043	-.209	-.230
2.	Sense of coherence – Understanding	r	.006	-	.514**	.733**	-.103	-.097

3.	Sense of coherence – Control	r	-.136	.514**	-	.895**	.314	.299
4.	Global sense of coherence	r	-.043	.733**	.895**	-	.214	.160
5.	CPCN-Attributional Style	r	-.209	-.103	.314	.214	-	.546**
6.	Hopefulness	r	-.230	-.097	.299	.160	.546**	-

Note. N=31

*Correlation is significant at the 0.01 level (2-tailed)***

*Correlation is significant at the 0.05 level (2-tailed)**

Table 19 displays that there is only one statistically significant positive correlation between the endocrine and psychological data. This is the correlation between socioeconomic status and C reactivity (r_s (28) = .482, p = .01) (Appendix XXXVIII). In order to investigate whether socioeconomic status or threat/challenge cognitions predict C reactivity, a multiple regression analysis was performed.

Table 19

Spearman's Rank Matrix Correlation between Psychological data and Post-Competition C Reactivity

		1.	2.	3.	4.	5.	6.	7.
1.	Help with Funding Matters	-	.671**	-.203	-.562**	-.031	.576	.051
2.	Wellbeing Course	.671**	-	-.203	.416*	-.168	-.413*	.203
3.	Conscientiousness	-.203	-.203	-	-.348	.302	-.388*	-.184
4.	Neuroticism	.562**	.416*	-.348	-	.124	.433*	.245
5.	Threat/Challenge Cognitions	-.031	-.168	.302	.124	-	-.433*	-.237
6.	Socioeconomic status	.576**	.413*	-.388*	.433*	-.433*	-	.482*
7.	C reactivity	.051	.203	-.184	.245	-.237	.482**	-

Note. N=30

*Correlation is significant at the 0.01 level (2-tailed)***

*Correlation is significant at the 0.05 level (2-tailed)**

As with T, to test for multicollinearity between predictor variables, Pearson Product Moment correlations were calculated among the two predictors (Table 20). Again, the table demonstrates lack of closely related variables (<.80).

Table 20

Correlation Coefficients Between Predictor (status and cognitions) and Criterion Variables (cortisol reactivity)

	1.	2.	3.
1. Cortisol Reactivity	-	.421*	-.231
2. Status	.421	-	-.441
3. Threat/Challenge cognitions	-.231*	-.441*	-

Note. N=30

**Pearson Product Moment Correlations significant at ≤ 0.05*

Using the enter method it was found that socioeconomic status and threat/challenge perceptions did not significantly predict the C reactivity: F (2, 27) = 2.96, p = .07, R^2 = .18, R^2Adjusted = .12. The R^2 of .12 indicates that 12% of the variance in C reactivity is explained by the predictor variables. Closer examination of the relation coefficients reveals that the predictor variable – socioeconomic status had a higher impact on the criterion variable (β = .396, t = 2.04, p = .05) compared to threat/challenge cognitions (β = -.06, t = -.29, p = .77) (Appendix XXXIX). It has been noted that threat/challenge cognitions functioned as a suppressor variable. Thus, when controlled for it, a linear regression model with one predictor variable – status – revealed that status statistically significant predicts cortisol levels, F (1, 28) = 6.04, p = .02, R^2 = .18, R^2Adjusted = .15. The R^2 of .18 indicates that 18% of the variance in C reactivity is explained by the predictor variable.

6.1.5 Threat/Challenge cognitions moderatos of C reactivity

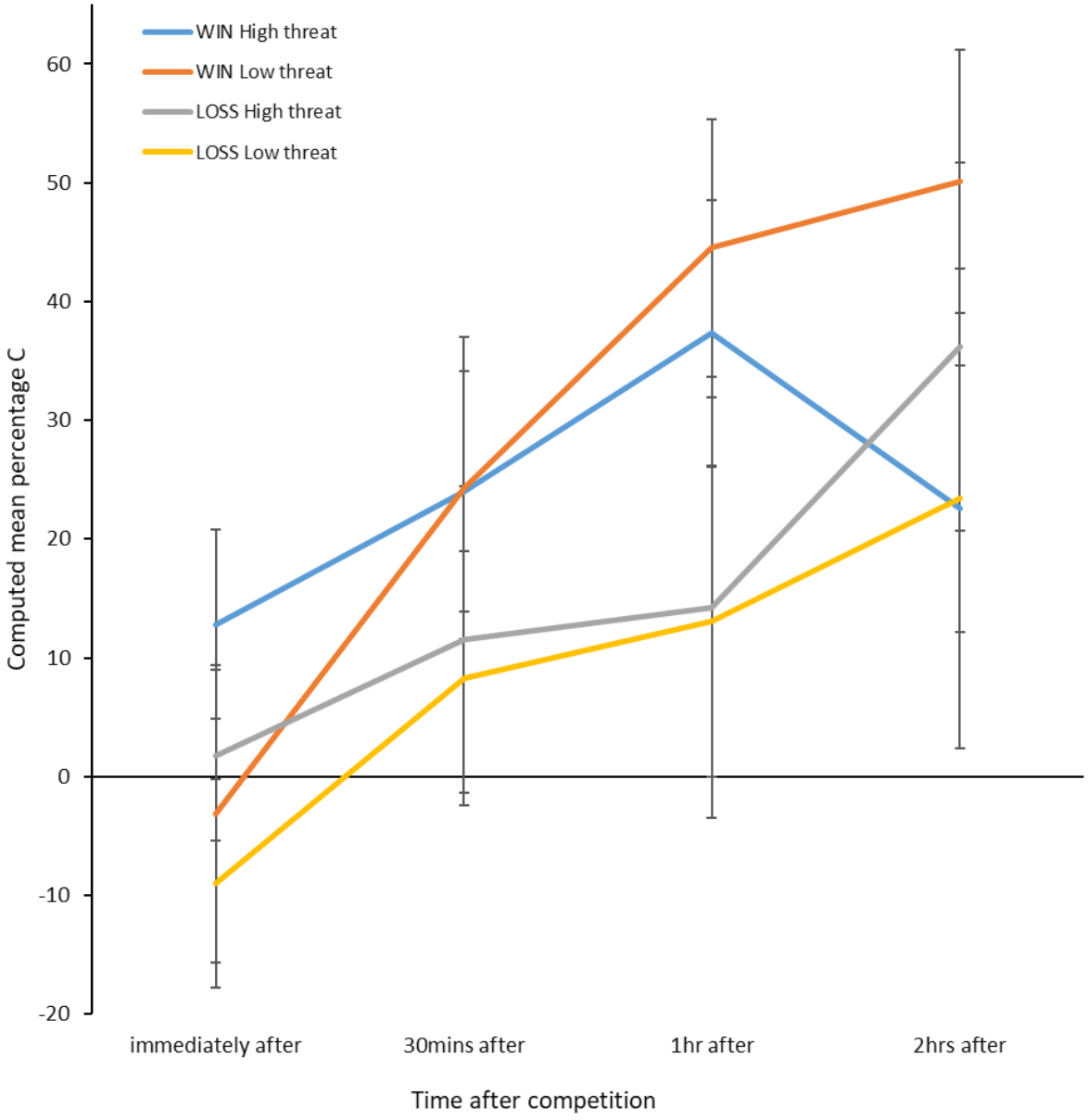
As with T, a custom ANCOVA has been performed on cortisol post-competition data in order to address how cognitive appraisal of threat/challenge moderate the

endocrine response. Again, threat/challenge scores have been recoded from the original 9-Likert point scale (where -4= 'most threatening'; 0= 'Neither'; and +4= 'Most Challenging') to High and Low Threat (0= 'low threat' and 1= 'high treat'). Whilst computed variables for the cortisol reactivity have also been created based on the subtraction between the mean C levels immediately before the competition and levels immediately after the competition. All assumptions for running the custom ANCOVA were tested and met prior to analysis.

Figure 20 illustrates the difference between C levels in winning and losing condition for low and high threat groups.

Figure 20

Computed Mean Percentage C by Post-Competition Outcome and Threat/Challenge Cognitions (Mean and ±SEM) (n=30)



In order to tests whether threat/challenge cognitions moderate C reactivity following victory/defeat a custom model ANCOVA analysis has been performed (n=30). Again, as with T, the model has been customised in order to include the interaction between the covariate and the categorical predictor. The independent variables were time

(withing-subject factor with 4 levels- time points) and outcome (between-subject factor with 2levels- win/loss). The dependent variable was computed mean percentage C levels. Covariate was threat/challenge cognitions. Descriptive statistics are presented at the table below (Table 21):

Table 21

Descriptive Statistics for Custom ANCOVA – Post-competition Computed Cortisol Difference by Outcome and Status (Mean, SEM)

	Win		Loss	
	High	Low	High	Low
Immediately post	12.8(7.96)	-3.14(12.51)	1.8(7.2)	-8.97(8.8)
30mins post	24.02(10.14)	24.3(12.7)	11.5(12.9)	8.3(10.7)
1hr post	37.3(11.2)	44.5(10.8)	14.2(17.7)	13.12(13.07)
2hr post	22.6(20.2)	50.1(11.1)	36.2(15.5)	23.4(11.2)

The analysis revealed a non-significant main effect of time, F (2.2, 56.04) = 2.41, p = .09 η^2 = .08, or outcome, F (1, 26) = 1.64, p = .21, η^2 = .06, after adjusting for the effects of threat/challenge cognitions. The covariate, threat/challenge cognitions, was also not significantly related to the C reactivity, F (1, 26) = .03, p = .86, η^2 = .001. The two-way interactions between time and outcome F (2.2, 56.04) = 2.6, p = .08, η^2 = .09, and outcome and threat, F (1, 26) = .17, p = .70, η^2 = .01, were not significant. However, the interaction between time and threat was just not significant, F (2.2, 56.04) = 2.96, p = .056, η^2 = .10. The three-way interaction time*outcome*threat was also not significant, F (2.2, 56.04) = 2.86, p = .06, η^2 = .1. Closer examination of the table of within-subject contrasts revealed that the two-way interaction time* threat appears significant only at a linear level (p = .03), meaning that the group mean C levels increase proportionally over time and depends on perceptions of threat/challenge (Appendix

XL). Notwithstanding, sample size caveats (e.g., false negatives – Type II error, and false positives due to low power) should be considered when the study results are interpreted. Indeed, considering the small sample size, it is very likely that the study did not have enough statistical power to detect higher order interactions (e.g., the three-way interaction).

6.1.6 Impact of Trait Affect and Competition Outcome on C Reactivity

As with T, the same statistical analysis has been performed to identify the impact of the competition outcome and trait affect on C reactivity in percentage. Descriptive statistics from the regression model outlined in Table 22.

Table 22

Descriptive Statistics for Computed Mean Percentage C Change, Competition Outcome and Trait Affect (Mean and SD)

Cortisol Percentage Change	-.84(27.5)
General Negative PANAS-X Score Change	-.13(4.5)
General Positive PANAS-X Score Change	1.9(6.8)
Competition Outcome	.52(.51)

Note N=29

The multiple linear regression analysis yielded significant results: F (3, 25) = 3.48, p = .03, R^2 = .30, R^2Adjusted = .21. Indicating that 21% of the variance in C reactivity is explained by positive and negative trait affects, and competition outcome. Closer examination of the relation coefficients reveals that the predictor variables positive and negative trait affects had a higher impact on C reactivity, (β = .42, t = 2.3, p = .03; β = .38, t = 2.2, p = .03, respectively) compared to competition outcome (β = .33, t = 1.8, p= .09) (Appendix XLI).

6.2 Endocrine Reactivity and Post Status Competition Motivation

Previous research on the reciprocal model of T (Mazur & Booth, 1998) demonstrates that status outcome is considered to trigger post-competition T reactivity. These T fluctuations, however, also produce a subsequent reciprocal effect, where future status seeking behaviours are influenced by T reactivity. For this reason, analysis of the effects of T change (calculated as T pre-competition minus T post-competition) on future motivation to compete were examined in this thesis (please refer back to Figure 16 for easier visualisation of the motivational questionnaire). A linear regression model (assumptions tested and met) was performed with a predictor variable T change and a criterion variable – motivational to complete another financial form, measured as one of the motivational scale's dimensions. The regression model only concentrates on one of the motivational questionnaire dimensions – complete another financial form- in order to utilise similar methodological methods as previous research (e.g., Mehta & Josephs, 2006) and minimise the probability of confounding variables when capturing motivational scores. For instance, the questionnaire dimension – apply for a loan – could not be more susceptible to individual's SES rather than post-competition T reactivity and competition outcome per se.

Table 23

Descriptive Statistics for Testosterone Percentage Change and Motivation

	Mean	SD	N
Testosterone Percentage Change	-.30	16.1	30
Motivation to complete another financial form	1.7	1.4	30

The results revealed that T change does not significantly predict motivation to complete the form again, F (1, 28) = .045, p = .83, R^2 = .002, R^2Adjusted = -.034. The R^2 of .002 indicates that 0.2% of the variance in motivation is explained by the predictor variable (Appendix XLII).

Further two linear regression models were separately run on the losing and the winning cohort (Appendix XLIII). The results, however, were again not significant (p= *.16; p* = .30, respectively). These results were further consolidated by the Spearman correlations between T reactivity and motivational questionnaire dimensions, performed earlier in this chapter.

Chapter 7

Discussion of Experimental Results

7 Introduction

The primary aim of this research was to examine the effects of social defeat on endocrine reactivity and further, to explore the endocrine consequences of defeat and position those effects within the context of subsequent motivational states. In describing the complexity of the relationship between the outcome of status encounters and hormones, a number of studies highlight the importance of cognitive interpretation of events (e.g., Blascovich & Mendes, 2000; Knight & Mehta, 2014) and psychosocial factors (Steptoe & Marmot, 2002). Consequently, the present study considered cognitive appraisals of threat/challenge as moderators in hormonal reactivity towards competition outcome, whilst psychosocial factors previously suggested to relate to the impact of SED on physiological responses – and thereby health (Steptoe & Marmot, 2002; Taylor & Seeman, 1999) – were also explored. The study also adopts a comprehensive salivary sampling (372 samples each for cortisol and testosterone or 744 in total), providing a more detailed, indepth and reliable picture of the dynamic hormone-competition relationship than prior studies have formed (e.g., Mazur et al., 1997 – 160 salivary samples).

This chapter discusses the analyses of the endocrine, psychosocial and cognitive moderators of endocrine response datasets, reported in chapters 4, 5 and 6 respectively. Firstly, the endocrine data analysis, performed in chapter 4, will be discussed allowing to address and answer RQ1 and H1:

RQ1: Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?

H1: Socio-economically disadvantaged population will demonstrate a dissimilar pattern of endocrine response to a social defeat stimulus compared to a higher SES population.

The chapter continues with a discussion of the psychosocial data. Albeit this discussion does not directly address any of the research questions and hypotheses, the section aims to provide an informative answer to whether SES groups differ in psychosocial factors in the predicted way (as outlined in the literature review and methodology sections). Moreover, unpacking potential differences in psychosocial factors between SES groups, assists in shedding some light on the complex relationship between experiences of SED, resilient/protective factors and physiological responses. For this reason, the analyses serve as a building block to addressing and answering RQ2 and H2:

RQ2: Are psychosocial variables related to endocrine reactivity?

H2: Psychosocial variables will be related to endocrine reactivity.

To fully address RQ2 and H2, these psychosocial factors are also analysed in relation to endocrine data in order to identify potential relationships between the factors and endocrine reactivity (as proposed by Steptoe & Marmot, 2002; Taylor & Seeman, 1999), before proceeding with the analysis of the cognitive moderators of endocrine response.

The chapter then proceeds with the moderators of endocrine response section where a discussion of the threat and challenge appraisals is provided. This discussion aims to address and answer the second RQ3 and H3:

RQ3: Does cognitive appraisal of threat/challenge moderate endocrine reactivity?

H3: Cognitive appraisals of threat/challenge will moderate endocrine reactivity in both SES populations.

Subsequently, the chapter engages with a discussion of the behavioural implications of endocrine reactivity to competition, whilst positioning these findings within the broader framework of socioeconomic disadvantage. This directly addresses and endeavours to answer the last RQ4 and H4:

RQ4: Does T reactivity correlate with a reduced motivation to engage socially?

H4: Reduced motivation to engage with subsequent social tasks will positively correlate with a decrease of T reactivity.

Finally, limitations of the study are explored and a short summary of all key findings is provided.

7.1 Endocrine Reactivity to Competition

Prior to looking at the psychosocial data and the moderating effects of threat/challenge cognitions for T and C reactivity towards competition outcome, T and C anticipatory and post-competition responses to the objective competition outcomes (win/loss) will be reviewed. The discussion of the anticipatory response of T and C reviewed in the two baseline and pre-competition sections respectively, aims to verify the validity of the experiment and to examine the credibility of the predicament of the challenge hypothesis/Mazur's biosocial model of status, whilst the discussion of post-competition T and C reactivity directly addresses RQ1 and H1.

7.1.1 Baseline and Pre-competition Testosterone

In order to assess the existence of an anticipatory T response to a social defeat task, salivary T at baseline and pre-competition, was determined (Table 2; Figure 9). The anticipatory response in T prior to the experimental task fails to reach statistical significance and therefore is not evident (pp. 173-174). A perhaps obvious interpretation would be the current research fails to support the first phase of the challenge hypothesis/Mazur's biosocial model of status, that T will rise prior to competition (Archer, 2006, Mazur, 1985). Moreover, this finding fails to provide

support for several earlier studies demonstrating an anticipatory response (e.g., Bateup et al. 2002; Casto et al. 2014; Edwards et al. 2006; Edwards and Kurlander 2010; Filaire et al. 2009; Gonzalez-Bono et al. 1999; Hamilton et al., 2009; Suay et al. 1999). It is important to Note that as this study applies a different statistical analysis and sample regime to previous studies, results should be interpreted with caution.

For instance, this study differed from the majority of previous studies by employing a comprehensive time-matched baseline 48hrs prior to the social defeat task, which allowed for a more insightful exploration of acute T changes compared against time-matched baseline samples. Hence, when the acute T changes appearing on the day of competition are contrasted with the time-matched baseline samples, a slight anticipatory response could be observed between 30mins and immediately prior to the contest. Nevertheless, this slight increase remains rather small and insufficient to render any support for the existence of an anticipatory rise in T prior to competition. Consequently, the current results appear to be in line with the "salivary T un-responsiveness to competition" observed in females in Sharp (2006, p. 118). The lack of anticipatory rise in T in response to a non-physical task in males has also been illustrated by Mazur and colleagues (1997), and Carré and colleagues (2013). Careful consideration of the results of this study are required in this regard. Despite the comprehensive multiple sampling adopted, the low statistical power of the study may also go some way towards accounting for the non-statistically significant T response to competition (i.e., Type II error).

Pre-competition rise in T has also been suggested to appear in relation to a 'fantasy rehearsal' (Kemper, 1990). Considering the number of animal and human studies interested in cognitive means such as watching physical contests, sport events and political elections and their relationships to T reactivity (Bernhardt et al., 1998; Stanton et al., 2009), the importance of 'fantasy rehearsal' for T implications should be considered. The nature of the current experiment did not however, allow (i.e., timewise) any imaginary techniques or visualisation procedures to be executed. Nor did any of the participants report engaging with those. This idea may explain the failure to reject the null hypothesis and thereby the lack of evidence supporting an

anticipatory response in T in the current research. However, it is noteworthy that limitations in sampling regimens and lack of meaningful baselines in previous research, could also impose a statistically artificial anticipatory response in T. Thus, indicating that a response may not necessarily exist and it is not that experimental conditions fail to capture it. Further, cognitive variables, issues of context, environmental factors (i.e., contest setting) and the simultaneous function of C reactivity in relation to anticipatory stress response have also been suggested as alternative explanations for the lack of rise in T prior to contest (Blascovich, 2008; Blascovich & Tomaka, 1996; Carré, 2009; Knight & Mehta, 2014; Mehta & Josephs, 2010; Sapolsky, 1998; Sharp, 2006). Thus, for instance, the dual-hormone hypothesis emphasises the importance of C function for T modification in response to competition, with individuals experiencing high levels of C not displaying a T rise due to the inhibitory function of C over T (Casto et al., 2019; Edwards & Casto, 2013; Mehta & Josephs, 2010; Mehta & Prasad, 2015; Pfattheicher, 2017; Ponzi et al., 2016; Sherman et al., 2016). The section on baseline and pre-competition C will thus focus on the discussion of whether an anticipatory C response was observed and if so, whether this could contribute for the partial explanation of the lack of statistical evidence supporting an anticipatory response in T.

Lastly, it should also be considered that more pronounced relationships could also be observed between T and physical competition (frequently comprising violence and aggression) compared to T response in non-physical domains (Carré et al., 2013; Mazur, 1997; Sapolsky, 1998; Sharp, 2006). This is because T could frequently be implicated in dominance behaviour through an overtly physical component (Sapolsky, 1999b). If this appears to be true and T remains heavily related to the preparation of biological systems to face physical danger/harm, then it could be argued the psychological competition faced in this research does not impose the same degree of danger (quite the opposite, it imposes minimal harm) and thereby hormonal response. That being said, the lack of statistically significant anticipatory T response however, could not be attributed to absence of task salience due to the fact that the financially oriented competition has been carefully methodologically and theoretically considered to facilitate a hormonal response. Whilst participants' self-reported responses also

indicated a high degree of task importance. In the light of these suppositions, what appears to become of interest are the cognitive, psychological and physiological structures upon which T might impact in order to facilitate status attainment in non-physical competitions. Some of these psychosocial and cognitive variables will be reviewed as either related to T reactivity or as moderators of the link between T reactivity to objective competition outcomes, whilst others will be reviewed in the light of post competition T reactivity and subsequent social behaviour (i.e., motivation).

7.1.2 Post Status Competition Testosterone

Post-experimental T reactivity to objective outcome (win/loss) was analysed against levels determined immediately before competition in both SES groups (low/high) (Figure 10, section 4.1.2). The results from the conducted 2 x 2 x 5 ANOVA reveal that the observed patterns of T reactivity across competition states fail to conform with the challenge hypothesis/Mazur's biosocial model of status (i.e., T rises following win and drops following loss). These findings thus might appear contrasting to previous results from several studies of competition, real-world sporting events, and laboratory settings (e.g., Mazur & Booth, 1998; Salvador & Costa, 2009; van Anders & Watson, 2007). Importantly though, the lack of sufficient statistical power in this study (stemming from the small sample size) could also account for the inability to detect small effects or higher order interactions, and perhaps fail to replicate previous findings. Indeed, a non-significant result might be a false negative due to insufficient test power.

Furthermore, rapid fluctuations in T, which do not neatly adhere to the narrative of increasing T following victory/decreasing T following defeat in status encounters illustrate the complexity of hormone dynamics and behaviour/cognition relationships. Previous studies investigating hormone-competition associations, have largely tended to utilize limited single-point measures of hormonal reactivity. Mazur et al., (1997), for example, obtained samples ranging from 1 and 2hrs before and after the competition to immediately before and after. Casto and Edwards (2016) and Sharp (2006) draw attention to the limitations this approach places on our ability to interpret the

complexities of hormonal reactivity and status encounters. And so, whilst the present research findings appear to be in line with findings from Gonzales-Bono and colleagues' (1999), Schultheiss and colleagues' (2005), Mehta and Josephs' (2006), and van Anders and Watson's (2007), where T reactivity does not significantly differ by competition outcome (win/loss), the difference in methodological (i.e., sampling regimes) and analytical approaches renders making straightforward comparisons with existing studies problematic.

Moreover, findings of acute T changes in both win/loss conditions (Figure 10), clearly demonstrate the limitations inherent in determination and interpretation of study results lacking comprehensive sampling regimens or utilising single-point hormone collection (i.e., Mazur et al., 1997 – single 30mins post competition sample). If T data were to be interpreted only at 30mins or 2hrs post-competition, findings would have made a strong case for Mazur and colleagues' (1997) results – i.e., T levels fall in both winners and losers 30mins post competition. However, if results were determined only at immediately after or 1hr post competition, an alternative interpretation would have presented itself. Multi time-point sampling, or lack of, has been put forward as an explanation for why some studies find an increase in T levels following win (i.e., "the winner effect", e.g., Booth et al., 1989; Jiménez et al., 2012; Oliveira et al., 2009), whilst others either do not establish this relationship (e.g., Schultheiss et al., 2005) or find a general "competition effect" on T regardless of match outcome (e.g., Casto et al., 2014; Edwards et al., 2006; Edwards & Kurlander, 2010; Gonzalez-Bono et al., 1999). Echoed by Sharp (2006), and Salvador and colleagues (2003), the authors argue that a more comprehensive examination of hormonal pattering is necessary, whilst previous research findings might require re-interpretation. The current post-competition results address this limitation by shedding some light on the complexity of T reactivity towards objective competition outcomes.

It is clear that endocrine reactivity to social defeat (or hormone status competitions more generally) is complex. For example, in their study of social identity threat, Scheepers and Ellemers (2005) found low and high-status groups exhibit different and complex physiological responses to status encounters. Consequently, in order to tease

out some of this complexity, the conducted 2 x 2 x 5 ANOVA in this study also explored the impact of social grouping (high/low SES) on circulating T levels. Results revealed a statistically significant main effect of socioeconomic status on circulating T levels. Indeed, the overall circulating T levels were higher in the high SES compared to the low SES group (for both competition conditions, Figure 10). Considering the lack of a significant three-way interaction effect between time, competition outcome and SES, however, it could be argued that in relation to T, populations experiencing socioeconomic disadvantage do not demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status – a key finding for this thesis, addressing RQ1 and H1:

> RQ1: Does a socio-economically disadvantaged population demonstrate a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher socioeconomic status?

> H1: Socio-economically disadvantaged population will demonstrate a dissimilar pattern of endocrine response to a social defeat stimulus compared to a higher SES population.

In summary, it could be argued that the present study produces results which fail to reject the null hypothesis, whilst mean T levels for all participants (n=31) appear to be unresponsive to competition outcome (section 4.1.2). Closer examination of the data, however, reveal a wide variety of individual hormonal pattering and differences between socioeconomic status groups (Figure 10). These findings suggest that amongst SES groups and individuals, participants differ in their steroid metabolism and time response to the competition outcome. Resultantly, some individuals might exhibit a rise in T at 30mins post competition win, whilst others might demonstrate the same pattern of endocrine activity at 1hr or 2hrs post competition win. This might implicate a reasonable explanation for the uncharacteristic and widely variable T response amongst competitors, echoed in the high standard error in the mean scores. Individual variability thus could be easily masked by the group mean scores, frequently translating into lack of statistical significance and biological meaningfulness (Booth et

al., 1989; Sharp, 2006). The lack of sufficient statistical power, stemming from the small sample size, could also account for the inability to detect small effects or higher order interactions in this study. For example, it is possible that non-significant result is a false negative due to insufficient test power.

Importantly, whilst the failure to detect significant difference in endocrine response between SES groups, yielded in these results, aligns with some previous findings (e.g., Mehta and Josephs, 2006; van Anders and Watson, 2007), the difference in methodological and analytical approaches, and statistical power renders making straightforward comparisons with existing studies problematic. Furthermore, the demonstrated differences in steroid metabolism and time response to the competition outcome amongst SES groups and individuals (potentially masked by the group mean score), should also be considered. Nevertheless, recent meta-analysis also reports that the existing support for the dual-hormone hypothesis is weak (Dekkers et al., 2019; Grebe, Del Giudice, et al., 2019). Indeed, the analysis suggests substantial heterogeneity in the direction and magnitude of the effect across studies (Casto et al., 2023). Given the ambiguity of research findings, there is a necessity for larger scale and more methodologically rigorous studies, providing greater theoretical clarity and specificity regarding testosterone and cortisol's relationships with status-relevant behaviour. Moreover, this incoherency of research outcomes suggests that potential implications of social defeat upon individuals of low status are indeed worthy of consideration in future research. This is drawn on the argument that despite whether testosterone is reduced following social defeat or status loss, or it is the other way around, individuals with low basal testosterone levels are more easily socially defeated because they appear less threatening to others (Björkqvist, 2001). Indeed, Mazur and Booth (1998), and Mehta and Josephs (2006) argue that T levels are not only influenced by status rise/drop but also produce a reciprocal effect by impacting subsequent status-related behaviours. The behavioural implications of social defeat will be further discussed in the general discussion of this chapter, allowing to address RQ4 and H4.

It is important to note that endocrine response only tells us so much, whilst win/loss are objective outcomes of a subjective experience. Previous research thus outlines the importance of cognitive and physiological variables moderating post-competition T reactivity (Knight & Mehta, 2014; Vermeer et al., 2016). With some studies suggesting implicit power motivation as a moderator of the effects of win/loss on T reactivity (Schultheiss et al., 2005), whilst others emphasise the importance of cognitive and affect in competition response (Salvador & Costa, 2009). Further, biological (basal hormone levels, Mehta & Josephs, 2010) and environmental factors (Carré, 2009; Carré et al., 2006; Fuxjager & Marler, 2010; Oyegbile & Marler, 2005), and personality traits (Maner et al., 2008) have also been proposed as factors modulating this relationship. In this line of reasoning, perceptions of threat/challenge as cognitive appraisals of the contest have been proposed, in this research, to moderate the link between endocrine reactivity and competition outcome, drawn on the Biopsychosocial model of stress (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996; Lazarus & Folkman, 1984). The extent to which those variables act as moderators of this relationship will be discussed in the 'moderators of endocrine response' section of this chapter. Where RQ3 and H3 will be addressed.

Prior to that, however, the baseline, pre- and post-competition cortisol reactivity will be discussed in relation to its susceptibility to objective competition outcomes, and in reconciliation of the T/C literature (outlined in the dual hormone hypothesis by Mehta & Josephs, 2010).

7.1.3 Baseline and Pre-Competition Cortisol

The significant drop in mean cortisol levels (Figure 11) from 9am until 1hr prior to competition (in both baseline and competition days) illustrates a typical, albeit not ubiquitous, circadian rhythm, where C levels are high early in the morning and dropping in the afternoon (Al-Dujaili & Sharp, 2012; Fries et al., 2009; Sharp, 2006). Results from the conducted repeated measures ANOVA (section 4.2.1) reveal a significant main effect of time and day but no time x day interaction effect. Indeed, participants display higher overall levels of circulating C levels on the day of the experiment compared to the baseline day. Whilst the significant difference in time lies

between the first (9am) collection point and the rest (1 hour later, 30 minutes later, and immediately pre-experimental exposure). In the light of those results, it could be argued that the current results fail to provide evidence for an anticipatory response of C in the face of a competition. Once the data is plotted (Figure 11 and plots in Appendix XVIII), however, the graphs illustrate that individuals exhibit some dissimilarities in C response between 30 minutes prior and immediately prior on the day of the competition. Therefore, it could be concluded that because the study is underpowered (due to the small sample size), the research might fail to detect higher order interaction effects and therefore the non-significant interaction result might be a false negative (i.e., Type II error). It should also be noted that there is only so much variance that a repeated measures ANOVA could account for in such a small sample size. Notwithstanding, low power studies (as the present one) also present the risk of false positives (Button et al., 2013), thereby again suggesting that results should be interpreted with caution.

The current research results thus contrast some previous findings indicating a rise in mean C levels (anticipatory response) prior to physical and non-physical competitions (Dickerson & Kemeny, 2004; Hare et al., 2013; Meyer et al., 2015; van Paridon et al., 2017). These conflicting findings could be attributed to a number of causes. Potential false negative results in the current study could stem from differences in analytical strategies and sampling regimes across studies. For that reason, but also the increased risk of false positives, findings should be interpreted with caution. Moreover, as with T limitations in sampling regimes and lack of meaningful baselines in previous research could also impose a statistically artificial anticipatory response in C. This may indicate that a response may not necessarily exist, rather than experimental conditions falsely 'discovering' one.

The lack of statistical evidence supporting a change in acute mean C levels therefore cannot account for the absence of T response prior to the competition. Indeed, these findings fail to provide evidence for consolidating the argument about the inhibitory function of C on T levels, and its relationship to dominance-related behaviours, initially stated in the dual-hormone hypothesis (Mehta & Josephs, 2010). Lastly, mean

circulating C levels might be moderated by cognitive appraisals of the competition task (i.e., threat/challenge appraisals) as documented in the biopsychosocial model of threat and challenge, where cognitive appraisals have implications not only for the performance outcomes but also on the physiological responses to the competition (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996). Sections 7.3.2 and 7.3.3 will reveal the moderating effects of those cognitive variables on endocrine reactivity.

7.1.4 Post Status Competition Cortisol

As with T, this section commences with analysis of the post-experimental C reactivity to objective competition outcome against levels determined immediately before competition in both SES groups (Figure 12, section 4.2.2). Similarly, to the T data, the differences in circulating C levels across SES groups (following competition outcome) did not reach the threshold for statistical significance when analysed in the three-way mixed ANOVA model (pp.182-183). As a result, this stuidy found that SES factor did not have a statistically significant impact on mean C levels; high SES individuals did not differ in their overall C circulating levels compared to low SES. Moreover, the three-way interaction between time, outcome and status was not significant, meaning that high SES winners and losers did not differ in their C reactivity to competition victory/defeat compared to their low SES counterparts. Arguably, in relation to C and on a statistical level, individuals experiencing socioeconomic disadvantage did not demonstrate a dissimilar pattern of endocrine reactivity to social defeat stimulus compared to those of higher socioeconomic status. Thereby, C data is complimenting the T data in addressing RQ1 and H1 in this study, suggesting that the current research fails to reject the null hypothesis.

These results reveal an observed pattern of post-competition C reactivity that stands in contrast with some previous non-human studies (e.g., Carney et al., 2010; Sapolsky, 1999a) and research investigating C reactivity in human competition contexts (Bateup et al., 2002; Jiménez et al., 2012; Stanton et al., 2010). Indeed, the lack of evidence to support the hypothesised significant differences between SES cohorts across competition outcome and time in the present research appear to differ from some previous studies demonstrating dissimilarities in glucocorticoid response to laboratory

psychosocial stressors between SES groups (e.g., Adler & Ostrove, 1999; Adler et al., 2000; Fiocco et al., 2007; Kristenson et al., 1998). For instance, Rosmond and Björntorp (2000b) report that low SES individuals not only display a cortisol hyperactivity compared to the high SES group but also reduce cortisol concentrations less effectively, post-competition. Results from the current study, however, differ from Rosmond and Björntorp's (2000b) findings. And yet, the current research appears to be in line with several studies demonstrating lack of evidence to reject the null relationship between cortisol levels and competition outcome (e.g., Hasegawa-Ohira et al., 2011; Oliveira et al., 2009) or even the reversed relationship – higher glucocorticoid response in winners (Suay et al., 1999). The author presents four possible explanations as to why differences in findings exist.

Firstly, the current study applies a different analytical strategy and sampling regime to previous research. Secondly, and of great importance, the research is a feasibility study therefore lacking sufficient power to detect higher order interactions and small effects and running the risk of producing false negative results. Thirdly, available evidence across multiple studies should not necessarily be based on a simple tally of whether studies reported significant effects or not. Some studies may be underpowered and thus not report significant effects (as is the case in the present research), whilst still showing effects consistent with predictions. Meta-analyses are one potential solution for this interpretative challenge, showing the 'true' magnitude of effect sizes and considering sample sizes. Indeed, a more evidenced synthesis across studies is needed to have a more accurate estimate of the effects. In conjunction, further work is needed to identify what the smallest effect size of interest would be. However, even this approach does not come without its limitations due to potential publication bias and bias in collection of unpublished studies. Similarly, issues around confirmation bias in past and this research should also be considered. Fourthly and finally, as with T, inconsistencies in findings may suggest that other cognitive factors also moderate the dynamic relationship between glucocorticoid response and competition outcome (Knight & Mehta, 2014).

For instance, Wirth and colleagues' (2006) findings support the importance of implicit power motives for this link by illustrating that competition loss is associated with higher glucocorticoid response only when individuals are motivated to achieve high status (i.e., individuals with high power motive). Simultaneously though, individuals who do not express an interest in achieving a higher status rank might perceive high status as a stressful experience, thereby exhibiting a high anticipatory glucocorticoid response (Wirth et al., 2006). In this line of reasoning, the current post-competition C results, just like post-competition T reactivity, might be associated with threat/challenge moderators between competition status and glucocorticoid response. The extent to which threat/challenge appraisals moderate cortisol reactivity to competition outcome will be further discussed in the next sections (e.g., section 7.3.3).

In summary, C reactivity post-competition further supports T findings, illustrating that in this population the low SES group do not display a dissimilar pattern of endocrine reactivity to a social defeat stimulus compared to those of higher SES (Figure 12). Notwithstanding this, results should be interpreted with caution due to various limitations of previous bio-behavioural literature and the present research, such as lack of sufficient power, dichotomization of results based on p values, differences in sampling regimes and analytical strategies, and lack of evidenced synthesis across studies showing what a more accurate estimate of the effects might be. One potential way of mitigating some of those issues (i.e., dichotomization of results based on p values) is by plotting data. Indeed, Figure 12 illustrates potential differences across SES and competition outcome groups which may not have reached the threshold for significance due to various reasons (e.g., insufficient study power) but remain important. It is important to note that those differences are not simply based on visual inspection of data, which could sometimes be misleading, but are also evident upon evaluation of raw endocrine parameters (i.e., differences in raw cortisol testosterone levels between groups). This once again emphasises the tensions between tests of statistical significance and biological consequence. Indeed, considered within the broader framework of SED, these results are important; not only in reference to the biological implications of living in disadvantage (chronically elevated cortisol levels) but with regards to the creation and design of policies which aim to eradicate

health inequalities, which should not simply be informed by research providing statistically significant results. Social and public policy which strives for long-term positive effects on health, cognition, and behaviour should also take into consideration the issues of conflicting evidence based on dichotomization of study results.

The implications of these conclusions are important because they indicate potential differing life-experience related modulations of the stress response. That is, the effects of glucocorticoid reactivity might be insignificant for the organism in the short term, or if exposure to stressors is infrequent and short. However, in the case of repeated exposure, an accumulative effect might occur, resulting in chronically elevated C levels, augmented or reduced C reactivity to stressors, subsequent slow and sluggish recovery, and eventually an inability of the individual to return to homeostasis (Hellhammer et al., 2004; Sapolsky, 2004). These chronically elevated levels of cortisol and augmented/reduced cortisol reactivity to stressors, have very definite cognitive, behavioural and health implications, being associated with long-term complications such as higher morbidity rates, shorter life expectancy, greater percentage of life spent in ill health, and lower educational and occupational attainment (Finch & Crimmins, 2004; McEwen & McEwen, 2017; Rodrigues et al., 2009). These outcomes are described in the literature review, where the implications of chronically elevated C are not only discussed as consequences but also suggested to partially account for the antecedents of SED.

7.2 Psychological Data

Psychosocial data have been analysed in relation to socioeconomic status in order to investigate any potential differences in sense of coherence, attributional style, personality style, and threat/challenge cognition scores between the groups. Following this, the data have been used to analyse potential relationships between endocrine response to competition, psychological variables, and objective competition outcome (win/loss). By doing so, this section aims to evaluate the links and impact of all aforementioned psychosocial factors on endocrine response which would allow to

address and answer RQ2 and H2. Further, the discussion of psychosocial data analysis allows to investigate whether SES groups differ in psychosocial factors in the predicted way (e.g., differences in personality traits, SoC and ASQ- please refer back to the literature review, sections 2.5.2; 2.5.5; 2.3.4), and facilitates later discussions aiming to directly address and answer the rest of the RQs and Hs. The evaluation of psychosocial factors in relation to endocrine data also allows to establish the strongest relationships, which could be used as statistical foundations for the previously suggested links between physiological responses and psychosocial factors. This would allow consideration of the extent to which psychosocial factors account for the complex relationship between experiences of SED, hormonal response and health.

7.2.1 Sense of Coherence

Sense of coherence (SoC) has been strongly associated with mental health, perceived health status, health-related behaviours, psychopathology, crime and anxiety (Antonovsky, 1987; Coward, 1996; Edwards & Besseling, 2001; Eriksson & Lindström, 2005; Eriksson & Lindström, 2007; Eriksson & Lindström, 2006; Flensborg-Madsen et al., 2005; Gibson, 2003). The impact of SoC also extends to particular physical illnesses and health outcomes (e.g., diabetes and mortality rates – Agardh et al., 2002; Ray et al., 2003; Ristner et al., 2000), albeit this relationship is not as straightforward as with the aforementioned variables (Coward, 1996). SoC has also been proposed as a potential explanatory framework for the excessive mortality and health disparities in Scotland (Scottish Government, 2008, 2009). Further, there is not only an explicit link between SoC and learned helplessness/hopelessness (i.e., arguably, the behavioural implications of social defeat), but also protective/resilience factors, where strong SoC is associated with greater generalised resistance resources (GRRs – described in section 2.5.5 of the literature review chapter) towards negative life events (Antonovsky, 1987).

In the light of these findings, this study uses the SoC-29 scale to: 1) measure any potential differences in SoC scores between SES groups; 2) indicate any potential differences in SoC between the groups perceiving the competition as either threatening or challenging (i.e., whether SoC has any implications for threat/challenge

appraisals of the competition) and 3) to indicate whether SoC relate in any way to the endocrine response towards competition victory/defeat.

Firstly, scores of the SoC scale have been analysed in relation to SES (Figure 13) in order to identify any potential differences in a concept presumably important for building resilience and further contributing to the cognitive appraisals of status competition related environmental threats between SES groups. Findings from the current study revealed non statistically significant differences between the SES groups on the linear combination of the SoC subscales (Figure 13). Closer examination of the sub-domains, however, revealed that the two groups differ in scores on some of the sub-scales. For example, individuals in the low SES group displayed lower scores in the sub-scales *perceived understanding of existence* and, most importantly, *global sense of coherence*, thus suggesting weaker SoC. These findings are in line with the results obtained by Packard and colleagues (2012) on a Glasgow population, where individuals from more deprived backgrounds displayed lower overall SoC-13 (a shorter version of the original SoC scale) scores compared to the affluent group. Low SoC scores have also been associated with particular population subgroups, i.e., substance abusers, unemployed individuals with psychopathologies, and single parents (Berg & Brevik, 1998; Eklund et al., 2001; Gottlieb, 1998); all of which are experiences frequently associated with living in socioeconomic disadvantage (Haushofer, 2011). Further, SoC has been associated with other measures of SES (Eriksson & Lindström, 2006; Ing & Reutter, 2003; Kalimo & Vuori, 1990; Larsson & Kallenberg, 1996; Volanen et al., 2004), strengthening the credibility of the scale as an indicator of health disparities (Eriksson & Lindström, 2006).

Walsh and colleagues (2014), however, display slightly different findings observed in the Glaswegian population, where high SoC remains associated with lower likelihoods of self-reported bad/very bad health outcomes. The authors suggest two potential explanations for the paradox (high SoC but also relatively worse mortality rates). Either the utilised version of the SoC scale (i.e., SoC-13) in Walsh and colleagues (2014) does not fully encompass the concept of SoC (as further argued by Flensborg-Madsen et al., 2005) and is susceptible to cultural influences in self-reporting (Walsh et al.,

2014); or the measure might appear to have a greater credibility as a measure of social identity rather than as a predictor of mortality rates, health disparities and the associated protective factors. Potential support for the latter explanation comes from research on Glasgow population associating the community with relatively strong sense of identity (Perchard, 2013; Richards, 2004). The absence of significant difference between SES groups on a linear combination of the SoC sub-domains, within the Glaswegian population investigated in this study, echoes the established paradox by Walsh and colleagues (2014). These overlapping findings within the same population group poses a significant challenge to part of the "Salutogenesis" theory. That which establishes a link between strong SoC and generalized resistance resources but overlooks the instrument as a potential measure of social identity.

Antonovsky (1987), and Denton and colleagues (2004), further propose that individuals high on SoC are more likely to perceive events as challenging rather than threatening. Indeed, stronger SoC has been associated with a more effective and suitable tension management system to cope with stressors (Antonovsky, 1987). In the light of this argument, the variable has been analysed in relation to threat/challenge appraisals of the experimental task (i.e., competition). The sample size, thus, has been split into two groups, based on individuals' perception of the competition – threatening, or challenging condition (Figure 14). Results yielded non-significant differences between the group appraising the competition as threatening and those perceiving it as challenging. Based on Antonovsky's (1979) argument it was further hypothesised that the group appraising the event as more threatening will also be the group scoring lower on SoC scale. However, closer examination of the findings demonstrated that in contrast to the hypothesised results, the group that appraised the event as more threatening surprisingly displayed higher scores on two of the SoC sub-domains (i.e., *perceived ability to control life events* and *global sense of coherence*) compared to the group that appraised the competition as challenging (Figure 14). Therefore, the direction of the current results does not align with the hypothesised direction of the relationship between SoC and threat/challenge conditions proposed by Antonovsky (1987), and Denton and colleagues (2004).

Potential explanation for the controversy (i.e., why the group appraising the event as more threatening also scores higher on the SoC subdomain- *perceived ability to control life events*) comes directly from Antovonsky. Indeed, the author argues that strong SoC does not necessarily imply that individuals are in control of their life (Antonovsky, 1979). More recently, Flensborg-Madsen and colleagues (2005) further propose that predictability, associated with control over life, should be removed from the scale since unpredictability is not by all means inherently unhealthy. These arguments could explain the reversed relationship between perceptions of threat and higher SoC. However, this cannot account for the stronger *global sense of coherence* displayed by the group appraising the event as threatening and not challenging. Hence, it could be argued that these findings might provide further support for the earlier argument about the questionable credibility of SoC as a measure of generalized resistance resources. It should be noted, however, that results should be interpreted with caution due to the increased risk of false positive and false negative results, stemming from multiple comparisons (and no applied corrections for those such as a Bonferroni correction), and insufficient study power, respectively. Additionally, whilst corrections such as Bonferroni have not been applied, therefore increasing the risk of Type I error in this analysis, such techniques may well be considered conservative and indeed inflate the rate of false negatives. For that reason, one might consider alternative approaches such as controlling for the false discovery rate where the overall rate of false positives can be maintained without inflating the rate of false negatives unnecessarily.

Finally, the SoC subscales (i.e., perceived understanding of existence, perceived control, and global sense of coherence) on which threat and challenge, and low and high SES groups differed significantly, have been further included in a statistical model evaluating the relationship between those subscales and endocrine reactivity (T and C) to competition outcome (Table 12 and 18). Results did not discover any significant relationships between SoC and endocrine reactivity towards victory/defeat, thereby imposing that the variable does not in any straightforward manner relate to the link between hormonal changes and competition outcome (this partially addresses and answers RQ2 and H2, by failing to reject the null hypothesis).

7.2.2 Attributional Style

Analysis of the attributional style questionnaire (ASQ) has been used to identify whether scores between SES groups vary, and thereby, establish any potential relationship between experiences of socioeconomic disadvantage, perceived control and learned helplessness/hopelessness. As previously discussed in section 2.3.4 of Chapter 2, attributional style lays at the foundations of the three concepts – learned helplessness, hopelessness depression and locus of control – as part of the reformulated "learned helplessness" theory (Abramson et al., 1978). Learned helplessness/hopelessness are recognised as the brain's default state when an individual is unable to exhibit control over reoccurring uncontrollable life events and fails to learn to escape from those circumstances (Maier & Seligman, 2016). Importantly, socioeconomic disadvantage is frequently associated with more stressful, uncontrollable and unavoidable negative life events (Dohrenwend & Dohrenwend, 1970; Kessler, 1979; Kim et al., 2018). Arguably then, experiences of socioeconomic disadvantage could potentially yield learned helplessness/hopelessness due to a high degree of uncontrollability of events and the lack of control low SES groups often experience (Marmot, 2004, 2015). Subsequently, resulting in default passive behaviour in response to aversive stimuli and failure to learn to escape (Maier & Seligman, 2016). Considering the well-established link between learned helplessness and uncontrollability of negative life outcomes in animal and human literature (Maier & Seligman, 2016; please also refer to section 2.3.4 for an overview of those studies) within laboratory and non-laboratory settings, the current study explored whether this relationship could be expanded to a broader framework of SES. To my knowledge, there are no other studies exploring this relationship.

Results from the ASQ (Figure 15), however, did not yield any significant differences between the SES groups, on a linear combination of the ASQ subdomains (i.e., *Composite Negative Attributional Style*; *Composite Positive Attributional Style*; *Composite Positive minus Composite Negative*, *Hopelessness*, and *Hopefulness*). This means that, overall, the low SES group did not exhibit higher scores on the subscales associated with internal-global-stable negative attributional style, and thereby

exhibited no higher overall score for learned helplessness/hopelessness and lack of control compared to their more affluent counterparts. Closer examination of the results though, allowed the identification of a significant difference between the groups in the mean scores of one of the ASQ sub-domains – *hopefulness*, with high SES individuals scoring higher in this domain compared to the low SES group. Generally, these findings appear to differ from the hypothesised link between low SES and higher risk of learned helplessness/hopelessness and lack of control. However, the significant difference in hopefulness scores, a central for this thesis concept, between SES groups appear to support, at least to some extent, previous research findings associating learned helplessness with uncontrollability of negative life outcomes (Fosco & Geer, 1971; Gatchel & Proctor, 1976; Glass & Singer, 1972; Hiroto, 1974; Hiroto & Seligman, 1975). By demonstrating that the low SES group in this study, which may arguably face more frequent stressors and have less control over negative life events (Kim et al., 2018; Marmot, 2004), appears to be less hopeful than the high SES group. A significant limitation of these findings, however, remains the fact that in the current study, low SES has not been empirically associated with higher number of stressors and uncontrollable life events compared to their more affluent counterparts. Thus, limiting the credibility of this argument to hypothetical grounds, whilst emphasising the importance of future research.

