Plotting the Intertwining Psychological and Biological Pathways Linking Stress and Health

by

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Declaration

I hereby declare that this thesis:

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Summary

The ability to maintain psychological well-being in the face of exposure to stressors is a crucial capacity that has implications for human health. How a person responds to adverse conditions is contingent on a multitude of interwoven biological and psychological factors. It is my goal in this thesis to outline a broad framework derived closely from the reserve capacity model (Gallo & Matthews, 2003) that specifies and provides empirical support for these relations. I do this by extending the current reserve capacity model and the methodology used to test its core tenets. Specifically, I demonstrate via empirical studies (i) how methodological advancements for the measurement of affect and psychobiological functioning can be used to test and provide support for the model (chapters 2 and 3), (ii) how feedback loops from health to emotion can be addressed in the model (chapters 4 and 5), and (iii) how genetic factors can be incorporated into the reserve capacity model (chapters 6 and 7). This is followed by a general discussion of the main findings, chapter 8.

Chapter 2 investigates the relation between affect levels and cardiovascular activity in day-to-day settings using a primary data source of 186 people who completed the Day Reconstruction Method (Kahneman, Krueger, Schkade, Schwarz, & Stone, 2004) and underwent baseline physiological testing and were monitored naturalistically for an entire day. Negative affect was found to predict an elevated ambulatory heart rate and tiredness predicted a lower heart rate. Chapter 3 examines whether a psychosocial resource, self-control, modulates patterns of emotion with likely implications for psychobiological functioning. High trait self-control was associated with stable emotional patterns which partially mediated cortisol and heart rate levels. This study indicated that the capacity to sustain stable patterns of affect across diverse contexts appears to be an important pathway through which self-control relates to health.

Chapters 4 and 5 address the idea that health conditions and psychobiological processes can influence well-being. Firstly, morning cortisol levels were shown to predict a steep increase in positive affect from morning through to the evening, particularly among the distressed. Next, in a study based on secondary data from two samples (N = 8190), obesity-related inflammation, as indexed by the acute phase reactant C-reactive protein, was shown to mediate between the presence of obesity and the neurovegetative symptoms of depression. Both of these studies suggest that it may be beneficial to extend the reserve capacity model to incorporate reciprocal relations and reverse feedback processes.

Chapters 6 and 7 aimed to investigate if genetic factors can modulate the emotional and health response to life-stressors (based on secondary data, N = 755). The first genetic study tested the role of the apolipoprotein E gene in moderating the influence of an exogenous stressor, an earthquake, on health. Those who experienced damage to their property or were forced to move from their homes as a result of the earthquake had low self-rated health a year later, only if they were apolipoprotein ε4 carriers (a dysfunctional lipid transporter). This study indicates support for the proposed extension to include genetic factors in the reserve capacity model. The second gene-stressor interaction study aimed to test if the stress of illness can be modified by both psychosocial and genetic factors. It did this by showing the number of chronic illnesses a person has been diagnosed with interacted with perceived control and variation in the apolipoprotein E gene to predict psychological adjustment. High levels of perceived control appeared to dampen genetic sensitivity to the adverse psychological effects of illness. As discussed in Chapter 8, the results of this thesis support a bidirectional resource model of health where one's genetic endowment, exposure to stressors, and psychological resources interact to produce patterns of emotion and psychobiological functioning which may lead to the exacerbation of illness.

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Publications arising from the present work

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Daly, M., Delaney, L., Doran, P., & MacLachlan, M. (under review). Awakening cortisol and diurnal rhythms of affect in psychological distress.

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Chapter 1

1. Introduction

This thesis aims to contribute to the literature which examines how stressful events can have mental and physical health effects. Various theoretical frameworks have been proposed that each outline different ways that stressors interact with factors like stable individual differences in personality, coping, health behaviour, and psychobiological functioning to predict health outcomes. In the first part of this general introductory chapter I provide an overview of the theoretical and empirical foundations of contemporary models of the stress-health link. Following this, I summarize key stress models and describe the reserve capacity model (Gallo & Matthews, 2003). I then explain how the reserve capacity model integrates the insights of existing models and provides a framework which is strongly supported by a wide-array of epidemiological and experimental research. In the later sections I outline vital gaps within the reserve capacity model and suggest areas where important relationships may exist but are not given appropriate attention in the current framework. Finally, I chart advancements in the measurement of *experience in context* and then suggest how these developments could help specify how stress links to health within the reserve capacity model. I conclude this general introduction by outlining the aims of the study which are addressed in six subsequent empirical chapters and a general discussion of the main findings.

1.1 Stress: A brief history of physiological balance and environmental demands

The model of stress used in this thesis follows closely from the reserve capacity model proposed by Gallo and Matthews (2003) who draw from the landmark work of stress researchers such as Steven Hobfoll (1989) and Richard Lazarus and Suzanne Folkman (1984). However, the theoretical and empirical antecedents of such models can

be traced back over a century to the work of three prominent physiologists: Claude Bernard, Walter Cannon and Hans Seyle.

Claude Bernard's work on the release of glucose from the liver, heat regulation, and the control of blood flow by sympathetic nerves formed the background from which his theory of how internal bodily states are maintained emerged. He described the "*milieu interieur"* as sustaining a balance through compensatory changes in numerous physiological processes. Canon labeled this trend towards physiological regulation for the purpose of stability as "homeostasis". He proposed that the levels of a wide array of physiological systems (e.g. blood glucose, oxygenation levels) are constantly monitored and maintained within acceptable ranges. All physiological endpoints were proposed to have an ideal level (e.g. an ideal blood pressure level, an ideal level of blood glucose) and the optimal state of functioning arises when as many such endpoints are at their ideal level (Goldstein & Kopin, 2007). This process depends largely on negative feedback loops which deactivate corrective mechanisms once appropriate levels of a particular physiological variable have been achieved. There are numerous "stressors" which can threaten homeostasis such as hypothermia, hypoglycemia, or physical illnesses. When such disruptions occur the body takes steps to ameliorate the physiological changes which have occurred by initiating a "stress response".

The negative feedback process implicated in homeostasis is often compared to the maintenance of a consistent temperature level by the thermostat in the home. Deviations from the temperature set-point are monitored by the thermostat which can affect change in the monitored variable (e.g. by turning on an air-conditioning unit) until temperature returns to normal levels upon which this fact is detected and the change is discontinued. Body heat is monitored in a similar way with high temperature levels corrected through the

activation of sweating and other processes which lead to heat loss, followed by a suspension of these processes when an appropriate temperature range is achieved.

Cannon made the key creative insight that disruptions to homeostasis may occur as a result of psychosocial threats and that negative feedback loops may correct such disruptions. This is because the psychological response to threat can activate the sympathetic adrenomedullary system (a process Cannon termed "the fight-or-flight" response) and the later explicated hypothalamic-pituitary-adrenal axis which can influence homeostasis. The autonomic nervous system is made up of the sympathetic and parasympathetic nervous systems. The parasympathetic branch of the autonomic nervous system is linked to recuperative processes such as digestion, the slowing of the heartbeat and an increase in heart rate variability. The sympathetic nervous system energizes the heart and vasculature, releasing norepinephrine and innervating the adrenal medulla, which releases epinephrine the "fight or flight" hormone that readies the body for action by raising blood sugar levels, dilating pupils, and suppressing the immune system. Corticotrophin-releasing hormone from the paraventricular nucleus of the hypothalamus then activates the hypothalamic-pituitary-adrenocortical axis which stimulates the release of corticotrophin from the pituitary, thus prompting glucocorticoid output, primarily from the zona fasiculata in the adrenal cortex (Lupien, Maheu, Fiocco, & Schramek, 2007). Together the neuroendocrine and cardiovascular system allow the body to react to short-term and enduring stressors. Following Canon's insights regarding the role of psychosocial stressors, theorists began to model stress processes as relations between environmental demands and physiological reactivity, forming the theoretical basis for later models which introduced psychological mediators and modifiers of this process.