It is worth noting here though, that the mean score difference between the SES groups On the reversed subscale for *hopefulness – hopelessness* – was statistically non-significant, implying a certain degree of statistical artefact for the aforementioned results (Figure 13). Importantly, the scores in the *hopelessness* subdomain were also reversed to those of the *hopefulness* subscale (low SES scoring lower on the hopelessness scale compared to high SES group) making the results even more challenging to interpret. Interestingly, mean score differences between SES groups were also very close to significance on another ASQ subscale, the *Composite Positive minus Composite Negative Attributional Style* (CPCN), - a score indicating how positively or negatively individuals react to all events – thus potentially contributing to the credibility of the *hopefulness* subscale results. Lastly, it is important to consider that most of the participants, classified as of low SES, have been going through a series

of mindfulness courses and interventions. Techniques, unquestionably linked to positive psychology focused on the restoration of control and the brain's hope circuits (Seligman, 1991).

Finally, results of the ASQ were analysed in relation to endocrine response. And more precisely, whether any of the two ASQ subdomains (*CPCN* and *hopefulness*) appear to correlate with the endocrine response towards competition outcome. This analysis has been considered and performed on the basis of an arguable link between learned helplessness and HPA axis activation (Camacho, 2012; Maier & Seligman, 2016). More specifically, in their recent work, Maier and Seligman (2016) review previous literature reporting a link between various neural circuits and learned helplessness, but also between learned helplessness, SES and disease outcome. The current study did not find any significant correlations between the ASQ subdomains – *hopefulness* and *CPCN* – and endocrine (T and C) reactivity to competition outcome (Table 12 and 18). Consequently, the findings fail to provide evidence in support of H2. This, however, appear to be in line with the conclusion made by Maier & Seligman (2016) that learned helplessness, and specifically control, are not in any direct manner related to the HPA axis response to stress. Nevertheless, as with the SoC scale, the conducted multiple comparisons and insufficient power of the study could introduce a degree of uncertainty regarding the interpretation of the present study's results. Additionally, as earlier pointed in this discussion chapter (p.231), firm conclusions based on dichotomously compared findings (i.e., significant or non-significant results) drawn from previous research and the current study, should not be made. This thesis thus suggests some contrasts and similarities amongst previous findings and the current research, however also cautiously accounts for the concern that low statistical power studies of this sort have a reduced chance of detecting a true effect. Low powered studies also reduce the likelihood that a statistically significant result reflects a true effect.

7.2.3 Personality Style

Personality style has been suggested as a potential filter of the experiences of status rank (Sapolsky, 2017). Meaning that the experiences of SED might be, to a certain

extent, regulated by personality. This, of course, also appears applicable for the experiences of affluence (Afshar et al., 2015; Pereira-Morales et al., 2018; Sapolsky, 2004, 2017). More particularly, this echoes in the role of personality in the status/health relationship, where personality acts as a buffer of the influences of stressors on health outcomes (Cohen & Edwards, 1989). Arguably then, individuals experiencing higher SES, but characterised with particular maladaptive personality traits (i.e., low scores on openness to novelty experiences and extraversion, and high neuroticism), display less healthy outcomes regardless of their more affluent status (Bolger & Schilling, 1991; Sapolsky, 2004, 2017). The opposite could also be true, with adaptive personality traits (e.g., high extraversion and conscientiousness) acting as a buffer of the experiences of SED (Vollrath & Torgersen, 2000).

Further, personality traits (e.g., trait anxiety) also appear to have a very prominent role in the relationship between status win/loss and steroid reactivity (i.e., T reactivity), frequently illustrated in competition settings, accurately representing naturalistic dominance contests (Maner et al., 2008; Norman et al., 2015). In support, Schultheiss and colleagues (1999) further argue that personality and not simply competition outcome might be the key determinant of who is and who is not displaying an endocrine response towards status encounter. Considering the aforementioned findings and arguments, the present study aimed to shed some light on the link between personality and endocrine response to competition within low and high SES groups, thereby addressing RQ2 and H2.

Firstly, the findings were analysed in relation to whether there are any significant differences in personality traits between the two high and low SES groups (Figure 16). Results displayed that the two groups were statistically different on a linear combination of the five NEO-FFI- 3 personality traits explored in this study (i.e., *neuroticism*, *extraversion*, *openness to experiences*, *agreeableness*, and *conscientiousness*). Closer examination of the results displayed that the statistically significant differences between the SES groups lie in the scores of *neuroticism* and *conscientiousness*. With low SES individuals displaying higher scores for *neuroticism* and lower scores for *conscientiousness* compared to their more affluent counterparts

(Figure 16). These results appear to be in line with the bulk of previous literature documenting the same association between SES, *conscientiousness*, and *neuroticism* (Jonassaint et al., 2011; Lahey, 2009). Furthermore, Damian and colleagues (2015) propose that *conscientiousness* relate to individual SES, over the lifespan. This relates to individuals experiencing SED, and who score high on *conscientiousness*, being more likely to experience socioeconomic mobility in later life compared to individuals experiencing the same socioeconomic background but displaying low *conscientiousness*. Potential explanation for this relationship comes from Ludwig and colleagues (2019) and Roberts and colleagues (2007) suggesting that high consciousness within low SES groups frequently encompass higher tendency for long-term planning, but also encouragement of selective behaviours during early life translating into higher professional achievements, respectively. Contrary, the lack of recourses, intrinsically associated with the experiences of SED, have been suggested to account for higher anxiety and depression, subsequently translating into higher neuroticism, in lower SES cohorts (Santiago et al.,2011). Finally, it has also been argued that higher neuroticism scores not only further increase the risk of mood disorders but also the risk of all-cause mortality rates amongst groups experiencing SED (Chapman et al., 2009; Jokela et al., 2011).

The findings for *openness to experiences*, *agreeableness* and *extraversion* also did not reach the threshold for statistical significance. However, the trend for *openness to experiences* (higher scores for the higher SES group) appear to be in line with previous research findings (Jonassaint et al., 2011). The results for *extraversion* and *agreeableness*, however, stand in contrast to background literature associating higher SES groups with higher scores on these two domains (Chapman et al., 2009; Jonassaint et al., 2011). These reversed findings could be interpreted in the light of two arguments. Firstly, the current low SES group went through a number of positive psychology interventions (i.e., mindfulness), proven to have an impact on personality traits (e.g., reducing neuroticism and thereby psychological distress), on coping strategies, and emotional regulation (Hanley et al., 2019). Secondly, there are some very definite relationships between SES and personality traits such as neuroticism and consciousness, however, research on the link between SES and other personality

domains remains equivocal (Hughes et al., 2021). Thirdly, concerns around the implications of low statistical power on detecting true effects (i.e., the present study is underpowered and therefore increases the risk of false negatives) and the low likelihood that a statistically significant result reflects a true effect (in small sample studies of this sort) should also be considered when comparing and contrasting findings. This principle is applicable, however, to previous research utilising small or non-representative samples.

Lastly, the current findings fail to provide further evidence for the argument that personality is related to endocrine response (Maner et al., 2008) and thereby links to the experiences of SED and health outcomes (Cohen and Edwards, 1989). In this study, even personality traits such as *consciousness* and *neuroticism*, demonstrating significant differences across SES, did not correlate with T and C reactivity to competition outcome (Table 13 and 19). Once again, considering the low power of the study and the conducted multiple comparisons, the inflated rates of Type I and II errors should be taken into consideration interpreting results, and drawing comparions to previous findings.

7.2.4 Threat/Challenge Appraisals

Another key finding for this thesis lies in the analyses of the cognitive appraisals (threat/challenge) of the status competition task. In the first instance, and in this subsection, a discussion of the analysis of cognitive appraisals by social grouping (SES) will be provided. Whilst in the moderators of endocrine response subsections, the discussion will be dedicated to the analysis of these threat/challenge appraisals as moderators of endocrine reactivity to competition outcome.

As stated in the Chapter 3 (section 3.6.2), a financially oriented competition task has been carefully, methodologically and theoretically, chosen to facilitate a framing effect by producing threat cognitions. This has been drawn from the theoretical position of Mullainathan and Shafir (2013), arguing that individuals experiencing SED and lacking financial resources might realistically be expected to experience bandwidth overload when engaging with finance-related tasks. This argument was further supported by

evidence illustrating that lower SES groups frequently experience higher levels of stress and perceive more events as threatening rather than challenging compared to higher SES populations (Kim et al., 2018, Marmot, 2004; Scheepers, 2009). Thereby, it was hypothesised that threat cognitions would be predominantly associated with the lower SES population in this study, although this was by no means a certainty or necessity for the relationship to be necessarily present (Marmot, 2004). Illustrating the potentially fragile nature of this proposed relationship, it is worth mentioning here that Scheepers (2009) notes perceptions of threat only prevail amongst lower SES groups if individuals are part of a stable hierarchical group. In case of unstable hierarchical social relations, it is often the higher status groups experiencing more threats to their current status rank compared to lower status groups.

This argument appears almost parallel to the observations in animal species within stable and unstable dominance hierarchies (Sapolsky, 2005). There, low ranked animals display higher levels of stress-related physiology in stable hierarchical systems but are superseded by their higher ranking peers when those structures become unstable. As noted earlier, however, not all individuals experiencing SED or of a low SES would also experience elevated levels of stress and increased number of threat cognitions compared to high SES groups (Marmot, 2004; Sapolsky, 2017). This stems from the fact that multiple factors such as resilience, subjective experiences of SES, and type of hierarchical systems individuals participate in, play a critical role in the formation of those cognitions and thus for the implications of SED for health (Adler et al., 2000; Scheepers & Ellemers, 2005; Wilkinson & Pickett, 2018).

Findings from this study appear to support the hypothesised results by demonstrating a statistically significant difference between SES groups in their scores of threat/challenge appraisals of the experimental task. As such, the low SES group appraised the competition as more threatening, whilst the high SES group perceived the event as more challenging (Figure 17). Whilst an examination of the number of daily threats experienced by lower SES groups lays outwith the scope of this thesis, the current results yield significant differences between SES populations and thereby arguably provide some evidence for Mullainathan and Shafir's (2013) argument.

Notwithstanding this, results should be carefully interpreted whilst accounting for the study's low power which leads to an increase in likelihood of identifying a statistically significant result that does not reflect a true effect.

Importantly though, these cognitions impact the way individuals respond to stress (Blascovich & Mendes, 2000, please refer to section 2.7.4 of the literature review for a more detailed discussion). This raises the important question how threat/challenge appraisals moderate endocrine reactivity to an environmental stressor – such as a status competition in this case. Before testing the moderating power of threat/challenge cognitions for endocrine reactivity, the appraisals, similarly to the previously explored psychosocial variables, were inserted into correlation models evaluating the relationships between the cognitions and endocrine reactivity (Table 13 and 19).

Results demonstrated that out of all psychosocial variables explored in this study, cognitive appraisals appeared to have the highest correlation with T post-competition reactivity (Table 13), whilst in the correlation model of C reactivity, threat/challenge cognitions were only dominated by another variable – objective SES (Table 19). Based on these results, it could be argued that in the present study, cognitive appraisals might display the strongest relationship to endocrine response, but only for T and not C. Nevertheless, threat/challenge appraisals remain the second strongest predictor after objective SES in C reactivity. In seeking to further unpack the complex relationships between threat/challenge cognitions, SES and endocrine response, socioeconomic status and cognitive appraisals have been further included into two separate regression models, where the appraisals were tested as potential predictors of the endocrine response (T and C) towards competition outcome (Table 14 and 20). Results from both models yielded non-significant results. Interestingly, closer examination of C reactivity data revealed that the model remained non-significant only when both variables threat/challenge cognitions and SES were included in the regression. Once threat/challenge appraisals were removed from the statistical model, SES became a significant predictor of C reactivity towards competition outcome.

Similar closer examination of the T regression model was performed. The results once again indicated that SES was a stronger predictor of T reactivity compared to threat/challenge cognitions. The results of the regression, however, unlike with C, remained insignificant even after controlling for the threat/challenge cognitions variable. Even though initially challenging to interpret, these findings only consolidate the hypothesise that threat/challenge appraisals might not directly impact upon endocrine response but rather moderate the link between competition outcome and endocrine reactivity. For that reason, threat/challenge appraisals were evaluated as moderators of the link between endocrine response and competition outcome via custom ANCOVA models. These analyses sufficiently address RQ3 and H3. Additionally, of significant interest, remains the question whether these cognitions will have a stronger moderating effect for T compared to C, and whether indeed objective SES is of greater importance for C reactivity rather than threat/challenge appraisals. The discussions provided in the future moderators of endocrine response subsections aim to shed some light on these issues.

Lastly, all of the aforementioned correlations and regressions should be interpreted with caution considering the inflated risk of Type I error (due to multiple comparisons) and false negatives (Type II error) as a result of the study's low power.

7.2.5 Task Workload and Threat/Challenge Appraisals

Considering the importance of threat/challenge appraisals for stress response, and endocrine response to status competition more particularly, the study aimed to better understand how these cognitions were constructed and the potential variables that might impact this construction. For this reason, similarly to SoC, another psychological variable (task workload) has been explored as a potential contributing factor for the construction of these cognitive appraisals. Task workload was measured on the NASA-TLX Raw questionnaire, examining how physically and mentally demanding the individuals perceive the competition task. The relationships between threat/challenge cognitions and task workload were explored in a correlation analysis (Table 11). The produced results were then used to analyse whether a relationship between threat/challenge appraisals and task workload (physical/mental task

demand) exists. Results yielded a non-statistically significant correlation between task workload and threat/challenge appraisals (Table 11). As a result, the study fails to provide evidence for SoC and Task Workload as contributing factors towards the formation of threat/challenge appraisals. Considering the low power of this study, however, the current results might be false negatives. For that reason, the relationship between SoC, Task Workload and cognitive appraisals should be explored on a larger scale. Additionally, these findings suggest that there might be other potential psychosocial factors related to the formation of threat/challenge cognitions, however, they appear to be beyond the scope of this study. Future research thus needs to explore other psychosocial factors, over and above SoC and Task Workload, that might potentially contribute towards the formation of threat/challenge cognitions.

7.2.6 Trait Affect and Competition Outcome

Finally, the present research was concerned with the psychological variable trait affect and its potential role in endocrine response to competition outcome (Gladue et al., 1989; Josephs et al., 2006; Mazur & Lamb, 1980). In contrast to the previously explored psychosocial variables, trait affect was not investigated in relation to SES but only as a variable potentially affecting endocrine reactivity to competition outcome. This is because, in the present research, trait affect has not been hypothesised to differ across SES groups, but to be amongst the variables related to or impacting endocrine response, as previously suggested by multiple authors (e.g., Gladue et al., 1989; Knight & Mehta, 2014; Sharp, 2006). Indeed, Mazur and Lamb (1980) argue that: "… if an individual with rising status felt emotional elation, his testosterone would rise; but if the change in status were not accompanied by a change in mood, there would be little change in testosterone." (p.237). This has been echoed by Gladue and colleagues (1989) suggesting that "…a status change with no mood change would have no associated endocrine change." (p.411).

The chosen research designs and strategies of these authors' studies, however, do not come without limitations. For instance, albeit Mazur and Lamb's (1980) research findings appear to support their aforementioned statement, results should be interpreted with caution considering the choice of sampling strategy (i.e., blood

samples, issues of which have been reviewed in the sections 3.2.1, 3.2.2, and 3.2.3) and adopted procedure (single point mood evaluation of participants, 48hrs after the event). More specifically, the magnitude of male T fluctuations and late mood evaluation in their participants, imply a degree of uncertainty about the validity of the established relationship. Gladue and colleagues (1989) also refer to their results as ambiguous due to design limitations (mood has only been accessed post-competition and not prior to it), thereby limiting opportunities to identify mood changes as a result of the experimental event. Similarly to Gladue and colleagues (1989), Josephs and colleagues' (2006) methodology also comprises limitations related to the utilised sample regime (one-post competition salivary sample) and mood tests. Conclusively, albeit previous research generally supporting the relationship between T and mood (where T levels increase in conjunction with mood), the described methodological and design limitations of the studies necessarily imply serious concerns about the validity of their results.

In contrast, more recent research (e.g., Sharp, 2006; Zillioli & Watson, 2013) adopts comprehensive sampling regimes and mood evaluation procedure. In particular, Sharp (2006) applies a multiple-point sampling regime allowing the researcher to closely investigate endocrine changes emerging as a result of the experimental task. The author, however, evaluates participants' mood only once during the entire experiment. Similarly, Zillioli & Watson (2013), utilise a one-point, post-competition evaluation of mood, and only two (pre and post competition) salivary collection points to capture the complex dynamic nature of endocrine response. Whilst Sharp's (2006) results do not support the relationship between T and mood, Zillioli & Watson's (2013) findings further support it. The current study, thus, aimed to further shed some light on the T/mood relationship by adopting a comprehensive sampling regime and mood evaluation procedure (endocrine response was measure once before the competition and four times post, whilst mood was measured immediately before and after it).

In attempt to measure the relationship between T and mood, a multiple linear regression was utilised, following the example of previous studies (e.g., Sharp, 2006; Zillioli & Watson, 2013). In line with Sharp's (2006) findings, the present results

demonstrated that positive and negative trait affects, and competition outcome do not significantly predict T reactivity (Chapter 6, section 6.1.3). Closer examination of the regression model, however, also revealed that the strongest predictor of T reactivity (yet not significant) was negative trait affect. Because of the lack of any statistically significant relationship between the variables competition outcome and T reactivity, one might argue that competition outcome should be excluded from the regression model and new statistical evaluation of the relationship between T reactivity and negative and positive trait affect should be performed. Indeed, the researcher took this argument into consideration and explored the relationship between trait affect and T reactivity without including competition outcome into the model (section 6.1.4). The results once again yielded non-significant relationship between negative and positive trait affects and post-competition T reactivity.

Negative and Positive trait affects was also examined in relation to post competition C reactivity (Chapter 6, section 6.1.6). The results yielded that positive and negative trait affects significantly predict C reactivity, with negative and positive affects being stronger predictors compared to competition outcome, in contrast to T data. It is worth noting here however that, as with the T data, sample size caveats (and thereby increased risk of false positives) should also be considered in the interpretation of those results.

Potential explanations for the difference between the current findings and previous research supporting the relationship between T and mood, lies in the choices of psychometric tools utilised to measure mood in the studies, different sample regimes, and potential risk of false negative and positive results due to low study power (as in the present study). For instance, Sharp (2006) utilised POMS to measure mood, whilst Gladue and colleagues (1989) applied the Multiple Affect Adjective Check List (MAACL). The current research, however, adopted PANAS-X instrument due to its stronger discriminant validity (Zillioli & Watson, 2013). Moreover, it has also been suggested that PANAS-X not only measures state mood but also encompasses individual trait variability (Zillioli & Watson, 2013). This, arguably, provides another explanation for the potential differences between previous findings and the current

research. Nevertheless, the overall current findings, appear to contrast Zillioli and Watson's (2013) results and other studies (e.g., Maner et al., 2008; Norman et al., 2015) who also utilised PANAS-X to establish a relationship between positive/negative trait affect and endocrine reactivity (particularly T). In conclusion, the limitations relating to statistical power and the likelihood of false positives and false negatives, inflated Type I error due to multiple comparisons, the lack of consistency in analytical strategies, and inconsistency in methodologies renders comparisons to existing studies problematic and complex.

7.3 Moderators of Endocrine Response

As already demonstrated the strongest correlations between psychosocial data and endocrine reactivity towards competition outcome appear to be between threat/challenge appraisals, SES and T/C reactivity. However, when analysed as predictors, threat/challenge appraisals did not predict post-competition endocrine response. Thereby, their impact has been considered within the boundaries of moderators of relationship between endocrine reactivity to competition. This argument is further supported by evidence drawn from non-human primate literature, which suggests that cognitive appraisals play a moderating role in post-competition endocrine response (Bernstein et a., 1983). Further, several authors stress on the importance of cognitive factors for the dynamic and reciprocal endocrine/status link in humans (Knight & Mehta, 2014; Salvador et al., 2003; Salvador & Costa, 2009). This section, thus, will explore the moderating power of threat/challenge appraisals on endocrine response to competition. The section commences with analysis of threat/challenge in relation to T reactivity and proceeds with the C data.

7.3.1 Threat/Challenge Appraisals as Moderators of T Reactivity Towards Win/Loss

Drawing on the theoretical argument that not all individuals experiencing low SES would also experience elevated levels of stress and threat cognitions (Adler et al., 2000; Marmot, 2004; Sapolsky, 2004, 2017), threat/challenge appraisals were analysed in a custom covariate model (ANCOVA), irrespective of SES. In the model, the cognitions operationalised as moderators (covariates) of T response towards competition outcome. Resultantly, the sample was divided into four groups: winners

who appraised the event as a threat (or high threat); winners who appraised the event as a challenge (or low threat); losers who appraised the event as threat; and losers who appraised the event as a challenge (Figure 19). The results from the custom ANCOVA yielded non-significant main effects of time and objective competition outcome, when controlled for threat/challenge cognitions. Moreover, the effects of the threat/challenge appraisals on post-competition T reactivity also did not reach statistical significance. Further, significance was not reached by any of the two-way interactions, e.g., between time and competition outcome, when accounted for the threat/challenge; between time and threat/challenge appraisals; and between outcome and threat/challenge appraisals, reached significance. Nevertheless, the three-way linear interaction between time, outcome and threat/challenge appraisals was significant.

To unpack this three-way interaction, two separate follow-up ANOVAs were performed. These examined the threat condition, and the challenge condition respectively. Results from both ANOVAs revealed non-significant main effects of time in both threat and challenge conditions (section 6.1.3). The main effects of outcome in both ANOVAs (based on threat and challenge grouping) were also non-significant. Moreover, the two-way interactions between time and outcome for both groups (threat and challenge in the two subsequent ANOVAs) were also not significant. These puzzling and challenging results proved difficult to interpret. For that reason, this study adopted an approach to interpret complex three-way interactions proposed by Field (2018), resulting in the findings being visualised on graphs (Figure 19, and plots from the analysis outputs – Appendix XXXIV). This allows closer examination of T changes in both threatening and challenging conditions following competition victory/defeat.

The findings from Figure 19 and plots from Appendix XXXIV demonstrate that the four groups demonstrate very different endocrine responses towards competition outcome. Generally, these findings reiterate the complexity of the pattering endocrine response towards competition and the importance of comprehensive sampling protocols for the understanding of the dynamic endocrine/cognition relationship.

Furthermore, the plots display that the interaction effect that time and outcome have on T levels differs a lot among the levels of threat/challenge cognitions (as split between high and low threat groups). This perhaps could explain why the delegate two-way interactions appear non-significant. Indeed, these results suggest that it is very difficult to interpret what is driving the significant higher level three-way interaction observed in the custom ANCOVA analysis. One possible explanation is that the result may be a false positive considering the low study power (Button et al., 2013). For that reason, whilst the significant high order interaction is consistent with H3 and demonstrates some evidence in favour of rejecting the null hypothesis, issues of small sample size caveats should be considered:

> H3: Cognitive appraisals of threat/ challenge will moderate endocrine reactivity in both SES populations.

Moreover, given the non-significant results of the follow-up analyses, it is difficult to form firm conclusions around what is driving this high order interaction. It could be concluded thus that a potential relationship between threat/challenge cognitions, competition outcome and time exists, however, further work is needed to establish both whether the significant high order interaction reflects a somewhat reliable true effect (or it is a false positive), and if reliable, what is driving it.

Generally, however, the findings provide some exploratory support for the biopsychosocial model of challenge and threat, proposing a dichotomy in the physiological responses to stress between individuals perceiving events as threatening and or challenging (Blascovich & Mendes, 2000). Importantly, differences in appraisals of threat and challenge, subsequently affect physiological responses to the environmental stressors – in this case a status competition (as evident in the data), which in turn influences individual performance on the competition (Brown et al., 2021; Blascovich, 2008; Blascovich & Tomaka, 1996; Kelsey et al., 2000; Schneider et al., 2008; Tomaka e al., 1993; Tomaka et al., 1997). More concretely, the results of this study further indicate that SES may be a potential powerful factor of stress reactivity in social competition encounters via psychobiological pathways of threat perceptions

(Scheepers & Ellemes, 2005). Although it was outwith the scope of this thesis to analyse and compare actual frequency and severity of exposure to real-life stressors in the high and low SES groups respectively, low SES individuals in this sample reported a higher prevalence of threat appraisals towards the competition as compared to high SES individuals (see section 7.2.4). This is in support of more general evidence coupling low SES and more frequent stressor exposure towards a higher likelihood of expressing threat, rather than challenge cognitions towards stressors.

However, as will be discussed further in this chapter, threat/challenge cognitions may not comprise the full scope of potential factors moderating endocrine responses to psychosocial stressors, but indeed need to be discussed in close consideration to the parameters of matters of social status and hierarchy impacting upon endocrine reactivity and health in complex ways. Thus, the results from this study appear (to some degree) be in line with relevant recent literature stressing on the importance of cognitive factors for the dynamic and reciprocal endocrine/status link in humans (Knight & Mehta, 2014; Salvador et al., 2003; Salvador & Costa, 2009). Conclusively, this evidence, placed within the broader context of SED, suggests that implications of status for endocrine reactivity might be moderated by cognitive factors such as threat/challenge appraisals. These considerations are important for public health policies, targeting health inequalities, because of the previously outlined relationship between endocrine reactivity, behaviour, and health within the framework of social status.

Lastly, because of these relationships, developing interventions which target cognitions might be a pathway to reduce SES-related health-risk. The current findings, thus, pose the important question: To what extent an intervention tackling those cognitions would effectively impact endocrine response to status encounter, or social tasks more generally, that would further facilitate individual's performance on tasks, and benefit future stress appraisals? Alas, exploring these interventions and their wider implications for SES related health outcomes lies outwith the scope of this thesis and future research is needed to address the issue. Nevertheless, there are a range of other dimensions which need to be considered for designing effective interventions,

which may modulate the cognition-physiology axis, and thus all interventions require close-examination of the targeted populations. This is highlighted by Rith-Najarian and colleagues' (2014) study of adolescents, finding less of a link between cognitions and physiology. However, these findings may well be explained due to poor emotional self-awareness, and immaturity of neurobiological systems, attributed to this age stage (Rith-Najarian et al., 2014), and thus might not stand in contradiction of the abovementioned found relationship – they simply emphasise that no size fits all.

7.3.2 Threat/Challenge Cognitions as Moderators of C Reactivity Towards Win/Loss

Similarly to T reactivity, the moderating effects of threat/challenge on C reactivity towards competition victory/defeat were analysed in a custom covariate (ANCOVA) model, irrespective of SES. As with T, the sample was divided into four groups: two winning groups, appraising the event as either a high threat or a low threat (i.e., challenge); and two losing groups appraising the event as either a hight threat or a low threat (i.e., challenge) (Figure 20). In contrast to T results, however, none of the ANCOVA results reached significance: no main effects of competition outcome or time after adjusting for threat/challenge cognitions; no significant relationship between threat/challenge appraisals and C reactivity, and lack of two-way (time*outcome, outcome*threat, and time*threat) and three-way interactions (time*outcome*threat). Importantly, the two-way interaction between time and threat/challenge appraisals was very close to statistical significance (p = .056). Suggesting that, over the different collection points, post-competition C reactivity differed almost significantly depending on the appraisals of threat and challenge. Closer examination of the statistics allowed the detection of a more definite (i.e., linear) relationship between C response and threat/challenge. This means that group C reactivity increases proportionally over time depending on appraisals of threat and challenge. These findings, however, do not clarify which cognitions provoke what endocrine response; more specifically, which group exhibited an increase of C reactivity over time. Was it the one perceiving the event as threatening, or the one perceiving it as challenging? These results should also be interpreted in light of small sample size caveats (i.e., false negatives and false positives) and drawing firm conclusions from this data is ill-advised. Whilst follow-up statistical analyses have not

been performed as the relationship between time and threat/challenge appraisals remains non-significant, a visual illustration of the directions of the results are presented in Figure 20.

Indeed, Figure 20 displays that as with T findings, distribution of changes from mean C levels are relatively different across all four groups. Moreover, although all four groups exhibit a heightened levels of circulating C response following the competition, the magnitude of their reactivity differs. Conclusively, the current findings once again reiterate the pattering nature of the endocrine response and the complexity of the dynamic status/endocrine reactivity relationship, which, if insufficiently observed over different collection points, could be easily misinterpreted and mistakenly support or contrast previous research. Thus, for example, if the current results were interpreted simply at immediately after the competition, the findings would have been entirely in line with Mazur's biosocial model of status and the challenge hypothesis predictions. In this case, however, results offer a degree of complexity.

For instance, it was expected that high threat, in both losing and winning conditions, would be associated with higher C levels compared to losing and winning low threat conditions, respectively (as argued by Beilock & Gray, 2007; Dienstbier, 1989, 1992; Kemeny, 2003; Mattarella-Micke et al., 2011). The current research findings, however, stand in contrast with the hypothesised. Additionally, since the competition outcome was experimentally manipulated, it was expected that higher levels of threat cognitions would be exhibited by the losing groups compared to the winning groups, due to lower self-evaluations of resources allowing individuals to outperform their opponents (Oliveira et al., 2013). For this reason, and because threat cognitions are associated with higher C reactivity (Townsend et al., 2011), the current research expected to observe generally higher endocrine response in the high threat groups, thereby demonstrating a physiological response that echoes a cognitive state. The current results, however, demonstrate that only the losing high threat group displayed higher levels of C compared to the losing low threat group. Additionally, it could be argued that the reversed relationship has been observed, particularly for the winning conditions (i.e., win low threat group displayed higher levels compared to win high

threat group). These results thus echo the perplexing relationship between hormones and cognitions. This is because the current findings do not display a concrete answer to whether threat cognitions necessarily facilitate an endocrine response that corresponds to these appraisals (i.e., higher C levels). Indeed, what becomes apparent is that in some instances (i.e., in the winning conditions), it is the high threat group that display lower C levels compared to the high threat group. This trend, however, is not observed in the losing condition, where the high threat group is the one with generally higher C levels compared to the low threat. For this reason, it cannot be straightforwardly concluded that high threat perceptions are necessarily related to high C response, but perhaps that C levels could be related to both – competition outcome (win/loss) and threat/challenge appraisals.

One assertion that could tentatively be made from the directions of the current results suggests that an interaction between threat/challenge appraisals and competition outcome (losing and winning), and not the state of winning/losing or of perceiving events as threatening/challenging, idiosyncratically constitutes the regulator of C reactivity. This idea remains entirely hypothetical however, based on directions of results rather than significant level findings. For that reason and due to the increased risk of false positive and negative results in this research, these relationships should be explored on a larger scale which can achieve greater confidence in the 'true' effects of results and sufficient statistical power to identify effects and interactions.

Once statistical findings between the present research and previous studies are compared however, it becomes evident that there are some contrasts. For instance, Oliveira and colleagues (2013) report that in their research winners are associated with more challenging cognitions, whilst losers are associated with more threatening appraisals. These findings oppose to the lack of statistical interactions between threat/challenge appraisals, competition outcome and C reactivity in this study. It is worth noting here though, that Oliveira and colleagues' (2013) participants gave their threat/challenge appraisal evaluations following the exposure to the competition, leaving the possibility of participants downgrading the subjective importance of the competition after the outcome has become apparent to them. This possibility relates

to the theoretical argument that individuals often amend answers related to the importance of the task and cognitive appraisals following competition outcome (Sharp, 2006). Bearing this in mind/avoiding such post hoc amendments, the current study design prompted participants to fill in a threat/challenge appraisal form prior to the contest. However, because evaluation of threat cognitions was reported prior to the contest, the study was unable to capture the number of threat/challenge cognitions experienced in each cognition following competition victory/defeat.

Furthermore, Oliveira and colleagues' (2013) results should also be interpreted with caution considering that the study utilised only two endocrine collection sample points (i.e., one pre and one post competition). Future research, thus, should utilise a procedure allowing to evaluate threat/challenge appraisals both prior to and after a competition exposure, thereby directly linking endocrine response to anticipatory cognitions, but also controlling for the aforementioned design limitations. Finally, differences in analytical strategies and sample size caveats should also be taken into consideration when research findings are compared.

More generally, these findings are of importance due to the relationship between C reactivity and health outcomes outlined within the literature review. Indeed, despite the lack of any statistically significant effects and interactions (some that could be attributed to insufficient study power), inspection of raw hormonal levels across groups (not merely visual inspection of graphs that could be misleading) appear to demonstrate some dissimilarities in endocrine reactivity. For that reason, even though no definitive conclusion could be drawn due to the aforementioned limitations, when placed within the context of SED, these findings can convey important messages for policy makers. Considering the previously outlined association between low SES and potential experiences of more frequent stressors (also displayed in this research, section 7.2.4), public health policies targeting the reduction of the health gap between SES groups should once again consider the implications of the two variables (competition win/loss, representing loss and gain of status and threat/challenge appraisals) for health outcomes.

Lastly, C results should be discussed in relation to T findings. Indeed, the ANCOVA model for C reactivity reveals that, as opposed to T data, threat/challenge appraisals do not moderate the link between competition outcome and C reactivity. Although initially challenging to interpret, the results are potentially in line with earlier performed statistical analyses (e.g., correlation and regression models) identifying stronger relationships between C reactivity and SES, compared to C reactivity and threat/challenge cognitions. Thus, arguably, what appears to have a greater importance for C reactivity to competition outcome in this sample is the objective SES independent of threat/challenge appraisals. The present research findings appear to be in line with Oliveira and colleagues (2013) results, demonstrating a relationship between T reactivity and threat/challenge appraisals, but not between C reactivity and these appraisals. Thus, in the light of SES impacting on C reactivity, it is worth testing whether SES and not threat/challenge appraisals moderate post-competition C reactivity. This, however, falls outside the scope of this thesis, and therefore a further investigation of the moderating effects of SES on C reactivity to competition outcome is required.

Conclusively, the results from the moderators of C reactivity to competition outcome allow RQ3 and H3 to be addressed and compliment the T data. When C data was entered in statistical models, the analysis yielded non statistically significant differences, therefore failing to provide evidence for rejecting the null hypothesis and accepting H3. The interpretation of endocrine data in relation to cognitive appraisals and competition outcome is a far more complex task than merely dichotomizing results based on statistical significance. Indeed, issues and caveats around sample size make any straightforward conclusions problematic. For this reason, further research is required in order to expand and replicate these findings. Doing so will allow scholars to more closely examine the issues, and in doing so will shed more light on the complex interactions between endocrine response and cognitions.

7.4 Behavioural Implications of Social Defeat/Victory

The discussion within this section aims to address the behavioural implications of winning and losing status competitions, in relation to endocrine reactivity (particularly T). In doing so, the section addresses the last RQ4 and H4:

> RQ4: Does T reactivity correlate with a reduced motivation to engage socially?
>
> H4: Reduced motivation to engage with subsequent social tasks will positively correlate with a decrease of T reactivity.

The section then examines these findings and their implications within the broader framework of SED. This leads to discussion of the significance of the results upon policymaking, leading to policy recommendations.

7.4.1 Motivation and T reactivity

As this thesis has highlighted, a great number of biological, psychosocial, environmental and contextual factors may predict T reactivity following status change (Knight & Mehta, 2014). What appears of fundamental importance is that status-induced changes in T reactivity also impact future status-seeking behaviours (Knight & Mehta, 2014; Losecaat Vermeer et al., 2020; Mehta & Josephs, 2006). This bidirectional link between T reactivity and status has been supported by Mehta and Josephs (2006), whose first empirical study of humans tested the relationship between post-competition T reactivity and subsequent engagement with social tasks, or subsequent social behaviour more generally. In their study, competition outcome was not associated with significant differences in T reactivity. Their findings however, indicate that losing individuals who experienced rise in T concentrations were more willing to compete again in future competitive tasks than individuals who lost and experienced a drop in T post-competition levels (Mehta & Josephs, 2006). Previous studies by Carré (2009), and Carré and colleagues (2013), measured the relationship between reactive aggression, subsequent motivation and performance in future contests. Their findings were also consistent with Mehta and Josephs' results, further supporting the argument for the reciprocal model of T (bidirectional T/status link).

Most recently the relationship between T, competition outcome and willingness to compete again was reported by both Losecaat Vermeer and colleagues (2020), and Kutlikova and colleagues (2021).

Advancement in our understanding of this phenomenon had been made by this study, the findings of which appear to differ both Mehta and Josephs (2006), and Carré (2009). When utilising a linear regression model to enquire whether T change predicts future motivation to engage with a similar, or in this case identical social competitive task the statistical relationship was found to be insignificant (Chapter 6, section 6.2). This leads to the assertion that T change does not significantly predict future motivation to engage with competition. As earlier argued by Mehta & Josephs (2006), the relationship between post-competition T reactivity and future motivation in their study was not uniform, but only observed in participants from the losing condition. In the light of this argument, research results in the study were re-analysed separately to measure the potential effects of T change upon motivation within both winning and losing conditions. Findings, however, remained non-significant for either of the groups. Potential explanation for the dissimilarities in findings within existing literature and this study, once again lie in methodological differences (sampling regimes and analytical strategies), and sample size caveats (e.g., insufficient power to detect interactions in this research). Additionally, the studies undertaken by Mehta & Josephs (2006), and Carré (2009) both adopt sampling regimens with only two hormonal samples obtained pre- and post-competition. Sections 2.3.5, 3.2.1, 3.2.2, and 3.2.3 illustrate the limitations of these regimens and the risk for potential misinterpretation of the results. It is also of note that in line with the present findings, not all prior research establishes a relationship between T changes and post-competition motivation (Hirschenhauser et al., 2008, 2013). However, similar to the current research's limitations, it may be that false positives – thus not representing accurate estimate of the effects – could also be present in previous studies. Therefore, conclusions regarding contrasting or analogous results should be interpreted with caution, as results might indeed not differ or replicate.

Furthermore, competition outcomes frequently lack a clear-cut outcome, with results and performance between winners and losers often similar and having implications for future motivation (Vermeer et a., 2016). Indeed, the behavioural implications of close outcomes – also known as small losses or near-misses – have been associated with positive motivational outcomes (Reid, 1986). This has been supported by studies employing gambling and competition tasks, where individuals who nearly won (such as someone who came second in a competition), were far more motivated to compete again than the competitors who have clearly lost the contest by a large margin (Berger & Pope, 2011; Clark et al., 2009; Zilioli & Watson, 2014). Indeed, Zilioli and Watson's (2014) study further clarifies that a defeat in an unstable hierarchy is associated with higher post-competition T reactivity which serves the purpose of boosting motivation for future encounters and thereby improve performance.

In order to avoid results that had a clear outcome the present research informed individuals from both SES groups that their opponent was of the same SES background. Furthermore, the competition task was carefully chosen to adequately represent the abilities of both SES groups, so there were no individuals who might have been more skilled or talented in completing the task compared to others (van Anders & Watson, 2007). This was expected to create the feeling that participants from both groups have an equal chance to outperform their opponent. A verbal investigation of the issue (consisting of a brief conversation between the researcher and participants) after the end of experiment, confirmed that indeed none of the participants felt they were at a disadvantage with regards to the task or their competitor. Taking this into consideration and the fact that most of the participants evaluated the experimental task as important to them (measured as a separate question on the threat/challenge appraisals questionnaire, prior to the competition), it was hypothesised that: 1) T will be responsive towards competition outcome; and 2) there will be clear behavioural implications for the participants experiencing competition defeat. None of the hypotheses, however, has been supported with evidence from this study. It is important to noted that previous studies suggest behavioural and particularly, motivational changes appear shortly after the competition (within the first 10-20mins), similarly to post-competition T fluctuations

(Vermeer et al., 2016). Thus, in this study motivation has been evaluated within the first 15 min post-competition, consistent with research suggestions. Conclusively, the lack of significant findings in this study cannot be attributed to methodological limitations.

In seeking more clarity, motivational data were also analysed in relation to competition outcome, irrespective of T reactivity (Figure 18). The analysis thus aimed to identify whether motivation to engage in related financial activities (such as applying for a job, seeking monetary literacy support or applying for a loan) would significantly differ following competition victory/defeat. Findings from this analysis again yielded non statistically significant differences between the winning and losing groups on all motivation domains, apart from the one asking individuals for their motivation to *apply for a loan*. A reversal of the expected relationship was observed in this subdomain, with the losing group displaying a higher motivation to apply for a loan compared to the winning group (Figure 18). This reversed trend was also observed in two other motivational scale subdomains – *seek help with funding matters* and *join a finance wellbeing* course. However, in contrast to *apply for a loan,* the differences in these two subdomains were not statistically significant. Closer examination of Figure 18 and the descriptive statistics also revealed that although not statistically significant, the losing group indeed exhibited a lower motivation to compete in a future financial form filling contest again compared to the winning group.

A few conclusions could be made from this analysis of motivation by competition outcome. Firstly, the presence of consistent non-statistically significant findings in this and in earlier regression analysis suggests that the lack of differences in motivation scores following victory/defeat could not be attributed to methodological or design limitations (notwithstanding small sample size caveats). Secondly, although puzzling, the reversed relationship between losing and motivation, observed in the *apply for a loan*, *seek help with funding matters*, and *join a finance wellbeing course*, scores could potentially be explained by the populations SES background. This point will be expanded on in a later paragraph. Finally, although the preceding analysis appears to

fail to provide evidence in support of – "T reactivity correlates with a reduced motivation to engage socially", the current analysis of motivational scores by competition outcome suggests that within specific domains of the motivational questionnaire potential links between competition victory/defeat and future motivation could be drawn. Indeed, the results from this analysis demonstrate that the losing group exhibit a lower motivation to re-engage with the competition task. Based on these perplexing and ambiguous findings, it could be argued that future research, utilising a comprehensive sampling regime on a larger scale, is needed to clarify the results. Without doubt, as animal and human research progresses, a clearer picture about the relationship between T reactivity and post-competition behaviour will emerge. At this stage, however, it could be concluded that results from the current study fail to provide evidence in support of H4, answering RQ4:

> RQ4: Does T reactivity correlate with a reduced motivation to engage socially?
>
> H4: Reduced motivation to engage with subsequent social tasks will positively correlate with a decrease of T reactivity.

Lastly, in seeking answers to the earlier point that reversed relationships in the motivational questionnaire subdomains (*apply for a loan*, *seek help with funding matters*, and *join a finance wellbeing course*) could be attributed to SES backgrounds, the motivational data was analysed by social grouping (SES). The results from the analysis provided supporting evidence that the reversed relationships for the motivational subdomains could be (arguably) attributed to SES. This is because low SES indeed displayed higher scores in these domains compared to high SES group suggesting that, although validated in a pilot study prior to the experiment, the motivational questionnaire utilised in this study may have stronger sensitivity for SES than competition outcome (Figure 19 in Appendix XXIX). This finding reiterates the call for research which expands, replicates and more deeply examines these puzzling observations Furthermore, it is worthwhile noting that as this study ran multiple comparisons, thereby inflating the rates for Type 1 error, results should be interpreted with caution.

The motivational findings provided in this section are of importance because of the long-term effects of competition victory/defeat on future status-related behaviours and competitiveness, also known as the winner and loser effects (Vermeer et al., 2016). It has been suggested that competition defeat/victory, or social defeat/victory more generally, does not only impact upon future competitiveness but also on future chances of winning or losing competitions (Chase et al., 1994; Dugatkin, 1997). The effects of victory and defeat for future success/failures in encounters have been documented in multiple animal (Dugatkin, 1997; Fuxjager et al., 2011; Gleason et al., 2009; Hsu et al., 2006; Oyegbile & Marler, 2005) and human studies (Oliveira & Oliveira, 2014). Research also emphasises the importance of moderating factors upon the observation of the winner and loser effect (McGee & McGee, 2013; Chiviacowsky & Wulf, 2002, 2007; van Anders & Watson, 2007). For instance, it has been argued that certain psychological aspects such as effort might underpin the winner effect, as individuals who outperform their opponents usually put more effort into the task (McGee & McGee, 2013). Although it is important to be noted that this effect only emerges if the task outcome adequately corresponds to the performance, or in other words, the contest was not won by chance (McGee and McGee, 2013; van Anders and Watson, 2007).

Resultantly, these findings suggest that for motivation to take part in future competitions through the winner effect to emerge, an actual achievement should be experienced by the competitors (Vermeer et al., 2016). Chiviacowsky and Wulf (2002, 2007) further add that positive performance feedback, in tasks without a competitive element (i.e., cognitive or motor), also improve and boost performance. Furthermore, in order to harvest the positive effects of winning on future motivation, research should take into consideration the importance of the aforementioned psychological variables. Moreover, research should also consider the underpinning neurobiological changes emerging as a result of the victory/defeat (Fuxjager et al., 2011; Fuxjager & Marler, 2010; Oyegbile & Marler, 2005; Trainor et al., 2004; Zilioli & Watson, 2014).

The present research thus acknowledges that T represents an important underpinning neuroendocrine aspect for the behavioural implications of social defeat/victory, and considers these implications within the broader contextual framework of SED. Indeed, the current study aimed to explore the relationship between T reactivity and motivation precisely because of the proposed risk of more frequent repeated exposures to social defeat within low SES groups (Sapolsky, 2017). Besides, despite the multiple reasons for those frequent exposures to social defeat (i.e., structural violence or societal inequity) amongst low SES groups, the behavioural and biological implications for future motivational states have been documented (Knight & Mehta, 2014). Within the framework of SED, these motivational states could be associated with desires to break the cycle of poverty. For this reason, even though the relationship between T reactivity and motivational states have not been statistically supported by this study's findings, this thesis argues that any interventions targeting the loser effect and aiming to harvest the winner effect in low SES groups should take cognisance of underpinning neuroendocrine and psychological moderators. Finally, this thesis emphasises the need for policy rearding eradication of health inequalities to carefully consider the interpretation of study results. Decisions should not be informed by contributions to knowledge which provide conflicting evidence solely based on dichotomization of study results.

7.5 Limitations

7.5.1 Participants cohort and Power

A calculated power analysis suggests an optimal sampling size of 188 participants in this study, based on (alpha= 0.05; effect size f= 0.19). Due to many challenging circumstances the study was only able to recruit 20 participants classified as higher SES and 11 classified as lower SES. It must be noted here thus, that for the study to reach a reliable power to detect an effect (i.e., 0.80), a much larger sample size was required. For that reason, whilst this study offers a comprehensive sampling regime (i.e., multiple sampling) and makes an empirical contribution to the theoretical paradigms offered within of Marmot's 'Status Syndrome' (2004) and Wilkinson and Pickett's 'The Inner Level' (2018), the work outlined in this thesis should be considered a feasibility study which requires further investigation on a larger scale. Moreover, the

results should be interpreted with caution due to the study being underpowered, thereby running an increased risk of inflated Type I and Type II errors and false positive results that do not reflect true effects.

Moreover, the smaller sample size for the low SES group compared to the high SES stems from the challenges experienced with the recruitment of low SES individuals, including the inability of the researcher to make and sustain contact with the individuals, greater percentage of withdrawal from the experiment in this group and exclusion of contaminated salivary samples (when more than 3 samples were contaminated) thereby resulting in an exclusion of the participant from the sample size.

The challenges experienced in this research have also been documented in previous literature suggesting that groups experiencing SED or of low SES appear to be underrepresented in public health and medical research (Bonevski et al., 2014; Sateren et al., 2002). Exclusion of low SES groups from health and medical research frequently occurs due to issues with sampling and recruitment, as outlined in this study (Bonevski et al., 2014). This in turn, impacts upon the accuracy and validity of research, and the ability to generalise from data (Bonevski et al., 2014). Bonevski and colleagues (2014), thereby argue that random population sampling restricts the ability to target and produce large experimental cohorts of low SES groups. Hence, an alternative and more advantageous sampling approach might be to target these groups through community organisations which could provide access to individuals from low SES groups, enlarge engagement by increasing the level of trust and by reducing the fear of research across vulnerable populations (Derose et al., 2000; Escobar-Chaves et al., 2002; Hoppitt et al., 2012). Following these suggestions, the present research utilised the exact same strategy where gatekeepers were used as support networks to gain access to more vulnerable groups whilst building and promoting trusting relationships. This approach, however, seemed to be insufficient in the endeavour to reach the targeted sample size. Future research programmes therefore need to address the challenge of including more vulnerable groups whilst making the experimental procedure easy to comply with. Moreover, to tackle some of the statistical issues

related to low study power (e.g., type II error and false positives) the present findings should be 271alifornia271d on a larger scale. Potential strategies might include longer recruitment time, techniques encouraging gatekeeper support, more enhanced media and social networking, and community research partnerships. Finally, an alternative for future research dedicated to bio-behavioural investigations might be the usage of secondary datasets gathered by specialist organisations.

7.5.2 Baselines

Despite utilising a relatively comprehensive sampling regimen compared to previous studies, the present research runs a degree of risk in its interpretation of the post-competition endocrine data. This is because baseline comparison was only applied to the pre-competition data, making the post-competition endocrine reactivity extremely difficult to interpret. Lack of meaningful baseline, as discussed in section 2.3.5 of Chapter 2, thus runs the risk of unexplained variability in T and C that results neither from competition outcome *per* se, nor from the moderating cognitive variables examined in this research. Sharp (2006) further emphasises the importance of establishing circadian profiles in order to reliably address post-competition hormonal changes. Adding to this complexity, the post-competition samples in the present study were also collected in the afternoon hours, making endocrine reactivity even more challenging to detect and interpret. This is because hormonal levels are lower and less reactive in the afternoon compared to the morning. Furthermore, considering the degree of non-compliance with the study design amongst the SES group (i.e., activities interfering with hormonal levels, sample contamination and lack of adherence to the collection schedule) and the lack of experimental control over the last sample collection points (1hr and 2 hrs post competition), data should be interpreted with caution.

In order to address this limitation, future research programmes should consider utilising a sampling regime comprising a meaningful baseline that encompasses the whole experimental duration (i.e., two baselines – one for the pre-competition phase and one for the post-competition data). Furthermore, in order to address the magnitude of hormonal changes (e.g., high hormonal variability during experimentally

uncontrolled sampling times) research could apply a couple of strategies. This may consist of more frequent post-competition samples (such as every 15 minutes up to the first 45 minutes) in the first critical periods after the competition outcome, as these time periods have been suggested to experience the greatest post-competition hormonal variability (Vermeer et al., 2016). Alternative, future studies could seek more control over participants' compliance, procedure, and environment. This may lead to lower ecological validity, however. Finally, it is important to note that the current sampling regime (12 samples over the course of two non-consecutive days) appeared to be challenging for participants, particularly for the individuals from lower SES backgrounds. Future research should therefore consider the most effective strategies through which comprehensive sampling regimes would appear attractive and easy to comply with for participants. Perhaps, smaller aliquots/samples (i.e., smaller that the 5ml), collected more frequently could be the key.