In the early 20th century the concept of stress had meaning mainly in areas such as engineering where it was used to describe the ability of a material to react, resist, and

restore form or become damaged under physical strain. Hans Seyle (1956) developed this heuristic model into a framework that delineated the stages of physiological reactivity to the demands placed on the body. Seyle outlined a series of physiological stress responses he called the general adaptation syndrome. He proposed that three universal stages occur when stress on the body is prolonged. The initial state is an alarm reaction which is similar to the "fight-or-flight" response to an emergency mediated principally by the activation of the sympathetic nervous system. The body is mobilized to contend with a stressor and arousal levels are increased as indexed by physiological changes such as elevated blood pressure, heart rate, and skin conductance. If stress continues a resistance stage follows whereby the stressor causes bodily arousal levels to be consistently elevated. In this state the body must provide resources to replenish the hormones and the capacity to respond to additional stressors may be impaired. Seyle proposed that in the resistance stage people are vulnerable to "diseases of adaptation" such as impaired immune functioning and the development of ulcers. The third stage is characterized by exhaustion, a depletion of bodily resources, disease proneness, and damage to internal organs potentially leading to organismic death.

Through his experimental work Seyle observed that very different stressors could produce the same pathological changes, namely, the development of ulcers, an increase in the size of the adrenal glands and a reduction in the size of immune organs. Whilst Canon's experiments focused on the sympathetic control of the secretion of epinephrine and norepinephrine, Seyle's laboratory research unveiled a link between stressors, physical pathology, and the secretion of steroid hormones called glucocorticoids by the adrenal glands. Both Canon and Seyle substantially advanced the work of Claude Bernard and laid the foundations for modern perspectives on stress. A variety of other endocrine secretions have since been linked to the physiological stress response including raised levels of vasopressin, prolactin, and glucagon as well as diminished levels of androgens and insulin

(Sapolsky, 2007). This wide array of physiological adjustments made by the body in times of stress ensure survival through numerous mechanisms including the mobilization of energy resources and shifting of blood flow to the muscles rather than less essential areas such as the intestines. In prehistoric times it is likely that these changes were often necessary for short-term survival and the physiological costs to restoring homeostasis were worthwhile. However, stress researchers have demonstrated that recovery from explosive changes in energy expenditure and dramatic shifts in physiology can take their toll especially when such dramatic changes are repeated continuously.

Sterling and Eyer (1988) introduced the term "allostasis" which is an estimate of the level of activity required by the organism to adapt or to "maintain stability through change". Allostasis typically refers to the quick adjustments made by bodily systems to correct the impact of contemporaneous factors such as bodily position, interaction, movement, and food intake. Allostasis differs from homeostasis in that it focused on the maintenance of physiological stability by matching internal processes to environmental demands (McEwen & Wingfield, 2003). Health is viewed as a whole body adaptation to context and fixed ideal physiological set-points in the homeostasis model are replaced by dynamic set-points which respond to changing criteria in the allostasis model. For example, elevating glucose levels to a higher than normal level during exercise represents an adaptive temporarily altered set-point (Goldstein & Kopin, 2007).

If environmental demands are consistently high or if there are inefficiencies in the process of allostasis (e.g. a consistently high level of one physiological parameter requiring a similarly raised level of an antagonistic parameter), "allostatic load" or wear-and-tear to the body can occur. This idea integrates key insights from prior work, such as Canon's recognition that environmental demands can have physiological effects and Seyle's model of stress resistance as causing long-term health effects. Allostatic load is considered a

calculable index of the long-term cost of adaptation which has predictable implications for subsequent health.

Numerous mechanisms have been documented whereby chronic activation of the stress system can damage the brain and body of distressed people (Juster, McEwen, & Lupien, 2009). Cortisol and the stress hormones epinephrine and norepinephrine in combination with inflammatory cytokines are considered to be the primary mediators of allostatic disruption. Metabolic, cardiovascular, and immune influences are then influenced to produce secondary allostatic outcomes which contribute to allostatic overload, leading eventually to disease states. For example, sympathetic nervous system activation as indexed by elevated blood pressure and heart rate may contribute to the development of heart failure and consistently high levels of activation of the HPA could lead to insulin resistance (Goldstein & Kopin, 2007). Thus, the stress response itself can damage the body if activated over prolonged periods.

Typical physical conditions which may elicit a stress response such as cold exposure or bodily harm due to hemorrhage or wounding are likely to be far more damaging than the stress response itself. Such stressors are also likely to end relatively quickly or to cause the death of the organism. Thus, it appears that high impact physical stressors may not lead to prolonged activation of the stress system and the associated detrimental health effects. Instead, stress-related diseases are likely to result from extended psychological rather than physical pressures (Sapolsky, 2007). This may be a problem in the modern age as the physiological processes which were designed for fleeing or fighting a lethal predator or rival appear to remain activated as a result of the slights and aggravations of contemporary daily life. There is a broad spectrum of potential causes of the physiological stress response and allostatic load ranging from overarching macro-level factors such as the current political system and social norms to intermediate systems of

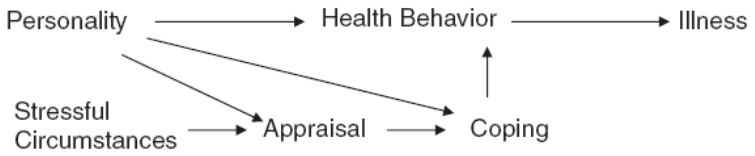
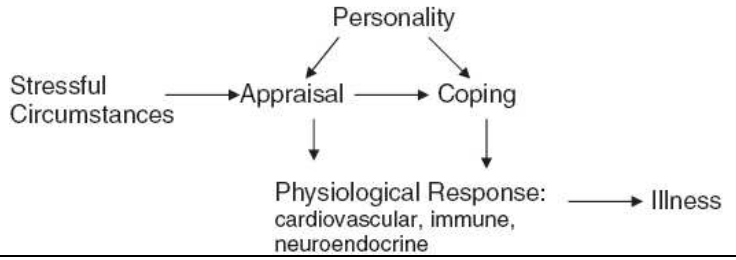
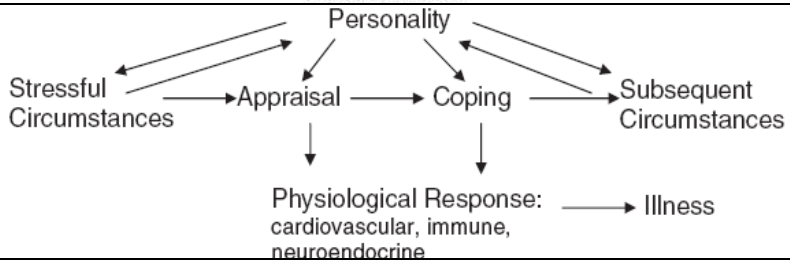
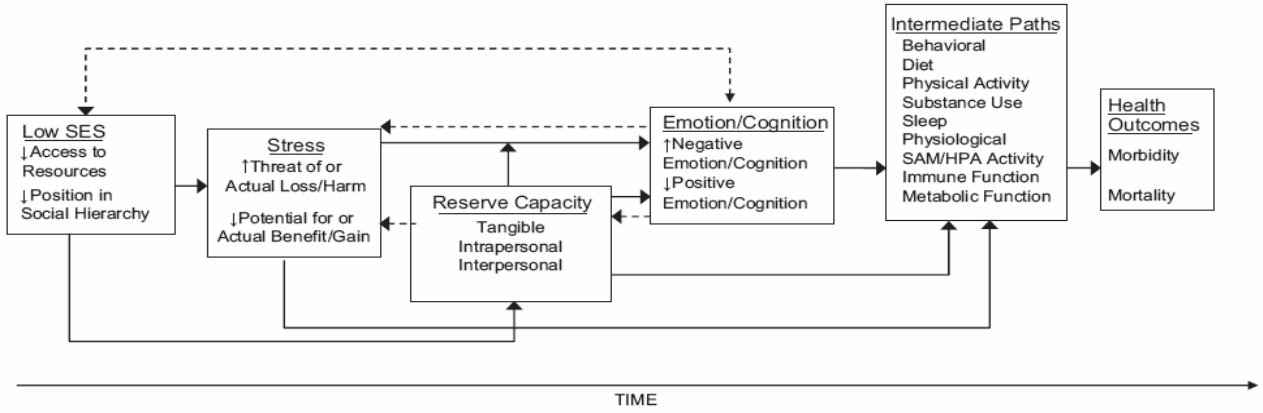
social networks and mass media and then to peer groups, the family and school and work environments which in turn impinge of the individual.

To summarize, when faced with an acute physical challenge the body responds rapidly, mobilizing a coordinated biological stress response to ensure survival. However, whilst multi-system stress-related changes are adaptive in the short-term, their chronic activation is not protective and instead carries a vast array of physical health risks. As physical stressors are usually acute in nature it is psychologically activated stress responses which often persist long enough to cause pathophysiological changes. A broad-set of contemporary models have been proposed to gauge the relation between psychological stress and poor-health, the foremost of which are outlined in brief below.