7.5.3 Sex of the participants

This study did not consider female endocrine response to competition outcome, cognitive moderators of this relationship and related psychosocial factors. In order to test whether the challenge hypothesis/Mazur's biosocial model of status could be applied within the context of SES, the present study concentrated on male participants solely. This is because the challenge hypothesis/Mazur's biosocial model of status have been predominantly tested in male species (Archer, 2006). This results from the argument that higher levels of testosterone reactivity and dominance behaviour are associated with males rather than females within the context of competition (Campbell, 1999, in Sharp, 2006). More recent research, however, demonstrates that the effects of competition have a very similar impact on the endocrine response in females (Bateup et al. 2002; Casto et al. 2014; Edwards & Kurlander 2010; Filaire et al. 2009; Hamilton et al., 2009). Yet, Sharp (2006) argues that further research is required in order to develop any firm position over the role of biology in female social dominance behaviour. In support to this argument even some recent scholarship continues to still fail to establish the relationship completely (see Carré et al., 2013). This in turn leads to ambiguity in the literature and lack of consensus even in the most recent bio-behavioural research. For this reason, the present study only tested the

robustness of the challenge hypothesis/Mazur's biosocial model of status for SES within male populations. Considering that some theoretical understanding of the issue could be drawn from the findings in this study, future research should aim to further expand and test the reliability of these findings amongst females.

Finally, this study explored and explained part of the neuroendocrine mechanisms underpinning the social gradient in health, in the form of androgen and corticosteroid systems. The results from the study aim to enhance future health and public policies by providing biological and psychosocial exploratory frameworks that have not been previously considered in the existing health inequalities literature. However, these findings are somewhat limited as the conclusions from this research relate only to male populations (within a very restricted sample size). Robust epidemiological research and policy making requires comprehensive, universal studies that can be generalised and applied to different populations. In order to achieve that, examination of the challenge hypothesis/Mazur's biosocial model of status within groups of females and on a larger scale is of fundamental importance. Only this will contribute towards evidence-based policymaking which aims to reduce the ever-widening health gaps affecting contemporary Scottish society.

7.5.4 Summary

Numerous findings emerge from this study. First, the study fails to provide evidence of an anticipatory response in competition T. As a consequence, it appears that in the present research paradigm T is unresponsive during pre-competition. Similarly, an anticipatory C response was not observed during pre-competition. Consequently, the present research fails to provide evidence to support the suggestion that change in mean C levels could partially explain the absence of T response prior to the competition (see Mehta & Josephs (2010) for further discussion of the dual-hormone hypothesis). Notwithstanding this, these results should be interpreted with caution considering the low power of the study and subsequent risk of false negative results. Indeed, this is particularly relevant for the C pre-competition data where the direction of results (based on plots and raw endocrine data inspection) suggests some evidence

for dissimilarities of endocrine response on the day of the competition compared to baseline.

Post-competition data were challenging to interpret, but revealed the complexities of endocrine reactivity upon psychosocial stressors. Post-competition T levels did not demonstrate statistically significant differences of circulating T over the different collection points and across competition outcomes (win/loss) or status groups (low/high). The same findings emerged from the analysis of C data (i.e., the change of C levels over time was not significantly different between competition outcomes or status groups). Thus, the present research fails to reject the null hypothesis and provide evidence in support of H1. Findings, however, should once again be interpreted with caution due to small sample sizes. Indeed, when results directions are visualised and explored on plots, and raw endocrine data is inspected, the findings suggest some potential dissimilarities across groups (winners and losers, and high/low SES). Consequently, whilst findings should be interpreted with caution due to potential false negatives and positives emerging from low study power, results should also not merely be compared to previous research based on dichotomous categories (i.e., statistical significance). Additionally, the comprehensive salivary collection protocol and ensuing complexity of endocrine reactivity highlights the importance of sampling protocols when critiquing previous research. Results could be easily misinterpreted if conclusions are drawn merely on observations undertaken at individual collection points. Finally, data relating to cognitive measurements (i.e., threat/challenge cognitions), illustrates slightly different results, thus supporting the possibility that the event was perceived to be of higher importance for the low SES group. The data suggests that individuals from lower SES groups appraised the event as more threatening compared to the higher SES group.

When the relationship between those psychosocial factors believed to interact with physiological responses – and thereby potentially account for some of the health disparities observed between SES groups (Steptoe & Marmot, 2002; Taylor & Seeman, 1999) – and endocrine responses were explored, findings yielded mixed results. Indeed, whilst there were some differences between low and high SES groups in some

of the psychosocial factors (e.g., personality style), differences between other variables such as sense of coherence and attributional style (sense of control) remained non-significant. However, when explored more closely, the direction of results indicated that most psychosocial variables the groups (low/high SES) differed in the predicted way (e.g., high SES displaying higher sense of control/sense of coherence than low SES). Furthermore, once the relationships between those psychosocial variables and endocrine response were explored through correlational and regression models results demonstrated non-significant findings. For that reason, it could be concluded that whilst some differences on psychosocial measurements were observed between SES groups, the lack of statistically significant findings fail to provide evidence for rejecting the null hypothesis. The study therefore does not provide evidence in support of H2.

Considering Sharp's (2006) findings which shows the impact of mood upon endocrine reactivity, an analysis evaluation of the effects of trait affects and competition outcome on T and C post-competition reactivity was conducted. The results for T revealed that positive and negative trait affects and competition outcome do not significantly predict the T reactivity. The C data demonstrated dissimilar results, where the predictors – general negative and positive trait affects and competition outcome significantly impact on C response. These results are in line with Sharp's (2006) findings from female non-physical hormone competition. Nevertheless, all psychosocial results should be carefully interpreted within the context of statistical issues related to Type I, Type II error and low study power.

Importantly, in line with the Biopsychosocial model of threat and challenge, cognitive variables appear to have some significant role in moderating the endocrine reactivity to victory/defeat. In particular, cognitive appraisals of threat/challenge exert a significant moderating effect upon T reactivity following the outcome of competition. Follow-up statistical analyses unpacking the complex high-order interaction between threat/challenge cognitions, competition outcome and time for circulating T levels, however, demonstrate non-statistically significant results. Therefore whilst the significant high order interaction is consistent with H3, given the non-significant results of the follow-up analyses, it is difficult to form firm conclusions around what is

driving this high order interaction. Conclusively, a potential relationship between threat/challenge cognitions, competition outcome and time occurs, however further work (on a larger scale) is needed to establish both whether the significant high order interaction reflects a somewhat reliable true effect (or it is a false positive), and if reliable, what drives this dynamic. Finally, these effects are not extended to C, however. Given that win/loss are objective terms of a subjective experience, cognitive interpretation of the outcome of competition – rather than the outcome *per se* – plays an important role in endocrine response. Future research should test the reliability of those findings within female populations, as well as revealing why the moderating effect is only limited to T reactivity.

Endocrine reactivity and response to status encounter, and the cognitive variables moderating that link, are important because of their long-term behavioural implications upon future competitiveness, as well as motivation to engage in competitive tasks in the future. Applied to the broader contextual framework of SED, low SES individuals are at greater risk of continuous and repetitive exposure to social defeat. They also experience the most detrimental aspects of social defeat such as lower motivation for future social tasks, or the loser effect which manifests itself through lower chances of success in future social tasks. Furthermore, the effects of social defeat also have accumulative biological (i.e., toxic stress, consecutive continuously elevated C levels and thereby higher risk of disease and mortality) and neuroendocrine consequences (i.e., low T levels) which subsequently become a significant element of the reciprocal model of T and status. Public and health policy interventions should pay particular attention to the behavioural and biological implications of social defeat within lower SES groups in order to minimise those consequences, and encourage positive health and behavioural outcomes such as the winner effect.

Furthermore, the theoretical frameworks explored in this research (the challenge hypothesis/Mazur's biosocial model of status and the BPS model of threat/challenge) do not appear to have been examined within the context of SES and endocrine reactivity to social defeat before. Marmot's (2004) and Wilkinson & Picket's (2018)

epidemiological research of health inequalities explore the psychological phenomena underpinning the social gradient in health, however neither of those theoretical positions empirically test specific biopsychosocial explanatory mechanisms. In examining an under-researched explanatory mechanism, whilst utilising a comprehensive endocrine sampling regime, this study makes a valuable contribution to the understanding of androgenic and glucocorticoid mechanisms underpinning the gradient in health. Although this research has not demonstrated any statistically significant differences in endocrine reactivity and very limited evidence for the relationship between cognitive appraisals and competition outcome amongst SES groups, the direction of the current exploratory findings provides some evidence of potential neuroendocrine mechanisms' involvement in status syndrome and status disparities in health. Indeed, further research on a larger scale is needed to either consolidate or perhaps provide an alternative argument to the suggested. Notwithstanding these caveats, these findings make a significant contribution to the existing knowledge regarding health inequalities and their relationship to status.

Exposure to chronic stress and elevated levels of corticoids have received considerable attention within recent literature, highlighting the deleterious effects of social inequalities upon health. Low SES has also been associated with a constellation of behaviours and cognitions such as aggression, hostility, risk taking, delinquency, status-seeking, fear, and impulsivity (Pepper & Nettle, 2017) which may be better explained by changes in levels of T (Mazur & Booth, 1998; Knight & Mehta, 2014). Nevertheless, little meaningful work has been conducted on the relationship between T and poverty, and that which has been conducted contradicts the cortisol literature (Haushofer, 2011; Mehta & Josephs, 2010). For this reason, this study aimed to explore the function of both hormones, their relationship with SES and implications for health and wellbeing. Alas, findings from the current study cannot concluded whether T and C each play a significant role in social defeat, nor have necessary implications for health outcomes, cognitions and behaviours associated with SES. Indeed, results should be explored with caution and within the context of sample size limitations and the potential statistical concerns stemming from this issue. Nevertheless, the study makes an empirical contribution towards a better understanding of the link between

status, androgens and health, and thereby enriches the existing health inequalities literature.

Moreover, the comprehensive sampling regimen adopted by this study enabled the measurement of chronobiological changes in T and C rather than static basal levels. For this reason, this study makes a robust contribution towards methodological approaches and knowledge; building upon existing bio-behavioural research whilst addressing the major limitations associated with single-point sampling. Investigating chronobiological hormonal changes rather than static basal levels also afforded the opportunity to consider the manner in which modifications of endocrine parameters in relation to cognitions (i.e., threat/challenge) impact upon behaviours which may reinforce aspects of SED. As outlined earlier, these findings appear of importance for evidence-based approaches and public policies tackling health disparities as they bring insights of the specific biopsychosocial pathways through which SED could be reinforced and related to health outcomes.

By utilising the theoretical framework of threat/challenge cognitions within this research, the study asked – and answered – the question: why don't individuals who experience SED also experience the negative health connotations associated with it? The findings indicated that both SES groups differ in their threat/challenge appraisals, but also that the cognitive elements perhaps play a moderating role in endocrine reactivity to status encounter (particularly T). This evidence, placed within the broader context of SED, suggests that implications of status upon endocrine reactivity are partially moderated by cognitive factors such as threat/challenge appraisals. The extent to which these findings can be replicated has yet to be determined. These findings add to our existing theoretical understanding of status syndrome due to the previously outlined relationship between endocrine reactivity, behaviour and health.

Finally, it is hoped that the knowledge produced by this study can fuel and inspire future research which further explores the implications of these findings upon larger cohorts. Moreover, this could lead to effective behavioural and psychological interventions which reduce the widening health inequalities which blights Western

industrialised societies. To conclude, the following chapter will summarise the thesis and propose approaches that could further advance scholarship in this field.

Chapter 8

Conclusion

8 Introduction

In attempting to explore chronobiological endocrine reactivity to threat/challenge cognitions within a bio-cultural theoretical framework, this thesis has been centrally concerned with investigating dissimilarities in the endocrine reactivity towards a non-physical competition in both high and low SES groups. In doing so, cognitive factors moderating the relationship between endocrine response and competition outcome were explored. Moreover, the study accounted for psychosocial factors which had been identified as enjoygin some relationship to physiological response. They may therefore ne involved in the relationship between endocrine response, SES and health outcomes (Cohen et al., 1999; Kaplan, 1999; Kraus et al., 2009; Phillips & Klein, 2010; Steptoe & Marmot, 2002). Whilst investigating this relationship, important questions regarding salivary sample regimes and measurement practises were raised. Finally, the behavioural implications of social defeat within the context of competition and the application of it to the broader framework of SES was addressed.

Whilst this thesis makes a contribution to knowledge concerning the scarcely explored biological mechanisms underpinning the social gradient in health, further research is required to fully consolidate the findings presented here. Despite the extensive and sophisticated body of literature focusing on explanatory models of health inequalities, it may be worth reflecting on studies that will shed further light on the inter-related biomarkers and cognitions associated with SED. This in turn will facilitate an integrated, cogent and coherent narrative, on the relationship between micro and macro elements of SED and aid selecting the nature and timing of potential interventions endeavouring to close the health gap. The following section summarises the content of previous chapters and proposes directions for future research.

8.1 Chapter Summaries

In *Chapter 1*, elements of the neuroendocrine mechanisms underpinning the social gradient in health were discussed by addressing the androgenic and glucocorticoid systems. The chapter started with a discussion of poverty and its implications upon health, whilst gradually moving towards the phenomenon of socioeconomic inequalities as a more appropriate explanation of the widening health disparities observed in Western industrialised societies (Marmot, 2004; 2015). Once establishing that the suggested theoretical explanation for the gradient in health is status syndrome (Marmot, 2004; Wilkinson & Pickett, 2018), the chapter then focused on the biological mechanisms underpinning status syndrome explored in this study. The chapter also considered the predominant theories and perspectives utilised to investigate health within the context of social sciences, and the plausible limitations of those. Finally, the chapter presents the study rationale, research questions, and aims and objectives.

Chapter 2 provided an extensive review of the literature relating to health inequalities and the biological implications of social status within animal and human hierarchies, subsequently drawing attention to the complexities of the human hierarchical structures. The chapter also discussed the evolutionary foundations and purposes of these ranking systems by providing examples from both human and animal research. Once acknowledging the importance and omnipresence of hierarchical structures within most species and the implications of rank for the individual in a hierarchy, the proposed neuroendocrine mechanisms underpinning status rank were discussed. Whilst utilising the challenge hypothesis/Mazur's biosocial model of status as a framework to explore the dynamic relationship between status encounter and endocrine reactivity, the chapter also acknowledged the wide variability of contextual and cognitive factors influencing this relationship (with the most central for this thesis being threat/challenge appraisals).

Chapter 3 proceeded with a discussion of the methodology of the present research and the study aims and objectives. Methodological limitations of chronobiological studies were discussed and the raised issues considered for the design of the present study. In

this chapter, the appropriate epistemological and ontological positions are also discussed. Finally, the chapter then discussed the utilised methods, materials and procedure and analytical strategies.

Chapter 4 empirically tested whether a dissimilar pattern of endocrine reactivity towards competition victory/defeat would develop within lower SES groups in comparison to their higher status counterparts. The results described in this chapter thus enabled this study to address RQ1 and H1. Whilst result directions yielding some differences in the post-competition endocrine response between SES groups, these results did not reach the threshold for statistical significance. Indeed, the study failed to provide evidence of a dissimilar pattern of endocrine reactivity between the competition outcome within the lower and higher SES groups. Notwithstanding this, these results should be interpreted with caution due to caveats relating to small sample size.

Chapter 5 paid significant attention to the wide range of cognitive and psychosocial variables hypothesised to relate to or moderate individual's endocrine response. Psychosocial variables were analysed by social grouping (SES), in order to examine any potential differences between SES groups. In doing so, the chapter questioned whether those variables could be addressed as protective factors, and queried why some individuals experiencing SED do not necessarily experience negative health outcomes (Marmot, 2004).

Chapter 6 combined the psychosocial and endocrine data in an attempt to evaluate potential existing links between psychosocial factors and endocrine response, thus addressing RQ2 and H2. The chapter paid particular attention to the moderating power of threat/challenge appraisals for endocrine reactivity to competition outcome, as the variable was of central focus for this thesis; by doing so, the chapter addressed RQ3 and H3. The findings indicated a significant moderating effect of threat/challenge appraisals on the post-competition testosterone reactivity. This analysis addressed RQ3 and H3: "Does cognitive appraisal of threat/challenge moderate endocrine reactivity?"; "Cognitive appraisals of threat/ challenge will moderate endocrine

reactivity in both SES populations.". The chapter then proceeded with evaluation of the behavioural implications of competition victory and defeat. By doing so, the chapter addressed research question and hypothesis 4: "Does T reactivity correlate with a reduced motivation to engage socially?"; "Reduced motivation to engage with subsequent social tasks will positively correlate with a decrease of T reactivity.".

Chapter 7 provided a discussion of all results from *Chapters 4,5 and 6,* and placed them within the context of SED. The chapter also discussed the advantages of the study methods. For instance, the comprehensive sampling protocol and reliable measurement practices allowed this research to shed some light on the complexity of the hormone/competition relationship, whilst also illustrating methodological limitations and thereby questioning the degree to which previous research findings were interpreted correctly. Finally, the chapter discussed the importance of continuous exposure to social defeat, not only within the context of competition but within the broader SES framework, upon individual's behaviour, endocrine reactivity towards defeat, and future motivation and engagement with social/status-related tasks. The chapter then ends with a summary of the research findings and their implications for the broader social policy context.

8.2 Future Research Directions

In the attempt to understand how testosterone is implicated in status behaviour, the present study paid significant attention to the wide variability of psychological and behavioural processes. Whilst accounting for cortisol in this relationship, it fell outside the scope of the current thesis to explore the effects and explanatory power of other endocrine parameters such as oxytocin or oestradiol and biomarkers such as serotonin, dopamine or DHEA-S in this dynamic hormone-competition interaction. Indeed, future research programmes testing the robustness of the current findings should pay particular attention to oestradiol and oxytocin; markers suggested to play a more fundamental role in female status seeking and dominant behaviours (Schultheiss, 2007; Stanton & Schultheiss, 2009). Additionally, more recent literature demonstrates that females are equally concerned with status and dominance (Al-

Dujaili & Sharp, 2012; Sharp, 2006). Alas, the present research was only tested amongst men. Given the potential relevance of this thesis' findings upon evidence-based health policies which aim to reduce health inequalities within the Scottish population, it is essential to test the robustness of these results amongst female cohorts.

Moreover, considering the range of methodological limitations within existing research, and the novel approaches strategies adopted within this study, future scholarship should consider the adoption of similarly comprehensive sampling regimes. This in turn will produce more reliable results and perhaps put some order into the equivocal findings emerging from past psychoneuroendocrine literature. Furthermore, this will allow policies which hope to ameliorate or eradicate health disparities to build on more reliable and consistent results. If future studies were to apply comprehensive sampling regimes and meaningful baselines on a larger scale and across gender groups, it could provide profound knowledge that enhances the efficacy of psychosocial and behavioural interventions to tackle disparities in health. Additionally, applying rigorous interpretation of the current results and those of previous studies (i.e., interpreting result directions and acknowledging true effects rather than drawing conclusions based on dichotomous categories of significance) could also allow policies targeting eradication of health inequalities to be better informed. One way of tackling this interpretative challenge would be through thorough meta-analyses of previous studies.

The results of the study also aimed to enhance future health and public policies by providing biological and cognitive exploratory frameworks that have not been previously considered within the existing health inequalities literature, namely the challenge hypothesis/Mazur's biosocial model of status, and Biopsychosocial model of threat and challenge. Whilst this could contribute towards evidence-based policymaking which aims to reduce the ever-widening health gaps affecting contemporary Scottish society, future research might also wish to consider the application of other theoretical frameworks. Moreover, research on a larger scale could also take into consideration different moderators of the link between endocrine

response, social status and health such as bandwidth taxation, social capital, opportunities in life or number and frequency of social threats.

Moreover, the present study explored the androgenic and glucocorticoid systems as potential underpinning biological mechanisms for status syndrome. Given the prevalent role of testosterone and cortisol in the SES/hormone relationship, it is entirely appropriate to examine their role in psycho-social status contexts. Future research, however, should also examine the role of the serotonergic and dopaminergic systems as underpinning mechanisms of the social gradient in health due to their significant function in reward-seeking behaviours (Schmidt et al., 2020) and thereby potential involvement in competitive behaviour and status attainment.

Potential implications of social defeat upon individuals of low status are worthy of consideration. It may be that these implications are of significant relevance to the behavioural and motivational factors which maintain transgenerational cycle of SED. Were this to be the case, policies and interventions should strive to minimise negative life exposures. Indeed, policies relating to form-filling exercises could be created to minimise the risk of social defeat whilst increasing the promotion of more positive behavioural and biological outcomes (i.e., small wins). This may take the form of specific procedures, practices and methods which allow individuals applying for welfare support to get additional assistance and help with the process. More particularly, these practices could include: providing individuals with the required assistance whilst filling in the forms; offering a checking service prior to form submission; offering appointments when forms have been filled in incorrectly in order to assist people in submitting a correct application rather than simply rejecting and/or returning the forms back. This in turn, could contribute to individuals being more successful in the application process, thereby fostering greater feelings of motivation, control and desire to break the cycle of SED.

8.3 Conclusion

The theoretical conclusions reached by this research emphasize potential important contribution of neuroendocrine reactivity upon stress-related health gradient. Albeit a significant body of literature is concerned with the SES/cortisol relationship, there has hitherto been no research which endeavoured to elucidate the underpinning biological mechanisms of the theoretical model of status syndrome (Marmot, 2004). For this reason, by utilising a comprehensive sampling regime and by testing the endocrine component of Marmot (2004) and Wilkinson and Pickett's (2018) theoretical position on status syndrome, this research represents a significant step forward in our knowledge and understanding of bio-behavioural research and the social gradient in health. It makes advances on both methodological and theoretical grounds. Indeed, by addressing the first, second and third RQs, the research provides an empirical contribution to the better understanding of the mechanisms underpinning status syndrome and the link between health and status.

Furthermore, the findings addressing the last RQ, allow the study to put into a broader applied social policy context the importance of endocrine reactivity to status encounter and the cognitive variables moderating that link. The study thus emphasises the implications of social defeat upon individuals having low status within society, and the extent to which these implications could transfer into behavioural and motivational drives for the maintenance of the transgenerational cycle of SED. Considering the higher prevalence of negative life events and stressors amongst low SES groups (Gibson, 2020; Kim et al., 2018) – and an increased risk of social defeat (Sapolsky, 2017) which stems from repeated exposures to structural violence or societal inequities – policy initiatives should strive to minimise those exposures.

Importantly, despite the existing and significant attention paid to the chronic stress and corticoids literature, the present study aimed to explain further issues related to low SES such as aggression, hostility, risk taking, delinquency, status-seeking, fear, and impulsivity (Pepper & Nettle, 2017) adopting a dual-hormone approach. The findings of the study fail to demonstrate that glucocorticoid and androgenic systems each have implications for the relationship between health and status. Yet these

results remain exploratory and ought to be considered within the framework of significant sample size limitations. For that reason, further research on a larger scale is needed to examine the complex relationship between endocrine reactivity and health. Nevertheless, by exploring this topic and providing some preliminary findings, the present research made another substantial contribution to the literature of health inequalities. Finally, investigating chronobiological hormonal changes rather than static basal levels also allowed the research to address how modifications of endocrine parameters in relation to cognitions (i.e., threat/challenge) impact upon behaviours which may reinforcing aspects of SED. Considering the importance of the study findings for the evidence-based approaches and public policies tackling health disparities, it could be concluded that the study makes a substantial empirical and methodological contribution of knowledge to the field.

The biological bases of health inequalities are however, by no means easy to explicate and even harder to eradicate or even ameliorate due to their multifaceted causal nature and deep-seated roots within societal constructs. Future research that aims to close the health gap between poor and rich thus needs to firstly either consolidate or challenge the present preliminary findings within male and female populations, and reflect on the most appropriate psychological and environmental interventions. Perhaps future research might start with exploring the number of threatening events faced by lower SES groups on a daily basis and the implications of this for health, thereby testing whether SED individuals are firstly psychologically and physically disproportionately affected by social inequalities. This concern of course, could be further expanded to the theme of resilience and protective factors which can later translate into the key question concerning the extent to which biological parameters might be modified through behavioural or cognitive intervention in a manner that is enduring and improves quality of life. Furthermore, when, where and in whom should the intervention be developed? Any attempt to develop a coherent strategy for intervention has to be set on a solid foundational understanding of the neurobiology of health inequalities and SED. This study has demonstrated the extent to which these two issues are inextricably entwined. Focusing upon one without the other would merely confuse and obscure the true nature of their dynamic relationship and impact.

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APPENDICES

Appendix I

Demographics Questionnaire

SCHOOL OF SOCIAL WORK & SOCIAL POLICY

University of Strathclyde Humanities & Social Sciences

Demographics Questionnaire

Participant name: Postcode:

Participant ID: Date:

Age:

PLEASE INITIAL THE BOXES BELOW AND PROVIDE INFORMATION FOR THE OPEN-ANSWERED QUESTIONS

1. Please specify your current employment status and the duration of employment:

_____

_____

_____

2. Please choose your highest educational qualification:

- ☐ Primary Education
- ☐ Secondary education
- ☐ College qualification
- ☐ Undergraduate qualification
- ☐ Postgraduate qualification

3. Please indicate your annual income:

- ☐ £0-£4,999
- ☐ £5,000-£9,999
- ☐ £10,000-£19,999
- ☐ £20,000-£29,999
- ☐ £30,000-£39,999
- ☐ £40,000-£49,999
- ☐ £50,000 and over

4. Please specify your marital status:

- ☐ Single
- ☐ In a relationship
- ☐ In a civil partnership
- ☐ Married
- ☐ Divorced
- ☐ Widowed

5. Please identify the number of working individuals in your current family and their annual income (if applicable):

_____

_____

_____

Appendix II
Financial Questionnaire
(Experimental Task)

SCHOOL OF SOCIAL WORK & SOCIAL POLICY

University of Strathclyde
Humanities & Social Sciences

Version 2
23 May 2019
Appendix F

Financial Questionnaire

READ THESE INSTRUCTIONS BEFORE COMPLETING THE QUESTIONNAIRE

You are being asked to provide a clear, complete and accurate picture of your current and recent financial situation.This must reflect the situation of anyone who shares financial responsibility with you. Please notify investigator once form is completed.

HOW TO COMPLETE THIS FORM

Please ensure you only write inside the white boxed areas. **Do not mark or strike through any other areas of the form**. Please write in **BLACK INK** using **BLOCK LETTERS**. If an answer will not fit in the space provided, please enter your answer on the continuation sheet at the back of this form, giving the number of the question you are referring to. If you make a mistake, please **do not correct it but delete it by striking it through**. Please do not use correcting fluid.

Ensure you answer **ALL** the questions. You can use the abbreviation **NA (Not Applicable)**. **NA - If a question does not apply to you**, write **NA in the first two boxes only of the relevant question**. **Not Known - If you do not know the answer**, or **you cannot provide the information needed**, write **NOT KNOWN** in the first line **only** of the **relevant question**. Please **provide** an **explanation** why the information is unknown to you in the appropriate boxes or on the continuation sheet. **Unanswered questions** or **Not Known replies** may result in you failing this task.

1. Your Details

If you have been given a reference number to quote, please enter it here:

a. Title (Mr/ Mrs/ Ms/ Miss/ Dr/ Prof/ Rev/ Mx

b. Full Forenames c. Surname (now)

d. Date of Birth: *dd* *mm* *yyyy*

e. Staff or Service Number (if any)

f. Your National Insurance Number
This will be on your last payslip. For example, JH 21 90 42 D

g. Please specify your employment type:
(if you have more than one employment type (for example if you are a civil servant but also a military reservist), please give both.)

Royal Navy Army RAF MOD Civilian Other Civil Servant

Police Officer/ Civilian Staff Civil Nuclear Employee Civil Nuclear Contractor

Contractor Other

2. Your Address Details

Please provide details of your current address plus any other addresses at which you currently live, for example student accommodation or lodgings away from home.

Please also provide details of any other addresses at which you have lived during the last 6 years.

Please provide your current permanent home address first and work backwards.

Address 1:

Type of address:	Current or previous?	Date from:	Date to:
UK	Current	*mm* *yyyy*	*mm* *yyyy*
OVERSEAS	Previous		
BFPO			
No Fixed Abode			

Please fill in the fields which are applicable:

Flat number: **House number:** **House name:**

(BFPO addresses only) **Name/Rank/Number:** **BFPO Code:**

Address Line 1:

Address Line 2:

Address Line 3:

Town:

Country (or Regioun/Area/State for overseas addresses):

Postcode *(or equivalent, where applicable, for overseas addresses):*

Country *(if not UK or a BFPO address):*

Address 2:

Type of address: Current or previous? Date from: Date to:

UK Current *mm* *yyyy* *mm* *yyyy*

OVERSEAS Previous

BFPO

No Fixed Abode

Please fill in the fields which are applicable:

Flat number: **House number:** **House name:**

(BFPO addresses only) **Name/Rank/Number:** **BFPO Code:**

Address Line 1:

Address Line 2:

Address Line 3:

Town:

Country (or Region/Area/State for overseas addresses):

Postcode *(or equivalent, where applicable, for overseas addresses):*

Country *(if not UK or a BFPO address):*

Address 3:

Type of address:	Current or previous?	Date from:	Date to:
UK	Current	*mm* *yyyy*	*mm* *yyyy*
OVERSEAS	Previous		
BFPO			
No Fixed Abode			

Please fill in the fields which are applicable:

Flat number: **House number:** **House name:**

(BFPO addresses only) **Name/Rank/Number:** **BFPO Code:**

Address Line 1:

Address Line 2:

Address Line 3:

Town:

Country (or Region/Area/State for overseas addresses):

Postcode *(or equivalent, where applicable, for overseas addresses):*

Country *(if not UK or a BFPO address):*

Financial Information

3. General Circumstances

In the following questions you are being asked to provide a clear, complete and accurate picture of your current and recent financial situation. This must reflect the situation of anyone who shares financial responsibility with you.

Independent Credit Reference Checks and investigations into your financial circumstances will be carried out.

a. Are you living with someone? **Yes** **No**

b. If you have answered 'Yes', Does the person you live with share financial responsibility with you or make a contribution to your financial situation? **Yes** **No**

If you have answered 'Yes', it will be necessary to explore the impact of this person's contribution on your financial situation. Please include the requested information in the relevant sections of this form.

c. For whom do you have financial responsibility, other than yourself? *(For example, partner, children, parent etc.)*

d. What major financial changes to your budget do you anticipate in the next 12 months?

e. Have you been the victim of 'identity theft' or credit card fraud? **Yes** ☐ **No** ☐
If you have answered 'Yes', please provide details.

4. Financial Circumstances- Household Monthly Income

To enable an accurate assessment of household income, please provide a clear picture of ALL sources of earned and unearned income (unearned income being income derived from private means rather than from work, for example interest from bank accounts and dividends on shares etc.). If you live with more than one person who contributes to your financial situation, please add up their contributions and give them as a single figure against each of the categories under "Other Person(s)".

Do not leave fields blank. If they are not applicable, please enter zero.

	You	Other Person(s)
Total **annual** salary (gross)	£	£
Usual **annual** bonus (gross)	£	£
Annual car allowance (gross)	£	£
Monthly net salary (including average bonus and any allowances)	£	£
Monthly income from state benefits	£	£

Monthly income from pensions and annuities	£	£
Monthly income from savings and investments (averaged)	£	£
Monthly net income from rented properties	£	£
Monthly net income from rented properties	£	£
Monthly maintenance received	£	£
Total **Monthly** income	£	£
Total Household Income	£	£

Has all income been declared for tax purposes? **Yes** ☐ **No** ☐

(For example, rental income etc.)

If a fuller explanation of your income purposes would be helpful to us, please provide details here:

5. Financial Circumstances- Monthly Household Outgoing

Please provide details of your monthly household outgoings for yourself and your partner (if applicable).

Mortgage / Rent / Board & lodging	£
Service charge / Ground rent / Management fees	£
Council tax	£
Housekeeping *For example, food and household necessities*	£
Motoring costs (including tax, insurance, petrol & maintenance) / commuting and travel costs	£

Utility bills (gas, electricity, oil, water bills, telephones and mobile phones) £

Credit card repayments £

Loan repayments £

Satellite / Cable / TV licence / Internet access £

Maintenance payment for children £

Nursery fees / school fees /parental contribution to student costs £

Pensions / insurance (all insurance - excluding car insurance) £

Holidays (average per month of annual costs) £

Entertainment (including sports and gym club memberships, trips out etc.) £

Other regular commitments £
(e.g., lottery tickets, mail order catalogue, hire purchase agreements: please detail below)

Please provide details:

Total monthly outgoings: £

6. Financial Circumstances- Properties Owned

a. How many properties do you own, or partly own, in the UK and/or overseas?
This includes those with mortgages outstanding, buy-to-let properties and timeshares.

b. Please provide details of all the properties you own, or partly own. Use the continuation page to add extra properties.

First Property

Address:

Flat number: **House number:** **House name:**

Address Line 1:

Address Line 2:

Address Line 3:

Town:

County (or Region/Area/State for overseas addresses):

Postcode *(or equivalent, where applicable, for overseas addresses):*

Country:

Date of purchase: *mm* *yyyy* **Purchase price (£):**

Current market valuation (£): **Is this property mortgaged? Yes** **No**

If the property is mortgaged:

How many mortgages do you have on it?

For each mortgage, please state:

Mortgage 1

Type of mortgage	Mortgage lender	Date taken out	Term (months)	Initial amount (£)	Balance outstanding (£)
Repayment		*mm* *yyyy*			
Interest only		*mm* *yyyy*			
Flexible		*mm* *yyyy*			
Buy to let		*mm* *yyyy*			
Other		*mm* *yyyy*			

Mortgage 2

Type of mortgage	Mortgage lender	Date taken out	Term (months)	Initial amount (£)	Balance outstanding (£)
Repayment		*mm* *yyyy*			
Interest only		*mm* *yyyy*			
Flexible		*mm* *yyyy*			

Buy to let			mm	yyyy			
Other			mm	yyyy			

Mortgage 3

Type of mortgage	Mortgage lender	Date taken out	Term (months)	Initial amount (£)	Balance outstanding (£)
Repayment		mm yyyy			
Interest only		mm yyyy			
Flexible		mm yyyy			
Buy to let		mm yyyy			
Other		mm yyyy			

Does this property have any secured loans (other than mortgages) on it? Yes ☐ **No** ☐

If there are any loans secured on the property, please provide details of each loan:

	Lender	Date taken out	Initial amount (£)	Balance outstanding (£)
First loan:		mm yyyy		
Second loan:		mm yyyy		
Third loan:		mm yyyy		

Second Property

Address:

Flat number: **House number:** **House name:**

Address Line 1:

Address Line 2:

Address Line 3:

Town:

County (or Region/Area/State for overseas addresses):

Postcode *(or equivalent, where applicable, for overseas addresses):*

Country:

Date of purchase: mm yyyy Purchase price (£):

Current market valuation (£): Is this property mortgaged? Yes No

If the property is mortgaged:

How many mortgages do you have on it?

For each mortgage, please state:

Mortgage 1

Type of mortgage	Mortgage lender	Date taken out	Term (months)	Initial amount (£)	Balance outstanding (£)
Repayment		mm yyyy			
Interest only		mm yyyy			
Flexible		mm yyyy			
Buy to let		mm yyyy			
Other		mm yyyy			

Mortgage 2

Type of mortgage	Mortgage lender	Date taken out	Term (months)	Initial amount (£)	Balance outstanding (£)
Repayment		mm yyyy			
Interest only		mm yyyy			
Flexible		mm yyyy			
Buy to let		mm yyyy			
Other		mm yyyy			

Mortgage 3

Type of mortgage	Mortgage lender	Date taken out	Term (months)	Initial amount (£)	Balance outstanding (£)
Repayment		mm yyyy			
Interest only		mm yyyy			
Flexible		mm yyyy			
Buy to let		mm yyyy			
Other		mm yyyy			

Does this property have any secured loans (other than mortgages) on it? Yes ☐ No ☐

If there are any loans secured on the property, please provide details of each loan:

	Lender	Date taken out	Initial amount (£)	Balance outstanding (£)
First loan:		*mm* ☐ *yyyy* ☐		
Second loan:		*mm* ☐ *yyyy* ☐		
Third loan:		*mm* ☐ *yyyy* ☐		

7. Financial Circumstances – Savings and Other Assets

a. Do you (and your partner) have finances in savings accounts, ISAs, bonds, premium bonds, invested in shares, funds etc? **Yes ☐ No ☐**

If 'Yes', how much? **£** ☐

If 'No', please provide a brief explanation as to why you do not have any savings or investments:

b. How often do you save? **Regularly ☐ Occasionally ☐ Hardly ever ☐**

c. What is the valuation of your (and your partner's) vehicles? **£** ☐

d. What is the valuation of your (and your partner's) other assets? **£** ☐
(For example, jewellery, antiques, collector's memorabilia, artwork etc.)

e. Have you ever received a lump sum worth more than £5,000? **Yes ☐ No ☐**

If 'Yes', please provide details:

TOTAL OF ALL ASSETS

Please add up:

- **Total market valuation of all properties owned - Section 6c**
- **The value of your/your partner's savings accounts, ISAs, etc. – Section 7a**
- **The value of your/your partner's vehicles - Section 7c**
- **The value of your/your partner's other assets – Section 7d**

Total Assets (£)

8. Financial Circumstance- Loans

a. How many loan agreements (including student loans) do you have outstanding?
This figure should not include mortgages or loans secured on your properties.

b. Please provide details of each loan.

First loan:

Lender:	Purpose:	Amount: £	Date taken out:	Expiry date (end date of the loan):
			mm yyyy	mm yyyy

Monthly repayment: £ **Balance outstanding: £**

Second loan:

Lender:	Purpose:	Amount: £	Date taken out:	Expiry date (end date of the loan):
			mm yyyy	mm yyyy

Monthly repayment: £ **Balance outstanding: £**

Third loan:

Lender:	Purpose:	Amount: £	Date taken out:	Expiry date (end date of the loan):
			mm yyyy	mm yyyy

Monthly repayment: £ **Balance outstanding: £**

c. Total of loan balances outstanding: £

9. Financial Circumstances – Credit Cards

a. How many credit, store and charge card accounts do you (and your partner) have?
Please provide details of each card. If necessary, please use the continuation page.

Name of Issuer	Type		Credit limit (£)	Average monthly payment (£)	Balance outstanding(£)
1.	Credit card				
	Store card				
	Charge card				
2.	Credit card				
	Store card				
	Charge card				
3.	Credit card				
	Store card				
	Charge card				
4.	Credit card				
	Store card				
	Charge card				

b. Do you transfer balances between credit cards? Yes ☐ No ☐
(For example, 0% interest deals)

c.Total of Average Monthly Payments (including any listed on the continuation page): £

d.Total of card balances outstanding (including any listed on the continuation page): £

10. Financial Circumstances- Current Accounts: Overdrafts

a. In the last 5 years, how many times have you exceeded your overdraft limit(s)?

b. How many current accounts do you have which were overdrawn in the previous month?

TOTAL OF LIABILITIES

- **Total of mortgage/secured loan balances outstanding (£) – Section 6e**
- **Total of loan balances outstanding (£) – Section 8c**
- **Total of card balances outstanding (£) – Section 9d**

Total of All Liabilities (£):

11. Financial Circumstances- Accommodation Charges

a. Do you pay rent for any property? **Yes** ☐ **No** ☐

If you have answered 'No', please go to Section 12. If you have answered 'Yes':

b. How much do you pay per month? £

c. What other costs are you liable for (e.g. service charge, agent's fees, etc.)?

d. Do you share rent and utility costs with other tenants? **Yes** ☐ **No** ☐

e. Do you receive accommodation free of charge? **Yes** ☐ **No** ☐

If you have answered 'Yes', please provide details:

12. Financial Circumstances - Financial Obligations

a. Do you have any form of financial obligation in excess of £1,000 that has not been disclosed elsewhere on this form? **Yes** ☐ **No** ☐

If you have answered 'Yes', please provide details:

b. Have you ever lent or given anyone (including family members) more than £1,000? Yes ☐ No ☐

If you have answered 'Yes', please provide details:

c. If you stated at Section 3b that you share financial responsibility with another person, do they (to your knowledge) have debts in their sole name in excess of £5,000? Yes ☐ No ☐

If you have answered 'Yes', please provide details:

13. Financial Circumstances- Financial History/Arrangements

a. In the last 10 years have you and/or your partner.....

i. Had a credit or store card application declined? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

ii. Had a personal loan application declined? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

iii. Sought a reduction in monthly repayments with a lender? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

iv. Ever consolidated debt onto a new financial arrangement? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

v. Had a default or termination notice issued to you by a bank or creditor? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

vi. Had a credit or store card withdrawn? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

vii. Consulted a debt management company or advisory service? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

viii. Arranged a debt management plan (either formal or informal)? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

ix. Been subject to a county court judgement? Yes ☐ No ☐

if 'Yes': Number of times: ☐ Most recent year: ☐

x. Been subject to an attachment of earnings order? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

b. Have you and/or your partner ever:

i. Had repossession proceedings commenced against you? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

ii. Been investigated by HMRC or DWP? Yes ☐ No ☐

If 'Yes': Number of times: ☐ Most recent year: ☐

iii. Applied for an Individual Voluntary Arrangement (IVA), a Debt Relief Order (DRO) or (in Scotland only) Yes ☐ No ☐

a Protected Trust Deed or Minimal Asset Process (MAP) Bankruptcy or Low Income Low Asset (LILA) Bankruptcy?

If 'Yes': Number of times: Most recent year:

iv. Been declared bankrupt? Yes No

If 'Yes': Number of times: Most recent year:

v. Do you have any financial interests that may conflict with your duties as a Civil Servant, member of HM Forces, or as a Government contractor? Yes No

(For example, your partner runs a company which tenders for contracts that you are responsible for placing etc.)

If 'Yes', please provide details:

vi. Are there any details or explanations that would help us to understand your financial position better? Yes No

If 'Yes', please provide details:

14. Financial Circumstances- Self-Employed Contractors: Additional Information

a. Are you a self-employed contractor? Yes No

If you have answered 'No', please go to the final declaration. If you have answered 'Yes', please answer the following questions:

b. Are you being employed through an agency for the contracted work? Yes No

c. Are you self-employed and submit invoices in your personal/trading name? Yes No

d. Are your invoices submitted by a company,

of which you are the Yes primary director or shareholder? **Yes** ☐ **No** ☐

If you have answered 'No', please go to the final declaration. If you have answered 'Yes', please answer the remaining questions in Section 14.

Company name:

Company number: **Accounting date:** *mm* *yyyy*

e. Are your annual returns to Companies House up to date? **Yes** ☐ **No** ☐

If you have answered 'No', please provide details:

f. Are your annual accounts registered at Companies House up to date? **Yes** ☐ **No** ☐

If you have answered 'No', please provide details:

g. Are payments of VAT, tax and social security paid up to date, by their due date? **Yes** ☐ **No** ☐

If you have answered 'No', please provide details:

h. Has a late filing penalty been imposed on the company at any time? **Yes** ☐ **No** ☐

If you have answered 'Yes', please provide details:

i. Have the directors been fined for non-filing of documents at any time? **Yes** ☐ **No** ☐

If you have answered 'Yes', please provide details:

j. Has the company been subject to a county court judgement? Yes ☐ No ☐

If you have answered 'Yes', please provide details:

k. Have you ever been involved with a company that has undertaken a voluntary liquidation or creditors' winding up? Yes ☐ No ☐

If you have answered 'Yes', please provide details:

l. Have you ever been disqualified from being a company director? Yes ☐ No ☐

If you have answered 'Yes', please provide details:

m. Has the company been investigated by HMRC or the DWP? Yes ☐ No ☐

If you have answered 'Yes', please provide details:

15. Below are a number of questions and statements. Please indicate how often they describe. Use the numbers for each of the boxes.

Never=1 Rarely=2 Sometimes=3 Often=4 Always=5

Education

I know how interest works on my current debts. ☐

I feel financially educated. ☐

I feel well informed about financial matters. ☐

Relationships

There are disagreements about money in my home. ☐

I tend to argue with others about money. ☐

Financial problems hurt my relationships. ☐

My relationships with others are affected by financial problems. ☐

Physical

Are you ever unable to sleep well because of financial worries? ☐

Do you ever get headaches from worry over money matters? ☐

Do your muscles get tense when you add up your bills? ☐

Does your financial situation cause you to feel heartburn or an upset stomach? ☐

Appendix III

Threat/Challenge Questionnaire

SCHOOL OF SOCIAL WORK & SOCIAL POLICY

University of Strathclyde Humanities & Social Sciences

Threat/Challenge and Task Importance Questionnaire
Pre-Task Exposure

Participant name: Postcode:

Participant ID: Date:

Age:

1. Please indicate on the scale below, how you feel prior to completing the task:

Where: Threat may be associated with lack of emotional, psychological, or physical resources to cope with the task. **For example:** I find job interviews very threatening. I can't cope with them. I perform very badly at interviews.

While, Challenge may be associated with slight anxiety and fear of the task, however, sufficient emotional, physical or psychological resources to cope with the task. **For example:** I find job interviews quite challenging, however, I know I am good at them and I have the appropriate skills to manage with it and get this position.

Threatened				**Neither**				**Challenged**
-4	-3	-2	-1	0	+1	+2	+3	+4

2. How important is the upcoming task to you?

Not at all	A little	Moderately	Quite a bit	Very much so
0	1	2	3	4

Appendix IV

Motivational Questionnaire

SCHOOL OF SOCIAL WORK & SOCIAL POLICY

University of Strathclyde Humanities & Social Sciences

Motivational Questionnaire

Participant name: Postcode:

Participant ID: Date:

Age:

PLEASE INDICATE ON THE SCALES BELOW, HOW MOTIVATED YOU CURRENTLY FEEL TO ENGAGE WITH THE FOLLOWING ACTIVITIES:

1. Apply for a job:

Not at all	A little	Moderately	Quite a bit	Very much so
0	1	2	3	4

2. Complete a financial form:

Not at all	A little	Moderately	Quite a bit	Very much so
0	1	2	3	4

3. Seek help for funding matters:

Not at all	A little	Moderately	Quite a bit	Very much so
0	1	2	3	4

4. Join a finance wellbeing course, in order to acquire knowledge of how to improve your financial situation:

Not at all	A little	Moderately	Quite a bit	Very much so
0	1	2	3	4

5. Apply for mortgage/credit/loan:

Not at all	A little	Moderately	Quite a bit	Very much so
0	1	2	3	4

Appendix V

Recruitment Poster

University of Strathclyde
Humanities & Social Sciences

RESEARCH PARTICIPANTS NEEDED!

We are recruiting participants to investigate how filling forms changes our biology

Participate in a study that requires you to collect saliva samples on two separate days at home and on university premises (each less than 40 secs). You will also be asked to complete an experiment at the University of Strathclyde. This will take just a few hours of your day (this includes most of the questionnaires).

Travel Expenses covered !!!

You have 50% chance to win £25

In order to participate you should:

- ✓ **BE a healthy male**
- ✓ **BE aged between 20 and 40 yrs old**
- ✓ **BE employed for more than 2 yrs OR Unemployed for more than 12 months**
- ✓ **NOT be visibly obese**
- ✓ **NOT have severe acne, diabetes, or high blood pressure**
- ✓ **NOT be on a strict diet or seriously restricting calorific intake**
- ✓ **NOT have previous history of kidney or liver disease**
- ✓ **NOT have consumed illicit drugs for a period of one month before taking part in the study**
- ✓ **NOT take natural supplements which alter cortisol levels (e.g. Ashwagandha, fish oil, Rhodiola Rosea, Bacopa, Ginkgo, Cordyceps, phosphatidylserine, L-Theanine, prebiotic fiber supplements)**

You will be asked to complete several questionnaires about things like personality, how you perceive life, how you interpret events in everyday life, mood, workload, and motivation. You will be also asked a few questions about yourself (your age and educational level, for example). Some of these can be answered at home! Each questionnaire will take you between 5 and 20 min. During the experimental phase, you will be asked to complete a financial form (less than 40 min to complete).

Consumption of alcohol and illicit drugs, sexual activity and severe exercise during and 24hrs prior to saliva collection is not allowed.

Interested???

Contact me at:
Konstantina.karastoyanova@strath.ac.uk

Appendix VI

Recruitment Advert

SCHOOL OF SOCIAL WORK & SOCIAL POLICY

University of Strathclyde
Humanities & Social Sciences

Recruitment Advert

Dear,

The current research is concerned with the question how our biology changes when we perform certain everyday tasks. Specifically, how levels of the chemical messengers testosterone and cortisol (which circulate in our blood) change over time in response to filling in forms.

The study requires you to collect saliva samples on two separate days at home (each less than 40 secs). You will also be asked to complete an experiment at the University of Strathclyde. This will take just a few hours of your day. The study requires completion of several questionnaires about things like personality, how you perceive life, how you interpret events in everyday life, mood, workload, and motivation. You will be also asked a few questions about yourself (your age and educational level, for example). Some of the questionnaires can be completed at home. Each questionnaire will take you between 5 and 20 min. On the day of the experiment, you will be asked to complete a financial form (less than 40 min) and most of the aforementioned questionnaires.

Measuring hormones in saliva is relatively straightforward but levels can be affected by many different things. Thus, if you are interested in participation, you should first make sure you meet the inclusion criteria:

- Male
- You should be between 20 and 40 yrs old
- **Either** long-term employed (for at least 2 yrs with an income per annum of at least £30,000) **or** long-term unemployed.
- Not visibly obese
- You should not have severe acne, diabetes, or high blood pressure
- You should not be on a strict diet or seriously restricting calorific intake
- You should not have previous history of kidney or liver disease
- You should not have consumed illicit drugs for a period of one month before taking part in the study.
- You should not administer any natural supplements which alter cortisol levels (e.g., Ashwagandha, Rhodiola Rosea, Bacopa, Ginkgo, fish oil, phosphatidylserine, L-Theanine, prebiotic fiber supplements). If you are not sure, please ask the researcher.
- During saliva collection, you will be asked not to consume alcohol and illicit drugs for the previous 24 hrs and also refrain from sexual activity.

SCHOOL OF SOCIAL WORK & SOCIAL POLICY

University of Strathclyde
Humanities & Social Sciences

If you are interested, please contact me at the address below. If you participate, you have the chance to win £25! You would be under no obligation to take part in the study.

Investigator: Konstantina V. Karastoyanova

Email: Konstantina.karastoyanova@strath.ac.uk

Appendix VII

Consent Form

Consent Form

Name of department: Social Policy
Title of the study: Form Filling: Does it Change Our Biology?

- I confirm that I have read and understood the Participant Information Sheet for the above project and the researcher has answered any queries to my satisfaction.
- I confirm that I have read and understood the Privacy Notice for Participants in Research Projects and understand how my personal information will be used and what will happen to it (i.e., how it will be stored and for how long).
- I understand that my participation is voluntary and that I am free to withdraw from the project at any time, up to the point that the study is completed, without having to give a reason and without any consequences.
- I understand that anonymised data (i.e., data that do not identify me personally) cannot be withdrawn once they have been included in the study.
- I understand that any information recorded in the research will remain confidential and no information that identifies me will be made publicly available.
- I consent to being a participant in the project.
- I consent to the taking of salivary samples from me and understand that they will be the property of the University of Strathclyde.
- I consent to my data being used for research purposes including: academic conferences, scientific journal publications and be used for researcher's PhD thesis
- I consent to my anonymised psychological and demographic data to be submitted to Hogrefe Ltd in scored form.

To be completed before participation in the investigation:

(PRINT NAME)	
Signature of Participant:	Date:

Appendix VIII
Baseline Collection Schedule

SCHOOL OF SOCIAL WORK & SOCIAL POLICY

University of Strathclyde
Humanities & Social Sciences

Baseline Collection Schedule

Participant name: Postcode:

Participant's ID: Date:

Age:

1. These baseline measurements should be taken 48 hours prior task exposure. For example, if your task exposure is on a **Friday** these measurements should be taken on the **preceding Wednesday**.

2. To work out sample collection times determine when your task exposure is likely to start (**e.g., 3pm on a Friday**). This means you will have to collect your first saliva sample at 9am. Then, the next samples will be at 1hr prior the expected task exposure **(e.g., 2pm),** 30min prior (**e.g. 2.30pm)** and immediately prior expected task (**e.g. 2.59pm)**.

3. Please follow the saliva collection procedure detailed earlier **(check General Instructions for Collecting Saliva)**.

4. Please ensure that the **number**, **day** and **time** on the **collection container** correspond to the **collection event**- you will be supplied with a bag marked **BASELINE**.

5. Please return the collection tubes to the investigator as soon as possible. **Samples should be refrigerated until their return.**

6. Please **do not exercise**, **engage in sexual activity**, or **consume alcohol** prior to or during the saliva collection.

Sample one	Sample two	Sample three	Sample four
9am	1 hr prior to task exposure	30 mins prior to task exposure	Immediately prior to Start of Task

Use empty cells to indicate time of actual saliva collection

Appendix IX

General Instructions for Collecting Salivary Samples

University of Strathclyde
Humanities & Social Sciences

General Instructions for Collecting Salivary Samples

Participant name: Postcode:

Participant's ID: Date:

Age:

Thank you for agreeing to participate in this study investigating the salivary hormonal fluctuation throughout the course of a day. The study will involve you collecting a total of **12 small saliva samples**. The following sheets contain **all the information** you require to participate in this study. **Please** accurately **follow the step-by-step procedures** detailed below.

Conditions of Collection

On the days that you collect saliva there are **two conditions** you will need to adhere to. Your testosterone levels are affected, in an **unpredictable manner**, by both **exercise** and **alcohol**. Consequently, it is **extremely** important that you **do not exercise** or **drink alcohol** in the **24 hours** before collection or during collection of saliva samples themselves. Exercise is taken to mean any **organised physical activity** such as **swimming**, **cycling**, **yoga** or **going to the gym**.

Collection of Samples

Each time you collect saliva **the procedure** should be **identical.** The steps you should follow are outlined below. In order to prevent tiny cuts or micro-abrasions to the mouth please **do not eat** or **brush your teeth** for approximately **30 minutes** prior to saliva collection.

Step 1. Rinse and swill mouth out thoroughly three times with tap/filter water.

Step 2. Wait three minutes for the environment in your mouth to normalise.

Step 3. Chew on quarter of a stick of the sugar free gum provided. This will aid you in producing saliva and the step **must** be included.

Step 4. While retaining the gum in your mouth, spit away the first mouthful of saliva. This will get rid of unwanted cellular elements in the gum.

Step 5. Continue chewing on gum and spitting in the tube until you have deposited 5mL of saliva into collection container. Please then ensure caps are replaced tightly.

You will also be provided with a **timetable** for collecting samples. Although it is not a problem if you begin collecting **5 minutes either side of the specified time** please try and get as close to them as possible and mark down the time you collected the sample. If any event occurs in the two hours **prior** to each collection, which stresses or upsets you, which makes you especially happy or which might in any way be considered put of the ordinary then **please make a note** of it in the right hand comments column. This might include events such as **unavoidable physical exercise**, **arguments**, **stress** and

sexual activity. Finally, please **ensure** that the **number**, **day** and **time** on the collection container correspond to the collection event.

Storage of Samples

Because you may wish to collect samples whilst out and about you will need to give a little thought to how the samples are stored. Once you have collected a sample (**for example 11am**) you can do one of two things with it. Either, place it in a **fridge immediately**, or keep it with you in a **cool place** (preferably below room temperature). If you are going to choose this option then please keep the sample **out of direct sunlight** and **don't let it get too hot** (radiators are definitely to be avoided). Once you have collected all the samples for a particular day they will need to be placed in a **fridge overnight** or until you are ready to return them.

Handing Samples Back

Please contact **Konstantina Karastoyanova** via **email** to arrange a suitable time for sample **collection/delivery**. Should you experience any difficulties with the procedure then please do not hesitate to contact me.

Payment

You will be paid £25 if you complete the experimental task successfully and return all saliva samples to the investigator

Investigator's Email: Konstantina.karastoyanova@strath.ac.uk

Appendix X

Questions (daily activities) for Baseline Collection

Questions (daily activities) for Baseline Collection

Participant name: Postcode:

Participant ID: Date:

Age:

1. Have you drunk alcohol at any point during the previous **twenty-four hours** (if yes, how much)?

_____

_____

_____

2. Have you engaged in any sexual activity **during** the collection times?

_____

_____

_____

3. Have you fasted during the **course** of the **day**?

_____

_____

_____

4. Has your diet **changed** from that of a normal day?

_____

_____

_____

Appendix XI

Debrief Sheet

Debrief Sheet

Name of department: Social Policy
Title of the study: Form Filling: Does it Change Our Biology?

Thank you very much for participation in the current experiment.

As you know, the aim of the study was to observe how levels of hormones (specifically testosterone and cortisol) change in response to a form-filling task. It was anticipated the task would be quite stressful once participants learned they would be competing against another person and they would see it as either threatening or challenging. The experiment deliberately utilised deception in order to trigger an acute physiological stress response amongst participants. Any information about the competition, given prior the day of task exposure, could have potentially jeopardized any endocrine response to the form-filling competition; this results from participants' cognitions about status. Additionally, the participants were not being deliberately mislead about the nature of the study (i.e., the study actually does concern endocrine reactivity and financial form completion). Deception was an integral part of the study design and it was adapted to promote scientific validity. However, if you are not comfortable with the experimental deception, you have the right to withdraw from the experiment and all your data will be immediately destroyed.

The competitor was actually a confederate of the researcher who was instructed to manipulate the outcome of the study so that only half of the participants were successful. We expect to find that those who were successful demonstrate a rise in testosterone and levels in those who were unsuccessful will fall. The difference in the levels – high or low – should be related to how participants felt after the experimental task.

To look at differences in whether participants saw this task as a threat or challenge the unemployed group, also classified as socio-economically disadvantaged (based on several poverty indicators from the demographics questionnaire), were compared to a group who were employed and had fewer financial pressures (poverty indicators also applied). It is possible that cortisol (which is a stress hormone) and testosterone (which is related to drive/motivation/desire to compete) will differ between the two groups and the researcher is interested in whether this is related to how the task was understood (threat or challenge).

How does this information allow us to help you? Form-filling related to unemployment benefit can be stressful. We know that long term stress is not good for peoples mental or physical health. The first phase of designing policies that may help the unemployed lower socioeconomic status groups by reducing stress is to demonstrate the entire process has a negative effect on biology (or positive if they are successful at tasks).

The £25 cash reward was there to increase feeling of social status following success. To ensure that no participants were actually disadvantaged those who were in the unsuccessful condition were also given £25 after all data had been collected.

All your physical and electronic data will be securely stored and safeguarded by passwords for the next 5 yrs (at university premises). The information that you have completed as part of the finance form will be immediately destroyed via secure shredding at the university confidential waste service. None of the data provided in the finance form will be used for the investigation.

If any negative emotions of psychological distress were experienced during your participation in this investigation, or, if you would like to discuss with someone your wellbeing you may refer to the below-mentioned **contacts for further support:**

- For Health Information and Self-Care Advice, please contact **NHS 24** on **111**.
- Please contact **Samaritans Glasgow** on **0141 248 4488**, by **email** jo@samaritans.org , or just visit **www.samaritans.org** for emotional support for anyone in emotional distress, struggling to cope or at risk of suicide.
- Please contact **Citizens Advice** on **0141 552 5556** or at **3 Mitchell Library, 201 North Street, Glasgow, G3 7DN**, for free confidential information and advice for money, legal, consumer and other problems.
- Please contact **Breathing Space** on **0800 83 85 87**, for free confidential phone service for anyone in Scotland experiencing low mood, depression or anxiety.
- In case of emergency, you can also contact **Campus Security** via calling **0141 548 2222**.

To be completed after participation in the investigation:

(PRINT NAME)	
Signature of Participant:	Date:

THANK YOU, Konstantina

Appendix XII

Participation Information Sheet

Participant Information Sheet

Name of department: Social Policy
Title of the study: Form Filling: Does it Change Our Biology?

Introduction
The investigation is being carried out by a doctoral student - Konstantina V. Karastoyanova - at the University of Strathclyde.

Before you decide whether or not to take part, it is important for you to understand what participation in the study will involve for you. Please take time to read the following information carefully. You can direct any questions you may have to the researcher conducting the study, who will be happy to answer any queries.

What is the purpose of this research?
You are invited to take part in a research study which is trying to understand how our biology changes when we perform certain everyday tasks. Specifically, how levels of the chemical messengers testosterone and cortisol (which circulate in our blood) change over time in response to filling in forms. These chemical messengers help the brain communicate with other parts of the body and can have an effect on our mental and physical health. This study aims to better understand these processes.

Do you have to take part?
No. It is entirely up to you to decide whether or not to take part in the investigation. You can withdraw from the study at any time, without giving a reason.

What will you do in the project?
You will be asked to collect saliva samples, complete questionnaires and fill in a form of a financial nature in order to help us understand how hormones and cognitions are in involved in and impact daily activities.

- You will be asked to complete several questionnaires about things like personality (e.g. do you worry about things, do you make friends easily), how you interpret events in everyday life (e.g. how trouble sleeping affects you), how you perceive life (e.g. how do you imagine your life in the future), mood (e.g. ranking how cheerful or sad you feel), workload (e.g. how mentally or physically hard you felt the experimental task was), motivation (e.g. how motivated you are to complete tasks). You will be also asked a few questions about yourself (age, education, marital status, for example). The estimated time for completion of each questionnaire is about 5-20 minutes over the course of two days, but this will vary individually.

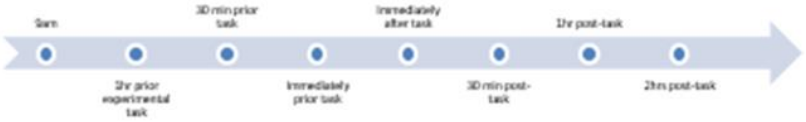
- You will be asked to provide several salivary samples (12 samples; approx. 3mL deposit each) over the course of two days. On the days that you collect saliva there are two conditions you will need to adhere to. It is extremely important that you do not exercise or drink alcohol in the 24 hours before collection or during collection of salivary samples. Exercise is taken to mean any organised physical activity such as swimming, cycling, or going to the gym. Each time you collect saliva the

procedure should be identical. The steps you should follow are outlined in the General Instructions for Collecting Salivary Samples. You will also be provided with a timetable for collecting samples. Baseline salivary measurements should be taken 48 hours prior the experimental task and thus, if your task is on a **Friday** these measurements should be taken on the **Wednesday**:

- To work out sample collection times determine when your task exposure is likely to start (**e.g., 3pm on a Friday**). This means you will always have to collect your first saliva sample at 9am. Then, the next samples will be at 1hr prior the expected task exposure **(e.g., 2pm),** 30min prior (**e.g., 2.30pm)** and immediately prior expected task (**e.g., 2.59pm)**. On the experimental day, you will collect samples at the same times as the baseline, plus additional 4 samples. Additional information and materials for collecting salivary samples (salivary collection tubes, sugar-free chewing gum to increase saliva levels, and plastic bags) will be provided by the investigator, after your consent.

First day (baseline), sample collection timeline:

Second day (experimental task exposure), sample collection timeline:



- You will be asked to come to the university to complete a financial form (it will take less than 40 min to complete and it comprises of questions related to your financial circumstances). If you complete the form successfully you will receive £25. Not everyone will manage this.

- To complete the experimental phase (complete questionnaires, fill in a financial form and collect all saliva samples on the second day), you will have to come to the University of Strathclyde, this will take approximately 4 hrs of your day. Each questionnaire will take you between 5 and 20 minutes to complete.

- Travel expenses (i.e., bus fare) will be provided.

Why have you been invited to take part?
Male participants from different socioeconomic and age groups are invited to participate in the study. To be

eligible you need to be long-term employed (for at least 2yrs) and earning more than £30 000 per annum which potentially makes you a suitable candidate for one of the groups. You will be asked about this in the exclusion criteria and this will be confirmed when you sign the informed consent form

Measuring hormones in saliva is relatively straightforward but levels can be affected by many different things. This means the study has several things that might not allow you to take part (exclusion criteria).

- Participants should be long-term employed (for at least 2yrs) and earning more than £30 000 per annum.
- Participants should be aged between 20 and 40 yrs old.
- Participants should not be visibly obese
- Participants should not have severe acne, diabetes, or high blood pressure
- Participants should not be on a strict diet or seriously restricting calorific intake
- Participants should not have previous history of kidney or liver disease
- Participants should not have consumed illicit drugs for a period of one month before taking part in the study.
- Participants should not administer any natural supplements which alter cortisol levels (e.g., Ashwagandha, Rhodiola Rosea, Bacopa, Ginkgo, Cordyceps, fish oil, phosphatidylserine, L-Theanine, prebiotic fibre supplements). If you are not sure, please ask the researcher.

During saliva collection, you will be asked not to consume alcohol or illicit drugs for the previous 24 hrs and also refrain from sexual activity. If you think you meet the criteria and want to participate in the study, please refer to the researcher in order to discuss and confirm your eligibility. Unfortunately, if you do not meet the criteria, you will be unable to further participate in the investigation.

What are the potential risks to you in taking part?
Apart from not being able to do the things listed earlier then there are no expected disadvantages to taking part in this study.

What information is being collected in the project?
The psychological and hormone data collected from you in this research will remain completely confidential. The data will be collected, stored and safeguarded on university computers which will be accessed only by the researcher. All data will be coded, which means you cannot be identified by anyone except the researcher. Data will be saved and stored for 5yrs and used for research purposes.

Who will have access to the information?
The researcher will be the only person with access to personally identifiable data. Each participant will be given a number and data stored with that number and not names. Anonymised psychological and demographic data will be shared with Hogrefe Ltd (Test Agency distributing psychological instruments across UK).

Where will the information be stored and how long will it be kept for?
The hormone data will be stored as a paper printout (with a number and not your name) in a locked cabinet at the University of Strathclyde for a period of 5 years. The data from the printout will be put into an electronic form which will be stored in a university network storage facility. You cannot be identified form this data. Physical copies of psychological data will also be stored in a locked cabinet at the university. Whilst electronic data will be de-identified and stored in university network storage. All data will be destroyed after 5 years using the Universities secure and confidential waste disposal service.

What happens next?
If you would like to participate in the study after reading this information sheet, please contact the researcher for additional information. This might be done either by email or personal contact. At the stage, you will be asked to give consent verbally and in writing to your voluntary participation. If you do not wish to participate, thank you for taking the time to read through this information sheet.

Once the study is completed the results will be presented at academic conferences, published in scientific journals, and be used for Konstantina's PhD thesis. Participants will not be able to see their individual data but a general information sheet will be provided describing and explaining the findings of the whole group.

Researcher contact details:
Name: Konstantina V. Karastoyanova
Address: 141 Saint James Road (Lord Hope Building), University of Strathclyde
Email: Konstantina.karastoyanova@strath.ac.uk

Chief Investigator details:
Name: Prof Ian Greener
Address: 141 Saint James Road
School of Social Work and Social Policy, Lord Hope Building, University of Strathclyde
Phone number: 0141 444 8754
Email: ian.greener@strath.ac.uk

This research was granted ethical approval by the University of Strathclyde Ethics Committee.

If you have any questions/concerns, during or after the research, or wish to contact an independent person to whom any questions may be directed or further information may be sought from, please contact:

Secretary to the University Ethics Committee
Research & Knowledge Exchange Services
University of Strathclyde
Graham Hills Building
50 George Street
Glasgow
G1 1QE

Telephone: 0141 548 3707
Email: ethics@strath.ac.uk

Appendix XIII

Optimised Testosterone and Cortisol Assay Protocols

Final cortisol and testosterone assay protocols have been optimised as follows:

Samples Preparation

Step 1. Once collected, samples are stored and preserved at –80 °C for at least 24hrs.

Step 2. Frozen samples are thawed (overnight), centrifuged at 3000rpm for 10mins, and prepared for aliquoting, each sample in duplicates of 1mL Eppendorf tube.

Step 3. Aliquots are then stored and preserved at -80°C.

Step 4. Prior to analysis, aliquots stand at room temp for 30mins and finally vortex mix to equilibrate.

Plate Preparation

(2) Cortisol

Step 1. Coat each well on plate with C conjugate (**100µl** of **5µl/ml)** GAM in sodium bicarbonate buffer (pH 9.6- coating buffer) and leave to incubate overnight at 4 °C.

Step 2. Wash plate 2 times with wash buffer (300mL) and dry.

Step 3. Block each well of plate with **220µl** of 0.5% bovine serum albumin (BSA) in phosphate buffer (PBS) solution, pH 7.4, for 1 hour at room temperature with shaking.

Step 4. Wash plate 2 times with wash buffer.

(2) Testosterone

Step 1. Coat plates with T conjugate in sodium bicarbonate coating buffer (pH 9.6), and leave to incubate overnight at 4°C.

Step 2. Wash 2 times with wash buffer and dry.

Step 3. Block for one hour at room temperature.

Step 4. Wash plate 2 times with wash buffer.

Elisa Procedure

(2) Cortisol

Step 1. Pipette **20µl** of previously extracted sample or standard and **80µl** of 1 in 4000 of Cortisol-HRP conjugate in 0.1% BSA PBS (assay buffer) to each well according to plate plan. Samples were added in duplicate. Standards are run at zero, 0.78, 1.56, 3.12, 6.25, 12.5, 25.0, 50.0 **ng/mL**. Utilise programme 24 on the Hamilton Microlab diluter.

Step 2. Mix briefly and then add **50µl** of mouse antibody at concentration 1:20,000 in 0.1% assay buffer (**6mL** of assay buffer and **3µl** of antibody).

Step 3. Incubate for 2 hours at 28°C with shaking.

Step 4. Wash plate 4 times with wash buffer.

Step 5. Add **120µl** of tetramethylbenzidine (at room temperature) to each well. Cover and leave in the dark for 15 minutes with shaking *(Expected o oD = 1.0-1.1)*.

Step 6. Add **80µl** of 1N sulphuric acid to each well to stop the reaction.

Step 7. Read with SoftMax Pro (Version 7.1, Molecular Devices) ELISA reader with filter of 450nm.

(2) Testosterone

Step 1. Pipette **50µl** of previously extracted sample or standard and **50µl** of Testo-HRP (ASTRA Biotech) in diluted assay buffer to each well according to plate plan. Samples were added in duplicate. Standards are run at zero, 20, 40, 80, 160, 320, 640, 1280 **pg/mL**. Utilise programme 24 on the Hamilton Microlab diluter.

Step 2. Mix briefly and then add **50µl** of Testo-Ab (R3S07-259, Meridian Life Science) in diluted assay buffer.

Step 3. Shake and incubate at 28˚C for 2hrs.

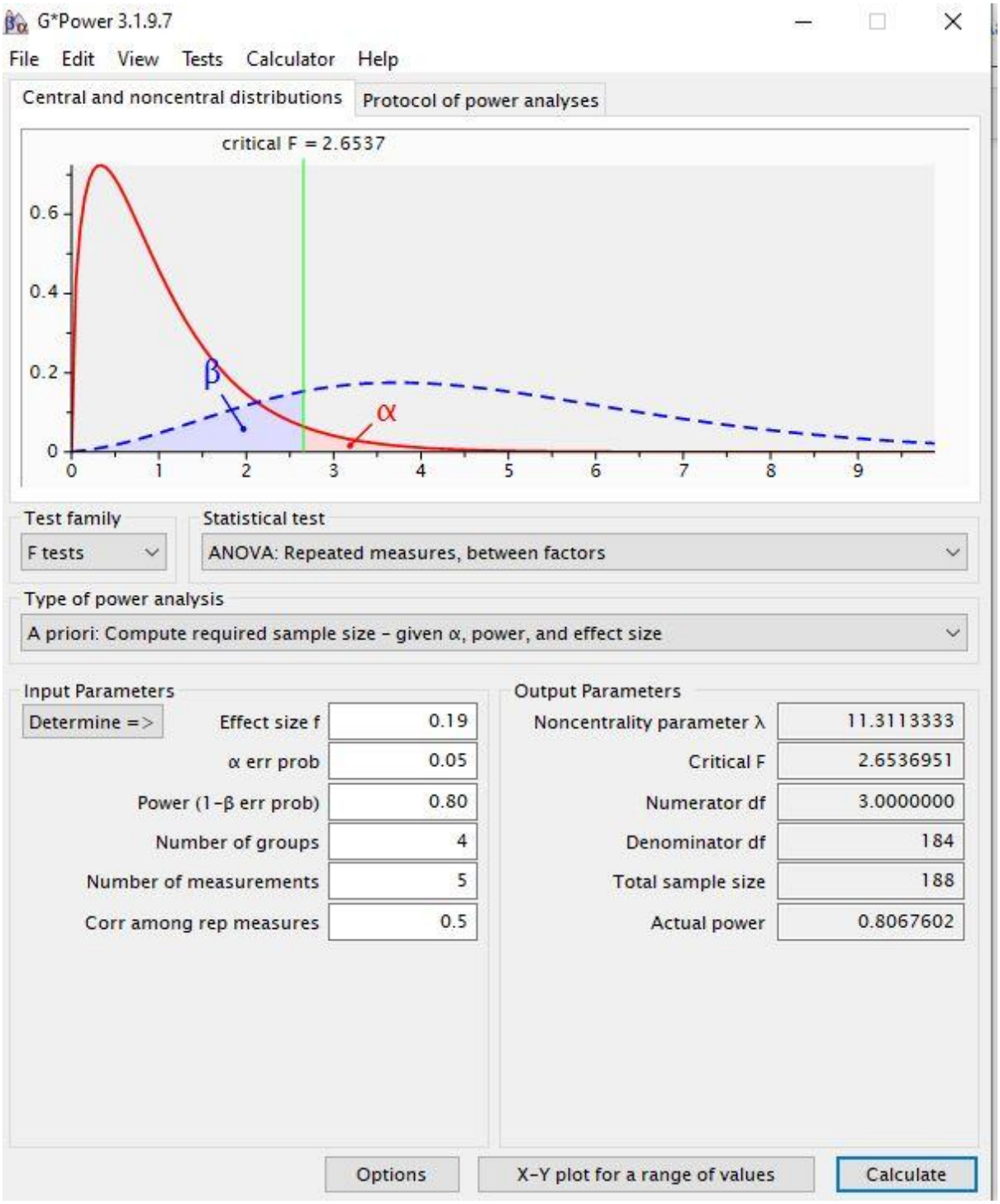
Step 4. Discard and wash 4 times

Step 5. Add **120µl** of tetramethylbenzidine (at room temperature) to each well. Cover and leave in the dark for 15 minutes with shaking.

Step 6. Add **80µl** of 1N sulphuric acid to each well to stop the reaction.

Step 7. Read with SoftMax Pro (Version 7.1, Molecular Devices) ELISA reader with filter of 450nm.

Appendix XIV
Power Analysis


G*Power 3.1.9.7
File Edit View Tests Calculator Help
Central and noncentral distributions
Protocol of power analyses
critical F = 2.6537
0.6
0.4
0.2
0
β
α
0 1 2 3 4 5 6 7 8 9
Test family
F tests
Statistical test
ANOVA: Repeated measures, between factors
Type of power analysis
A priori: Compute required sample size – given α, power, and effect size
Input Parameters
Determine =>
Effect size f 0.19
α err prob 0.05
Power (1-β err prob) 0.80
Number of groups 4
Number of measurements 5
Corr among rep measures 0.5
Output Parameters
Noncentrality parameter λ 11.3113333
Critical F 2.6536951
Numerator df 3.0000000
Denominator df 184
Total sample size 188
Actual power 0.8067602
Options
X-Y plot for a range of values
Calculate

Appendix XV
Ethics Approval

From: Ethics
Sent: 03 July 2019 10:47
To: Konstantina Karastoyanova; Philip Winn
Cc: Ian Greener; Henry Burns; Ethics
Subject: RE: UEC19/18 Greener/Burns/Karastoyanova: Salivary testosterone and cortisol reactivity to threat/challenge cognitions amongst unemployed and employed males

Dear Konstantina

Your application has now been approved and you will receive the formal approval email shortly.

The Committee made the following comments:

- Point 5: The poster could be more user-friendly and it is suggested that you make it more appealing and easier to understand. You may find this guide useful: http://www.ethicsguidebook.ac.uk/Writing-information-leaflets-166
- Point 7: It is suggested that you keep track of how participants come to you so that you can check whether there are any outcome differences that are explained by route-of-recruitment.

Let me know if you need any more information.

Regards

Angelique

Appendix XVI
Baseline and Task Phase Testosterone Data Analysis

Within-Subjects Factors

Measure: T_levels

time	phase	Dependent Variable
1	1	Baseline_9am_testo_normalised
	2	Exp_9am_testo_normalised
2	1	Baseline_1hr_testo_normalised
	2	Exp_1hr_testo_normalised
3	1	Baseline_30min_testo_normalised
	2	Exp_30min_testo_normalised
4	1	Baseline_imm_testo_normalised
	2	Exp_imm_testo_normalised

Descriptive Statistics

	Mean	Std. Deviation	N
9am baseline testo	.9125	.13488	28
9am day of competition testo	.9663	.07482	28
1hr prior baseline testo	.7570	.17011	28
1hr prior competition testo	.7297	.17946	28
30 min prior baseline testo	.7449	.22698	28
30 min prior competition testo	.7040	.17124	28
immediately before baseline testo	.6955	.21167	28
immediately before competition testo	.7301	.16564	28

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.567	10.910[b]	3.000	25.000	.000	.567	32.730	.997
	Wilks' Lambda	.433	10.910[b]	3.000	25.000	.000	.567	32.730	.997
	Hotelling's Trace	1.309	10.910[b]	3.000	25.000	.000	.567	32.730	.997
	Roy's Largest Root	1.309	10.910[b]	3.000	25.000	.000	.567	32.730	.997
phase	Pillai's Trace	.001	.031[b]	1.000	27.000	.862	.001	.031	.053
	Wilks' Lambda	.999	.031[b]	1.000	27.000	.862	.001	.031	.053
	Hotelling's Trace	.001	.031[b]	1.000	27.000	.862	.001	.031	.053
	Roy's Largest Root	.001	.031[b]	1.000	27.000	.862	.001	.031	.053
time * phase	Pillai's Trace	.126	1.202[b]	3.000	25.000	.330	.126	3.605	.282
	Wilks' Lambda	.874	1.202[b]	3.000	25.000	.330	.126	3.605	.282
	Hotelling's Trace	.144	1.202[b]	3.000	25.000	.330	.126	3.605	.282
	Roy's Largest Root	.144	1.202[b]	3.000	25.000	.330	.126	3.605	.282

a. Design: Intercept
Within Subjects Design: time + phase + time * phase

b. Exact statistic

c. Computed using alpha =

Mauchly's Test of Sphericity[a]

Measure: T_levels

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.556	15.103	5	.010	.741	.810	.333
phase	1.000	.000	0	.	1.000	1.000	1.000
time * phase	.897	2.796	5	.732	.933	1.000	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept
Within Subjects Design: time + phase + time * phase

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: T_levels

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Sphericity Assumed	1.925	3	.642	19.992	.000	.425	59.977	1.000
	Greenhouse-Geisser	1.925	2.222	.866	19.992	.000	.425	44.429	1.000
	Huynh-Feldt	1.925	2.431	.792	19.992	.000	.425	48.594	1.000
	Lower-bound	1.925	1.000	1.925	19.992	.000	.425	19.992	.991
Error(time)	Sphericity Assumed	2.599	81	.032					
	Greenhouse-Geisser	2.599	60.003	.043					
	Huynh-Feldt	2.599	65.627	.040					
	Lower-bound	2.599	27.000	.096					
phase	Sphericity Assumed	.001	1	.001	.031	.862	.001	.031	.053
	Greenhouse-Geisser	.001	1.000	.001	.031	.862	.001	.031	.053
	Huynh-Feldt	.001	1.000	.001	.031	.862	.001	.031	.053
	Lower-bound	.001	1.000	.001	.031	.862	.001	.031	.053
Error(phase)	Sphericity Assumed	1.261	27	.047					
	Greenhouse-Geisser	1.261	27.000	.047					
	Huynh-Feldt	1.261	27.000	.047					
	Lower-bound	1.261	27.000	.047					
time * phase	Sphericity Assumed	.090	3	.030	1.737	.166	.060	5.211	.438
	Greenhouse-Geisser	.090	2.798	.032	1.737	.170	.060	4.859	.421
	Huynh-Feldt	.090	3.000	.030	1.737	.166	.060	5.211	.438
	Lower-bound	.090	1.000	.090	1.737	.199	.060	1.737	.246
Error(time*phase)	Sphericity Assumed	1.394	81	.017					
	Greenhouse-Geisser	1.394	75.533	.018					
	Huynh-Feldt	1.394	81.000	.017					
	Lower-bound	1.394	27.000	.052					

a. Computed using alpha =

Tests of Within-Subjects Contrasts

Measure: T_levels

Source	time	phase	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear		1.367	1	1.367	27.458	.000	.504	27.458	.999
	Quadratic		.476	1	.476	13.926	.001	.340	13.926	.949
	Cubic		.081	1	.081	6.604	.016	.197	6.604	.698
Error(time)	Linear		1.345	27	.050					
	Quadratic		.923	27	.034					
	Cubic		.332	27	.012					
phase		Linear	.001	1	.001	.031	.862	.001	.031	.053
Error(phase)		Linear	1.261	27	.047					
time * phase	Linear	Linear	.004	1	.004	.213	.648	.008	.213	.073
	Quadratic	Linear	.086	1	.086	3.752	.063	.122	3.752	.463
	Cubic	Linear	.000	1	.000	.027	.870	.001	.027	.053
Error(time*phase)	Linear	Linear	.451	27	.017					
	Quadratic	Linear	.618	27	.023					
	Cubic	Linear	.326	27	.012					

a. Computed using alpha =

Tests of Between-Subjects Effects

Measure: T_levels

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	136.284	1	136.284	3139.830	.000	.991	3139.830	1.000
Error	1.172	27	.043					

a. Computed using alpha =

Estimated Marginal Means

1. time

Estimates

Measure: T_levels

time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.939	.019	.901	.978
2	.743	.023	.696	.791
3	.724	.028	.667	.782
4	.713	.029	.654	.772

Pairwise Comparisons

Measure: T_levels

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	.196*	.036	.000	.094	.299
	3	.215*	.041	.000	.098	.332
	4	.227*	.041	.000	.109	.344
2	1	-.196*	.036	.000	-.299	-.094
	3	.019	.023	.959	-.045	.083
	4	.031	.031	.911	-.057	.118
3	1	-.215*	.041	.000	-.332	-.098
	2	-.019	.023	.959	-.083	.045
	4	.012	.027	.999	-.064	.087
4	1	-.227*	.041	.000	-.344	-.109
	2	-.031	.031	.911	-.118	.057
	3	-.012	.027	.999	-.087	.064

Based on estimated marginal means

*. The mean difference is significant at the

b. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.567	10.910[a]	3.000	25.000	.000	.567	32.730	.997
Wilks' lambda	.433	10.910[a]	3.000	25.000	.000	.567	32.730	.997
Hotelling's trace	1.309	10.910[a]	3.000	25.000	.000	.567	32.730	.997
Roy's largest root	1.309	10.910[a]	3.000	25.000	.000	.567	32.730	.997

Each F tests the multivariate effect of time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha =

2. phase

Estimates

Measure: T_levels

			95% Confidence Interval	
phase	Mean	Std. Error	Lower Bound	Upper Bound
1	.777	.021	.734	.821
2	.783	.019	.744	.821

Pairwise Comparisons

Measure: T_levels

					95% Confidence Interval for Difference[a]	
(I) phase	(J) phase	Mean Difference (I-J)	Std. Error	Sig.[a]	Lower Bound	Upper Bound
1	2	-.005	.029	.862	-.064	.054
2	1	.005	.029	.862	-.054	.064

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.001	.031[a]	1.000	27.000	.862	.001	.031	.053
Wilks' lambda	.999	.031[a]	1.000	27.000	.862	.001	.031	.053
Hotelling's trace	.001	.031[a]	1.000	27.000	.862	.001	.031	.053
Roy's largest root	.001	.031[a]	1.000	27.000	.862	.001	.031	.053

Each F tests the multivariate effect of phase. These tests are based on the linearly independent pairwise comparisons among the

estimated marginal means.

a. Exact statistic

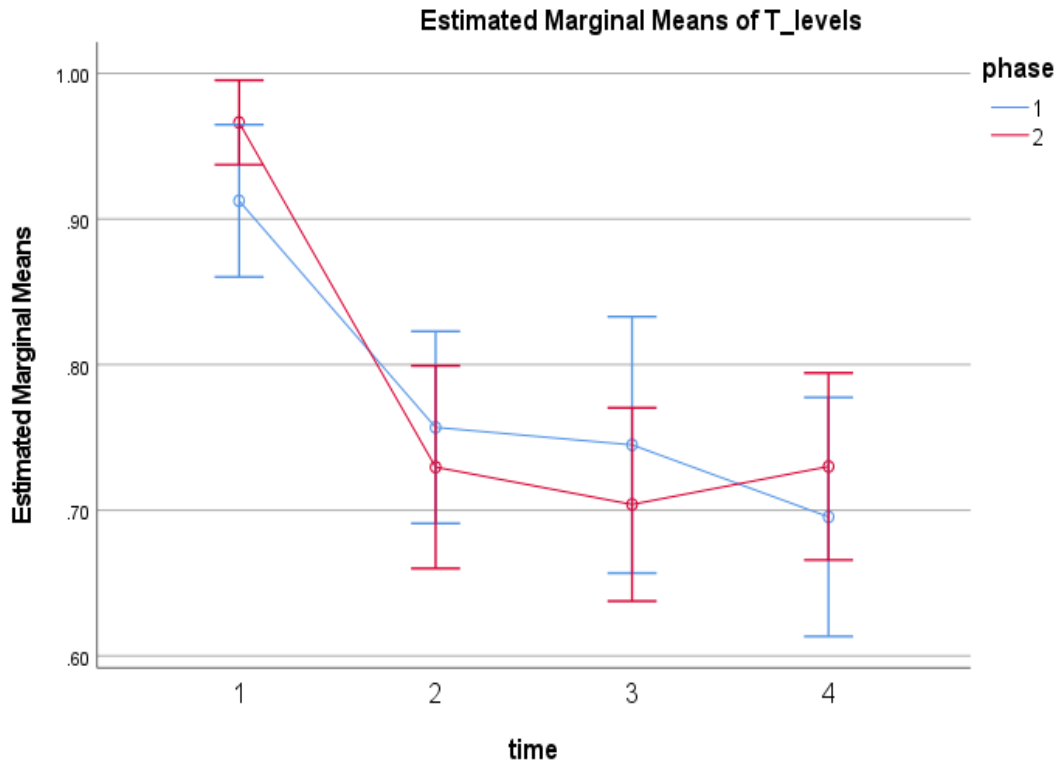
b. Computed using alpha =

3. time * phase

Measure: T_levels

time	phase	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
1	1	.913	.025	.860	.965
	2	.966	.014	.937	.995
2	1	.757	.032	.691	.823
	2	.730	.034	.660	.799
3	1	.745	.043	.657	.833
	2	.704	.032	.638	.770
4	1	.695	.040	.613	.778
	2	.730	.031	.666	.794

Profile Plots



Appendix XVII

Post-Competition Testosterone Data by Outcome and SES

General Linear Model

[DataSet2]

Warnings

Post hoc tests are not performed for Status because there are fewer than three groups.
Post hoc tests are not performed for Outcome because there are fewer than three groups.

Within-Subjects Factors

Measure: T_normalised

time	Dependent Variable
1	Pre_imm_testo_normalised
2	Post_imm_testo_normalised
3	Post_30min_testo_normalised
4	Post_1hr_testo_normalised
5	Post_2hrs_testo_normalised

Between-Subjects Factors

		Value Label	N
Status	.00	high	20
	1.00	low	11
Outcome	0	loss	15
	1	win	16

Descriptive Statistics

	Status	Outcome	Mean	Std. Deviation	N
Pre_imm_testo_normalised	high	loss	.8028	.14550	10
		win	.8706	.10203	10
		Total	.8367	.12716	20
	low	loss	.7349	.36614	5
		win	.7658	.19602	6
		Total	.7517	.27036	11
	Total	loss	.7801	.23024	15
		win	.8313	.14766	16
		Total	.8065	.19056	31
Post_imm_testo_normalised	high	loss	.8591	.16703	10
		win	.8957	.13999	10
		Total	.8774	.15116	20
	low	loss	.7415	.20260	5
		win	.6661	.10963	6
		Total	.7004	.15484	11
	Total	loss	.8199	.18154	15
		win	.8096	.17010	16
		Total	.8146	.17284	31
Post_30min_testo_normalised	high	loss	.8009	.08673	10
		win	.8445	.13511	10
		Total	.8227	.11275	20
	low	loss	.7162	.24934	5
		win	.7219	.20095	6
		Total	.7193	.21229	11
	Total	loss	.7726	.15590	15
		win	.7985	.16785	16
		Total	.7860	.16001	31
Post_1hr_testo_normalised	high	loss	.8853	.13613	10
		win	.8741	.15072	10
		Total	.8797	.13990	20
	low	loss	.6858	.23663	5
		win	.7251	.17817	6
		Total	.7072	.19670	11
	Total	loss	.8188	.19337	15
		win	.8182	.17252	16
		Total	.8185	.17981	31
Post_2hrs_testo_normalised	high	loss	.8674	.11030	10
		win	.8600	.08808	10
		Total	.8637	.09723	20

	Total	[illegible]	[illegible]	[illegible]
low	loss	.5314	.24305	5
	win	.7970	.19532	6
	Total	.6762	.24888	11
Total	loss	.7554	.22712	15
	win	.8364	.13552	16
	Total	.7972	.18694	31

Box's Test of Equality of Covariance Matrices[a]

Box's M	36.321
F	.765
df1	30
df2	964.005
Sig.	.814

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Status + Outcome + Status * Outcome Within Subjects Design: time

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.035	.220[b]	4.000	24.000	.924	.035	.881	.089
	Wilks' Lambda	.965	.220[b]	4.000	24.000	.924	.035	.881	.089
	Hotelling's Trace	.037	.220[b]	4.000	24.000	.924	.035	.881	.089
	Roy's Largest Root	.037	.220[b]	4.000	24.000	.924	.035	.881	.089
time * Status	Pillai's Trace	.146	1.022[b]	4.000	24.000	.416	.146	4.087	.273
	Wilks' Lambda	.854	1.022[b]	4.000	24.000	.416	.146	4.087	.273
	Hotelling's Trace	.170	1.022[b]	4.000	24.000	.416	.146	4.087	.273
	Roy's Largest Root	.170	1.022[b]	4.000	24.000	.416	.146	4.087	.273
time * Outcome	Pillai's Trace	.164	1.178[b]	4.000	24.000	.346	.164	4.710	.312
	Wilks' Lambda	.836	1.178[b]	4.000	24.000	.346	.164	4.710	.312
	Hotelling's Trace	.196	1.178[b]	4.000	24.000	.346	.164	4.710	.312
	Roy's Largest Root	.196	1.178[b]	4.000	24.000	.346	.164	4.710	.312
time * Status * Outcome	Pillai's Trace	.230	1.788[b]	4.000	24.000	.164	.230	7.153	.463
	Wilks' Lambda	.770	1.788[b]	4.000	24.000	.164	.230	7.153	.463
	Hotelling's Trace	.298	1.788[b]	4.000	24.000	.164	.230	7.153	.463
	Roy's Largest Root	.298	1.788[b]	4.000	24.000	.164	.230	7.153	.463

a. Design: Intercept + Status + Outcome + Status * Outcome
Within Subjects Design: time

b. Exact statistic

c. Computed using alpha = .05

Mauchly's Test of Sphericity[a]

Measure: T_normalised

					Epsilon[b]		
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.629	11.778	9	.227	.834	1.000	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Status + Outcome + Status * Outcome
Within Subjects Design: time

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: T_normalised

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Sphericity Assumed	.022	4	.005	.215	.930	.008	.860	.095
	Greenhouse-Geisser	.022	3.336	.006	.215	.903	.008	.717	.091
	Huynh-Feldt	.022	4.000	.005	.215	.930	.008	.860	.095
	Lower-bound	.022	1.000	.022	.215	.647	.008	.215	.073
time * Status	Sphericity Assumed	.069	4	.017	.684	.605	.025	2.734	.216
	Greenhouse-Geisser	.069	3.336	.021	.684	.579	.025	2.281	.198
	Huynh-Feldt	.069	4.000	.017	.684	.605	.025	2.734	.216
	Lower-bound	.069	1.000	.069	.684	.416	.025	.684	.125
time * Outcome	Sphericity Assumed	.088	4	.022	.873	.483	.031	3.490	.270
	Greenhouse-Geisser	.088	3.336	.026	.873	.468	.031	2.911	.245
	Huynh-Feldt	.088	4.000	.022	.873	.483	.031	3.490	.270
	Lower-bound	.088	1.000	.088	.873	.359	.031	.873	.147
time * Status * Outcome	Sphericity Assumed	.156	4	.039	1.552	.192	.054	6.209	.466
	Greenhouse-Geisser	.156	3.336	.047	1.552	.202	.054	5.179	.420
	Huynh-Feldt	.156	4.000	.039	1.552	.192	.054	6.209	.466
	Lower-bound	.156	1.000	.156	1.552	.223	.054	1.552	.225
Error(time)	Sphericity Assumed	2.722	108	.025					
	Greenhouse-Geisser	2.722	90.085	.030					
	Huynh-Feldt	2.722	108.000	.025					
	Lower-bound	2.722	27.000	.101					

a. Computed using alpha = .05

Tests of Within-Subjects Contrasts

Measure: T_normalised

Source	time	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear	.009	1	.009	.324	.574	.012	.324	.085
	Quadratic	.000	1	.000	.007	.932	.000	.007	.051
	Cubic	.003	1	.003	.148	.704	.005	.148	.066
	Order 4	.009	1	.009	.382	.542	.014	.382	.092
time * Status	Linear	.036	1	.036	1.275	.269	.045	1.275	.193
	Quadratic	.000	1	.000	.005	.943	.000	.005	.051
	Cubic	.009	1	.009	.410	.527	.015	.410	.095
	Order 4	.024	1	.024	.993	.328	.035	.993	.161
time * Outcome	Linear	.026	1	.026	.921	.346	.033	.921	.153
	Quadratic	.049	1	.049	1.826	.188	.063	1.826	.256

	Cubic	.000	1	.000	.005	.942	.000	.005	.051
	Order 4	.012	1	.012	.513	.480	.019	.513	.106
time * Status * Outcome	Linear	.108	1	.108	3.786	.062	.123	3.786	.467
	Quadratic	.047	1	.047	1.732	.199	.060	1.732	.246
	Cubic	3.955E-5	1	3.955E-5	.002	.966	.000	.002	.050
	Order 4	.002	1	.002	.068	.796	.003	.068	.057
Error(time)	Linear	.770	27	.029					
	Quadratic	.730	27	.027					
	Cubic	.580	27	.021					
	Order 4	.641	27	.024					

a. Computed using alpha = .05

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
Pre_imm_testo_normalised	Based on Mean	12.646	3	27	.000
	Based on Median	1.530	3	27	.229
	Based on Median and with adjusted df	1.530	3	6.958	.289
	Based on trimmed mean	11.749	3	27	.000
Post_imm_testo_normalised	Based on Mean	2.004	3	27	.137
	Based on Median	.610	3	27	.615
	Based on Median and with adjusted df	.610	3	25.424	.615
	Based on trimmed mean	1.852	3	27	.162
Post_30min_testo_normalised	Based on Mean	1.957	3	27	.144
	Based on Median	1.329	3	27	.286
	Based on Median and with adjusted df	1.329	3	14.828	.303
	Based on trimmed mean	1.899	3	27	.154
Post_1hr_testo_normalised	Based on Mean	.707	3	27	.556
	Based on Median	.359	3	27	.783
	Based on Median and with adjusted df	.359	3	22.902	.783
	Based on trimmed mean	.700	3	27	.560
Post_2hrs_testo_normalised	Based on Mean	2.858	3	27	.056
	Based on Median	1.667	3	27	.198
	Based on Median and with adjusted df	1.667	3	8.839	.244
	Based on trimmed mean	2.701	3	27	.065

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Status + Outcome + Status * Outcome

Within Subjects Design: time

Tests of Between-Subjects Effects

Measure: T_normalised

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observe Power
Intercept	86.395	1	86.395	2366.849	.000	.989	2366.849	1
Status	.768	1	.768	21.031	.000	.438	21.031	
Outcome	.055	1	.055	1.513	.229	.053	1.513	
Status * Outcome	.007	1	.007	.181	.674	.007	.181	
Error	.986	27	.037					

a. Computed using alpha = .05

Estimated Marginal Means

1. time

Estimates

Measure: T_normalised

time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.794	.036	.719	.868
2	.791	.029	.731	.851
3	.771	.030	.709	.832
4	.793	.031	.728	.857
5	.764	.028	.706	.822

Pairwise Comparisons

Measure: T_normalised

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.003	.030	1.000	-.089	.094
	3	.023	.050	1.000	-.129	.174
	4	.001	.048	1.000	-.146	.148
	5	.030	.038	.997	-.088	.147
2	1	-.003	.030	1.000	-.094	.089
	3	.020	.041	1.000	-.106	.146
	4	-.002	.045	1.000	-.140	.137

	5	.027	.036	.998	-.083	.136
3	1	-.023	.050	1.000	-.174	.129
	2	-.020	.041	1.000	-.146	.106
	4	-.022	.047	1.000	-.165	.122
	5	.007	.042	1.000	-.120	.134
4	1	-.001	.048	1.000	-.148	.146
	2	.002	.045	1.000	-.137	.140
	3	.022	.047	1.000	-.122	.165
	5	.029	.041	.999	-.095	.153
5	1	-.030	.038	.997	-.147	.088
	2	-.027	.036	.998	-.136	.083
	3	-.007	.042	1.000	-.134	.120
	4	-.029	.041	.999	-.153	.095

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.035	.220[a]	4.000	24.000	.924	.035	.881	.089
Wilks' lambda	.965	.220[a]	4.000	24.000	.924	.035	.881	.089
Hotelling's trace	.037	.220[a]	4.000	24.000	.924	.035	.881	.089
Roy's largest root	.037	.220[a]	4.000	24.000	.924	.035	.881	.089

Each F tests the multivariate effect of time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha = .05

2. Status

Estimates

Measure: T_normalised

			95% Confidence Interval	
Status	Mean	Std. Error	Lower Bound	Upper Bound
high	.856	.019	.817	.895
low	.709	.026	.655	.762

Pairwise Comparisons

Measure: T_normalised

(I) Status	(J) Status	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
high	low	.147*	.032	.000	.081	.213
low	high	-.147*	.032	.000	-.213	-.081

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Sidak.