1.2. Psychological models of stress and health and the reserve capacity model

A set of stress models and the mechanisms suggested to explain the link between exposure to stressors and a decline in health are illustrated in Table 1. The *health behaviour model* suggests that more or less favorable health-relevant behaviours such as exercising or smoking are central to explaining the role of psychological factors in stress-health relationship. Personality can modify the extent to which stressors are appraised as stressful and subsequently lead to indulgent health behaivour. For example, there is clear evidence that certain personality traits like conscientiousness are closely associated with the foods people choose to eat, the amount of alcohol they consume, and other health habits. Conscientious people make healthier choices and live longer lives (Kern & Friedman, 2008). However, the link between personality traits like conscientiousness and important health outcomes such as mortality cannot be fully accounted for by health behaviour (Smith, 2006). The groundbreaking work of Bernard, Cannon, and Seyle suggests that the physiological stress response way be an alternative way that stress can cause adverse health outcomes. Personality could influence how stressors are appraised

Table 1. Models of the processes linking stress, personality, and health

Health Behaviour Model	
Interactional Stress Moderation Model	
Transactional Stress Moderation Model	
Reserve Capacity Model	

and how people manage appraisals of threat, harm, or loss when they occur. In this way personality traits could modify physiological reactivity to stress as indexed by cardiovascular, neuroendocrine, and immune functioning, as proposed in the *interactional stress moderation model* (Smith, Glazer, Ruiz, & Gallo, 2004). Indeed, personality traits like hostility, neuroticism, and trait positive affect have been shown to exaggerate or attenuate psychobiological reactivity and recovery to an experimental stressor, as discussed in detail later in the introduction (e.g. Chida & Steptoe, 2009a).

The *transactional model of stress* (Lazarus & Cohen, 1977) proposes that as well as modifying the physiological effects of stressors, personality traits also determine what kinds of situations a person encounters and the frequency and severity of the stressors those situations provoke. For example, a neurotic person may appraise a social situation as threatening and fail to pick up on social cues thus potentially provoking indifference or hostility. Traits like agreeableness and openness to experience may assist in resolving stressful situations when they occur thus altering the duration of the stressor and the circumstances which follow. As well as personality influencing the occurrence of stressors, stressful circumstances could contribute to the developmental trajectory of personality. For example, adverse early environments could promote neuroticism and diminish openness to experience and agreeableness, thus increasing the likelihood of stress in later life and making conflict resolution unlikely when stressful situations occur.

The reserve capacity model outlines: (i) how health behaviour can mediate between personality and health, (ii) how personality can influence the stress appraisal processes and the psychobiological reactivity and recovery that follows, (iii) and how personality can influence the extent to which stressors are encountered. It thus incorporates much of the health behaviour, interactional stress moderation, and transactional models of stress. In addition, the reserve capacity model distinguishes between personality traits which index

intrapersonal psychosocial resources (e.g. self-esteem, optimism, perceived control) and cognitive-emotional factors (e.g. negative affect, hostility, anxiety proneness, aggressiveness, trait positive affect). The model therefore generates predictions which go beyond prior models of stress and health. For example, the traits that make up intrapersonal resources are considered to induce change in cognitive-emotional factors and to modify the effect of stressors on cognitive-emotional factors. Attenuation or elevation in the intensity, frequency, or duration of emotional responses in turn leads to both behavioural and psychobiological changes which may influence health. Thus, the reserve capacity model clearly delineates the relations between stressful circumstances, well-being and health and shows how certain psychological factors can buffer these relations. I now turn to the special buffer role outlined for psychosocial resources in the reserve capacity model and specify how the reserve capacity model provides a framework that allows emotion, psychobiology, health behaviour and health to be examined in unison.