Univariate Tests

Measure: T_normalised

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	.154	1	.154	21.031	.000	.438	21.031	.993
Error	.197	27	.007					

The F tests the effect of Status. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha = .05

3. Outcome

Estimates

Measure: T_normalised

Outcome	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
loss	.763	.023	.714	.811
win	.802	.022	.757	.847

Pairwise Comparisons

Measure: T_normalised

(I) Outcome	(J) Outcome	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
loss	win	-.040	.032	.229	-.106	.026
win	loss	.040	.032	.229	-.026	.106

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Univariate Tests

Measure: T_normalised

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	.011	1	.011	1.513	.229	.053	1.513	.220
Error	.197	27	.007					

The F tests the effect of Outcome. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha = .05

4. Status * Outcome

Measure: T_normalised

				95% Confidence Interval	
Status	Outcome	Mean	Std. Error	Lower Bound	Upper Bound
high	loss	.843	.027	.788	.899
	win	.869	.027	.814	.924
low	loss	.682	.038	.604	.760
	win	.735	.035	.664	.807

5. Status * time

Measure: T_normalised

				95% Confidence Interval	
Status	time	Mean	Std. Error	Lower Bound	Upper Bound
high	1	.837	.043	.748	.926
	2	.877	.035	.806	.949
	3	.823	.036	.750	.896
	4	.880	.037	.803	.956
	5	.864	.034	.795	.932
low	1	.750	.059	.630	.871
	2	.704	.047	.607	.800
	3	.719	.048	.620	.818
	4	.705	.051	.602	.809
	5	.664	.045	.571	.757

6. Outcome * time

Measure: T_normalised

				95% Confidence Interval	
Outcome	time	Mean	Std. Error	Lower Bound	Upper Bound
loss	1	.769	.053	.660	.878
	2	.800	.043	.713	.888

	3	.759	.044	.669	.848
	4	.786	.046	.692	.879
	5	.699	.041	.615	.784
win	1	.818	.050	.716	.921
	2	.781	.040	.699	.863
	3	.783	.041	.699	.867
	4	.800	.043	.711	.888
	5	.828	.039	.749	.908

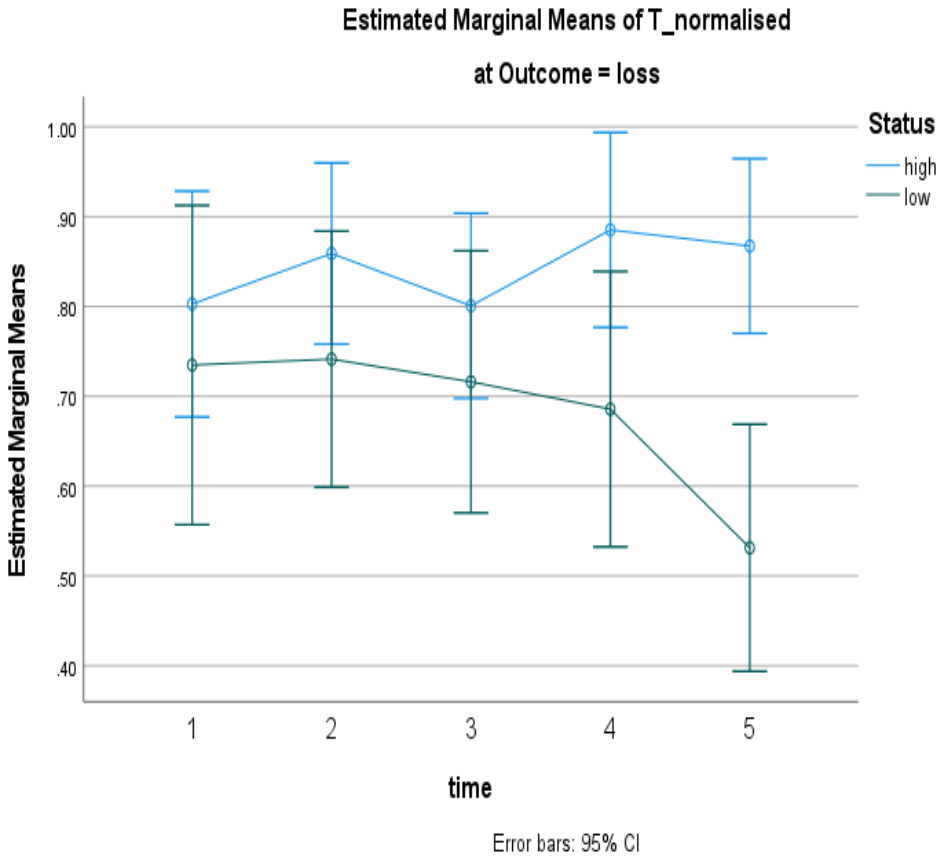
7. Status * Outcome * time

Measure: T_normalised

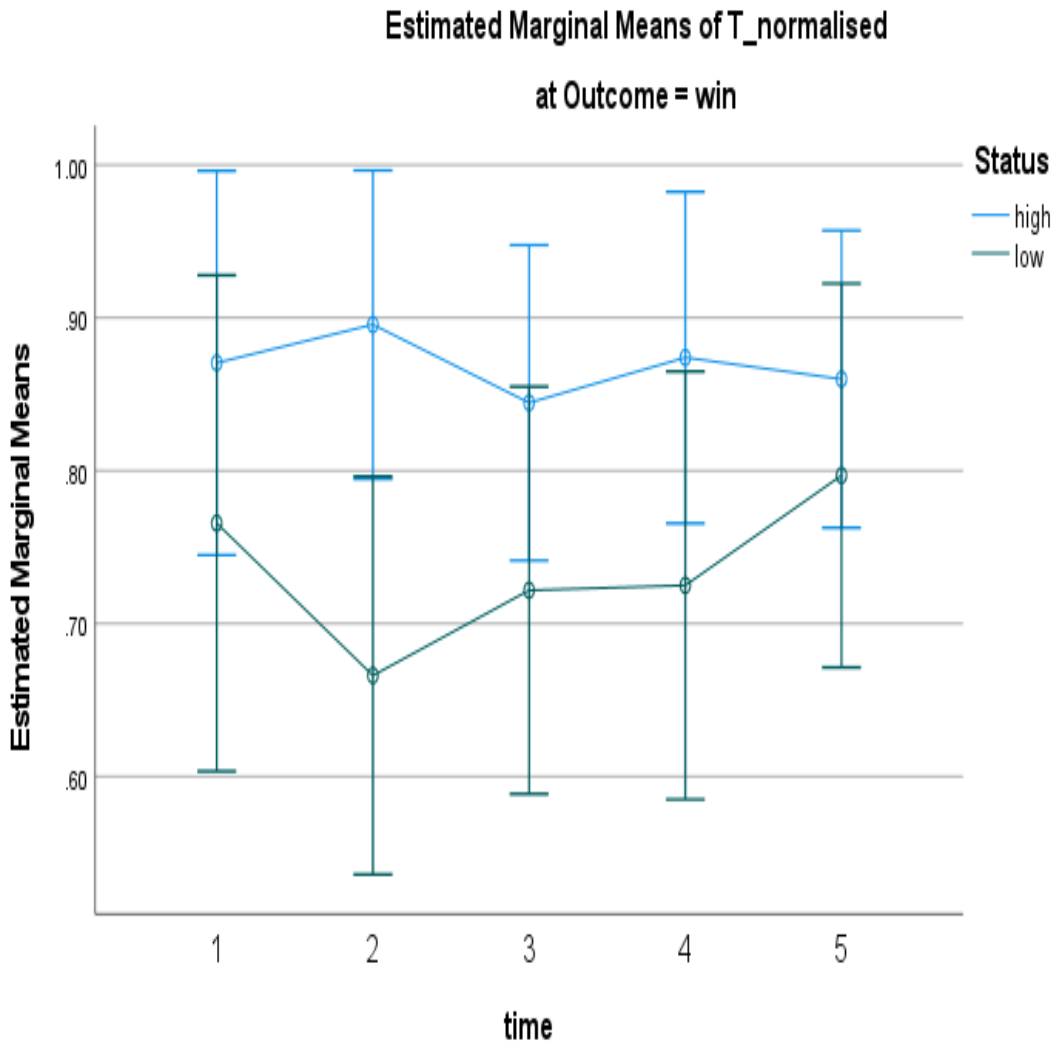
					95% Confidence Interval	
Status	Outcome	time	Mean	Std. Error	Lower Bound	Upper Bound
high	loss	1	.803	.061	.677	.928
		2	.859	.049	.758	.960
		3	.801	.050	.698	.904
		4	.885	.053	.777	.994
		5	.867	.047	.770	.965
	win	1	.871	.061	.745	.996
		2	.896	.049	.795	.996
		3	.845	.050	.741	.948
		4	.874	.053	.766	.983
		5	.860	.047	.763	.957
low	loss	1	.735	.087	.557	.913
		2	.741	.069	.599	.884
		3	.716	.071	.570	.862
		4	.686	.075	.532	.839
		5	.531	.067	.394	.669
	win	1	.766	.079	.604	.928
		2	.666	.063	.536	.796
		3	.722	.065	.589	.855
		4	.725	.068	.585	.865
		5	.797	.061	.671	.922

Profile Plots

time * Status * Outcome



Error bars: 95% CI



Error bars: 95% CI

Appendix XVIII

Baseline and Task Phase Cortisol Data Analysis

General Linear Model

Warnings

The HOMOGENEITY specification in the PRINT subcommand will be ignored because there are no between-subjects factors.

Within-Subjects Factors

Measure: Cortisol_percentage

time	day	Dependent Variable
1	1	Cortisol_9am_baseline_percentage
	2	Cortisol_9am_experimental_percentage
2	1	Cortisol_1hr_baseline_percentage
	2	Cortisol_1hr_experimental_percentage
3	1	Cortisol_30min_baseline_percentage
	2	Cortisol_30min_experimental_percentage
4	1	Cortisol_imm_baseline_percentage
	2	Cortisol_imm_experimental_percentage

Descriptive Statistics

	Mean	Std. Deviation	N
Cortisol_9am_baseline_percentage	41.1692	26.05749	28
Cortisol_9am_experimental_percentage	30.9328	30.73836	28
Cortisol_1hr_baseline_percentage	-6.0200	18.24207	28

Cortisol_1hr_experimental_percentage	-10.5254	16.68592	28
Cortisol_30min_baseline_percentage	-16.7528	18.37896	28
Cortisol_30min_experimental_percentage	-14.3143	17.16840	28
Cortisol_imm_baseline_percentage	-18.3964	11.65070	28
Cortisol_imm_experimental_percentage	-6.0931	28.51870	28

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.743	24.069[b]	3.000	25.000	.000	.743	72.208	1.000
	Wilks' Lambda	.257	24.069[b]	3.000	25.000	.000	.743	72.208	1.000
	Hotelling's Trace	2.888	24.069[b]	3.000	25.000	.000	.743	72.208	1.000
	Roy's Largest Root	2.888	24.069[b]	3.000	25.000	.000	.743	72.208	1.000
day	Pillai's Trace	.238	8.456[b]	1.000	27.000	.007	.238	8.456	.800
	Wilks' Lambda	.762	8.456[b]	1.000	27.000	.007	.238	8.456	.800
	Hotelling's Trace	.313	8.456[b]	1.000	27.000	.007	.238	8.456	.800
	Roy's Largest Root	.313	8.456[b]	1.000	27.000	.007	.238	8.456	.800
time * day	Pillai's Trace	.207	2.170[b]	3.000	25.000	.117	.207	6.509	.486
	Wilks' Lambda	.793	2.170[b]	3.000	25.000	.117	.207	6.509	.486
	Hotelling's Trace	.260	2.170[b]	3.000	25.000	.117	.207	6.509	.486
	Roy's Largest Root	.260	2.170[b]	3.000	25.000	.117	.207	6.509	.486

a. Design: Intercept
Within Subjects Design: time + day + time * day

b. Exact statistic

c. Computed using alpha =

Mauchly's Test of Sphericity[a]

Measure: Cortisol_percentage

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.391	24.155	5	.000	.644	.692	.333
day	1.000	.000	0	.	1.000	1.000	1.000
time * day	.856	4.000	5	.550	.902	1.000	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept
Within Subjects Design: time + day + time * day

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: Cortisol_percentage

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Sphericity Assumed	98522.911	3	32840.970	43.076	.000	.615	129.229	1.000
	Greenhouse-Geisser	98522.911	1.931	51014.914	43.076	.000	.615	83.191	1.000
	Huynh-Feldt	98522.911	2.077	47428.443	43.076	.000	.615	89.482	1.000
	Lower-bound	98522.911	1.000	98522.911	43.076	.000	.615	43.076	1.000
Error(time)	Sphericity Assumed	61753.782	81	762.392					
	Greenhouse-Geisser	61753.782	52.144	1184.295					
	Huynh-Feldt	61753.782	56.087	1101.036					
	Lower-bound	61753.782	27.000	2287.177					
day	Sphericity Assumed	2.834E-12	1	2.834E-12	6.486	.017	.194	6.486	.690
	Greenhouse-Geisser	2.834E-12	1.000	2.834E-12	6.486	.017	.194	6.486	.690
	Huynh-Feldt	2.834E-12	1.000	2.834E-12	6.486	.017	.194	6.486	.690
	Lower-bound	2.834E-12	1.000	2.834E-12	6.486	.017	.194	6.486	.690
Error(day)	Sphericity Assumed	1.180E-11	27	4.369E-13					
	Greenhouse-Geisser	1.180E-11	27.000	4.369E-13					
	Huynh-Feldt	1.180E-11	27.000	4.369E-13					
	Lower-bound	1.180E-11	27.000	4.369E-13					
time * day	Sphericity Assumed	3953.582	3	1317.861	2.585	.059	.087	7.755	.616
	Greenhouse-Geisser	3953.582	2.705	1461.394	2.585	.065	.087	6.993	.583
	Huynh-Feldt	3953.582	3.000	1317.861	2.585	.059	.087	7.755	.616
	Lower-bound	3953.582	1.000	3953.582	2.585	.120	.087	2.585	.341
Error(time*day)	Sphericity Assumed	41295.167	81	509.817					
	Greenhouse-Geisser	41295.167	73.044	565.343					
	Huynh-Feldt	41295.167	81.000	509.817					
	Lower-bound	41295.167	27.000	1529.451					

a. Computed using alpha =

Tests of Within-Subjects Contrasts

Measure: Cortisol_percentage

Source	time	day	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear		64817.305	1	64817.305	48.783	.000	.644	48.783	1.000
	Quadratic		31737.328	1	31737.328	49.245	.000	.646	49.245	1.000
	Cubic		1968.278	1	1968.278	6.268	.019	.188	6.268	.675
Error(time)	Linear		35874.376	27	1328.681					
	Quadratic		17400.794	27	644.474					
	Cubic		8478.612	27	314.023					
day		Linear	.000	1	.000	.000	1.000	.000	.000	.050
Error(day)		Linear	1.273E-11	27	4.716E-13					
time * day	Linear	Linear	3891.731	1	3891.731	6.103	.020	.184	6.103	.663
	Quadratic	Linear	59.809	1	59.809	.113	.740	.004	.113	.062
	Cubic	Linear	2.042	1	2.042	.006	.941	.000	.006	.051
Error(time*day)	Linear	Linear	17216.976	27	637.666					
	Quadratic	Linear	14333.954	27	530.887					
	Cubic	Linear	9744.236	27	360.898					

a. Computed using alpha =

Tests of Between-Subjects Effects

Measure: Cortisol_percentage

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	.000	1	.000	.000	1.000	.000	.000	.050
Error	1.091E-11	27	4.042E-13					

a. Computed using alpha =

Estimated Marginal Means

1. time

Estimates

Measure: Cortisol_percentage

time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	36.051	4.461	26.899	45.203
2	-8.273	2.466	-13.332	-3.214
3	-15.534	2.027	-19.693	-11.374
4	-12.245	3.280	-18.975	-5.515

Pairwise Comparisons

Measure: Cortisol_percentage

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	44.324*	5.986	.000	27.336	61.311
	3	51.585*	5.899	.000	34.843	68.326
	4	48.296*	7.057	.000	28.268	68.324
2	1	-44.324*	5.986	.000	-61.311	-27.336
	3	7.261	2.978	.123	-1.191	15.712
	4	3.972	4.523	.947	-8.866	16.810
3	1	-51.585*	5.899	.000	-68.326	-34.843
	2	-7.261	2.978	.123	-15.712	1.191
	4	-3.289	3.690	.944	-13.760	7.182
4	1	-48.296*	7.057	.000	-68.324	-28.268
	2	-3.972	4.523	.947	-16.810	8.866
	3	3.289	3.690	.944	-7.182	13.760

Based on estimated marginal means

*. The mean difference is significant at the

b. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.743	24.069[a]	3.000	25.000	.000	.743	72.208	1.000
Wilks' lambda	.257	24.069[a]	3.000	25.000	.000	.743	72.208	1.000
Hotelling's trace	2.888	24.069[a]	3.000	25.000	.000	.743	72.208	1.000
Roy's largest root	2.888	24.069[a]	3.000	25.000	.000	.743	72.208	1.000

Each F tests the multivariate effect of time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha =

2. day

Estimates

Measure: Cortisol_percentage

day	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	-8.929E-11	.000	-8.929E-11	-8.929E-11
2	-2.023E-7	.000	-3.837E-7	-2.082E-8

Pairwise Comparisons

Measure: Cortisol_percentage

(I) day	(J) day	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	2.022E-7*	.000	.025	2.785E-8	3.765E-7
2	1	-2.022E-7*	.000	.025	-3.765E-7	-2.785E-8

Based on estimated marginal means

*. The mean difference is significant at the

b. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.166	5.362[a]	1.000	27.000	.028	.166	5.362	.608
Wilks' lambda	.834	5.362[a]	1.000	27.000	.028	.166	5.362	.608
Hotelling's trace	.199	5.362[a]	1.000	27.000	.028	.166	5.362	.608
Roy's largest root	.199	5.362[a]	1.000	27.000	.028	.166	5.362	.608

Each F tests the multivariate effect of day. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha =

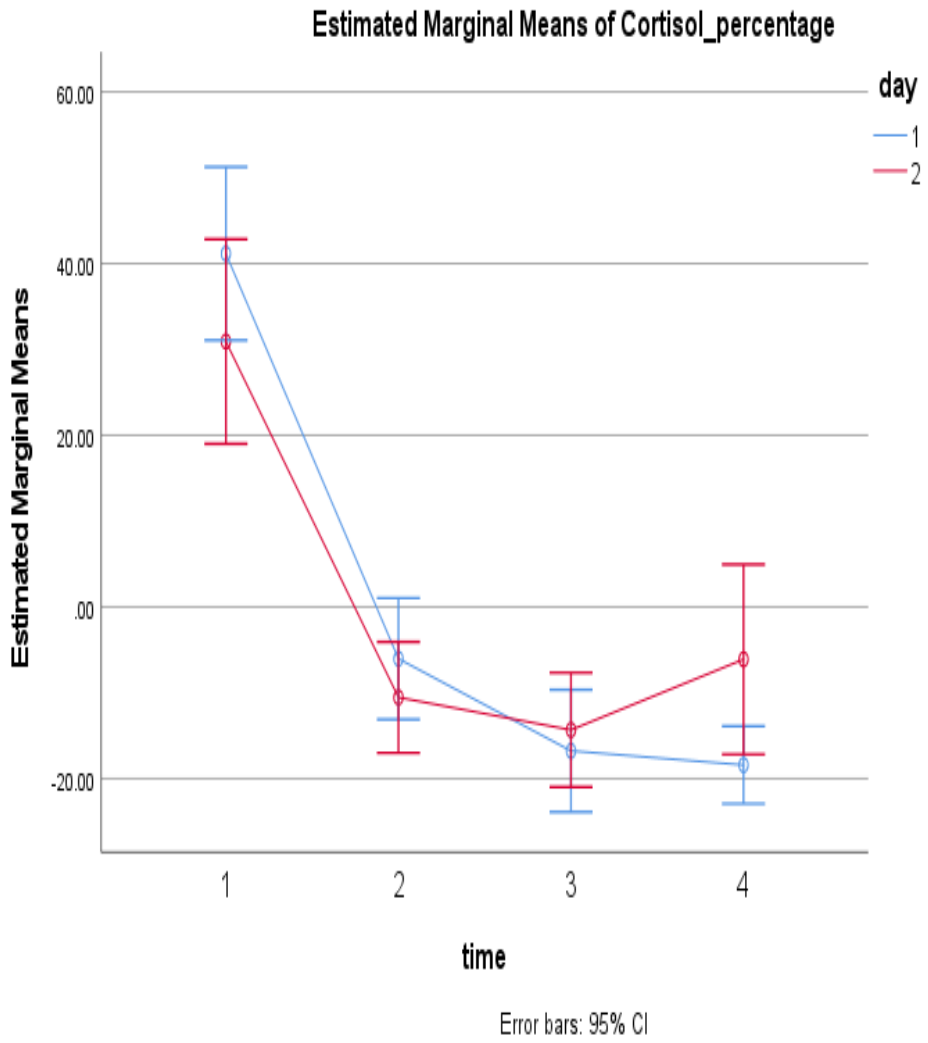
3. time * day

Measure: Cortisol_percentage

time	day	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
1	1	41.169	4.924	31.065	51.273
	2	30.933	5.809	19.014	42.852
2	1	-6.020	3.447	-13.094	1.054
	2	-10.525	3.153	-16.996	-4.055
3	1	-16.753	3.473	-23.879	-9.626
	2	-14.314	3.245	-20.972	-7.657
4	1	-18.396	2.202	-22.914	-13.879

4	1	-18.396	2.202	-22.914	-13.879
	2	-6.093	5.390	-17.152	4.965

Profile Plots



Appendix XIX

Post-Competition Cortisol Data by Outcome and SES

Within-Subjects Factors

Measure: Cortisol

time	Dependent Variable
1	Pre_imm_cortisol_normalised
2	Post_imm_cortisol_normalised
3	Post_30min_cortisol_normalised
4	Post_1hr_cortisol_normalised
5	Post_2hrs_cortisol_normalised

Between-Subjects Factors

		Value Label	N
Status	.00	high	18
	1.00	low	8
Outcome	0	loss	13
	1	win	13

Descriptive Statistics

	Status	Outcome	Mean	Std. Deviation	N
Pre_imm_cortisol_normalised	high	loss	.8152	.18086	9
		win	.8233	.23425	9
		Total	.8192	.20306	18
	low	loss	.9885	.02299	4
		win	1.0000	.00000	4
		Total	.9943	.01626	8
	Total	loss	.8685	.16991	13
		win	.8776	.20926	13
		Total	.8731	.18681	26
Post_imm_cortisol_normalised	high	loss	.9283	.09238	9
		win	.9610	.08227	9
		Total	.9447	.08651	18
	low	loss	.9260	.08776	4
		win	.7177	.04736	4

	Total	loss	.9276	.08727	13
		win	.8861	.13686	13
		Total	.9069	.11443	26
Post_30min_cortisol_normalised	high	loss	.6869	.18084	9
		win	.6828	.26025	9
		Total	.6849	.21741	18
	low	loss	.6819	.21098	4
		win	.5663	.22069	4
		Total	.6241	.20920	8
	Total	loss	.6854	.18148	13
		win	.6470	.24588	13
		Total	.6662	.21263	26
Post_1hr_cortisol_normalised	high	loss	.6089	.20591	9
		win	.4310	.22838	9
		Total	.5200	.22994	18
	low	loss	.6210	.24542	4
		win	.4194	.11408	4
		Total	.5202	.20738	8
	Total	loss	.6126	.20822	13
		win	.4274	.19508	13
		Total	.5200	.21908	26
Post_2hrs_cortisol_normalised	high	loss	.5428	.20881	9
		win	.3725	.20566	9
		Total	.4576	.21932	18
	low	loss	.3996	.33381	4
		win	.4857	.09488	4
		Total	.4427	.23180	8
	Total	loss	.4987	.24830	13
		win	.4073	.18278	13
		Total	.4530	.21864	26

Box's Test of Equality of Covariance Matrices[a]

Box's M	12.554
F	.546
df1	15
df2	1030.737
Sig.	.915

Tests the null hypothesis that the observed

covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Status + Outcome + Status * Outcome Within Subjects Design: time

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.811	20.406[b]	4.000	19.000	.000	.811	81.625	1.000
	Wilks' Lambda	.189	20.406[b]	4.000	19.000	.000	.811	81.625	1.000
	Hotelling's Trace	4.296	20.406[b]	4.000	19.000	.000	.811	81.625	1.000
	Roy's Largest Root	4.296	20.406[b]	4.000	19.000	.000	.811	81.625	1.000
time * Status	Pillai's Trace	.325	2.292[b]	4.000	19.000	.097	.325	9.167	.553
	Wilks' Lambda	.675	2.292[b]	4.000	19.000	.097	.325	9.167	.553
	Hotelling's Trace	.482	2.292[b]	4.000	19.000	.097	.325	9.167	.553
	Roy's Largest Root	.482	2.292[b]	4.000	19.000	.097	.325	9.167	.553
time * Outcome	Pillai's Trace	.287	1.913[b]	4.000	19.000	.150	.287	7.652	.471
	Wilks' Lambda	.713	1.913[b]	4.000	19.000	.150	.287	7.652	.471
	Hotelling's Trace	.403	1.913[b]	4.000	19.000	.150	.287	7.652	.471
	Roy's Largest Root	.403	1.913[b]	4.000	19.000	.150	.287	7.652	.471
time * Status * Outcome	Pillai's Trace	.264	1.701[b]	4.000	19.000	.191	.264	6.804	.422
	Wilks' Lambda	.736	1.701[b]	4.000	19.000	.191	.264	6.804	.422
	Hotelling's Trace	.358	1.701[b]	4.000	19.000	.191	.264	6.804	.422
	Roy's Largest Root	.358	1.701[b]	4.000	19.000	.191	.264	6.804	.422

a. Design: Intercept + Status + Outcome + Status * Outcome
Within Subjects Design: time

b. Exact statistic

c. Computed using alpha = .05

Mauchly's Test of Sphericity[a]

Measure: Cortisol

Source	time	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear	3.609	1	3.609	75.528	.000	.774	75.528	1.000
	Quadratic	3.340E-6	1	3.340E-6	.000	.993	.000	.000	.050
	Cubic	.161	1	.161	7.563	.012	.256	7.563	.748
	Order 4	.034	1	.034	1.951	.176	.081	1.951	.267
time * Status	Linear	.037	1	.037	.765	.391	.034	.765	.133
	Quadratic	.126	1	.126	3.330	.082	.131	3.330	.415
	Cubic	.105	1	.105	4.940	.037	.183	4.940	.566
	Order 4	.006	1	.006	.367	.551	.016	.367	.089
time * Outcome	Linear	.023	1	.023	.490	.491	.022	.490	.103
	Quadratic	.044	1	.044	1.157	.294	.050	1.157	.177
	Cubic	.013	1	.013	.600	.447	.027	.600	.115
	Order 4	.041	1	.041	2.322	.142	.095	2.322	.308
time * Status * Outcome	Linear	.072	1	.072	1.515	.231	.064	1.515	.218
	Quadratic	.100	1	.100	2.653	.118	.108	2.653	.344
	Cubic	.005	1	.005	.214	.648	.010	.214	.073
	Order 4	.008	1	.008	.474	.498	.021	.474	.101
Error(time)	Linear	1.051	22	.048					
	Quadratic	.832	22	.038					
	Cubic	.469	22	.021					
	Order 4	.388	22	.018					

a. Computed using alpha = .05

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
Pre_imm_cortisol_normalised	Based on Mean	4.255	3	22	.016
	Based on Median	2.493	3	22	.087
	Based on Median and with adjusted df	2.493	3	13.339	.105
	Based on trimmed mean	3.722	3	22	.026
Post_imm_cortisol_normalised	Based on Mean	.945	3	22	.436
	Based on Median	.356	3	22	.785
	Based on Median and with adjusted df	.356	3	18.373	.785
	Based on trimmed mean	.902	3	22	.456
Post_30min_cortisol_normalised	Based on Mean	.175	3	22	.912
	Based on Median	.120	3	22	.948
	Based on Median and with adjusted df	.120	3	18.548	.947
	Based on trimmed mean	.114	3	22	.951
Post_1hr_cortisol_norma	Based on Mean	.578	3	22	.635

Post_30min_cortisol_normalised	Based on Mean	.175	3	22	.912
	Based on Median	.120	3	22	.948
	Based on Median and with adjusted df	.120	3	18.548	.947
	Based on trimmed mean	.114	3	22	.951
Post_1hr_cortisol_normalised	Based on Mean	.578	3	22	.635
	Based on Median	.311	3	22	.817
	Based on Median and with adjusted df	.311	3	18.547	.817
	Based on trimmed mean	.558	3	22	.648
Post_2hrs_cortisol_normalised	Based on Mean	1.620	3	22	.213
	Based on Median	.935	3	22	.441
	Based on Median and with adjusted df	.935	3	15.340	.448
	Based on trimmed mean	1.520	3	22	.237

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Status + Outcome + Status * Outcome
Within Subjects Design: time

Tests of Between-Subjects Effects

Measure: Cortisol

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	51.663	1	51.663	929.732	.000	.977	929.732	1.000
Status	.001	1	.001	.011	.918	.000	.011	.051
Outcome	.151	1	.151	2.725	.113	.110	2.725	.352
Status * Outcome	.004	1	.004	.067	.798	.003	.067	.057
Error	1.222	22	.056					

a. Computed using alpha = .05

Estimated Marginal Means

1. Status

Estimates

Measure: Cortisol

Status	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
high	.685	.025	.634	.737
low	.681	.037	.603	.758

Pairwise Comparisons

Measure: Cortisol

(I) Status	(J) Status	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
high	low	.005	.045	.918	-.088	.098
low	high	-.005	.045	.918	-.098	.088

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Univariate Tests

Measure: Cortisol

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	.000	1	.000	.011	.918	.000	.011	.051
Error	.244	22	.011					

The F tests the effect of Status. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha = .05

2. Outcome

Estimates

Measure: Cortisol

Outcome	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
loss	.720	.032	.654	.786
win	.646	.032	.580	.712

Pairwise Comparisons

Measure: Cortisol

(I) Outcome	(J) Outcome	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
loss	win	.074	.045	.113	-.019	.167
win	loss	-.074	.045	.113	-.167	.019

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Univariate Tests

Measure: Cortisol

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	.030	1	.030	2.725	.113	.110	2.725	.352
Error	.244	22	.011					

The F tests the effect of Outcome. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha = .05

3. time

Estimates

Measure: Cortisol

time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.907	.038	.828	.985
2	.883	.018	.847	.920
3	.654	.047	.557	.752
4	.520	.045	.427	.613
5	.450	.046	.354	.546

Pairwise Comparisons

Measure: Cortisol

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	.023	.049	1.000	-.130	.177
	3	.252*	.058	.002	.073	.432
	4	.387*	.060	.000	.201	.572
	5	.457*	.062	.000	.264	.649
2	1	-.023	.049	1.000	-.177	.130
	3	.229*	.052	.002	.068	.390
	4	.363*	.049	.000	.210	.516
	5	.433*	.050	.000	.278	.588
3	1	-.252*	.058	.002	-.432	-.073
	2	-.229*	.052	.002	-.390	-.068
	4	.134*	.040	.026	.011	.257
	5	.204*	.064	.041	.006	.403
4	1	-.387*	.060	.000	-.572	-.201

	2	-.363*	.049	.000	-.516	-.210
	3	-.134*	.040	.026	-.257	-.011
	5	.070	.041	.665	-.058	.198
5	1	-.457*	.062	.000	-.649	-.264
	2	-.433*	.050	.000	-.588	-.278
	3	-.204*	.064	.041	-.403	-.006
	4	-.070	.041	.665	-.198	.058

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.811	20.406[a]	4.000	19.000	.000	.811	81.625	1.000
Wilks' lambda	.189	20.406[a]	4.000	19.000	.000	.811	81.625	1.000
Hotelling's trace	4.296	20.406[a]	4.000	19.000	.000	.811	81.625	1.000
Roy's largest root	4.296	20.406[a]	4.000	19.000	.000	.811	81.625	1.000

Each F tests the multivariate effect of time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha = .05

4. Status * Outcome

Measure: Cortisol

				95% Confidence Interval	
Status	Outcome	Mean	Std. Error	Lower Bound	Upper Bound
high	loss	.716	.035	.644	.789
	win	.654	.035	.581	.727
low	loss	.723	.053	.614	.833
	win	.638	.053	.529	.747

5. Status * time

Measure: Cortisol

				95% Confidence Interval	
Status	time	Mean	Std. Error	Lower Bound	Upper Bound
high	1	.819	.042	.732	.907
	2	.945	.020	.904	.985
	3	.685	.052	.576	.793

	4	.520	.050	.417	.623
	5	.458	.051	.351	.564
low	1	.994	.063	.863	1.125
	2	.822	.029	.761	.883
	3	.624	.078	.461	.787
	4	.520	.074	.366	.675
	5	.443	.077	.283	.603

6. Outcome * time

Measure: Cortisol

				95% Confidence Interval	
Outcome	time	Mean	Std. Error	Lower Bound	Upper Bound
loss	1	.902	.054	.791	1.013
	2	.927	.025	.875	.979
	3	.684	.067	.546	.823
	4	.615	.063	.484	.746
	5	.471	.066	.335	.607
win	1	.912	.054	.800	1.023
	2	.839	.025	.788	.891
	3	.625	.067	.486	.763
	4	.425	.063	.294	.556
	5	.429	.066	.293	.565

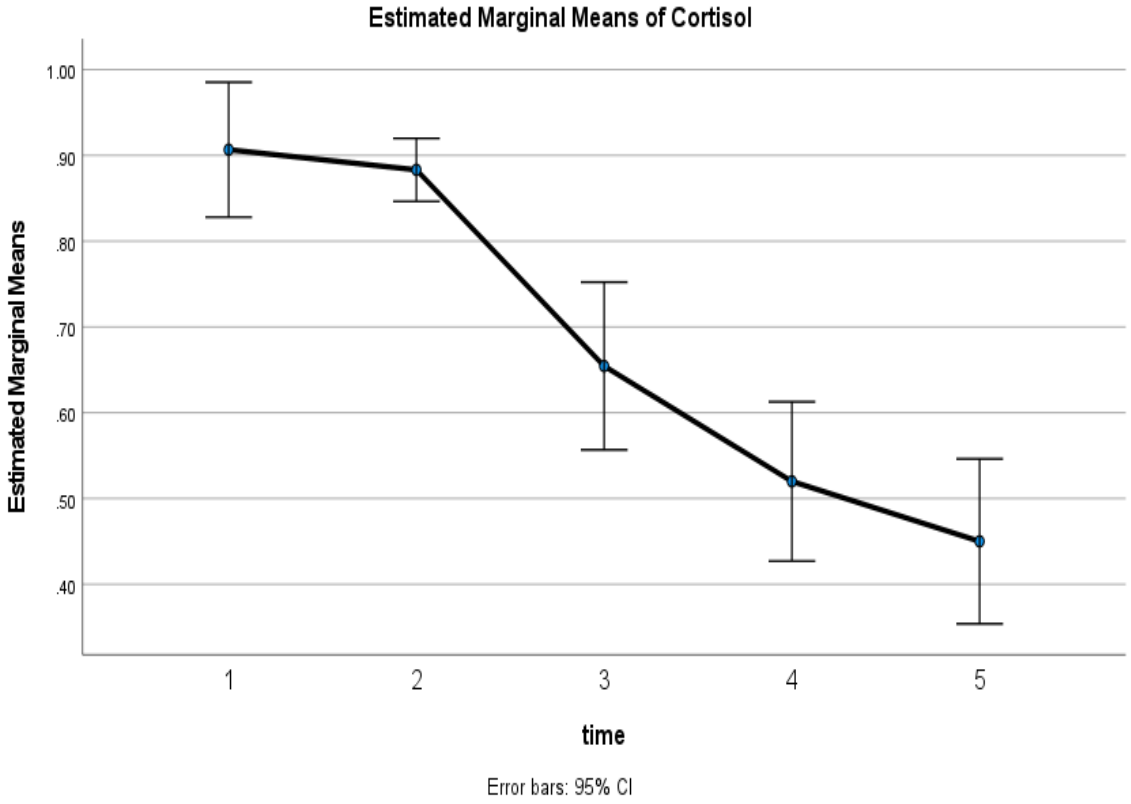
7. Status * Outcome * time

Measure: Cortisol

					95% Confidence Interval	
Status	Outcome	time	Mean	Std. Error	Lower Bound	Upper Bound
high	loss	1	.815	.060	.692	.939
		2	.928	.028	.871	.986
		3	.687	.074	.534	.840
		4	.609	.070	.463	.755
		5	.543	.073	.392	.694
	win	1	.823	.060	.700	.947
		2	.961	.028	.903	1.018
		3	.683	.074	.529	.836
		4	.431	.070	.285	.577
		5	.372	.073	.222	.523
low	loss	1	.989	.089	.803	1.174
		2	.926	.042	.840	1.012
		3	.682	.111	.452	.912

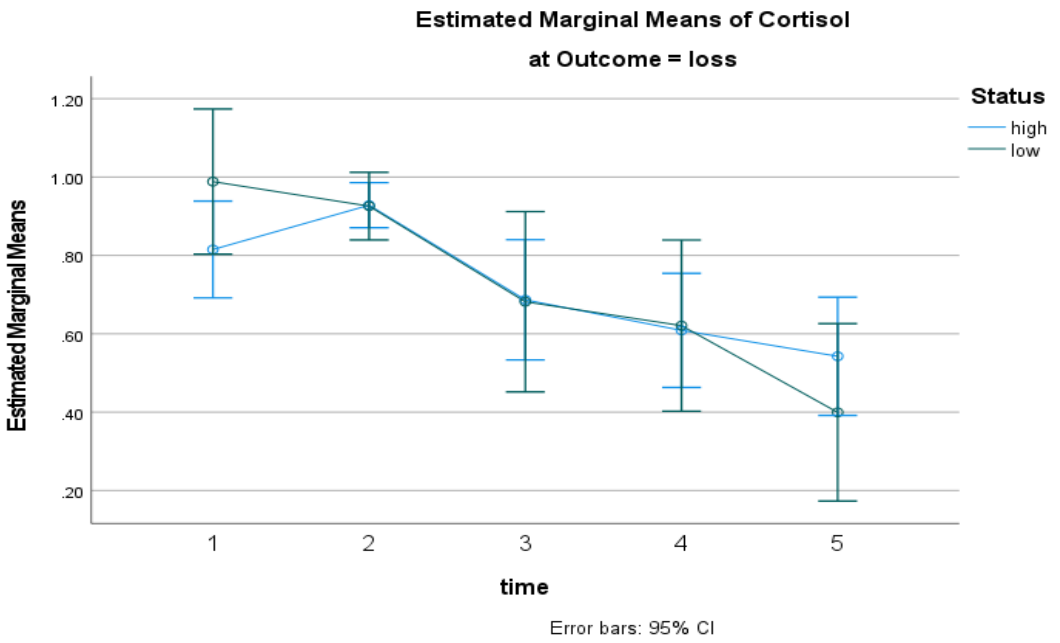
	4	.621	.105	.403	.839
	5	.400	.109	.173	.626
win	1	1.000	.089	.815	1.185
	2	.718	.042	.631	.804
	3	.566	.111	.336	.796
	4	.419	.105	.201	.638
	5	.486	.109	.259	.712

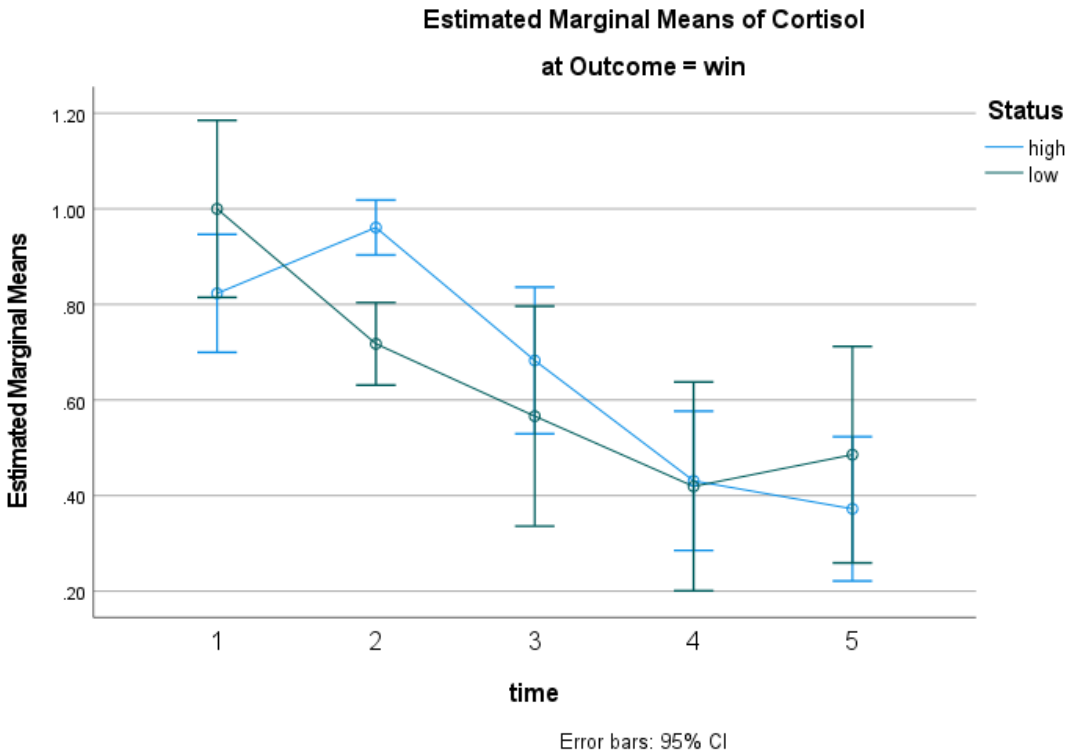
Profile Plots



Profile Plots

time * Status * Outcome





Appendix XX

MANOVA Analysis for Sense of Coherence

General Linear Model

Warnings

Post hoc tests are not performed for status because there are fewer than three groups.

Between-Subjects Factors

		Value Label	N
status	0	high status	20
	1	low-status	11

Descriptive Statistics

	status	Mean	Std. Deviation	N
SOC_Meaningful	high status	38.80	6.630	20
	low-status	37.09	11.086	11
	Total	38.19	8.336	31
SOC_PerceivedUnderstanding	high status	43.80	8.924	20
	low-status	36.00	4.919	11
	Total	41.03	8.538	31
SOC_PAControl	high status	49.50	8.426	20
	low-status	43.27	8.186	11
	Total	47.29	8.745	31
sense_of_coherence	high status	136.70	18.607	20
	low-status	121.09	21.659	11
	Total	131.16	20.815	31

Box's Test of Equality of Covariance Matrices[a]

Box's M	16.161
F	1.334
df1	10
df2	1978.637
Sig.	.206

Tests the null hypothesis that the observed

covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + status

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
Intercept	Pillai's Trace	.979	308.703[b]	4.000	26.000	.000	.979	1234.813	1.000
	Wilks' Lambda	.021	308.703[b]	4.000	26.000	.000	.979	1234.813	1.000
	Hotelling's Trace	47.493	308.703[b]	4.000	26.000	.000	.979	1234.813	1.000
	Roy's Largest Root	47.493	308.703[b]	4.000	26.000	.000	.979	1234.813	1.000
status	Pillai's Trace	.244	2.099[b]	4.000	26.000	.110	.244	8.395	.542
	Wilks' Lambda	.756	2.099[b]	4.000	26.000	.110	.244	8.395	.542
	Hotelling's Trace	.323	2.099[b]	4.000	26.000	.110	.244	8.395	.542
	Roy's Largest Root	.323	2.099[b]	4.000	26.000	.110	.244	8.395	.542

a. Design: Intercept + status

b. Exact statistic

c. Computed using alpha =

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
SOC_Meaningful	Based on Mean	6.069	1	29	.020
	Based on Median	1.766	1	29	.194
	Based on Median and with adjusted df	1.766	1	16.510	.202
	Based on trimmed mean	5.541	1	29	.026
SOC_PerceivedUnderstanding	Based on Mean	3.386	1	29	.076
	Based on Median	3.116	1	29	.088
	Based on Median and with adjusted df	3.116	1	24.254	.090
	Based on trimmed mean	3.448	1	29	.074
SOC_PAControl	Based on Mean	.270	1	29	.607
	Based on Median	.263	1	29	.612
	Based on Median and with adjusted df	.263	1	28.276	.612
	Based on trimmed mean	.313	1	29	.580
sense_of_coherence	Based on Mean	.280	1	29	.601

	Based on Median	.074	1	29	.787
	Based on Median and with adjusted df	.074	1	24.533	.788
	Based on trimmed mean	.250	1	29	.621

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + status

Tests of Between-Subjects Effects

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[e]
Corrected Model	SOC_Meaningful	20.730[a]	1	20.730	.291	.594	.010	.291	.082
	SOC_PerceivedUnderstanding	431.768[b]	1	431.768	7.134	.012	.197	7.134	.733
	SOC_PAControl	275.205[c]	1	275.205	3.953	.056	.120	3.953	.485
	sense_of_coherence	1729.084[d]	1	1729.084	4.450	.044	.133	4.450	.532
Intercept	SOC_Meaningful	40873.375	1	40873.375	574.256	.000	.952	574.256	1.000
	SOC_PerceivedUnderstanding	45192.542	1	45192.542	746.686	.000	.963	746.686	1.000
	SOC_PAControl	61080.367	1	61080.367	877.252	.000	.968	877.252	1.000
	sense_of_coherence	471624.310	1	471624.310	1213.681	.000	.977	1213.681	1.000
status	SOC_Meaningful	20.730	1	20.730	.291	.594	.010	.291	.082
	SOC_PerceivedUnderstanding	431.768	1	431.768	7.134	.012	.197	7.134	.733
	SOC_PAControl	275.205	1	275.205	3.953	.056	.120	3.953	.485
	sense_of_coherence	1729.084	1	1729.084	4.450	.044	.133	4.450	.532
Error	SOC_Meaningful	2064.109	29	71.176					
	SOC_PerceivedUnderstanding	1755.200	29	60.524					
	SOC_PAControl	2019.182	29	69.627					
	sense_of_coherence	11269.109	29	388.590					
Total	SOC_Meaningful	47306.000	31						
	SOC_PerceivedUnderstanding	54380.000	31						
	SOC_PAControl	71622.000	31						
	sense_of_coherence	546300.000	31						
Corrected Total	SOC_Meaningful	2084.839	30						
	SOC_PerceivedUnderstanding	2186.968	30						
	SOC_PAControl	2294.387	30						
	sense_of_coherence	12998.194	30						

a. R Squared = .010 (Adjusted R Squared = -.024)

b. R Squared = .197 (Adjusted R Squared = .170)

c. R Squared = .120 (Adjusted R Squared = .090)

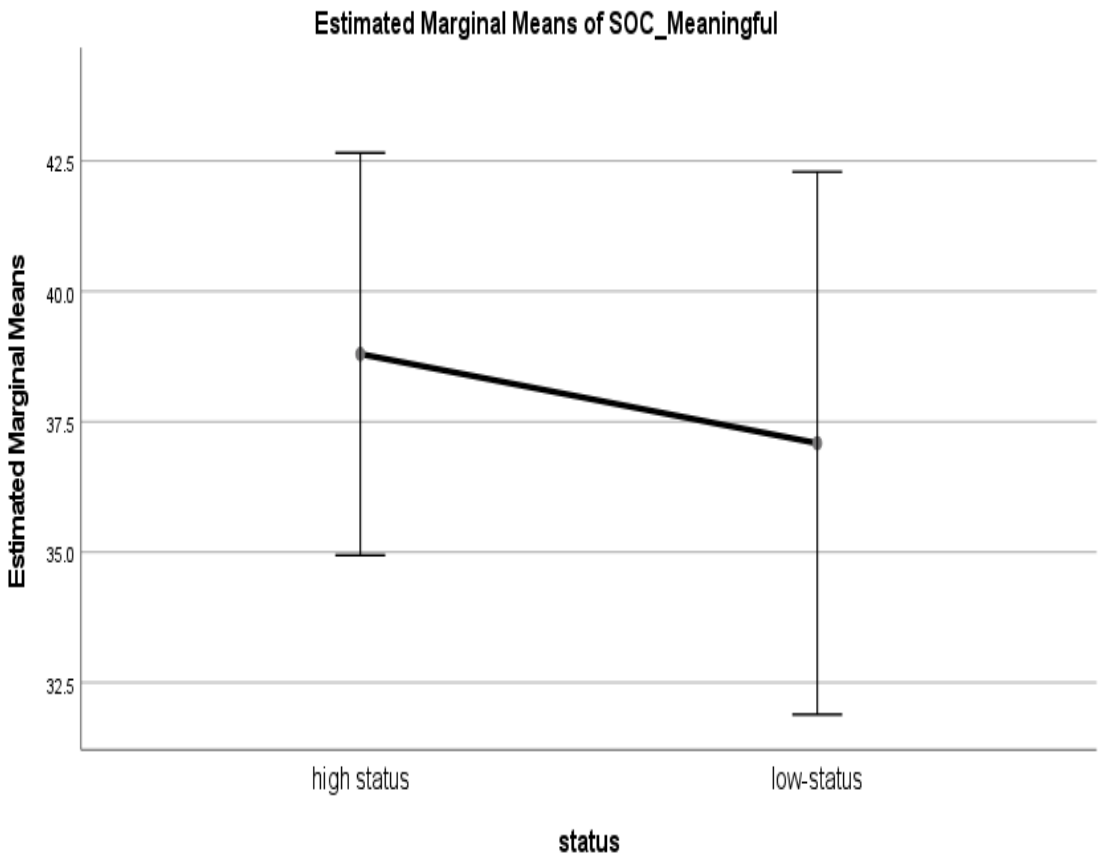
Estimated Marginal Means

status

Dependent Variable	status	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
SOC_Meaningful	high status	38.800	1.886	34.942	42.658
	low-status	37.091	2.544	31.888	42.293
SOC_PerceivedUnderstanding	high status	43.800	1.740	40.242	47.358
	low-status	36.000	2.346	31.203	40.797
SOC_PAControl	high status	49.500	1.866	45.684	53.316
	low-status	43.273	2.516	38.127	48.418
sense_of_coherence	high status	136.700	4.408	127.685	145.715
	low-status	121.091	5.944	108.935	133.247

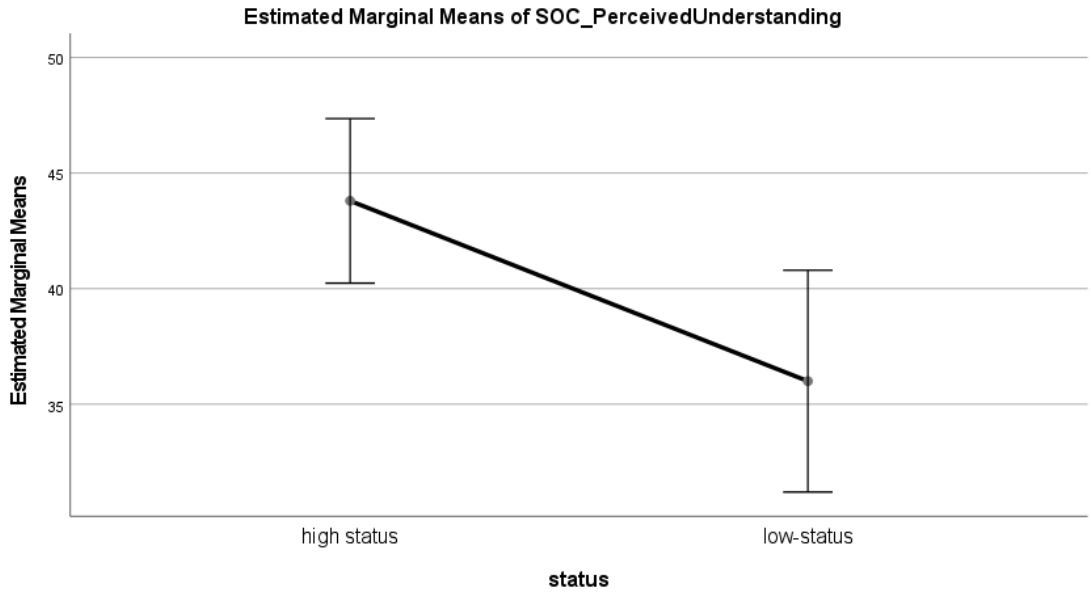
Profile Plots

SOC_Meaningful



Error bars: 95% CI

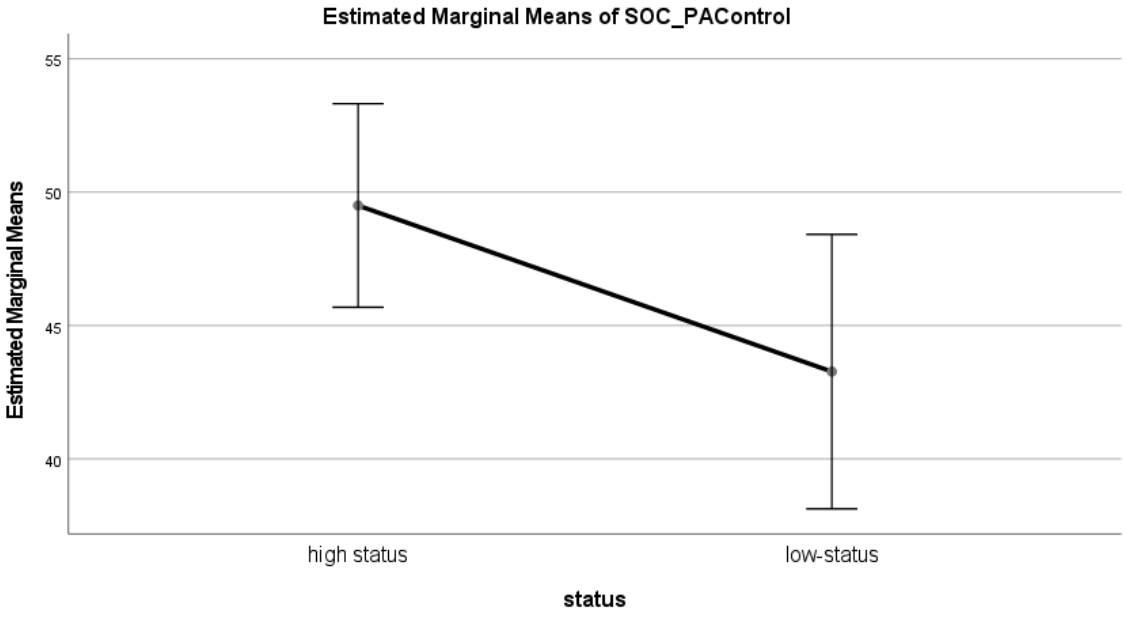
SOC_PerceivedUnderstanding



Error bars: 95% CI

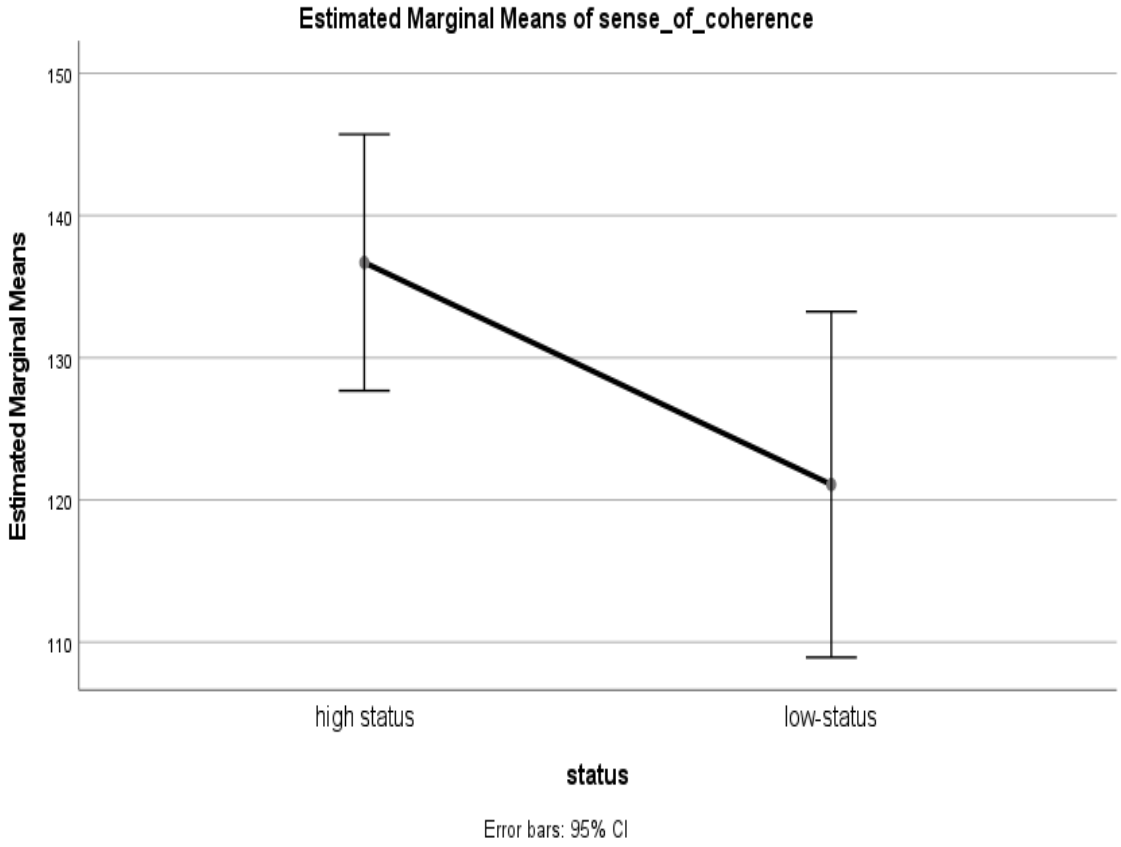
SOC_PAControl

SOC_PAControl



Error bars: 95% CI

sense_of_coherence



Appendix XXI

One-Way ANOVAs for Sense of Coherence

```
Your license renewal date has passed.  This product will stop working if a new license is not installed
soon.
ONEWAY SOC_Meaningful BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
  /POSTHOC=BONFERRONI ALPHA(0.05).
```

Oneway

```
[DataSet1] /Users/Koki/Desktop/OneDrive - University of Strathclyde/PhD/Data PhD/Master_Data.sav_completed.sav
```

Warnings

Post hoc tests are not performed for SOC_Meaningful because there are fewer than three groups.

Descriptives

SOC_Meaningful

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	38.80	6.630	1.483	35.70	41.90	26	48
low-status	11	37.09	11.086	3.342	29.64	44.54	17	51
Total	31	38.19	8.336	1.497	35.14	41.25	17	51

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
SOC_Meaningful	Based on Mean	6.069	1	29	.020
	Based on Median	1.766	1	29	.194
	Based on Median and with adjusted df	1.766	1	16.510	.202
	Based on trimmed mean	5.541	1	29	.026

ANOVA

SOC_Meaningful

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	20.730	1	20.730	.291	.594
Within Groups	2064.109	29	71.176		
Total	2084.839	30			

Robust Tests of Equality of Means

SOC_Meaningful

Robust Tests of Equality of Means

SOC_Meaningful

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	.218	1	14.036	.647

a. Asymptotically F distributed.

```
ONEWAY SOC_PerceivedUnderstanding BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
  /POSTHOC=BONFERRONI ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for SOC_PerceivedUnderstanding because there are fewer than three groups.

Descriptives

SOC_PerceivedUnderstanding

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	43.80	8.924	1.996	39.62	47.98	27	58
low-status	11	36.00	4.919	1.483	32.70	39.30	28	45
Total	31	41.03	8.538	1.533	37.90	44.16	27	58

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
SOC_PerceivedUnderstanding	Based on Mean	3.386	1	29	.076
	Based on Median	3.116	1	29	.088
	Based on Median and with adjusted df	3.116	1	24.254	.090
	Based on trimmed mean	3.448	1	29	.074

ANOVA

SOC_PerceivedUnderstanding

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	431.768	1	431.768	7.134	.012
Within Groups	1755.200	29	60.524		
Total	2186.968	30			

Robust Tests of Equality of Means

SOC_PerceivedUnderstanding

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	9.841	1	28.984	.004

a. Asymptotically F distributed.

```
ONEWAY SOC_PAControl BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
  /POSTHOC=BONFERRONI ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for SOC_PAControl because there are fewer than three groups.

Descriptives

SOC_PAControl

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	49.50	8.426	1.884	45.56	53.44	33	62
low-status	11	43.27	8.186	2.468	37.77	48.77	26	52
Total	31	47.29	8.745	1.571	44.08	50.50	26	62

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
SOC_PAControl	Based on Mean	.270	1	29	.607
	Based on Median	.263	1	29	.612
	Based on Median and with adjusted df	.263	1	28.276	.612
	Based on trimmed mean	.313	1	29	.580

ANOVA

SOC_PAControl

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	275.205	1	275.205	3.953	.056
Within Groups	2019.182	29	69.627		

Total	2294.387	30			

Robust Tests of Equality of Means

SOC_PAControl

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	4.022	1	21.251	.058

a. Asymptotically F distributed.

```
ONEWAY sense_of_coherence BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
  /POSTHOC=BONFERRONI ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for sense_of_coherence because there are fewer than three groups.

Descriptives

sense_of_coherence

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	136.70	18.607	4.161	127.99	145.41	105	172
low-status	11	121.09	21.659	6.530	106.54	135.64	75	150
Total	31	131.16	20.815	3.739	123.53	138.80	75	172

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
sense_of_coherence	Based on Mean	.280	1	29	.601
	Based on Median	.074	1	29	.787
	Based on Median and with adjusted df	.074	1	24.533	.788
	Based on trimmed mean	.250	1	29	.621

ANOVA

sense_of_coherence

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	1729.084	1	1729.084	4.450	.044

Within Groups	11269.109	29	388.590		
Total	12998.194	30			

Robust Tests of Equality of Means

sense_of_coherence

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	4.064	1	18.189	.059

a. Asymptotically F distributed.

Appendix XXII

MANOVA Analysis for Attributional Style

General Linear Model

```
[DataSet1] /Users/Koki/Desktop/OneDrive - University of Strathclyde/PhD/Data PhD/Master_Data.sav_completed.s
```

Warnings

Post hoc tests are not performed for status because there are fewer than three groups.

Between-Subjects Factors

		Value Label	N
status	0	high status	20
	1	low-status	11

Descriptive Statistics

	status	Mean	Std. Deviation	N
CoNeg_ASQ	high status	12.394	2.3001	20
	low-status	12.065	2.0731	11
	Total	12.277	2.1929	31
CoPos_ASQ	high status	15.200	2.0557	20
	low-status	14.226	1.8062	11
	Total	14.855	1.9970	31
CPCN_ASQ	high status	2.807	2.8910	20
	low-status	2.163	2.7400	11
	Total	2.578	2.8096	31
hopelessness	high status	4.0400	1.00058	20
	low-status	3.6900	.69477	11
	Total	3.9158	.90771	31
hopefulness	high status	5.2605	.75524	20
	low-status	4.5864	.74231	11
	Total	5.0213	.80773	31

Box's Test of Equality of Covariance Matrices[a]

Box's M	24.334
F	1.267
df1	15
df2	1712.504
Sig.	.215

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + status

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
Intercept	Pillai's Trace	.989	446.013[b]	5.000	25.000	.000	.989	2230.066	1.000
	Wilks' Lambda	.011	446.013[b]	5.000	25.000	.000	.989	2230.066	1.000
	Hotelling's Trace	89.203	446.013[b]	5.000	25.000	.000	.989	2230.066	1.000
	Roy's Largest Root	89.203	446.013[b]	5.000	25.000	.000	.989	2230.066	1.000
status	Pillai's Trace	.278	1.928[b]	5.000	25.000	.125	.278	9.642	.552
	Wilks' Lambda	.722	1.928[b]	5.000	25.000	.125	.278	9.642	.552
	Hotelling's Trace	.386	1.928[b]	5.000	25.000	.125	.278	9.642	.552
	Roy's Largest Root	.386	1.928[b]	5.000	25.000	.125	.278	9.642	.552

a. Design: Intercept + status

b. Exact statistic

c. Computed using alpha =

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
CoNeg_ASQ	Based on Mean	1.017	1	29	.322
	Based on Median	.932	1	29	.342
	Based on Median and with adjusted df	.932	1	28.116	.343
	Based on trimmed mean	1.027	1	29	.319
CoPos_ASQ	Based on Mean	.167	1	29	.686
	Based on Median	.238	1	29	.630
	Based on Median and with adjusted df	.238	1	28.686	.630

	Based on trimmed mean	.162	1	29	.690
CPCN_ASQ	Based on Mean	.410	1	29	.527
	Based on Median	.664	1	29	.422
	Based on Median and with adjusted df	.664	1	23.275	.423
	Based on trimmed mean	.548	1	29	.465
hopelessness	Based on Mean	2.513	1	29	.124
	Based on Median	1.838	1	29	.186
	Based on Median and with adjusted df	1.838	1	26.736	.187
	Based on trimmed mean	2.382	1	29	.134
hopefulness	Based on Mean	.001	1	29	.982
	Based on Median	.047	1	29	.831
	Based on Median and with adjusted df	.047	1	23.531	.831
	Based on trimmed mean	.001	1	29	.980

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + status

Tests of Between-Subjects Effects

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[f]
Corrected Model	CoNeg_ASQ	.768[a]	1	.768	.155	.696	.005	.155	.067
	CoPos_ASQ	6.728[b]	1	6.728	1.728	.199	.056	1.728	.246
	CPCN_ASQ	2.946[c]	1	2.946	.365	.550	.012	.365	.090
	hopelessness	.869[d]	1	.869	1.057	.312	.035	1.057	.169
	hopefulness	3.225[e]	1	3.225	5.721	.023	.165	5.721	.638
Intercept	CoNeg_ASQ	4245.262	1	4245.262	857.942	.000	.967	857.942	1.000
	CoPos_ASQ	6145.174	1	6145.174	1578.244	.000	.982	1578.244	1.000
	CPCN_ASQ	175.277	1	175.277	21.734	.000	.428	21.734	.995
	hopelessness	424.053	1	424.053	515.641	.000	.947	515.641	1.000
	hopefulness	688.108	1	688.108	1220.681	.000	.977	1220.681	1.000
status	CoNeg_ASQ	.768	1	.768	.155	.696	.005	.155	.067
	CoPos_ASQ	6.728	1	6.728	1.728	.199	.056	1.728	.246
	CPCN_ASQ	2.946	1	2.946	.365	.550	.012	.365	.090
	hopelessness	.869	1	.869	1.057	.312	.035	1.057	.169
	hopefulness	3.225	1	3.225	5.721	.023	.165	5.721	.638
Error	CoNeg_ASQ	143.498	29	4.948					
	CoPos_ASQ	112.917	29	3.894					
	CPCN_ASQ	233.875	29	8.065					
	hopelessness	23.849	29	.822					

	hopefulness	16.348	29	.564			
Total	CoNeg_ASQ	4816.560	31				
	CoPos_ASQ	6960.000	31				
	CPCN_ASQ	442.912	31				
	hopelessness	500.058	31				
	hopefulness	801.187	31				
Corrected Total	CoNeg_ASQ	144.265	30				
	CoPos_ASQ	119.644	30				
	CPCN_ASQ	236.821	30				
	hopelessness	24.718	30				
	hopefulness	19.573	30				

a. R Squared = .005 (Adjusted R Squared = -.029)

b. R Squared = .056 (Adjusted R Squared = .024)

c. R Squared = .012 (Adjusted R Squared = -.022)

d. R Squared = .035 (Adjusted R Squared = .002)

e. R Squared = .165 (Adjusted R Squared = .136)

f. Computed using alpha =

Estimated Marginal Means

status

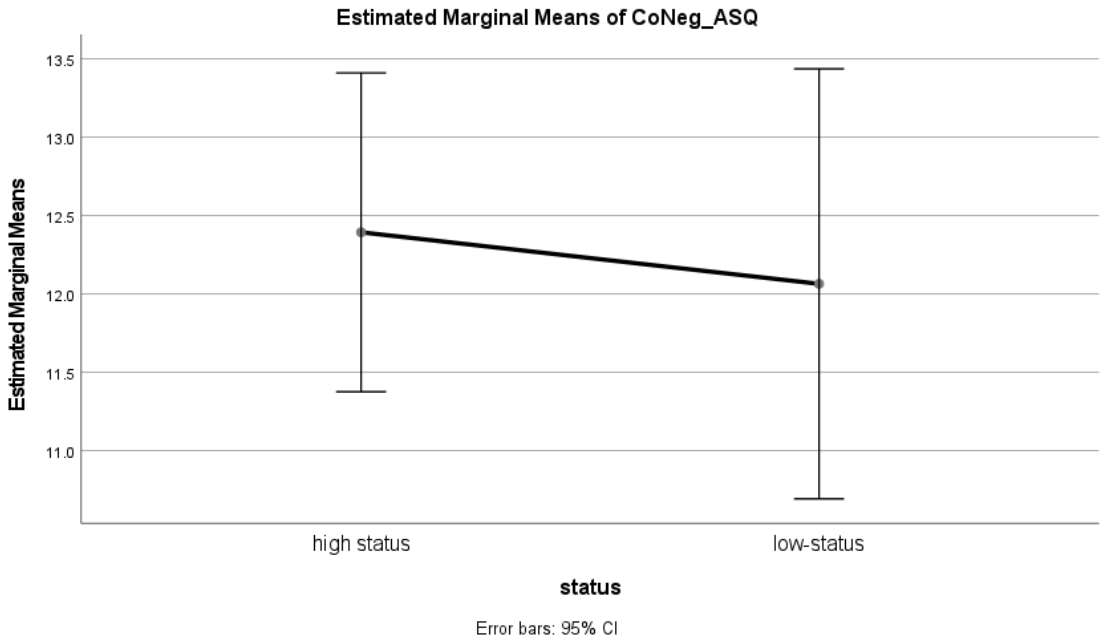
Dependent Variable	status	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
CoNeg_ASQ	high status	12.394	.497	11.376	13.411
	low-status	12.065	.671	10.693	13.436
CoPos_ASQ	high status	15.200	.441	14.298	16.102
	low-status	14.226	.595	13.010	15.443
CPCN_ASQ	high status	2.807	.635	1.508	4.106
	low-status	2.163	.856	.412	3.914
hopelessness	high status	4.040	.203	3.625	4.455
	low-status	3.690	.273	3.131	4.249
hopefulness	high status	5.261	.168	4.917	5.604
	low-status	4.586	.226	4.123	5.049

Profile Plots

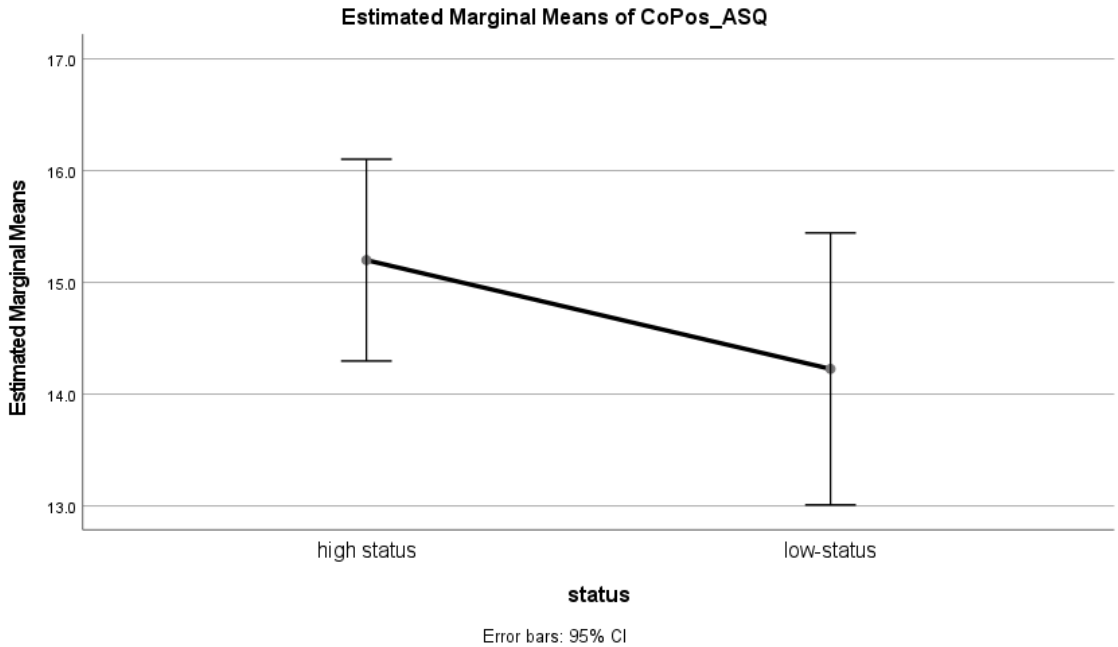
CoNeg_ASQ

Estimated Marginal Means of CoNeg_ASQ

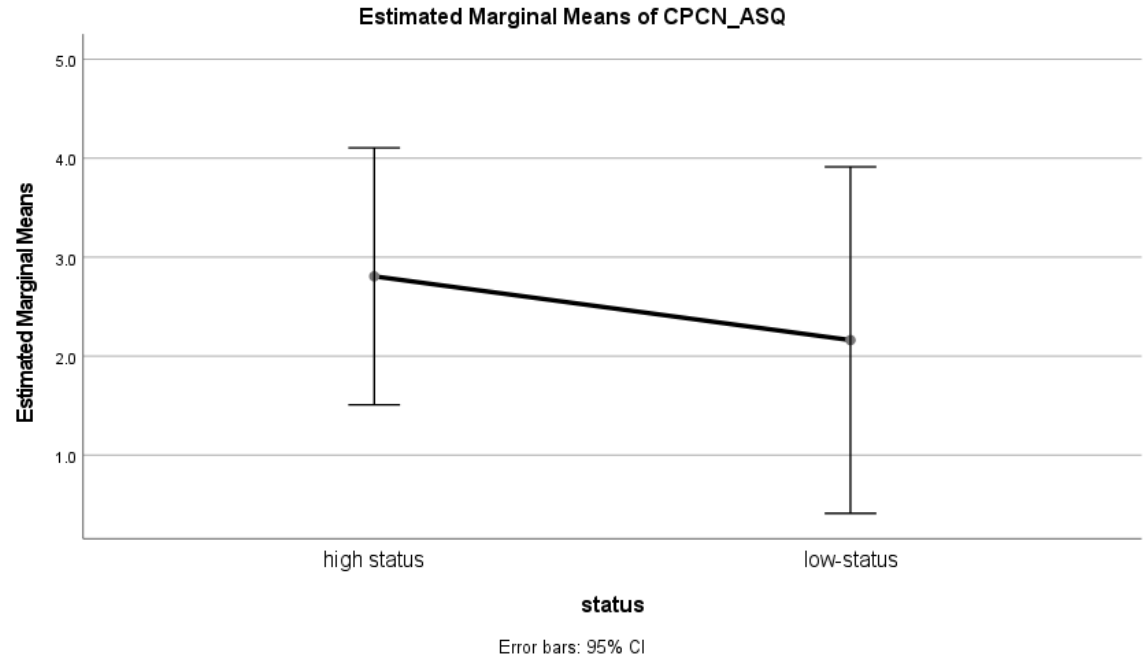
CoNeg_ASQ



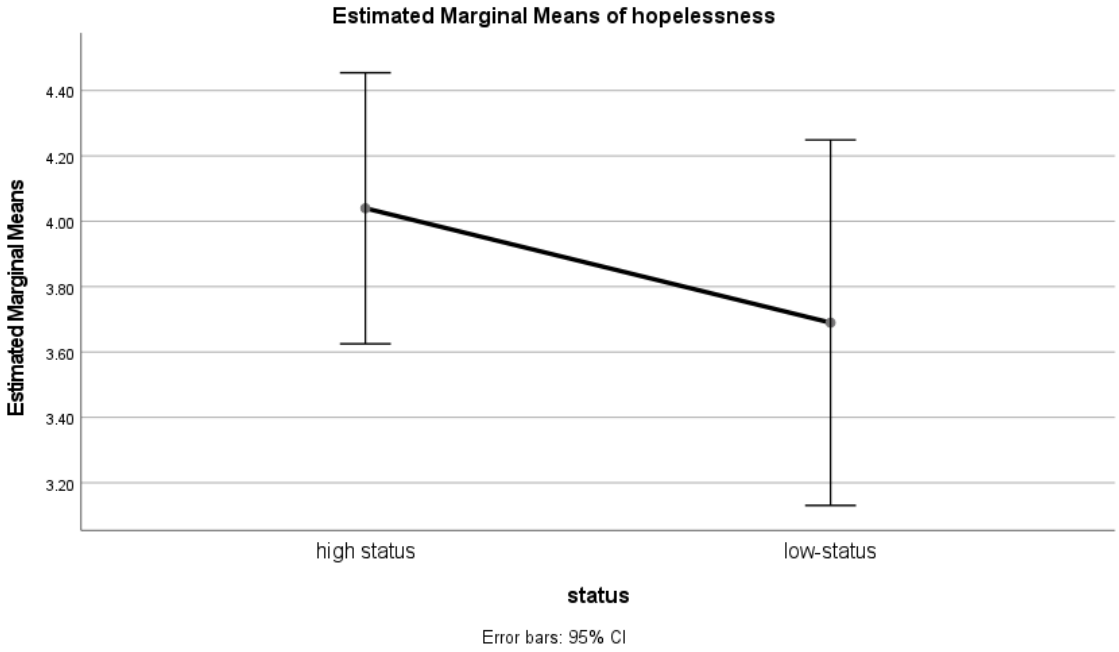
CoPos_ASQ



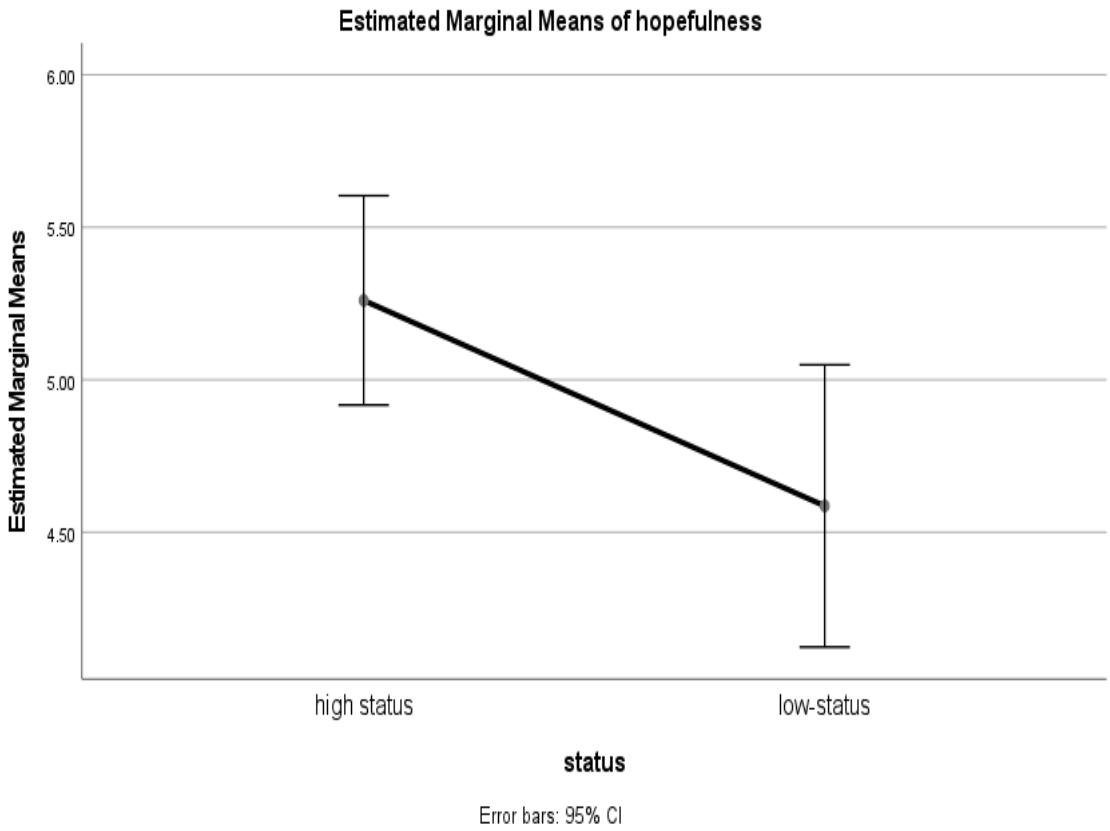
CPCN_ASQ



hopelessness



hopefulness



Appendix XXIII

One-Way ANOVAs for Attributional Style

Oneway

Warnings

Post hoc tests are not performed for CoNeg_ASQ because there are fewer than three groups.

Descriptives

CoNeg_ASQ

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	12.394	2.3001	.5143	11.317	13.470	9.0	16.0
low-status	11	12.065	2.0731	.6251	10.672	13.457	8.7	16.5
Total	31	12.277	2.1929	.3939	11.472	13.081	8.7	16.5

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
CoNeg_ASQ	Based on Mean	1.017	1	29	.322
	Based on Median	.932	1	29	.342
	Based on Median and with adjusted df	.932	1	28.116	.343
	Based on trimmed mean	1.027	1	29	.319

ANOVA

CoNeg_ASQ

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.768	1	.768	.155	.696
Within Groups	143.498	29	4.948		
Total	144.265	30			

Robust Tests of Equality of Means

CoNeg_ASQ

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	.165	1	22.659	.688

a. Asymptotically F distributed.

```
ONEWAY CoPos_ASQ BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
```

```
/POSTHOC=SCHEFFE ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for CoPos_ASQ because there are fewer than three groups.

Descriptives

CoPos_ASQ

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	15.200	2.0557	.4597	14.238	16.162	11.0	18.2
low-status	11	14.226	1.8062	.5446	13.013	15.440	11.7	17.2
Total	31	14.855	1.9970	.3587	14.122	15.587	11.0	18.2

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
CoPos_ASQ	Based on Mean	.167	1	29	.686
	Based on Median	.238	1	29	.630
	Based on Median and with adjusted df	.238	1	28.686	.630
	Based on trimmed mean	.162	1	29	.690

ANOVA

CoPos_ASQ

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	6.728	1	6.728	1.728	.199
Within Groups	112.917	29	3.894		
Total	119.644	30			

Robust Tests of Equality of Means

CoPos_ASQ

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	1.867	1	23.143	.185

a. Asymptotically F distributed.

```
ONEWAY CPCN_ASQ BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
  /POSTHOC=SCHEFFE ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for CPCN_ASQ because there are fewer than three groups.

Descriptives

CPCN_ASQ

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	2.807	2.8910	.6464	1.454	4.160	-1.3	8.2
low-status	11	2.163	2.7400	.8262	.322	4.004	-4.0	4.8
Total	31	2.578	2.8096	.5046	1.548	3.609	-4.0	8.2

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
CPCN_ASQ	Based on Mean	.410	1	29	.527
	Based on Median	.664	1	29	.422
	Based on Median and with adjusted df	.664	1	23.275	.423
	Based on trimmed mean	.548	1	29	.465

ANOVA

CPCN_ASQ

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	2.946	1	2.946	.365	.550
Within Groups	233.875	29	8.065		
Total	236.821	30			

Robust Tests of Equality of Means

CPCN_ASQ

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	.377	1	21.711	.545

a. Asymptotically F distributed.

```
ONEWAY hopelessness BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
  /POSTHOC=SCHEFFE ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for hopelessness because there are fewer than three groups.

Descriptives

hopelessness

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	4.0400	1.00058	.22374	3.5717	4.5083	2.58	6.34
low-status	11	3.6900	.69477	.20948	3.2232	4.1568	2.59	5.00
Total	31	3.9158	.90771	.16303	3.5829	4.2488	2.58	6.34

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
hopelessness	Based on Mean	2.513	1	29	.124
	Based on Median	1.838	1	29	.186
	Based on Median and with adjusted df	1.838	1	26.736	.187
	Based on trimmed mean	2.382	1	29	.134

ANOVA

hopelessness

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.869	1	.869	1.057	.312
Within Groups	23.849	29	.822		
Total	24.718	30			

Robust Tests of Equality of Means

hopelessness

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	1.304	1	27.199	.263

a. Asymptotically F distributed.

```
ONEWAY hopefulness BY status
  /STATISTICS DESCRIPTIVES HOMOGENEITY BROWNFORSYTHE
  /MISSING ANALYSIS
  /POSTHOC=SCHEFFE ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for hopefulness because there are fewer than three groups.

Descriptives

hopefulness

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	20	5.2605	.75524	.16888	4.9070	5.6140	3.67	6.42
low-status	11	4.5864	.74231	.22382	4.0877	5.0851	3.50	5.75
Total	31	5.0213	.80773	.14507	4.7250	5.3176	3.50	6.42

Test of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
hopefulness	Based on Mean	.001	1	29	.982
	Based on Median	.047	1	29	.831
	Based on Median and with adjusted df	.047	1	23.531	.831
	Based on trimmed mean	.001	1	29	.980

ANOVA

hopefulness

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	3.225	1	3.225	5.721	.023
Within Groups	16.348	29	.564		
Total	19.573	30			

Robust Tests of Equality of Means

hopefulness

	Statistic[a]	df1	df2	Sig.
Brown-Forsythe	5.781	1	21.039	.025

a. Asymptotically F distributed.

Appendix XXIV

Manova Analysis for NEO-FFI-3

General Linear Model

Warnings

Post hoc tests are not performed for status because there are fewer than three groups.

Between-Subjects Factors

		Value Label	N
status	0	high status	19
	1	low-status	11

Descriptive Statistics

	status	Mean	Std. Deviation	N
N_NEO_FFI_3	high status	1.89	1.243	19
	low-status	3.00	.894	11
	Total	2.30	1.236	30
E_NEO_FFI_3	high status	2.16	.958	19
	low-status	2.64	.924	11
	Total	2.33	.959	30
O_NEO_FFI_3	high status	2.53	.964	19
	low-status	2.45	.820	11
	Total	2.50	.900	30
A_NEO_FFI_3	high status	2.11	1.449	19
	low-status	1.91	1.221	11
	Total	2.03	1.351	30
C_NEO_FFI_3	high status	2.42	1.121	19
	low-status	1.45	1.128	11
	Total	2.07	1.202	30

Box's Test of Equality of Covariance Matrices[a]

Box's M	38.401
F	1.993
df1	15
df2	1747.751
Sig.	.013

Tests the null

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + status

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
Intercept	Pillai's Trace	.952	94.773[b]	5.000	24.000	.000	.952	473.864	1.000
	Wilks' Lambda	.048	94.773[b]	5.000	24.000	.000	.952	473.864	1.000
	Hotelling's Trace	19.744	94.773[b]	5.000	24.000	.000	.952	473.864	1.000
	Roy's Largest Root	19.744	94.773[b]	5.000	24.000	.000	.952	473.864	1.000
status	Pillai's Trace	.426	3.557[b]	5.000	24.000	.015	.426	17.784	.844
	Wilks' Lambda	.574	3.557[b]	5.000	24.000	.015	.426	17.784	.844
	Hotelling's Trace	.741	3.557[b]	5.000	24.000	.015	.426	17.784	.844
	Roy's Largest Root	.741	3.557[b]	5.000	24.000	.015	.426	17.784	.844

a. Design: Intercept + status

b. Exact statistic

c. Computed using alpha =

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
N_NEO_FFI_3	Based on Mean	1.006	1	28	.324
	Based on Median	.720	1	28	.403
	Based on Median and with adjusted df	.720	1	24.162	.405
	Based on trimmed mean	1.041	1	28	.316
E_NEO_FFI_3	Based on Mean	.155	1	28	.697
	Based on Median	.290	1	28	.595
	Based on Median and with adjusted df	.290	1	27.333	.595
	Based on trimmed mean	.221	1	28	.642
O_NEO_FFI_3	Based on Mean	.561	1	28	.460
	Based on Median	.027	1	28	.871
	Based on Median and with adjusted df	.027	1	27.162	.871

	Based on trimmed mean	.556	1	28	.462
A_NEO_FFI_3	Based on Mean	2.472	1	28	.127
	Based on Median	2.521	1	28	.124
	Based on Median and with adjusted df	2.521	1	25.760	.125
	Based on trimmed mean	2.461	1	28	.128
C_NEO_FFI_3	Based on Mean	.024	1	28	.877
	Based on Median	.002	1	28	.965
	Based on Median and with adjusted df	.002	1	27.934	.965
	Based on trimmed mean	.028	1	28	.867

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + status

Tests of Between-Subjects Effects

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[f]
Corrected Model	N_NEO_FFI_3	8.511[a]	1	8.511	6.658	.015	.192	6.658	.702
	E_NEO_FFI_3	1.595[b]	1	1.595	1.781	.193	.060	1.781	.252
	O_NEO_FFI_3	.036[c]	1	.036	.043	.838	.002	.043	.055
	A_NEO_FFI_3	.268[d]	1	.268	.142	.709	.005	.142	.065
	C_NEO_FFI_3	6.508[e]	1	6.508	5.153	.031	.155	5.153	.592
Intercept	N_NEO_FFI_3	166.911	1	166.911	130.583	.000	.823	130.583	1.000
	E_NEO_FFI_3	160.128	1	160.128	178.830	.000	.865	178.830	1.000
	O_NEO_FFI_3	172.836	1	172.836	206.247	.000	.880	206.247	1.000
	A_NEO_FFI_3	112.268	1	112.268	59.651	.000	.681	59.651	1.000
	C_NEO_FFI_3	104.641	1	104.641	82.863	.000	.747	82.863	1.000
status	N_NEO_FFI_3	8.511	1	8.511	6.658	.015	.192	6.658	.702
	E_NEO_FFI_3	1.595	1	1.595	1.781	.193	.060	1.781	.252
	O_NEO_FFI_3	.036	1	.036	.043	.838	.002	.043	.055
	A_NEO_FFI_3	.268	1	.268	.142	.709	.005	.142	.065
	C_NEO_FFI_3	6.508	1	6.508	5.153	.031	.155	5.153	.592
Error	N_NEO_FFI_3	35.789	28	1.278					
	E_NEO_FFI_3	25.072	28	.895					
	O_NEO_FFI_3	23.464	28	.838					
	A_NEO_FFI_3	52.699	28	1.882					
	C_NEO_FFI_3	35.359	28	1.263					
Total	N_NEO_FFI_3	203.000	30						
	E_NEO_FFI_3	190.000	30						
	O_NEO_FFI_3	211.000	30						

	A_NEO_FFI_3	177.000	30			
	C_NEO_FFI_3	170.000	30			
Corrected Total	N_NEO_FFI_3	44.300	29			
	E_NEO_FFI_3	26.667	29			
	O_NEO_FFI_3	23.500	29			
	A_NEO_FFI_3	52.967	29			
	C_NEO_FFI_3	41.867	29			

a. R Squared = .192 (Adjusted R Squared = .163)

b. R Squared = .060 (Adjusted R Squared = .026)

c. R Squared = .002 (Adjusted R Squared = -.034)

d. R Squared = .005 (Adjusted R Squared = -.030)

e. R Squared = .155 (Adjusted R Squared = .125)

f. Computed using alpha =

Estimated Marginal Means

status

Dependent Variable	status	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
N_NEO_FFI_3	high status	1.895	.259	1.363	2.426
	low-status	3.000	.341	2.302	3.698
E_NEO_FFI_3	high status	2.158	.217	1.713	2.603
	low-status	2.636	.285	2.052	3.221
O_NEO_FFI_3	high status	2.526	.210	2.096	2.957
	low-status	2.455	.276	1.889	3.020
A_NEO_FFI_3	high status	2.105	.315	1.461	2.750
	low-status	1.909	.414	1.062	2.756
C_NEO_FFI_3	high status	2.421	.258	1.893	2.949
	low-status	1.455	.339	.760	2.149

Appendix XXV

Separate One-Way ANOVAs for NEO-FFI-3

```
ONEWAY N_NEO_FFI_3 BY status
  /ES=OVERALL
  /STATISTICS DESCRIPTIVES HOMOGENEITY
  /MISSING ANALYSIS
  /CRITERIA=CILEVEL(0.95)
  /POSTHOC=SCHEFFE SIDAK ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for N_NEO_FFI_3 because there are fewer than three groups.

Descriptives

N_NEO_FFI_3

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	19	1.89	1.243	.285	1.30	2.49	0	4
low-status	11	3.00	.894	.270	2.40	3.60	2	4
Total	30	2.30	1.236	.226	1.84	2.76	0	4

Tests of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
N_NEO_FFI_3	Based on Mean	1.006	1	28	.324
	Based on Median	.720	1	28	.403
	Based on Median and with adjusted df	.720	1	24.162	.405
	Based on trimmed mean	1.041	1	28	.316

ANOVA

N_NEO_FFI_3

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	8.511	1	8.511	6.658	.015
Within Groups	35.789	28	1.278		
Total	44.300	29			

ANOVA Effect Sizes[a,b]

		Point Estimate	95% Confidence Interval Lower	95% Confidence Interval Upper
N_NEO_FFI_3	Eta-squared	.192	.007	.417
	Epsilon-squared	.163	-.029	.396
	Omega-squared Fixed-effect	.159	-.028	.388
	Omega-squared Random-effect	.159	-.028	.388

a. Eta-squared and Epsilon-squared are estimated based on the fixed-effect model.

b. Negative but less biased estimates are retained, not rounded to zero.

```
ONEWAY E_NEO_FFI_3 BY status
  /ES=OVERALL
  /STATISTICS DESCRIPTIVES HOMOGENEITY
  /MISSING ANALYSIS
  /CRITERIA=CILEVEL(0.95)
  /POSTHOC=SCHEFFE SIDAK ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for E_NEO_FFI_3 because there are fewer than three groups.

Descriptives

E_NEO_FFI_3

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean Lower Bound	95% Confidence Interval for Mean Upper Bound	Minimum	Maximum
high status	19	2.16	.958	.220	1.70	2.62	1	4
low-status	11	2.64	.924	.279	2.02	3.26	1	4
Total	30	2.33	.959	.175	1.98	2.69	1	4

Tests of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
E_NEO_FFI_3	Based on Mean	.155	1	28	.697
	Based on Median	.290	1	28	.595
	Based on Median and with adjusted df	.290	1	27.333	.595
	Based on trimmed mean	.221	1	28	.642

ANOVA

E_NEO_FFI_3

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	1.595	1	1.595	1.781	.193
Within Groups	25.072	28	.895		
Total	26.667	29			

ANOVA Effect Sizes[a,b]

		Point Estimate	95% Confidence Interval	
			Lower	Upper
E_NEO_FFI_3	Eta-squared	.060	.000	.268
	Epsilon-squared	.026	-.036	.242
	Omega-squared Fixed-effect	.025	-.034	.236
	Omega-squared Random-effect	.025	-.034	.236

a. Eta-squared and Epsilon-squared are estimated based on the fixed-effect model.

b. Negative but less biased estimates are retained, not rounded to zero.

```
ONEWAY O_NEO_FFI_3 BY status
  /ES=OVERALL
  /STATISTICS DESCRIPTIVES HOMOGENEITY
  /MISSING ANALYSIS
  /CRITERIA=CILEVEL(0.95)
  /POSTHOC=SCHEFFE SIDAK ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for O_NEO_FFI_3 because there are fewer than three groups.

Descriptives

O_NEO_FFI_3

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	19	2.53	.964	.221	2.06	2.99	1	4
low-status	11	2.45	.820	.247	1.90	3.01	1	4
Total	30	2.50	.900	.164	2.16	2.84	1	4

Tests of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
O_NEO_FFI_3	Based on Mean	.561	1	28	.460
	Based on Median	.027	1	28	.871
	Based on Median and with adjusted df	.027	1	27.162	.871
	Based on trimmed mean	.556	1	28	.462

ANOVA

O_NEO_FFI_3

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.036	1	.036	.043	.838
Within Groups	23.464	28	.838		
Total	23.500	29			

ANOVA Effect Sizes[a,b]

		Point Estimate	95% Confidence Interval	
			Lower	Upper
O_NEO_FFI_3	Eta-squared	.002	.000	.112
	Epsilon-squared	-.034	-.036	.081
	Omega-squared Fixed-effect	-.033	-.034	.078
	Omega-squared Random-effect	-.033	-.034	.078

a. Eta-squared and Epsilon-squared are estimated based on the fixed-effect model.

b. Negative but less biased estimates are retained, not rounded to zero.

```
ONEWAY A_NEO_FFI_3 BY status
  /ES=OVERALL
  /STATISTICS DESCRIPTIVES HOMOGENEITY
  /MISSING ANALYSIS
  /CRITERIA=CILEVEL(0.95)
  /POSTHOC=SCHEFFE SIDAK ALPHA(0.05).
```

Oneway

Warnings

Post hoc tests are not performed for A_NEO_FFI_3 because there are fewer than three groups.

Descriptives

A_NEO_FFI_3

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	19	2.11	1.449	.332	1.41	2.80	0	4
low-status	11	1.91	1.221	.368	1.09	2.73	0	4
Total	30	2.03	1.351	.247	1.53	2.54	0	4

Tests of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
A_NEO_FFI_3	Based on Mean	2.472	1	28	.127
	Based on Median	2.521	1	28	.124
	Based on Median and with adjusted df	2.521	1	25.760	.125
	Based on trimmed mean	2.461	1	28	.128

ANOVA

A_NEO_FFI_3

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.268	1	.268	.142	.709
Within Groups	52.699	28	1.882		
Total	52.967	29			

ANOVA Effect Sizes[a,b]

		Point Estimate	95% Confidence Interval	
			Lower	Upper
A_NEO_FFI_3	Eta-squared	.005	.000	.147
	Epsilon-squared	-.030	-.036	.116
	Omega-squared Fixed-effect	-.029	-.034	.113
	Omega-squared Random-effect	-.029	-.034	.113

a. Eta-squared and Epsilon-squared are estimated based on the fixed-effect model.

b. Negative but less biased estimates are retained, not rounded to zero.

```
ONEWAY C_NEO_FFI_3 BY status
  /ES=OVERALL
  /STATISTICS DESCRIPTIVES HOMOGENEITY
  /MISSING ANALYSIS
```

Warnings

Post hoc tests are not performed for C_NEO_FFI_3 because there are fewer than three groups.

Descriptives

C_NEO_FFI_3

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
high status	19	2.42	1.121	.257	1.88	2.96	0	4
low-status	11	1.45	1.128	.340	.70	2.21	0	3
Total	30	2.07	1.202	.219	1.62	2.52	0	4

Tests of Homogeneity of Variances

		Levene Statistic	df1	df2	Sig.
C_NEO_FFI_3	Based on Mean	.024	1	28	.877
	Based on Median	.002	1	28	.965
	Based on Median and with adjusted df	.002	1	27.934	.965
	Based on trimmed mean	.028	1	28	.867

ANOVA

C_NEO_FFI_3

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	6.508	1	6.508	5.153	.031
Within Groups	35.359	28	1.263		
Total	41.867	29			

ANOVA Effect Sizes[a,b]

		Point Estimate	95% Confidence Interval	
			Lower	Upper
C_NEO_FFI_3	Eta-squared	.155	.000	.381
	Epsilon-squared	.125	-.036	.359
	Omega-squared Fixed-effect	.122	-.034	.351
	Omega-squared Random-effect	.122	-.034	.351

a. Eta-squared and Epsilon-squared are estimated based on the fixed-effect model.

b. Negative but less biased estimates are retained, not rounded to zero.

Appendix XXVI

Threat/Challenge Appraisals by SES

NPar Tests

Descriptive Statistics

	N	Mean	Std. Deviation	Minimum	Maximum	Percentiles 25th	50th (Median)	75th
threat	30	1.20	1.472	-3	3	.00	2.00	2.00
status	31	.35	.486	0	1	.00	.00	1.00

Mann-Whitney Test

Ranks

	status	N	Mean Rank	Sum of Ranks
threat	high status	20	17.75	355.00
	low-status	10	11.00	110.00
	Total	30		

Test Statistics[a]

	threat
Mann-Whitney U	55.000
Wilcoxon W	110.000
Z	-2.079
Asymp. Sig. (2-tailed)	.038
Exact Sig. [2*(1-tailed Sig.)]	.049[b]

a. Grouping Variable: status

b. Not corrected for ties.

```
EXAMINE VARIABLES=Apply_Job_Motv Complete_Form_Motv Help_Funding_Motv Wellbeing_Course_Motv
    Apply_Loan_Motv BY status
  /PLOT BOXPLOT STEMLEAF HISTOGRAM NPPLOT
  /COMPARE GROUPS
  /STATISTICS DESCRIPTIVES EXTREME
  /CINTERVAL 95
  /MISSING LISTWISE
  /NOTOTAL.
```

Appendix XXVII

Task Workload and Threat/Challenge Appraisals

```
NONPAR CORR
  /VARIABLES=Total_NASA threat
  /PRINT=SPEARMAN TWOTAIL NOSIG
  /MISSING=PAIRWISE.
```

Nonparametric Correlations

Correlations

			Total_NASA	threat
Spearman's rho	Total_NASA	Correlation Coefficient	1.000	.289
		Sig. (2-tailed)	.	.121
		N	30	30
	threat	Correlation Coefficient	.289	1.000
		Sig. (2-tailed)	.121	.
		N	30	30

Appendix XXVIII

Multivariate Kruskal-Wallis for Motivational Questionnaire Data

```
NPTESTS
  /INDEPENDENT TEST (Apply_Job_Motv Complete_Form_Motv Help_Funding_Motv Wellbeing_Course_Motv A
  /MISSING SCOPE=ANALYSIS USERMISSING=EXCLUDE
  /CRITERIA ALPHA=0.05  CILEVEL=95.
```

Nonparametric Tests

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.	Decision
1	The distribution of Apply_Job_Motv is the same across categories of Outcome.	Independent-Samples Kruskal-Wallis Test	.966	Retain the null hypothesis.
2	The distribution of Complete_Form_Motv is the same across categories of Outcome.	Independent-Samples Kruskal-Wallis Test	.360	Retain the null hypothesis.
3	The distribution of Help_Funding_Motv is the same across categories of Outcome.	Independent-Samples Kruskal-Wallis Test	.451	Retain the null hypothesis.
4	The distribution of Wellbeing_Course_Motv is the same across categories of Outcome.	Independent-Samples Kruskal-Wallis Test	.255	Retain the null hypothesis.
5	The distribution of Apply_Loan_Motv is the same across categories of Outcome.	Independent-Samples Kruskal-Wallis Test	.031	Reject the null hypothesis.

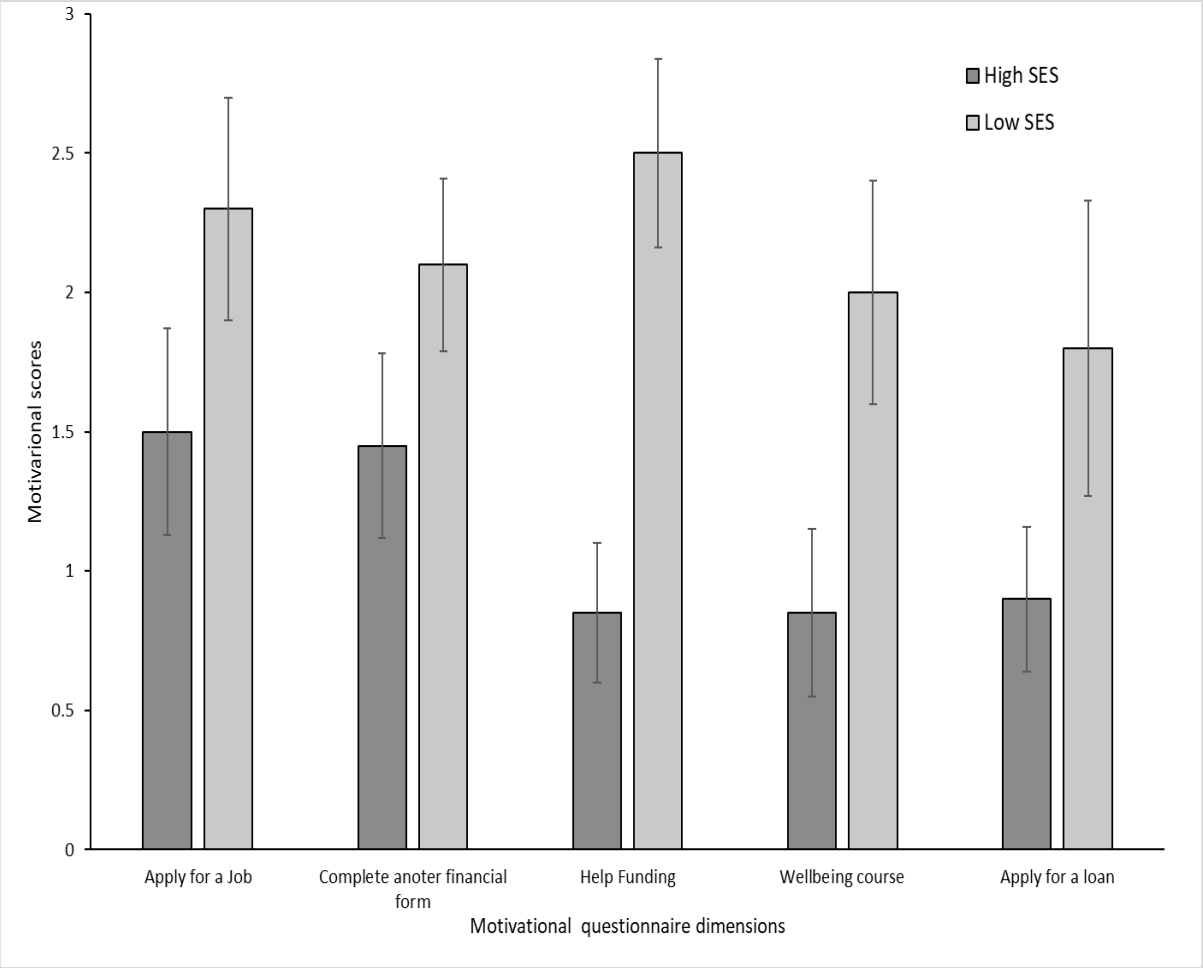
Asymptotic significances are displayed. The significance level is .05.

Appendix XXIX

Motivational Questionnaire by SES (Figure)

Figure 21

Motivational Questionnaire Scores Across SES Groups (n= 30) (Mean and ±SEM)



Appendix XXX

Pearson's R Matrix Correlation between Psychological data and Post-competition T Reactivity

Correlations

Correlations

		Testo_difference_immediately_percentage	SOC_PerceivedUnderstanding	SOC_PAControl	sense_of_coherence	CPCN_ASQ	hopefulness
Testo_difference_immediately_percentage	Pearson Correlation	1	-.201	-.213	-.227	-.044	-.119
	Sig. (2-tailed)		.279	.250	.219	.813	.523
	N	31	31	31	31	31	31
SOC_PerceivedUnderstanding	Pearson Correlation	-.201	1	.514**	.733**	-.103	-.097
	Sig. (2-tailed)	.279		.003	.000	.583	.605
	N	31	31	31	31	31	31
SOC_PAControl	Pearson Correlation	-.213	.514**	1	.895**	.314	.299
	Sig. (2-tailed)	.250	.003		.000	.085	.102
	N	31	31	31	31	31	31
sense_of_coherence	Pearson Correlation	-.227	.733**	.895**	1	.214	.160
	Sig. (2-tailed)	.219	.000	.000		.248	.390
	N	31	31	31	31	31	31
CPCN_ASQ	Pearson Correlation	-.044	-.103	.314	.214	1	.546**
	Sig. (2-tailed)	.813	.583	.085	.248		.001
	N	31	31	31	31	31	31
hopefulness	Pearson Correlation	-.119	-.097	.299	.160	.546**	1
	Sig. (2-tailed)	.523	.605	.102	.390	.001	
	N	31	31	31	31	31	31

**. Correlation is significant at the 0.01 level (2-tailed).

```
EXAMINE VARIABLES=Testo_difference_immediately_percentage
  /PLOT BOXPLOT STEMLEAF HISTOGRAM NPPLOT
  /COMPARE GROUPS
  /STATISTICS DESCRIPTIVES
  /CINTERVAL 95
  /MISSING LISTWISE
  /NOTOTAL.
```

Appendix XXXI

Spearman's Rank Matrix Correlation between Psychological Data and Post-Competition T Reactivity

Nonparametric Correlations

Correlations

			Help_Funding_Motv	Wellbeing_Course_Motv	C_NEO_FFI_3	N_NEO_FFI_3	threat	Status	Testo_difference_immediately_percentage
Spearman's rho	Help_Funding_Motv	Correlation Coefficient	1.000	.671**	-.203	.562**	-.031	.576**	.220
		Sig. (2-tailed)	.	.000	.291	.001	.870	.001	.244
		N	30	30	29	29	30	30	30
	Wellbeing_Course_Motv	Correlation Coefficient	.671**	1.000	-.203	.416*	-.168	.413*	.042
		Sig. (2-tailed)	.000	.	.292	.025	.375	.023	.826
		N	30	30	29	29	30	30	30
	C_NEO_FFI_3	Correlation Coefficient	-.203	-.203	1.000	-.348	.302	-.388*	-.056
		Sig. (2-tailed)	.291	.292	.	.059	.105	.034	.771
		N	29	29	30	30	30	30	30
	N_NEO_FFI_3	Correlation Coefficient	.562**	.416*	-.348	1.000	.124	.433*	.304
		Sig. (2-tailed)	.001	.025	.059	.	.512	.017	.103
		N	29	29	30	30	30	30	30
	threat	Correlation Coefficient	-.031	-.168	.302	.124	1.000	-.433*	-.351
		Sig. (2-tailed)	.870	.375	.105	.512	.	.015	.053
		N	30	30	30	30	31	31	31
	Status	Correlation Coefficient	.576**	.413*	-.388*	.433*	-.433*	1.000	.309
		Sig. (2-tailed)	.001	.023	.034	.017	.015	.	.091
		N	30	30	30	30	31	31	31
	Testo_difference_immediately_percentage	Correlation Coefficient	.220	.042	-.056	.304	-.351	.309	1.000
		Sig. (2-tailed)	.244	.826	.771	.103	.053	.091	.
		N	30	30	30	30	31	31	31

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

```
CORRELATIONS
  /VARIABLES=Testo_difference_immediately_percentage SOC_PerceivedUnderstanding SOC_PAControl
    sense_of_coherence CPCN_ASQ hopefulness
  /PRINT=TWOTAIL NOSIG
  /MISSING=PAIRWISE.
```

Appendix XXXII

Multiple Regression T, SES and Threat-Challenge Cognitions

Regression

Descriptive Statistics

	Mean	Std. Deviation	N
Testo_difference_immediately_percentage	-.8021	16.14741	31
Status	.3548	.48637	31
threat	1.03	1.722	31

Correlations

		Testo_difference_immediately_percentage	Status	threat
Pearson Correlation	Testo_difference_immediately_percentage	1.000	.277	-.225
	Status	.277	1.000	-.452
	threat	-.225	-.452	1.000
Sig. (1-tailed)	Testo_difference_immediately_percentage	.	.066	.112
	Status	.066	.	.005
	threat	.112	.005	.
N	Testo_difference_immediately_percentage	31	31	31
	Status	31	31	31
	threat	31	31	31

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	threat, Status[b]	.	Enter

a. Dependent Variable: Testo_difference_immediately_percentage

b. All requested variables entered.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.299[a]	.089	.024	15.95115	.089	1.371	2	28	.270

a. Predictors: (Constant), threat, Status

a. Predictors: (Constant), threat, Status

b. Dependent Variable: Testo_difference_immediately_percentage

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	697.873	2	348.936	1.371	.270[b]
	Residual	7124.295	28	254.439		
	Total	7822.167	30			

a. Dependent Variable: Testo_difference_immediately_percentage

b. Predictors: (Constant), threat, Status

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	-2.194	4.682		-.469	.643					
	Status	7.328	6.712	.221	1.092	.284	.277	.202	.197	.796	1.257
	threat	-1.171	1.896	-.125	-.618	.542	-.225	-.116	-.111	.796	1.257

a. Dependent Variable: Testo_difference_immediately_percentage

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions		
				(Constant)	Status	threat
1	1	1.791	1.000	.10	.08	.07
	2	1.000	1.338	.00	.22	.33
	3	.209	2.927	.90	.70	.60

a. Dependent Variable: Testo_difference_immediately_percentage

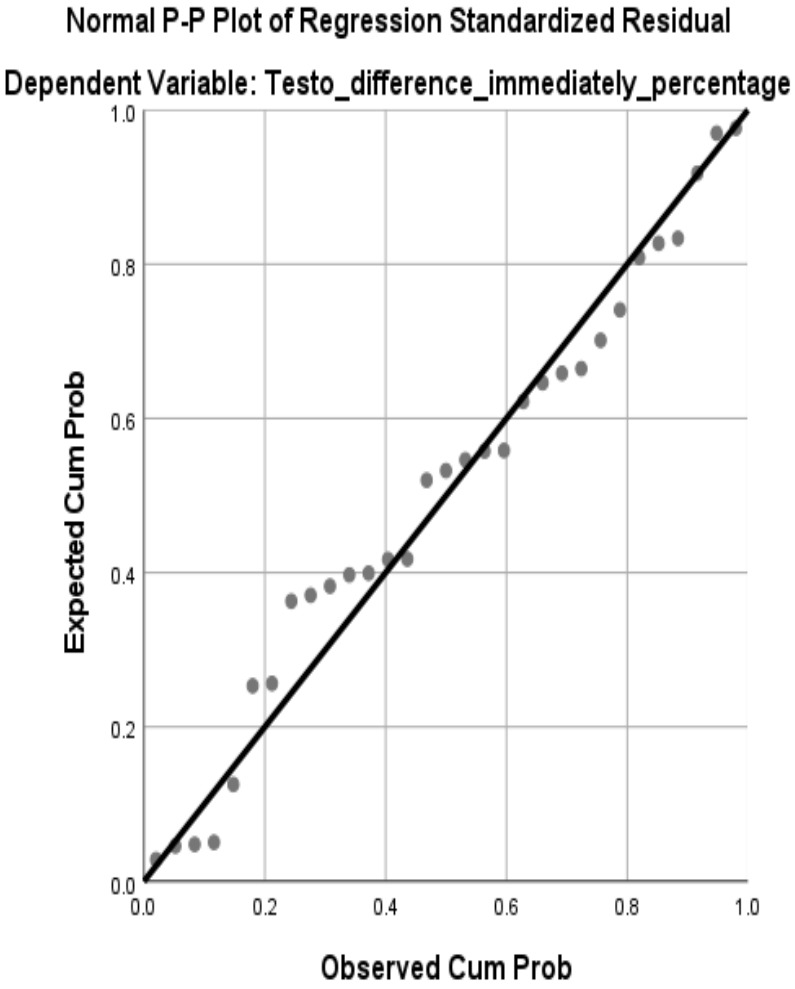
Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	-5.7061	9.8171	-.8021	4.82311	31
Std. Predicted Value	-1.017	2.202	.000	1.000	31
Standard Error of Predicted Value	3.646	8.979	4.756	1.438	31
Adjusted Predicted Value	-6.2792	22.3021	-.3673	6.25887	31
Residual	-30.42329	31.62534	.00000	15.41027	31
Std. Residual	-1.907	1.983	.000	.966	31
Stud. Residual	-2.041	2.037	-.012	1.029	31
Deleted Residual	-39.39757	33.36921	-.43480	17.60203	31

Deleted Residual	-39.39757	33.36921	-.43480	17.60203	31
Stud. Deleted Residual	-2.173	2.167	-.017	1.071	31
Mahal. Distance	.600	8.539	1.935	1.976	31
Cook's Distance	.000	.644	.051	.127	31
Centered Leverage Value	.020	.285	.065	.066	31

a. Dependent Variable: Testo_difference_immediately_percentage

Charts



Appendix XXXIII

Custom ANCOVA T

General Linear Model

[DataSet1] /Users/Koki/Desktop/OneDrive - University of Strathclyde/PhD/Data PhD/Endocrine Data/nor

Within-Subjects Factors

Measure: T_difference

time	Dependent Variable
1	Testo_difference_immediately_percentage
2	T_difference_30min_percentage
3	T_difference_1hr_percentage
4	T_difference_2hrs_percentage

Between-Subjects Factors

		Value Label	N
Outcome	0	loss	15
	1	win	16

Descriptive Statistics

	Outcome	Mean	Std. Deviation	N
Testo_difference_immediately_percentage	loss	-3.9726	15.40835	15
	win	2.1702	16.74552	16
	Total	-.8021	16.14741	31
T_difference_30min_percentage	loss	.7513	26.03568	15
	win	3.2770	25.02835	16
	Total	2.0549	25.12347	31
T_difference_1hr_percentage	loss	-3.8638	28.88881	15
	win	1.3078	21.24656	16
	Total	-1.1946	24.94139	31
T_difference_2hrs_percentage	loss	2.4764	24.60194	15
	win	-.5075	18.70062	16
	Total	.9363	21.43844	31

Box's Test of Equality of Covariance Matrices[a]

Box's M	5.062
F	.430
df1	10
df2	3977.868
Sig.	.933

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.064	.565[b]	3.000	25.000	.643	.064	1.696	.150
	Wilks' Lambda	.936	.565[b]	3.000	25.000	.643	.064	1.696	.150
	Hotelling's Trace	.068	.565[b]	3.000	25.000	.643	.064	1.696	.150
	Roy's Largest Root	.068	.565[b]	3.000	25.000	.643	.064	1.696	.150
time * Outcome	Pillai's Trace	.186	1.910[b]	3.000	25.000	.154	.186	5.731	.433
	Wilks' Lambda	.814	1.910[b]	3.000	25.000	.154	.186	5.731	.433
	Hotelling's Trace	.229	1.910[b]	3.000	25.000	.154	.186	5.731	.433
	Roy's Largest Root	.229	1.910[b]	3.000	25.000	.154	.186	5.731	.433
time * threat	Pillai's Trace	.054	.473[b]	3.000	25.000	.704	.054	1.420	.132
	Wilks' Lambda	.946	.473[b]	3.000	25.000	.704	.054	1.420	.132
	Hotelling's Trace	.057	.473[b]	3.000	25.000	.704	.054	1.420	.132
	Roy's Largest Root	.057	.473[b]	3.000	25.000	.704	.054	1.420	.132
time * Outcome * threat	Pillai's Trace	.299	3.547[b]	3.000	25.000	.029	.299	10.642	.717
	Wilks' Lambda	.701	3.547[b]	3.000	25.000	.029	.299	10.642	.717

	Wilks' Lambda	.701	3.547	3.000	25.000	.029	.299	10.642	.717
	Hotelling's Trace	.426	3.547[b]	3.000	25.000	.029	.299	10.642	.717
	Roy's Largest Root	.426	3.547[b]	3.000	25.000	.029	.299	10.642	.717

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

b. Exact statistic

c. Computed using alpha =

Mauchly's Test of Sphericity[a]

Measure: T_difference

					Epsilon[b]		
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.952	1.273	5	.938	.967	1.000	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: T_difference

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Sphericity Assumed	471.952	3	157.317	.685	.564	.025	2.056	.189
	Greenhouse-Geisser	471.952	2.900	162.746	.685	.559	.025	1.987	.186
	Huynh-Feldt	471.952	3.000	157.317	.685	.564	.025	2.056	.189
	Lower-bound	471.952	1.000	471.952	.685	.415	.025	.685	.126
time * Outcome	Sphericity Assumed	1240.810	3	413.603	1.802	.153	.063	5.405	.453
	Greenhouse-Geisser	1240.810	2.900	427.876	1.802	.156	.063	5.224	.444
	Huynh-Feldt	1240.810	3.000	413.603	1.802	.153	.063	5.405	.453
	Lower-bound	1240.810	1.000	1240.810	1.802	.191	.063	1.802	.254
time * threat	Sphericity Assumed	390.199	3	130.066	.567	.639	.021	1.700	.162
	Greenhouse-Geisser	390.199	2.900	134.555	.567	.633	.021	1.643	.160
	Huynh-Feldt	390.199	3.000	130.066	.567	.639	.021	1.700	.162
	Lower-bound	390.199	1.000	390.199	.567	.458	.021	.567	.112
time * Outcome * threat	Sphericity Assumed	2389.861	3	796.620	3.470	.020	.114	10.410	.757
	Greenhouse-Geisser	2389.861	2.900	824.111	3.470	.021	.114	10.063	.746
	Huynh-Feldt	2389.861	3.000	796.620	3.470	.020	.114	10.410	.757
	Lower-bound	2389.861	1.000	2389.861	3.470	.073	.114	3.470	.435
Error(time)	Sphericity Assumed	18595.848	81	229.578					
	Greenhouse-Geisser	18595.848	78.298	237.501					

	Greenhouse-Geisser	18595.848	78.298	237.501					
	Huynh-Feldt	18595.848	81.000	229.578					
	Lower-bound	18595.848	27.000	688.735					

a. Computed using alpha =

Tests of Within-Subjects Contrasts

Measure: T_difference

Source	time	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear	139.361	1	139.361	.655	.425	.024	.655	.122
	Quadratic	21.476	1	21.476	.091	.765	.003	.091	.060
	Cubic	311.116	1	311.116	1.294	.265	.046	1.294	.195
time * Outcome	Linear	1110.337	1	1110.337	5.219	.030	.162	5.219	.596
	Quadratic	61.101	1	61.101	.260	.615	.010	.260	.078
	Cubic	69.371	1	69.371	.288	.596	.011	.288	.081
time * threat	Linear	122.713	1	122.713	.577	.454	.021	.577	.113
	Quadratic	267.216	1	267.216	1.135	.296	.040	1.135	.177
	Cubic	.271	1	.271	.001	.973	.000	.001	.050
time * Outcome * threat	Linear	1334.098	1	1334.098	6.270	.019	.188	6.270	.675
	Quadratic	159.751	1	159.751	.678	.417	.025	.678	.125
	Cubic	896.012	1	896.012	3.725	.064	.121	3.725	.461
Error(time)	Linear	5744.549	27	212.761					
	Quadratic	6357.357	27	235.458					
	Cubic	6493.941	27	240.516					

a. Computed using alpha =

Levene's Test of Equality of Error Variances[a]

	F	df1	df2	Sig.
Testo_difference_immediately_percentage	.000	1	29	.983
T_difference_30min_percentage	.163	1	29	.689
T_difference_1hr_percentage	2.176	1	29	.151
T_difference_2hrs_percentage	.899	1	29	.351

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

Tests of Between-Subjects Effects

Tests of Between-Subjects Effects

Measure: T_difference

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	574.759	1	574.759	.439	.513	.016	.439	.098
Outcome	838.940	1	838.940	.641	.430	.023	.641	.121
threat	1130.166	1	1130.166	.863	.361	.031	.863	.146
Outcome * threat	380.851	1	380.851	.291	.594	.011	.291	.082
Error	35341.686	27	1308.951					

a. Computed using alpha =

Transformation Coefficients (M Matrix)

Average

Measure: T_difference

Transformed Variable: AVERAGE

Testo_difference_immediately_percentage	.500
T_difference_30min_percentage	.500
T_difference_1hr_percentage	.500
T_difference_2hrs_percentage	.500

time[a]

Measure: T_difference

	time		
Dependent Variable	Linear	Quadratic	Cubic
Testo_difference_immediately_percentage	-.671	.500	-.224
T_difference_30min_percentage	-.224	-.500	.671
T_difference_1hr_percentage	.224	-.500	-.671
T_difference_2hrs_percentage	.671	.500	.224

a. The contrasts for the within subjects factors are:
time: Polynomial contrast

Estimated Marginal Means

Outcome

Transformation Coefficients (M Matrix)

Dependent Variable	Measure T_difference
Testo_difference_immediately_percentage	.250
T_difference_30min_percentage	.250
T_difference_1hr_percentage	.250
T_difference_2hrs_percentage	.250

Estimates

Measure: T_difference

Outcome	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
loss	-1.543[a]	4.773	-11.336	8.249
win	2.944[a]	4.811	-6.928	12.815

a. Covariates appearing in the model are evaluated at the following values: threat = 1.03.

Pairwise Comparisons

Measure: T_difference

(I) Outcome	(J) Outcome	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
loss	win	-4.487	6.777	.514	-18.392	9.418
win	loss	4.487	6.777	.514	-9.418	18.392

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Univariate Tests

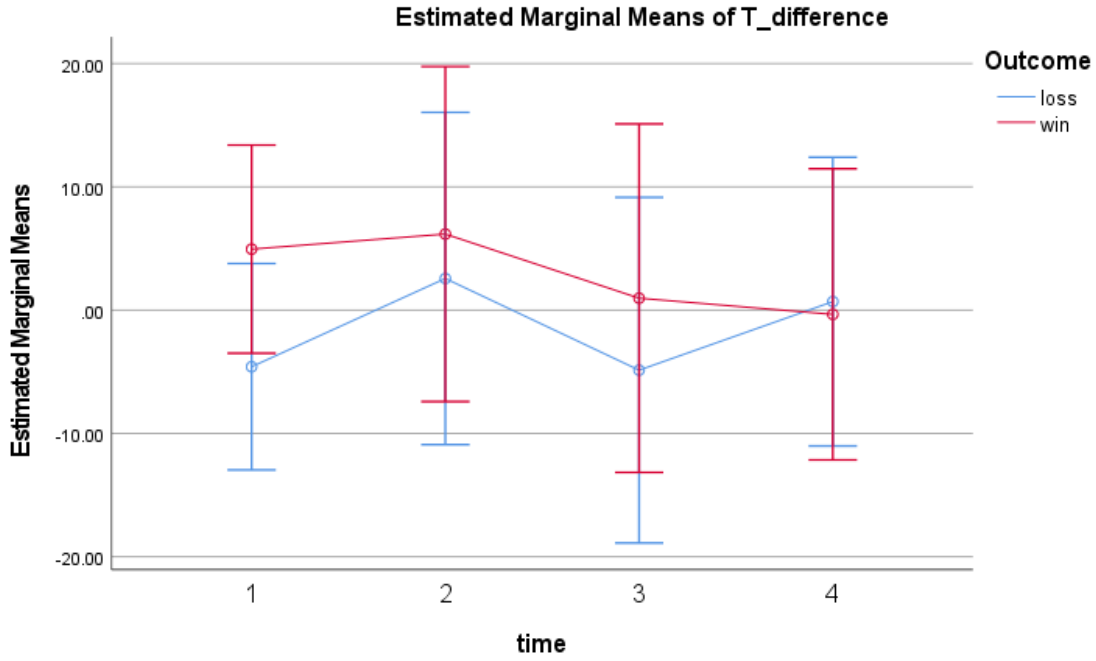
Measure: T_difference

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	143.450	1	143.450	.438	.514	.016	.438	.098
Error	8835.421	27	327.238					

The F tests the effect of Outcome. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha =

Profile Plots



Covariates appearing in the model are evaluated at the following values: threat = 1.03

Error bars: 95% CI

Appendix XXXIV

Follow-up ANOVAs for unpacking three-way interaction (T)

Post hoc tests are not performed for Outcome because there are fewer than three groups.

Within-Subjects Factors

Measure: T_difference

time	Dependent Variable
1	Testo_difference_immediately_percentage
2	T_difference_30min_percentage
3	T_difference_1hr_percentage
4	T_difference_2hrs_percentage

Between-Subjects Factors

		Value Label	N
Outcome	0	loss	7
	1	win	7

Descriptive Statistics

	Outcome	Mean	Std. Deviation	N
Testo_difference_immediately_percentage	loss	.1020	12.03274	7
	win	7.6527	14.65425	7
	Total	3.8773	13.46434	14
T_difference_30min_percentage	loss	-6.0664	34.96796	7
	win	8.5791	31.51274	7
	Total	1.2564	32.86989	14
T_difference_1hr_percentage	loss	-7.5403	33.55430	7
	win	-.3062	24.70232	7
	Total	-3.9232	28.55458	14
T_difference_2hrs_percentage	loss	10.1787	33.28719	7
	win	1.5008	25.52310	7
	Total	5.8398	28.85023	14

Box's Test of Equality of Covariance Matrices[a]

Box's M	14.922
F	.936
df1	10
df2	688.446
Sig.	.499

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Outcome Within Subjects Design: time

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.271	1.240[b]	3.000	10.000	.346	.271	3.720	.239
	Wilks' Lambda	.729	1.240[b]	3.000	10.000	.346	.271	3.720	.239
	Hotelling's Trace	.372	1.240[b]	3.000	10.000	.346	.271	3.720	.239
	Roy's Largest Root	.372	1.240[b]	3.000	10.000	.346	.271	3.720	.239
time * Outcome	Pillai's Trace	.275	1.267[b]	3.000	10.000	.338	.275	3.802	.243
	Wilks' Lambda	.725	1.267[b]	3.000	10.000	.338	.275	3.802	.243
	Hotelling's Trace	.380	1.267[b]	3.000	10.000	.338	.275	3.802	.243
	Roy's Largest Root	.380	1.267[b]	3.000	10.000	.338	.275	3.802	.243

a. Design: Intercept + Outcome
Within Subjects Design: time

b. Exact statistic

c. Computed using alpha = .05

Mauchly's Test of Sphericity[a]

Measure: T_difference

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Epsilon[b] Huynh-Feldt	Epsilon[b] Lower-bound
time	.738	3.263	5	.661	.838	1.000	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Outcome
Within Subjects Design: time

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: T_difference

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Sphericity Assumed	751.527	3	250.509	.807	.498	.063	2.422	.207
	Greenhouse-Geisser	751.527	2.514	298.980	.807	.481	.063	2.029	.190
	Huynh-Feldt	751.527	3.000	250.509	.807	.498	.063	2.422	.207
	Lower-bound	751.527	1.000	751.527	.807	.387	.063	.807	.132
time * Outcome	Sphericity Assumed	1020.168	3	340.056	1.096	.363	.084	3.287	.271
	Greenhouse-Geisser	1020.168	2.514	405.854	1.096	.358	.084	2.754	.246
	Huynh-Feldt	1020.168	3.000	340.056	1.096	.363	.084	3.287	.271
	Lower-bound	1020.168	1.000	1020.168	1.096	.316	.084	1.096	.162
Error(time)	Sphericity Assumed	11172.140	36	310.337					
	Greenhouse-Geisser	11172.140	30.164	370.385					
	Huynh-Feldt	11172.140	36.000	310.337					
	Lower-bound	11172.140	12.000	931.012					

a. Computed using alpha = .05

Tests of Within-Subjects Contrasts

Measure: T_difference

Source	time	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear	.351	1	.351	.001	.975	.000	.001	.050
	Quadratic	536.770	1	536.770	2.679	.128	.182	2.679	.325
	Cubic	214.406	1	214.406	.546	.474	.044	.546	.105
time * Outcome	Linear	550.711	1	550.711	1.630	.226	.120	1.630	.217
	Quadratic	463.146	1	463.146	2.311	.154	.162	2.311	.288
	Cubic	6.312	1	6.312	.016	.901	.001	.016	.052
Error(time)	Linear	4054.324	12	337.860					
	Quadratic	2404.462	12	200.372					

	Quadratic	2404.462	12	200.372					
	Cubic	4713.354	12	392.780					

a. Computed using alpha = .05

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
Testo_difference_immediately_percentage	Based on Mean	.346	1	12	.568
	Based on Median	.293	1	12	.598
	Based on Median and with adjusted df	.293	1	11.875	.598
	Based on trimmed mean	.311	1	12	.587
T_difference_30min_percentage	Based on Mean	.121	1	12	.734
	Based on Median	.080	1	12	.782
	Based on Median and with adjusted df	.080	1	11.940	.782
	Based on trimmed mean	.109	1	12	.747
T_difference_1hr_percentage	Based on Mean	.583	1	12	.460
	Based on Median	.289	1	12	.601
	Based on Median and with adjusted df	.289	1	10.399	.602
	Based on trimmed mean	.478	1	12	.502
T_difference_2hrs_percentage	Based on Mean	.918	1	12	.357
	Based on Median	.156	1	12	.700
	Based on Median and with adjusted df	.156	1	8.899	.702
	Based on trimmed mean	.844	1	12	.376

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Outcome
Within Subjects Design: time

Tests of Between-Subjects Effects

Measure: T_difference

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	173.970	1	173.970	.083	.779	.007	.083	.058
Outcome	376.829	1	376.829	.179	.680	.015	.179	.068
Error	25253.294	12	2104.441					

a. Computed using alpha = .05

Estimated Marginal Means

1. Outcome

Estimates

Measure: T_difference

Outcome	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
loss	-.831	8.669	-19.721	18.058
win	4.357	8.669	-14.532	23.246

Pairwise Comparisons

Measure: T_difference

(I) Outcome	(J) Outcome	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
loss	win	-5.188	12.260	.680	-31.901	21.525
win	loss	5.188	12.260	.680	-21.525	31.901

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Univariate Tests

Measure: T_difference

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	94.207	1	94.207	.179	.680	.015	.179	.068
Error	6313.324	12	526.110					

The F tests the effect of Outcome. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha = .05

2. time

Estimates

Measure: T_difference

time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	3.877	3.583	-3.930	11.685
2	1.256	8.896	-18.126	20.639
3	-3.923	7.874	-21.080	13.233
4	5.840	7.927	-11.432	23.111

Pairwise Comparisons

Measure: T_difference

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	2.621	6.757	.999	-18.606	23.848
	3	7.801	6.383	.815	-12.250	27.851
	4	-1.962	6.302	1.000	-21.760	17.835
2	1	-2.621	6.757	.999	-23.848	18.606
	3	5.180	8.041	.989	-20.079	30.438
	4	-4.583	7.082	.989	-26.830	17.663
3	1	-7.801	6.383	.815	-27.851	12.250
	2	-5.180	8.041	.989	-30.438	20.079
	4	-9.763	5.007	.373	-25.490	5.964
4	1	1.962	6.302	1.000	-17.835	21.760
	2	4.583	7.082	.989	-17.663	26.830
	3	9.763	5.007	.373	-5.964	25.490

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.271	1.240[a]	3.000	10.000	.346	.271	3.720	.239
Wilks' lambda	.729	1.240[a]	3.000	10.000	.346	.271	3.720	.239
Hotelling's trace	.372	1.240[a]	3.000	10.000	.346	.271	3.720	.239
Roy's largest root	.372	1.240[a]	3.000	10.000	.346	.271	3.720	.239

Each F tests the multivariate effect of time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha = .05

3. Outcome * time

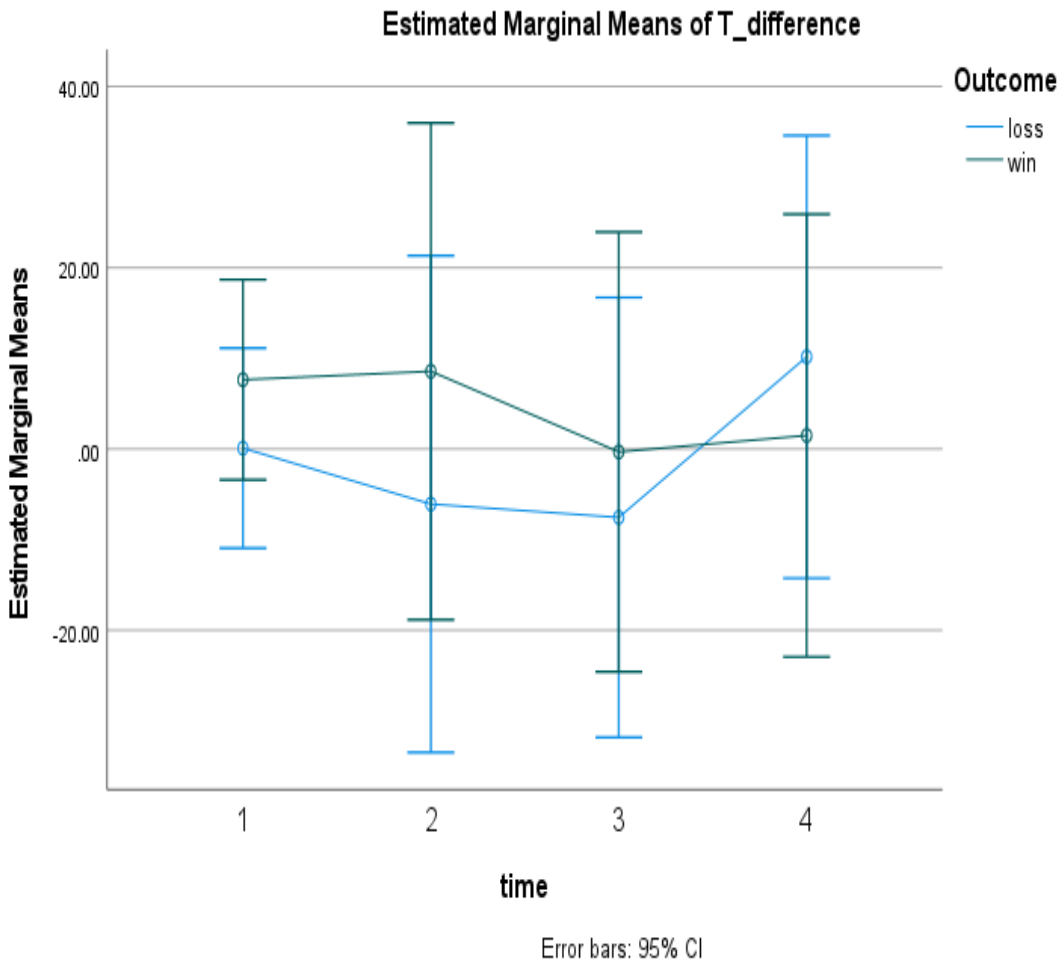
Measure: T_difference

Outcome	time	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
loss	1	.102	5.068	-10.939	11.143
	2	-6.066	12.581	-33.477	21.344
	3	-7.540	11.136	-31.803	16.723
	4	10.179	11.211	-14.247	34.604

win	1	7.653	5.068	-3.389	18.694
	2	8.579	12.581	-18.832	35.990
	3	-.306	11.136	-24.569	23.957
	4	1.501	11.211	-22.925	25.926

Model: Intercept + Outcome

Profile Plots



General Linear Model

Warnings

Post hoc tests are not performed for Outcome because there are fewer than three groups.

Within-Subjects Factors

Measure: T_difference

time	Dependent Variable
1	Testo_difference_immediately_percentage
2	T_difference_30min_percentage
3	T_difference_1hr_percentage
4	T_difference_2hrs_percentage

Between-Subjects Factors

		Value Label	N
Outcome	0	loss	8
	1	win	9

Descriptive Statistics

	Outcome	Mean	Std. Deviation	N
Testo_difference_immediately_percentage	loss	-7.5379	17.87743	8
	win	-2.0939	17.83134	9
	Total	-4.6558	17.51141	17
T_difference_30min_percentage	loss	6.7167	14.84873	8
	win	-.8469	19.64724	9
	Total	2.7124	17.45315	17
T_difference_1hr_percentage	loss	-.6469	26.05253	8
	win	2.5631	19.61376	9
	Total	1.0525	22.18161	17

	Total	1.0525	22.18161	17
T_difference_2hrs_percentage	loss	-4.2632	12.22822	8
	win	-2.0696	12.68331	9
	Total	-3.1018	12.12955	17

Box's Test of Equality of Covariance Matrices[a]

Box's M	24.327
F	1.708
df1	10
df2	1033.845
Sig.	.074

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Outcome Within Subjects Design: time

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.227	1.275[b]	3.000	13.000	.324	.227	3.824	.263
	Wilks' Lambda	.773	1.275[b]	3.000	13.000	.324	.227	3.824	.263
	Hotelling's Trace	.294	1.275[b]	3.000	13.000	.324	.227	3.824	.263
	Roy's Largest Root	.294	1.275[b]	3.000	13.000	.324	.227	3.824	.263
time * Outcome	Pillai's Trace	.189	1.011[b]	3.000	13.000	.419	.189	3.033	.215
	Wilks' Lambda	.811	1.011[b]	3.000	13.000	.419	.189	3.033	.215
	Hotelling's Trace	.233	1.011[b]	3.000	13.000	.419	.189	3.033	.215
	Roy's Largest Root	.233	1.011[b]	3.000	13.000	.419	.189	3.033	.215

a. Design: Intercept + Outcome Within Subjects Design: time

b. Exact statistic

c. Computed using alpha = .05

Mauchly's Test of Sphericity[a]

Measure: T_difference

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.825	2.639	5	.756	.888	1.000	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Outcome
Within Subjects Design: time

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: T_difference

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Sphericity Assumed	653.417	3	217.806	1.121	.351	.070	3.362	.282
	Greenhouse-Geisser	653.417	2.665	245.178	1.121	.348	.070	2.987	.264
	Huynh-Feldt	653.417	3.000	217.806	1.121	.351	.070	3.362	.282
	Lower-bound	653.417	1.000	653.417	1.121	.307	.070	1.121	.168
time * Outcome	Sphericity Assumed	420.416	3	140.139	.721	.545	.046	2.163	.191
	Greenhouse-Geisser	420.416	2.665	157.750	.721	.530	.046	1.922	.181
	Huynh-Feldt	420.416	3.000	140.139	.721	.545	.046	2.163	.191
	Lower-bound	420.416	1.000	420.416	.721	.409	.046	.721	.125
Error(time)	Sphericity Assumed	8745.185	45	194.337					
	Greenhouse-Geisser	8745.185	39.976	218.760					
	Huynh-Feldt	8745.185	45.000	194.337					
	Lower-bound	8745.185	15.000	583.012					

a. Computed using alpha = .05

Tests of Within-Subjects Contrasts

Measure: T_difference

Source	time	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear	7.481	1	7.481	.040	.844	.003	.040	.054
	Quadratic	597.269	1	597.269	2.831	.113	.159	2.831	.350
	Cubic	48.668	1	48.668	.262	.616	.017	.262	.077
time * Outcome	Linear	.221	1	.221	.001	.973	.000	.001	.050
	Quadratic	152.245	1	152.245	.722	.409	.046	.722	.125
	Cubic	267.950	1	267.950	1.444	.248	.088	1.444	.203

Error(time)	Linear	2796.536	15	186.436					
	Quadratic	3165.018	15	211.001					
	Cubic	2783.631	15	185.575					

a. Computed using alpha = .05

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
Testo_difference_immediately_percentage	Based on Mean	.001	1	15	.980
	Based on Median	.003	1	15	.954
	Based on Median and with adjusted df	.003	1	14.933	.954
	Based on trimmed mean	.001	1	15	.974
T_difference_30min_percentage	Based on Mean	.298	1	15	.593
	Based on Median	.057	1	15	.815
	Based on Median and with adjusted df	.057	1	12.069	.816
	Based on trimmed mean	.227	1	15	.641
T_difference_1hr_percentage	Based on Mean	.139	1	15	.715
	Based on Median	.190	1	15	.669
	Based on Median and with adjusted df	.190	1	13.879	.669
	Based on trimmed mean	.121	1	15	.732
T_difference_2hrs_percentage	Based on Mean	.009	1	15	.925
	Based on Median	.001	1	15	.976
	Based on Median and with adjusted df	.001	1	13.992	.976
	Based on trimmed mean	.011	1	15	.917

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Outcome
Within Subjects Design: time

Tests of Between-Subjects Effects

Measure: T_difference

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	70.823	1	70.823	.098	.758	.006	.098	.060
Outcome	11.419	1	11.419	.016	.902	.001	.016	.052
Error	10829.566	15	721.971					

a. Computed using alpha = .05

Estimated Marginal Means

1. Outcome

Estimates

Measure: T_difference

Outcome	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
loss	-1.433	4.750	-11.557	8.691
win	-.612	4.478	-10.157	8.933

Pairwise Comparisons

Measure: T_difference

(I) Outcome	(J) Outcome	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
loss	win	-.821	6.528	.902	-14.735	13.093
win	loss	.821	6.528	.902	-13.093	14.735

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Univariate Tests

Measure: T_difference

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	2.855	1	2.855	.016	.902	.001	.016	.052
Error	2707.391	15	180.493					

The F tests the effect of Outcome. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha = .05

2. time

Estimates

Measure: T_difference

time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	-4.816	4.337	-14.061	4.429
2	2.935	4.269	-6.165	12.034
3	.958	5.550	-10.872	12.789

4	-3.166	3.030	-9.625	3.293

Pairwise Comparisons

Measure: T_difference

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-7.751	4.085	.383	-20.112	4.610
	3	-5.774	5.597	.900	-22.708	11.160
	4	-1.650	4.622	1.000	-15.634	12.335
2	1	7.751	4.085	.383	-4.610	20.112
	3	1.977	4.750	.999	-12.395	16.348
	4	6.101	4.053	.631	-6.164	18.367
3	1	5.774	5.597	.900	-11.160	22.708
	2	-1.977	4.750	.999	-16.348	12.395
	4	4.124	5.413	.975	-12.253	20.502
4	1	1.650	4.622	1.000	-12.335	15.634
	2	-6.101	4.053	.631	-18.367	6.164
	3	-4.124	5.413	.975	-20.502	12.253

Based on estimated marginal means

a. Adjustment for multiple comparisons: Sidak.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.227	1.275[a]	3.000	13.000	.324	.227	3.824	.263
Wilks' lambda	.773	1.275[a]	3.000	13.000	.324	.227	3.824	.263
Hotelling's trace	.294	1.275[a]	3.000	13.000	.324	.227	3.824	.263
Roy's largest root	.294	1.275[a]	3.000	13.000	.324	.227	3.824	.263

Each F tests the multivariate effect of time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha = .05

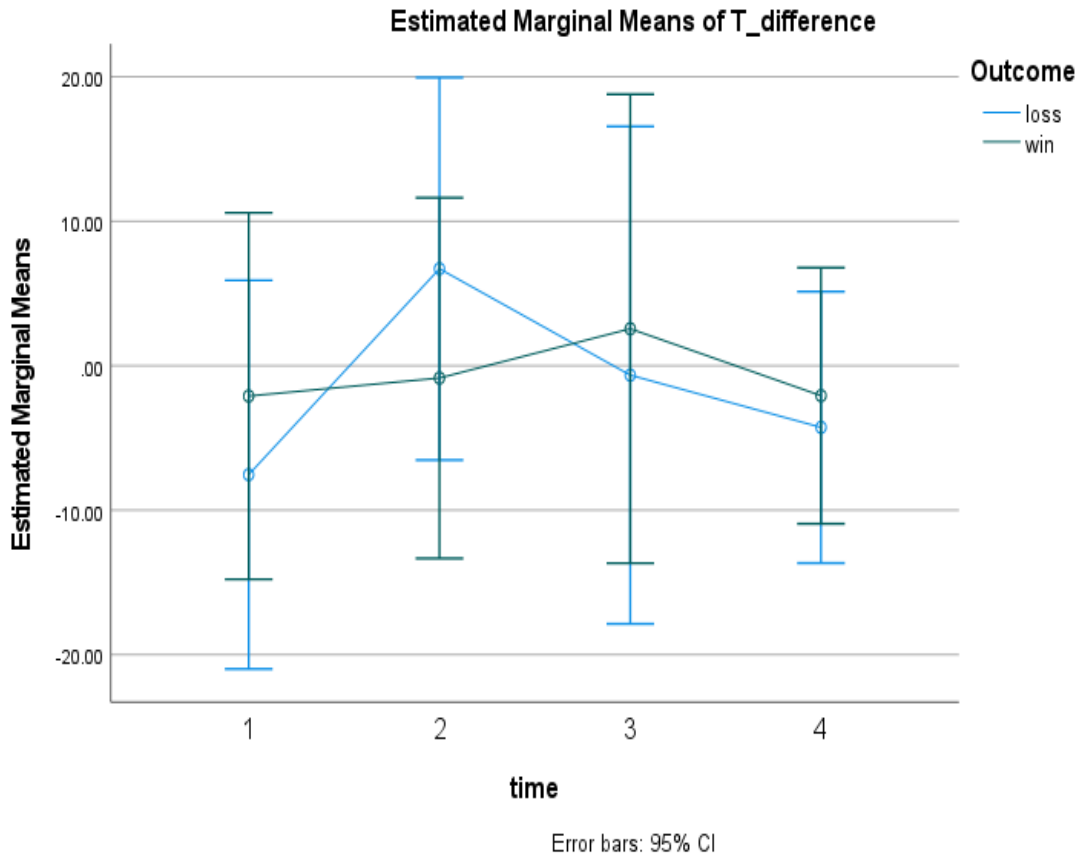
3. Outcome * time

Measure: T_difference

Outcome	time	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
loss	1	-7.538	6.312	-20.991	5.916
	2	6.717	6.213	-6.525	19.958

	2	6.717	6.213	-6.525	19.958
	3	-.647	8.077	-17.863	16.569
	4	-4.263	4.410	-13.663	5.136
win	1	-2.094	5.951	-14.778	10.590
	2	-.847	5.857	-13.331	11.638
	3	2.563	7.615	-13.668	18.794
	4	-2.070	4.158	-10.931	6.792

Profile Plots



Appendix XXXV

Rgeression Model for Trait Affect, Competition Outcome and T reactivity

Regression

Descriptive Statistics

	Mean	Std. Deviation	N
Testo_difference_immediately_percentage	-.2590	16.13290	30
GNA_PANAS_Difference	-.0333	4.48356	30
GPA_PANAS_Difference	2.0000	6.64883	30
Outcome	.53	.507	30

Correlations

		Testo_difference_immediately_percentage	GNA_PANAS_Difference	GPA_PANAS_Difference	Outcome
Pearson Correlation	Testo_difference_immediately_percentage	1.000	.316	.084	.164
	GNA_PANAS_Difference	.316	1.000	-.039	.008
	GPA_PANAS_Difference	.084	-.039	1.000	-.388
	Outcome	.164	.008	-.388	1.000
Sig. (1-tailed)	Testo_difference_immediately_percentage	.	.045	.330	.194
	GNA_PANAS_Difference	.045	.	.418	.483
	GPA_PANAS_Difference	.330	.418	.	.017
	Outcome	.194	.483	.017	.
N	Testo_difference_immediately_percentage	30	30	30	30
	GNA_PANAS_Difference	30	30	30	30
	GPA_PANAS_Difference	30	30	30	30
	Outcome	30	30	30	30

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	Outcome, GNA_PANAS_Difference, GPA_PANAS_Difference[b]	.	Enter

a. Dependent Variable: Testo_difference_immediately_percentage

b. All requested variables entered.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics					Durbin-Watson
					R Square Change	F Change	df1	df2	Sig. F Change	
1	.394[a]	.155	.058	15.65941	.155	1.593	3	26	.215	2.266

a. Predictors: (Constant), Outcome, GNA_PANAS_Difference, GPA_PANAS_Difference

b. Dependent Variable: Testo_difference_immediately_percentage

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	1172.197	3	390.732	1.593	.215[b]
	Residual	6375.649	26	245.217		
	Total	7547.846	29			

a. Dependent Variable: Testo_difference_immediately_percentage

b. Predictors: (Constant), Outcome, GNA_PANAS_Difference, GPA_PANAS_Difference

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B		Correlations		
		B	Std. Error	Beta			Lower Bound	Upper Bound	Zero-order	Partial	Part
1	(Constant)	-5.094	4.746		-1.073	.293	-14.850	4.661			
	GNA_PANAS_Difference	1.155	.649	.321	1.780	.087	-.179	2.490	.316	.330	.321
	GPA_PANAS_Difference	.454	.475	.187	.956	.348	-.522	1.431	.084	.184	.172
	Outcome	7.435	6.219	.234	1.195	.243	-5.349	20.219	.164	.228	.215

a. Dependent Variable: Testo_difference_immediately_percentage

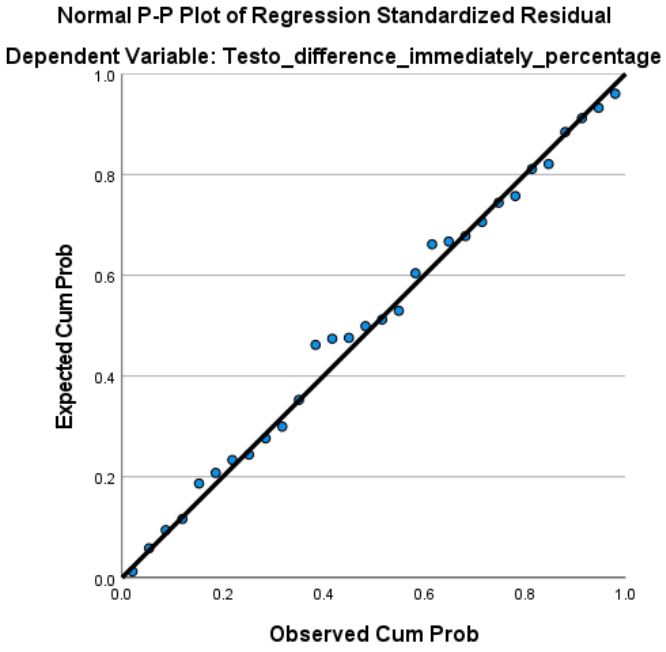
Residuals Statistics[a]

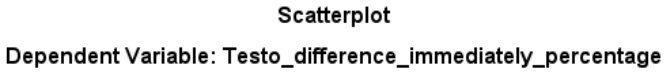
	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	-13.9614	19.1768	-.2590	6.35772	30
Std. Predicted Value	-2.155	3.057	.000	1.000	30
Standard Error of Predicted Value	4.074	9.603	5.467	1.704	30
Adjusted Predicted Value	-21.5344	26.8589	-.0543	8.03338	30
Residual	-35.27608	27.48752	.00000	14.82734	30
Std. Residual	-2.253	1.755	.000	.947	30
Stud. Residual	-2.541	1.847	-.006	1.028	30
Deleted Residual	-44.90030	30.43582	-.20468	17.58613	30
Stud. Deleted Residual	-2.875	1.943	-.015	1.073	30

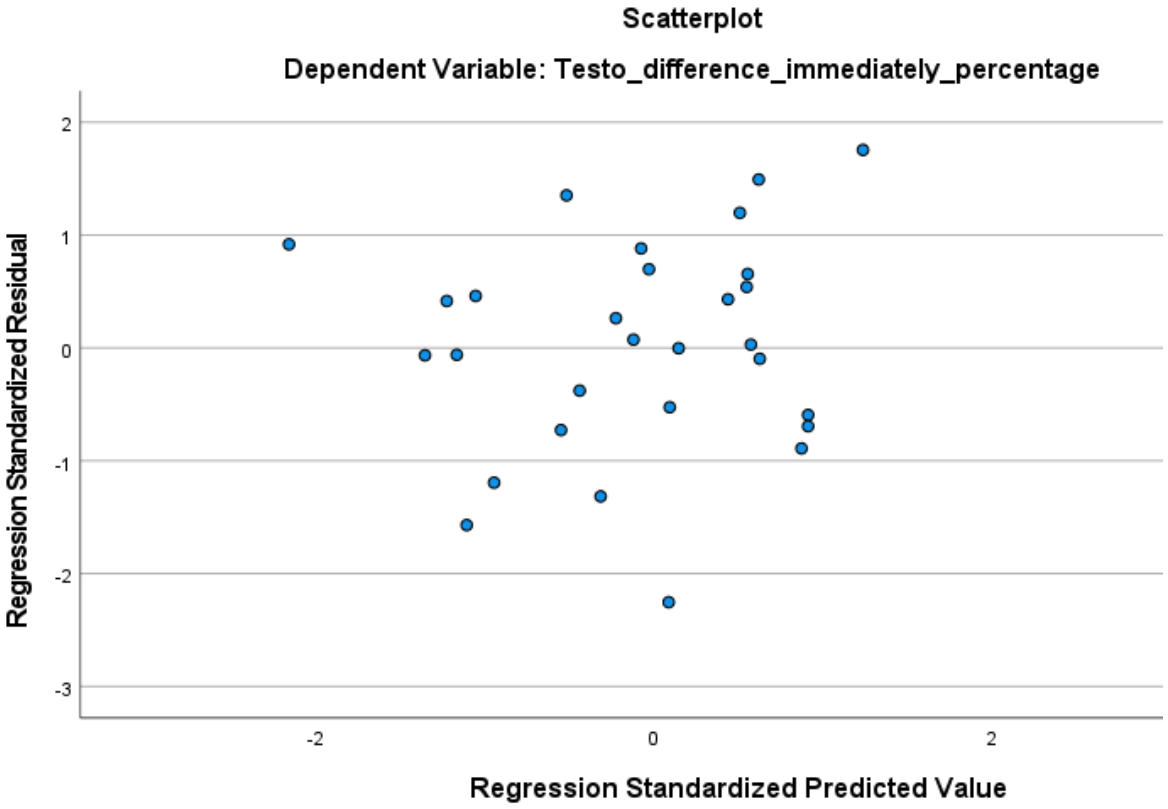
Stud. Deleted Residual	-2.875	1.943	-.015	1.073	30
Mahal. Distance	.996	9.940	2.900	2.652	30
Cook's Distance	.000	.441	.049	.091	30
Centered Leverage Value	.034	.343	.100	.091	30

a. Dependent Variable: Testo_difference_immediately_percentage

Charts






Scatterplot
Dependent Variable: Testo_difference_immediately_percentage
Regression Standardized Residual
Regression Standardized Predicted Value
2
1
0
-1
-2
-3
-2
0
2

Appendix XXXVI

Regression Trait Affect and T reactivity

Regression

Descriptive Statistics

	Mean	Std. Deviation	N
Testo_difference_immediately_percentage	-.2590	16.13290	30
GNA_PANAS_Difference	-.0333	4.48356	30
GPA_PANAS_Difference	2.0000	6.64883	30

Correlations

		Testo_difference_immediately_percentage	GNA_PANAS_Difference	GPA_PANAS_Difference
Pearson Correlation	Testo_difference_immediately_percentage	1.000	.316	.084
	GNA_PANAS_Difference	.316	1.000	-.039
	GPA_PANAS_Difference	.084	-.039	1.000
Sig. (1-tailed)	Testo_difference_immediately_percentage	.	.045	.330
	GNA_PANAS_Difference	.045	.	.418
	GPA_PANAS_Difference	.330	.418	.
N	Testo_difference_immediately_percentage	30	30	30
	GNA_PANAS_Difference	30	30	30
	GPA_PANAS_Difference	30	30	30

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	GPA_PANAS_Difference, GNA_PANAS_Difference[b]	.	Enter

a. Dependent Variable: Testo_difference_immediately_percentage

b. All requested variables entered.

Model Summary[b]

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics					Durbin-Watson
					R Square Change	F Change	df1	df2	Sig. F Change	
1	.330[a]	.109	.043	15.78337	.109	1.649	2	27	.211	2.197

a. Predictors: (Constant), GPA_PANAS_Difference, GNA_PANAS_Difference

b. Dependent Variable: Testo_difference_immediately_percentage

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	821.752	2	410.876	1.649	.211[b]
	Residual	6726.095	27	249.115		
	Total	7547.846	29			

a. Dependent Variable: Testo_difference_immediately_percentage

b. Predictors: (Constant), GPA_PANAS_Difference, GNA_PANAS_Difference

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B		Correlations		
		B	Std. Error	Beta			Lower Bound	Upper Bound	Zero-order	Partial	Part
1	(Constant)	-.688	3.014		-.228	.821	-6.871	5.495			
	GNA_PANAS_Difference	1.149	.654	.319	1.757	.090	-.193	2.492	.316	.320	.319
	GPA_PANAS_Difference	.234	.441	.096	.530	.601	-.671	1.139	.084	.101	.096

a. Dependent Variable: Testo_difference_immediately_percentage

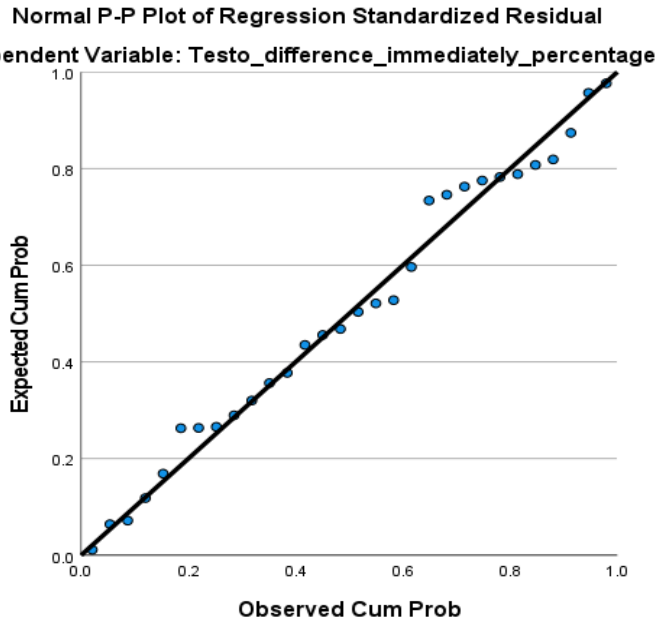
Residuals Statistics[a]

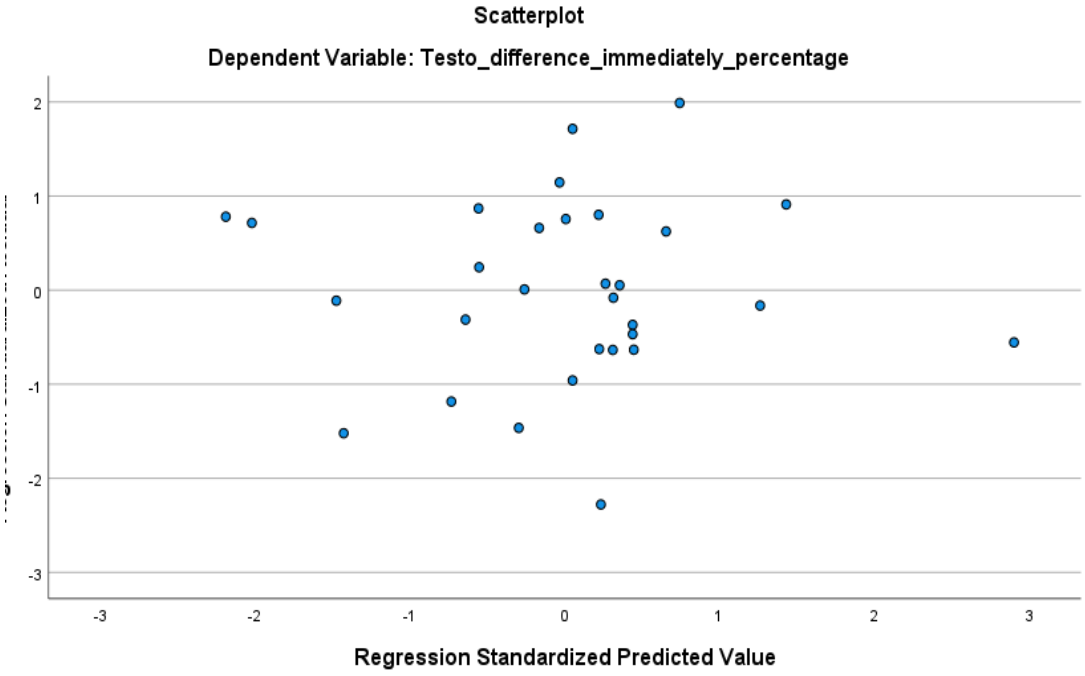
	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	-11.9084	15.1872	-.2590	5.32318	30
Std. Predicted Value	-2.188	2.902	.000	1.000	30
Standard Error of Predicted Value	2.882	9.144	4.586	2.004	30
Adjusted Predicted Value	-18.0615	19.5122	.0500	6.55579	30
Residual	-35.94517	31.41655	.00000	15.22939	30
Std. Residual	-2.277	1.990	.000	.965	30
Stud. Residual	-2.567	2.045	-.009	1.026	30
Deleted Residual	-45.67766	33.16847	-.30898	17.32564	30
Stud. Deleted Residual	-2.898	2.183	-.016	1.074	30
Mahal. Distance	.000	8.766	1.933	2.737	30
Cook's Distance	.000	.595	.049	.115	30

Centered Leverage Value	.000	.302	.067	.094	30

a. Dependent Variable: Testo_difference_immediately_percentage

Charts





Appendix XXXVII

Pearson's R Matrix Correlation between Psychological data and Post-Competition C Reactivity

Correlations

[DataSet1] /Users/Koki/Desktop/OneDrive - University of Strathclyde/PhD/Data PhD/Endocrine Data/normalised_and_percentage_data

Descriptive Statistics

	Mean	Std. Deviation	N
C_difference_immed_percentage	-.3576	27.16249	30
SOC_PerceivedUnderstanding	41.03	8.538	31
SOC_PAControl	47.29	8.745	31
sense_of_coherence	131.16	20.815	31
CPCN_ASQ	2.578	2.8096	31
hopefulness	5.0213	.80773	31

Correlations

		C_difference_immed_percentage	SOC_PerceivedUnderstanding	SOC_PAControl	sense_of_coherence	CPCN_ASQ	hopefulness
C_difference_immed_percentage	Pearson Correlation	1	.006	-.136	-.043	-.209	-.230
	Sig. (2-tailed)		.976	.472	.821	.268	.222
	N	30	30	30	30	30	30
SOC_PerceivedUnderstanding	Pearson Correlation	.006	1	.514**	.733**	-.103	-.097
	Sig. (2-tailed)	.976		.003	.000	.583	.605
	N	30	31	31	31	31	31
SOC_PAControl	Pearson Correlation	-.136	.514**	1	.895**	.314	.299
	Sig. (2-tailed)	.472	.003		.000	.085	.102
	N	30	31	31	31	31	31
sense_of_coherence	Pearson Correlation	-.043	.733**	.895**	1	.214	.160
	Sig. (2-tailed)	.821	.000	.000		.248	.390
	N	30	31	31	31	31	31
CPCN_ASQ	Pearson Correlation	-.209	-.103	.314	.214	1	.546**
	Sig. (2-tailed)	.268	.583	.085	.248		.001
	N	30	31	31	31	31	31
hopefulness	Pearson Correlation	-.230	-.097	.299	.160	.546**	1
	Sig. (2-tailed)	.222	.605	.102	.390	.001	
	N	30	31	31	31	31	31

**. Correlation is significant at the 0.01 level (2-tailed).

```
CORRELATIONS
  /VARIABLES=C_difference_immed_percentage threat C_NEO_FFI_3 N_NEO_FFI_3 Help_Funding_Motv
    Wellbeing_Course_Motv Status
  /PRINT=TWOTAIL NOSIG
  /STATISTICS DESCRIPTIVES
  /MISSING=PAIRWISE.
```

Correlations

Descriptive Statistics

	Mean	Std. Deviation	N
C_difference_immed_percentage	-.3576	27.16249	30
threat	1.03	1.722	31
C_NEO_FFI_3	2.07	1.202	30
N_NEO_FFI_3	2.30	1.236	30
Help_Funding_Motv	1.4000	1.35443	30
Wellbeing_Course_Motv	1.2333	1.27802	30
Status	.3548	.48637	31

Correlations

		C_difference_immed_percentage	threat	C_NEO_FFI_3	N_NEO_FFI_3	Help_Funding_Motv	Wellbeing_Course_Motv	Status
C_difference_immed_percentage	Pearson Correlation	1	-.231	-.204	.168	-.049	.118	.421*
	Sig. (2-tailed)		.219	.288	.385	.800	.544	.020
	N	30	30	29	29	29	29	30
threat	Pearson Correlation	-.231	1	.426*	.071	-.145	-.246	-.452*
	Sig. (2-tailed)	.219		.019	.711	.444	.191	.011
	N	30	31	30	30	30	30	31
C_NEO_FFI_3	Pearson Correlation	-.204	.426*	1	-.362*	-.239	-.259	-.394*
	Sig. (2-tailed)	.288	.019		.049	.212	.175	.031
	N	29	30	30	30	29	29	30
N_NEO_FFI_3	Pearson Correlation	.168	.071	-.362*	1	.563**	.377*	.438*
	Sig. (2-tailed)	.385	.711	.049		.001	.044	.015
	N	29	30	30	30	29	29	30
Help_Funding_Motv	Pearson Correlation	-.049	-.145	-.239	.563**	1	.681**	.584**
	Sig. (2-tailed)	.800	.444	.212	.001		.000	.001
	N	29	30	29	29	30	30	30

	N	29	30	29	29	30	30	30
Wellbeing_Course_Motv	Pearson Correlation	.118	-.246	-.259	.377*	.681**	1	.431*
	Sig. (2-tailed)	.544	.191	.175	.044	.000		.017
	N	29	30	29	29	30	30	30
Status	Pearson Correlation	.421*	-.452*	-.394*	.438*	.584**	.431*	1
	Sig. (2-tailed)	.020	.011	.031	.015	.001	.017	
	N	30	31	30	30	30	30	31

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Appendix XXXVIII

Spearman's Rank Matrix Correlation between Psychological data and Post-Competition C Reactivity

```
NONPAR CORR
  /VARIABLES=Help_Funding_Motv Wellbeing_Course_Motv C_NEO_FFI_3 N_NEO_FFI_3 threat Status
    C_difference_immed_percentage
  /PRINT=SPEARMAN TWOTAIL NOSIG
  /MISSING=PAIRWISE.
```

Nonparametric Correlations

Correlations

			Help_Funding_Motv	Wellbeing_Course_Motv	C_NEO_FFI_3	N_NEO_FFI_3	threat	Status	C_difference_immed_percentage
Spearman's rho	Help_Funding_Motv	Correlation Coefficient	1.000	.671**	-.203	.562**	-.031	.576**	.051
		Sig. (2-tailed)	.	.000	.291	.001	.870	.001	.793
		N	30	30	29	29	30	30	29
	Wellbeing_Course_Motv	Correlation Coefficient	.671**	1.000	-.203	.416*	-.168	.413*	.203
		Sig. (2-tailed)	.000	.	.292	.025	.375	.023	.290
		N	30	30	29	29	30	30	29
	C_NEO_FFI_3	Correlation Coefficient	-.203	-.203	1.000	-.348	.302	-.388*	-.184
		Sig. (2-tailed)	.291	.292	.	.059	.105	.034	.339
		N	29	29	30	30	30	30	29
	N_NEO_FFI_3	Correlation Coefficient	.562**	.416*	-.348	1.000	.124	.433*	.245
		Sig. (2-tailed)	.001	.025	.059	.	.512	.017	.200
		N	29	29	30	30	30	30	29
	threat	Correlation Coefficient	-.031	-.168	.302	.124	1.000	-.433*	-.237
		Sig. (2-tailed)	.870	.375	.105	.512	.	.015	.207
		N	30	30	30	30	31	31	30
	Status	Correlation Coefficient	.576**	.413*	-.388*	.433*	-.433*	1.000	.482**
		Sig. (2-tailed)	.001	.023	.034	.017	.015	.	.007
		N	30	30	30	30	31	31	30
	C_difference_immed_percentage	Correlation Coefficient	.051	.203	-.184	.245	-.237	.482**	1.000
		Sig. (2-tailed)	.793	.290	.339	.200	.207	.007	.
		N	29	29	29	29	30	30	30

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Appendix XXXIX

Multiple Regression C, SES and Threat/Challenge Cognitions

Regression

Descriptive Statistics

	Mean	Std. Deviation	N
C_difference_immed_percentage	-.3576	27.16249	30
Status	.3333	.47946	30
threat	1.07	1.741	30

Correlations

		C_difference_immed_percentage	Status	threat
Pearson Correlation	C_difference_immed_percentage	1.000	.421	-.231
	Status	.421	1.000	-.441
	threat	-.231	-.441	1.000
Sig. (1-tailed)	C_difference_immed_percentage	.	.010	.109
	Status	.010	.	.007
	threat	.109	.007	.
N	C_difference_immed_percentage	30	30	30
	Status	30	30	30
	threat	30	30	30

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	threat, Status[b]	.	Enter

a. Dependent Variable: C_difference_immed_percentage

b. All requested variables entered.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.424[a]	.180	.119	25.48930

a. Predictors: (Constant), threat, Status

b. Dependent Variable: C_difference_immed_percentage

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	3854.194	2	1927.097	2.966	.068[b]
	Residual	17542.023	27	649.705		
	Total	21396.217	29			

a. Dependent Variable: C_difference_immed_percentage

b. Predictors: (Constant), threat, Status

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Correlations			Collinearity Statistics	
		B	Std. Error	Beta			Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	-6.899	7.482		-.922	.365					
	Status	22.455	10.998	.396	2.042	.051	.421	.366	.356	.806	1.241
	threat	-.885	3.029	-.057	-.292	.772	-.231	-.056	-.051	.806	1.241

a. Dependent Variable: C_difference_immed_percentage

Collinearity Diagnostics[a]

Model	Dimension	Eigenvalue	Condition Index	Variance Proportions		
				(Constant)	Status	threat
1	1	1.783	1.000	.11	.08	.07
	2	1.000	1.335	.00	.25	.32
	3	.217	2.867	.89	.67	.61

a. Dependent Variable: C_difference_immed_percentage

Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	-9.5532	19.0952	-.3576	11.52837	30
Std. Predicted Value	-.798	1.687	.000	1.000	30
Standard Error of Predicted Value	5.827	14.553	7.701	2.419	30
Adjusted Predicted Value	-12.4482	21.6908	-.1149	11.78966	30
Residual	-58.83804	64.25305	.00000	24.59466	30
Std. Residual	-2.308	2.521	.000	.965	30
Stud. Residual	-2.404	2.589	-.004	1.000	30

Deleted Residual	-63.79377	67.79607	-.24270	26.45298	30
Stud. Deleted Residual	-2.660	2.931	-.003	1.059	30
Mahal. Distance	.549	8.487	1.933	2.021	30
Cook's Distance	.000	.162	.025	.036	30
Centered Leverage Value	.019	.293	.067	.070	30

a. Dependent Variable: C_difference_immed_percentage

Appendix XL

Custom ANCOVA C

General Linear Model

Within-Subjects Factors

Measure: MEASURE_1

time	Dependent Variable
1	C_difference_immed_percentage
2	C_difference_30min_percentage
3	C_difference_1hr_percentage
4	C_difference_2hrs_percentage

Between-Subjects Factors

		Value Label	N
Outcome	0	loss	15
	1	win	15

Descriptive Statistics

	Outcome	Mean	Std. Deviation	N
C_difference_immed_percentage	loss	-3.9471	22.24406	15
	win	3.2318	31.71584	15
	Total	-.3576	27.16249	30
C_difference_30min_percentage	loss	9.8052	30.97702	15
	win	24.1670	32.31509	15
	Total	16.9861	31.94863	30
C_difference_1hr_percentage	loss	13.6002	40.25099	15
	win	41.6524	29.64905	15
	Total	27.6263	37.55038	30
C_difference_2hrs_percentage	loss	29.3629	35.60325	15
	win	39.1261	41.24439	15
	Total	34.2445	38.18131	30

Box's Test of Equality of Covariance Matrices[a]

Box's M	13.764
F	1.161
df1	10
df2	3748.207
Sig.	.312

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
time	Pillai's Trace	.141	1.311[b]	3.000	24.000	.294	.141	3.933	.304
	Wilks' Lambda	.859	1.311[b]	3.000	24.000	.294	.141	3.933	.304
	Hotelling's Trace	.164	1.311[b]	3.000	24.000	.294	.141	3.933	.304
	Roy's Largest Root	.164	1.311[b]	3.000	24.000	.294	.141	3.933	.304
time * Outcome	Pillai's Trace	.392	5.163[b]	3.000	24.000	.007	.392	15.490	.874
	Wilks' Lambda	.608	5.163[b]	3.000	24.000	.007	.392	15.490	.874
	Hotelling's Trace	.645	5.163[b]	3.000	24.000	.007	.392	15.490	.874
	Roy's Largest Root	.645	5.163[b]	3.000	24.000	.007	.392	15.490	.874
time * threat	Pillai's Trace	.207	2.088[b]	3.000	24.000	.128	.207	6.265	.467
	Wilks' Lambda	.793	2.088[b]	3.000	24.000	.128	.207	6.265	.467
	Hotelling's Trace	.261	2.088[b]	3.000	24.000	.128	.207	6.265	.467
	Roy's Largest Root	.261	2.088[b]	3.000	24.000	.128	.207	6.265	.467
time * Outcome * threat	Pillai's Trace	.295	3.346[b]	3.000	24.000	.036	.295	10.038	.685
	Wilks' Lambda	.705	3.346[b]	3.000	24.000	.036	.295	10.038	.685
	Hotelling's Trace	.418	3.346[b]	3.000	24.000	.036	.295	10.038	.685

	Hotelling's Trace	.418	3.346[b]	3.000	24.000	.036	.295	10.038	.685
	Roy's Largest Root	.418	3.346[b]	3.000	24.000	.036	.295	10.038	.685

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

b. Exact statistic

c. Computed using alpha =

Mauchly's Test of Sphericity[a]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[b] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.380	23.932	5	.000	.718	.876	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

b. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Sphericity Assumed	3294.210	3	1098.070	2.413	.073	.085	7.238	.582
	Greenhouse-Geisser	3294.210	2.155	1528.402	2.413	.095	.085	5.200	.485
	Huynh-Feldt	3294.210	2.628	1253.583	2.413	.082	.085	6.340	.541
	Lower-bound	3294.210	1.000	3294.210	2.413	.132	.085	2.413	.322
time * Outcome	Sphericity Assumed	3475.953	3	1158.651	2.546	.062	.089	7.637	.607
	Greenhouse-Geisser	3475.953	2.155	1612.724	2.546	.083	.089	5.487	.508
	Huynh-Feldt	3475.953	2.628	1322.743	2.546	.071	.089	6.690	.566
	Lower-bound	3475.953	1.000	3475.953	2.546	.123	.089	2.546	.336
time * threat	Sphericity Assumed	4037.344	3	1345.781	2.957	.037	.102	8.871	.680
	Greenhouse-Geisser	4037.344	2.155	1873.190	2.957	.056	.102	6.373	.574
	Huynh-Feldt	4037.344	2.628	1536.375	2.957	.045	.102	7.770	.637
	Lower-bound	4037.344	1.000	4037.344	2.957	.097	.102	2.957	.381
time * Outcome * threat	Sphericity Assumed	3901.244	3	1300.415	2.857	.042	.099	8.572	.663
	Greenhouse-Geisser	3901.244	2.155	1810.045	2.857	.062	.099	6.158	.558
	Huynh-Feldt	3901.244	2.628	1484.584	2.857	.050	.099	7.508	.620
	Lower-bound	3901.244	1.000	3901.244	2.857	.103	.099	2.857	.370
Error(time)	Sphericity Assumed	35500.031	78	455.129					
	Greenhouse-Geisser	35500.031	56.039	633.493					

	Huynh-Feldt	35500.031	68.324	519.585
	Lower-bound	35500.031	26.000	1365.386

a. Computed using alpha =

Tests of Within-Subjects Contrasts

Measure: MEASURE_1

Source	time	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
time	Linear	2623.066	1	2623.066	3.479	.073	.118	3.479	.435
	Quadratic	655.322	1	655.322	1.307	.263	.048	1.307	.196
	Cubic	15.822	1	15.822	.144	.707	.006	.144	.065
time * Outcome	Linear	1176.316	1	1176.316	1.560	.223	.057	1.560	.225
	Quadratic	1727.426	1	1727.426	3.444	.075	.117	3.444	.432
	Cubic	572.211	1	572.211	5.206	.031	.167	5.206	.594
time * threat	Linear	3959.641	1	3959.641	5.252	.030	.168	5.252	.597
	Quadratic	30.968	1	30.968	.062	.806	.002	.062	.057
	Cubic	46.734	1	46.734	.425	.520	.016	.425	.096
time * Outcome * threat	Linear	2663.263	1	2663.263	3.532	.071	.120	3.532	.440
	Quadratic	1128.061	1	1128.061	2.249	.146	.080	2.249	.303
	Cubic	109.920	1	109.920	1.000	.327	.037	1.000	.161
Error(time)	Linear	19603.010	26	753.962					
	Quadratic	13039.137	26	501.505					
	Cubic	2857.884	26	109.919					

a. Computed using alpha =

Levene's Test of Equality of Error Variances[a]

	F	df1	df2	Sig.
C_difference_immed_percentage	1.097	1	28	.304
C_difference_30min_percentage	.111	1	28	.741
C_difference_1hr_percentage	.646	1	28	.428
C_difference_2hrs_percentage	.014	1	28	.906

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Outcome + threat + Outcome * threat
Within Subjects Design: time

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	26232.850	1	26232.850	8.209	.008	.240	8.209	.788
Outcome	5239.593	1	5239.593	1.640	.212	.059	1.640	.234
threat	106.161	1	106.161	.033	.857	.001	.033	.054
Outcome * threat	555.741	1	555.741	.174	.680	.007	.174	.069
Error	83084.665	26	3195.564					

a. Computed using alpha =

Custom Hypothesis Tests

Contrast Results (K Matrix)

Outcome Simple Contrast[a]			Averaged Variable MEASURE_1
Level 2 vs. Level 1	Contrast Estimate		18.861
	Hypothesized Value		0
	Difference (Estimate - Hypothesized)		18.861
	Std. Error		14.730
	Sig.		.212
	95% Confidence Interval for Difference	Lower Bound	-11.416
		Upper Bound	49.138

a. Reference category = 1

Test Results

Measure: MEASURE_1

Transformed Variable: AVERAGE

Source	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	1309.898	1	1309.898	1.640	.212	.059	1.640	.234
Error	20771.166	26	798.891					

a. Computed using alpha =

Estimated Marginal Means

1. Outcome

Estimates

Measure: MEASURE_1

Outcome	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
loss	12.621[a]	7.483	-2.760	28.003
win	28.106[a]	7.942	11.781	44.430

a. Covariates appearing in the model are evaluated at the following values: threat = 1.07.

Pairwise Comparisons

Measure: MEASURE_1

(I) Outcome	(J) Outcome	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
loss	win	-15.485	10.912	.168	-37.914	6.945
win	loss	15.485	10.912	.168	-6.945	37.914

Based on estimated marginal means

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Univariate Tests

Measure: MEASURE_1

	Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contrast	1608.806	1	1608.806	2.014	.168	.072	2.014	.277
Error	20771.166	26	798.891					

The F tests the effect of Outcome. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Computed using alpha =

2. time

Estimates

Measure: MEASURE_1

time	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	1.645[a]	5.125	-8.890	12.179
2	19.059[a]	6.208	6.299	31.820
3	29.135[a]	6.958	14.833	43.437
4	31.615[a]	7.532	16.133	47.096

a. Covariates appearing in the model are evaluated at the following values: threat = 1.07.

Pairwise Comparisons

Measure: MEASURE_1

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b]	
					Lower Bound	Upper Bound
1	2	-17.415*	5.724	.005	-29.180	-5.649
	3	-27.490*	6.970	.001	-41.818	-13.162
	4	-29.970*	6.980	.000	-44.318	-15.622
2	1	17.415*	5.724	.005	5.649	29.180
	3	-10.075*	3.956	.017	-18.208	-1.943
	4	-12.555	6.239	.055	-25.379	.268
3	1	27.490*	6.970	.001	13.162	41.818
	2	10.075*	3.956	.017	1.943	18.208
	4	-2.480	4.342	.573	-11.405	6.445
4	1	29.970*	6.980	.000	15.622	44.318
	2	12.555	6.239	.055	-.268	25.379
	3	2.480	4.342	.573	-6.445	11.405

Based on estimated marginal means

*. The mean difference is significant at the

b. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Pillai's trace	.426	5.928[a]	3.000	24.000	.004	.426	17.783	.918
Wilks' lambda	.574	5.928[a]	3.000	24.000	.004	.426	17.783	.918
Hotelling's trace	.741	5.928[a]	3.000	24.000	.004	.426	17.783	.918
Roy's largest root	.741	5.928[a]	3.000	24.000	.004	.426	17.783	.918

Each F tests the multivariate effect of time. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

b. Computed using alpha =

3. Outcome * time

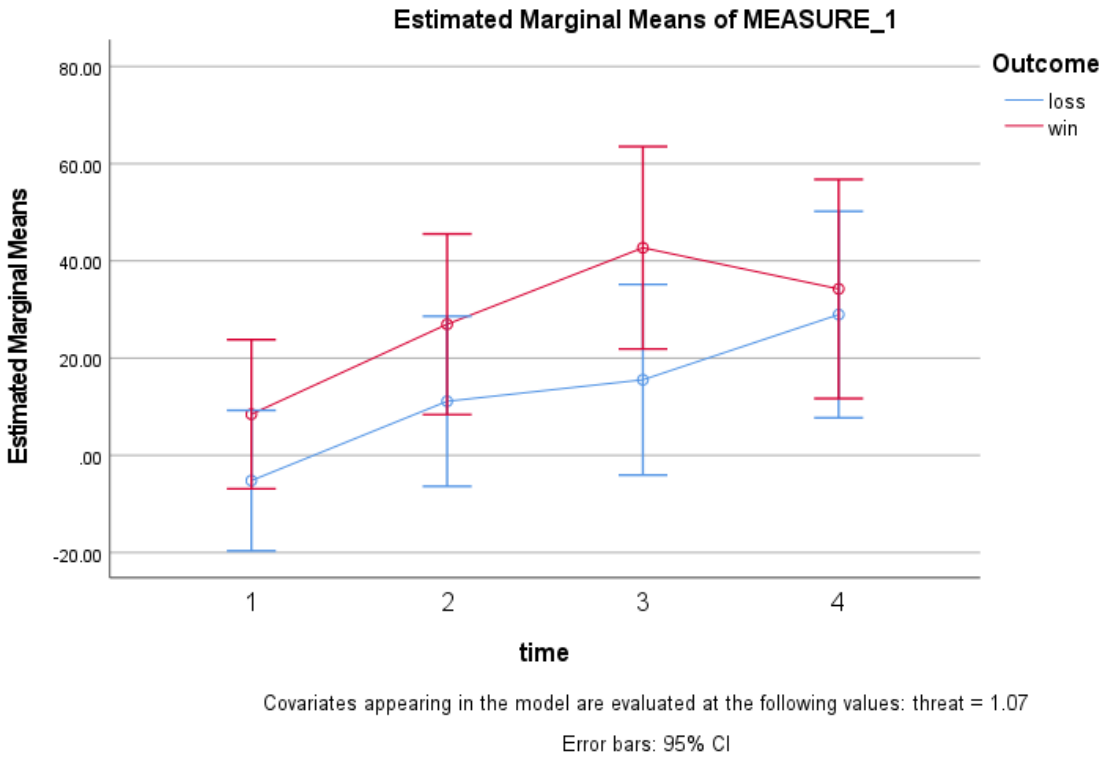
Measure: MEASURE_1

Outcome	time	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
loss	1	-5.183[a]	7.029	-19.631	9.266

	2	11.136[a]	8.514	-6.366	28.637
	3	15.554[a]	9.543	-4.061	35.170
	4	28.977[a]	10.330	7.743	50.211
win	1	8.472[a]	7.460	-6.862	23.806
	2	26.983[a]	9.036	8.409	45.557
	3	42.715[a]	10.128	21.897	63.533
	4	34.253[a]	10.963	11.717	56.788

a. Covariates appearing in the model are evaluated at the following values: threat = 1.07.

Profile Plots



Covariates appearing in the model are evaluated at the following values: threat = 1.07

Error bars: 95% CI

Appendix XLI

Regression Model For Trait Affect, Competition Outcome and C Reactivity

Regression

Descriptive Statistics

	Mean	Std. Deviation	N
C_difference_immed_percentage	-.8433	27.51036	29
GNA_PANAS_Difference	-.1379	4.52552	29
GPA_PANAS_Difference	1.9310	6.75559	29
Outcome	.52	.509	29

Correlations

		C_difference_immed_percentage	GNA_PANAS_Difference	GPA_PANAS_Difference	Outcome
Pearson Correlation	C_difference_immed_percentage	1.000	.354	.264	.156
	GNA_PANAS_Difference	.354	1.000	-.047	-.014
	GPA_PANAS_Difference	.264	-.047	1.000	-.405
	Outcome	.156	-.014	-.405	1.000
Sig. (1-tailed)	C_difference_immed_percentage	.	.030	.083	.209
	GNA_PANAS_Difference	.030	.	.404	.470
	GPA_PANAS_Difference	.083	.404	.	.015
	Outcome	.209	.470	.015	.
N	C_difference_immed_percentage	29	29	29	29
	GNA_PANAS_Difference	29	29	29	29
	GPA_PANAS_Difference	29	29	29	29
	Outcome	29	29	29	29

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	Outcome, GNA_PANAS_Difference, GPA_PANAS_Difference[b]	.	Enter

a. Dependent Variable: C_difference_immed_percentage

b. All requested variables entered.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics					Durbin-Watson
					R Square Change	F Change	df1	df2	Sig. F Change	
1	.543[a]	.295	.210	24.44992	.295	3.483	3	25	.031	2.701

a. Predictors: (Constant), Outcome, GNA_PANAS_Difference, GPA_PANAS_Difference

b. Dependent Variable: C_difference_immed_percentage

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	6245.996	3	2081.999	3.483	.031[b]
	Residual	14944.963	25	597.799		
	Total	21190.959	28			

a. Dependent Variable: C_difference_immed_percentage

b. Predictors: (Constant), Outcome, GNA_PANAS_Difference, GPA_PANAS_Difference

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B		Correlations		
		B	Std. Error	Beta			Lower Bound	Upper Bound	Zero-order	Partial	Part
1	(Constant)	-13.021	7.427		-1.753	.092	-28.316	2.274			
	GNA_PANAS_Difference	2.297	1.023	.378	2.246	.034	.190	4.403	.354	.410	.377
	GPA_PANAS_Difference	1.692	.749	.415	2.258	.033	.148	3.235	.264	.412	.379
	Outcome	17.840	9.944	.330	1.794	.085	-2.641	38.321	.156	.338	.301

a. Dependent Variable: C_difference_immed_percentage

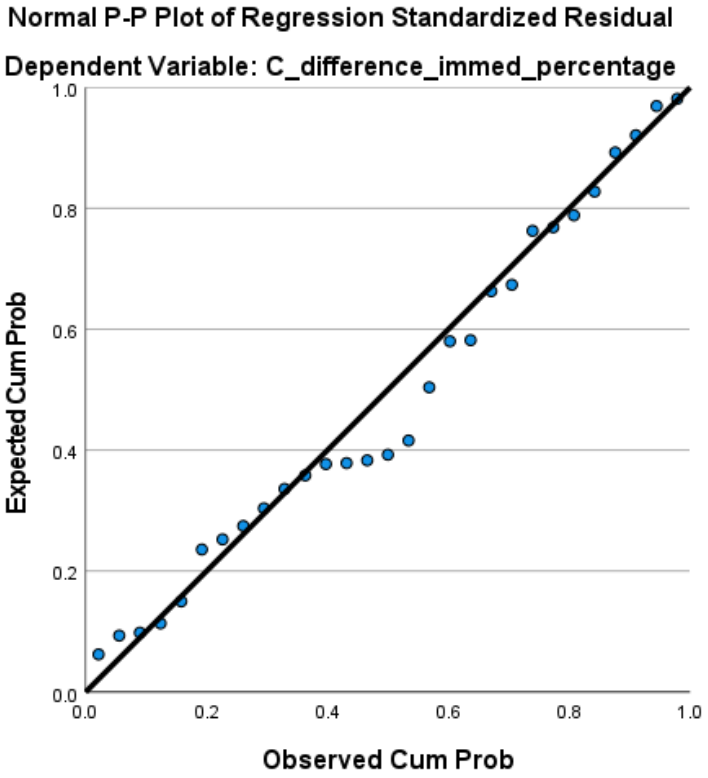
Residuals Statistics[a]

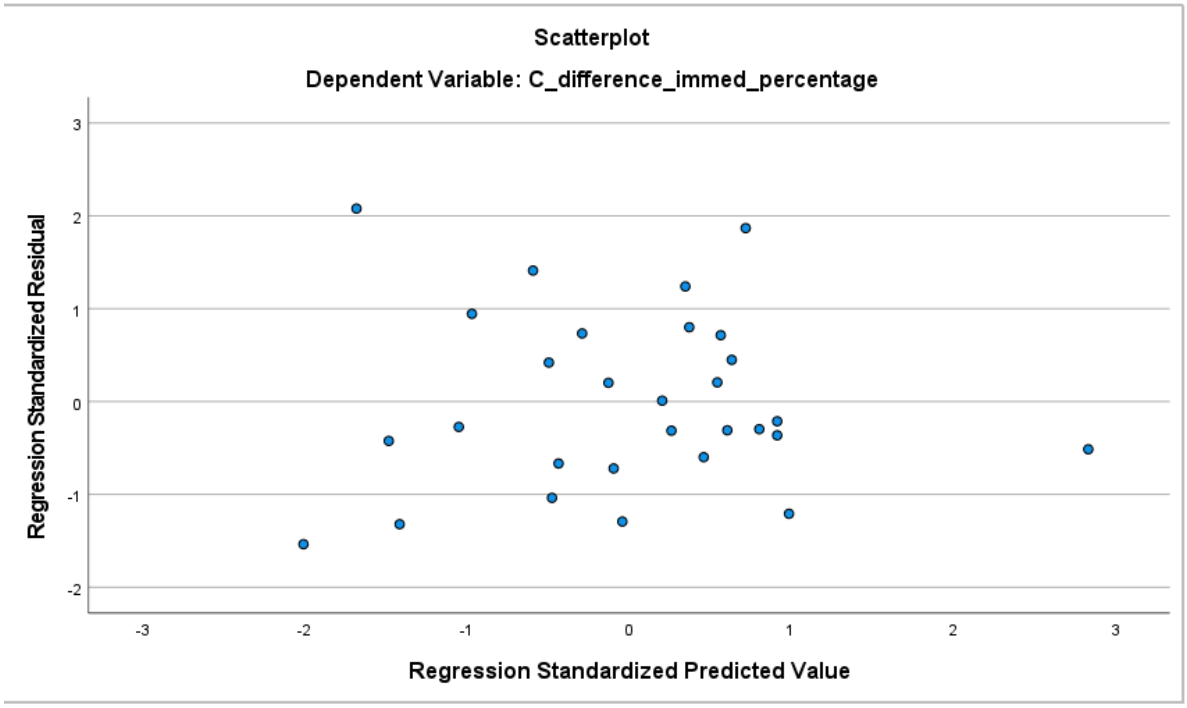
	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	-30.8318	41.4454	-.8433	14.93557	29
Std. Predicted Value	-2.008	2.831	.000	1.000	29
Standard Error of Predicted Value	6.585	15.487	8.686	2.694	29
Adjusted Predicted Value	-42.7075	49.8573	-.2092	16.12248	29
Residual	-37.55940	50.83329	.00000	23.10300	29
Std. Residual	-1.536	2.079	.000	.945	29
Stud. Residual	-1.888	2.397	-.011	1.048	29
Deleted Residual	-56.71569	67.59061	-.63415	28.68708	29
Stud. Deleted Residual	-1.997	2.677	-.001	1.094	29
Mahal. Distance	1.065	10.268	2.897	2.640	29

Mahal. Distance	1.065	10.268	2.897	2.640	29
Cook's Distance	.000	.474	.067	.133	29
Centered Leverage Value	.038	.367	.103	.094	29

a. Dependent Variable: C_difference_immed_percentage

Charts




Scatterplot
Dependent Variable: C_difference_immed_percentage
Regression Standardized Residual
Regression Standardized Predicted Value
3
2
1
0
-1
-2
-3
-2
-1
0
1
2
3

Appendix XLII

Regression T reactivity and Motivation

Regression

Descriptive Statistics

	Mean	Std. Deviation	N
Complete_Form_Motv	1.6667	1.34762	30
Testo_difference_immediately_percentage	-.2590	16.13290	30

Correlations

		Complete_Form_Motv	Testo_difference_immediately_percentage
Pearson Correlation	Complete_Form_Motv	1.000	.040
	Testo_difference_immediately_percentage	.040	1.000
Sig. (1-tailed)	Complete_Form_Motv	.	.417
	Testo_difference_immediately_percentage	.417	.
N	Complete_Form_Motv	30	30
	Testo_difference_immediately_percentage	30	30

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	Testo_difference_immediately_percentage[b]	.	Enter

a. Dependent Variable: Complete_Form_Motv

b. All requested variables entered.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Durbin-Watson
1	.040[a]	.002	-.034	1.37038	1.371

a. Predictors: (Constant), Testo_difference_immediately_percentage

b. Dependent Variable: Complete_Form_Motv

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	.084	1	.084	.045	.834[b]
	Residual	52.582	28	1.878		
	Total	52.667	29			

a. Dependent Variable: Complete_Form_Motv

b. Predictors: (Constant), Testo_difference_immediately_percentage

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval for B	
		B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound
1	(Constant)	1.668	.250		6.664	.000	1.155	2.180
	Testo_difference_immediately_percentage	.003	.016	.040	.212	.834	-.029	.036

a. Dependent Variable: Complete_Form_Motv

Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	1.5507	1.7849	1.6667	.05391	30
Residual	-1.75805	2.36542	.00000	1.34655	30
Std. Predicted Value	-2.151	2.192	.000	1.000	30
Std. Residual	-1.283	1.726	.000	.983	30

a. Dependent Variable: Complete_Form_Motv

Appendix XLIII

Regression Models for T reactvitiy and Motivation within winning and losing cohorts

Regression

[DataSet1] /Users/Koki/Desktop/OneDrive - University of Strathclyde/PhD/Data I

Descriptive Statistics

	Mean	Std. Deviation	N
Complete_Form_Motv	1.4286	1.34246	14
Testo_difference_immediately_percentage	-3.0353	15.53986	14

Correlations

		Complete_Form_Motv	Testo_difference_immediately_percentage
Pearson Correlation	Complete_Form_Motv	1.000	.400
	Testo_difference_immediately_percentage	.400	1.000
Sig. (1-tailed)	Complete_Form_Motv	.	.078
	Testo_difference_immediately_percentage	.078	.
N	Complete_Form_Motv	14	14
	Testo_difference_immediately_percentage	14	14

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	Testo_difference_immediately_percentage[b]	.	Enter

a. Dependent Variable: Complete_Form_Motv

b. All requested variables entered.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Durbin-Watson
1	.400[a]	.160	.090	1.28083	2.168

a. Predictors: (Constant), Testo_difference_immediately_percentage

a. Predictors: (Constant), Testo_difference_immediately_percentage

b. Dependent Variable: Complete_Form_Motv

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	3.742	1	3.742	2.281	.157[b]
	Residual	19.686	12	1.641		
	Total	23.429	13			

a. Dependent Variable: Complete_Form_Motv

b. Predictors: (Constant), Testo_difference_immediately_percentage

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	1.533	.349		4.390	.001	.772	2.294
	Testo_difference_immediately_percentage	.035	.023	.400	1.510	.157	-.015	.084

a. Dependent Variable: Complete_Form_Motv

Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	.3264	2.2843	1.4286	.53653	14
Residual	-1.61856	2.45192	.00000	1.23058	14
Std. Predicted Value	-2.054	1.595	.000	1.000	14
Std. Residual	-1.264	1.914	.000	.961	14

a. Dependent Variable: Complete_Form_Motv

Regression

Descriptive Statistics

	Mean	Std. Deviation	N
Complete_Form_Motv	1.8750	1.36015	16
Testo_difference_immediately_percentage	2.1702	16.74552	16

Correlations

		Complete_Form_Motv	Testo_difference_immediately_percentage
Pearson Correlation	Complete_Form_Motv	1.000	-.294
	Testo_difference_immediately_percentage	-.294	1.000
Sig. (1-tailed)	Complete_Form_Motv	.	.134
	Testo_difference_immediately_percentage	.134	.
N	Complete_Form_Motv	16	16
	Testo_difference_immediately_percentage	16	16

Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	Testo_difference_immediately_percentage[b]	.	Enter

a. Dependent Variable: Complete_Form_Motv

b. All requested variables entered.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Durbin-Watson
1	.294[a]	.087	.021	1.34557	1.682

a. Predictors: (Constant), Testo_difference_immediately_percentage

b. Dependent Variable: Complete_Form_Motv

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	2.402	1	2.402	1.327	.269[b]
	Residual	25.348	14	1.811		
	Total	27.750	15			

a. Dependent Variable: Complete_Form_Motv

b. Predictors: (Constant), Testo_difference_immediately_percentage

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	1.927	.339		5.677	.000	1.199	2.655
	Testo_difference_immediately_percentage	-.024	.021	-.294	-1.152	.269	-.068	.021

a. Dependent Variable: Complete_Form_Motv

Residuals Statistics[a]

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	1.0878	2.6880	1.8750	.40018	16
Residual	-1.70430	2.39728	.00000	1.29994	16
Std. Predicted Value	-1.967	2.032	.000	1.000	16
Std. Residual	-1.267	1.782	.000	.966	16

a. Dependent Variable: Complete_Form_Motv