1.3. Research supporting the reserve capacity model

1.3.1. Psychosocial resources, stress, and health

The reserve capacity model (see Figure 1; Gallo & Matthews, 2003) places resources at the centre of the stress management process drawing on the work of Stevan Hobfoll (1989) who proposed the conservation of resources model of stress. Hobfoll's model uses an economic metaphor, where people are proposed to actively accumulate and protect various types of resources including personal characteristics (e.g. self-mastery, personal control), conditions (e.g. commitments such as job contracts and marriage), energies (e.g. money, time), objects (e.g. home), and social support. Stress results from a potential threat to such valued resources. Resources can buffer against stress by aiding the accumulation of further resources, reducing the likelihood of resources being lost, offsetting the effect of unavoidable losses, and by preventing loss spirals.

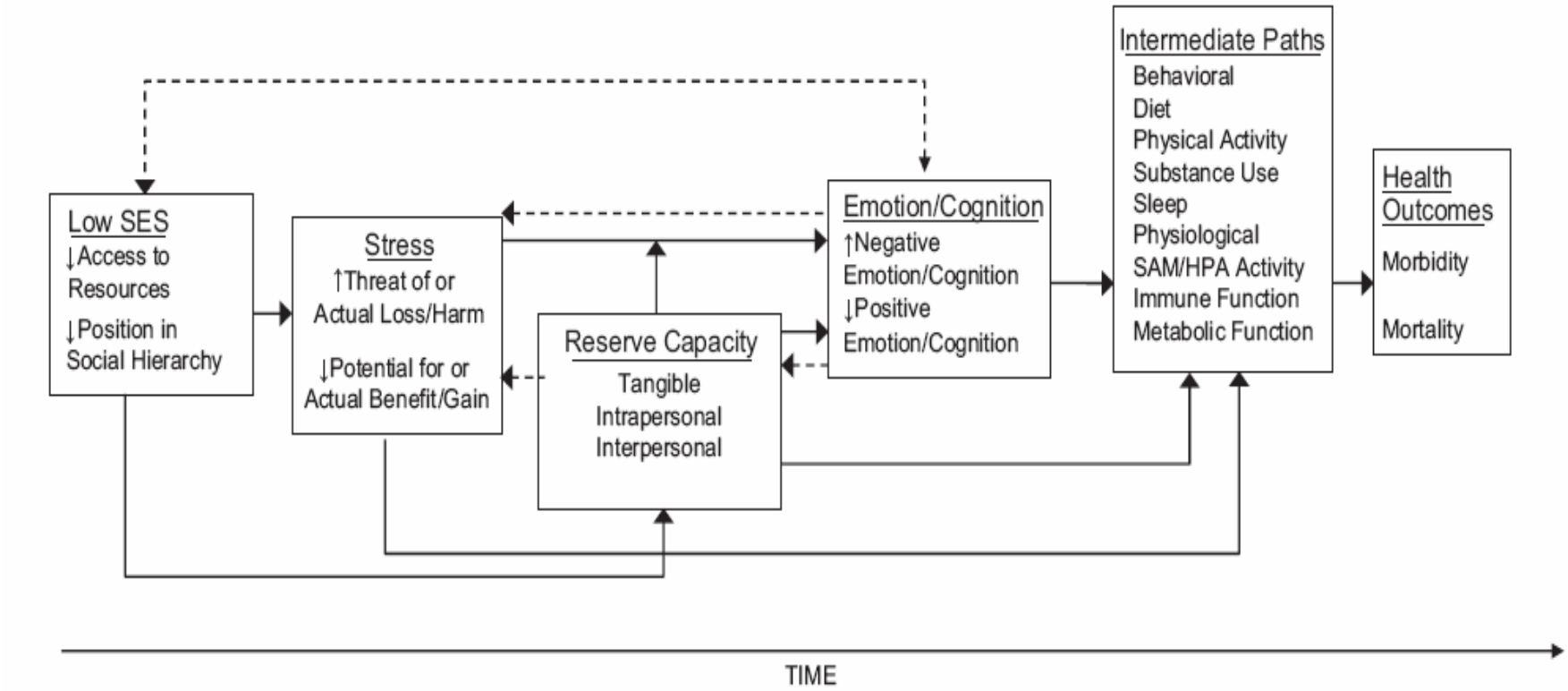


Figure 1. The Reserve Capacity Model (from Gallo, de los Monteros, & Shivpuri, 2009)

The reserve capacity (Gallo & Matthews, 2003; Gallo, de los Monteros, & Shivpuri, 2009) is composed of tangible resources (comparable to Hobfoll's energies, conditions, and objects) and intrapersonal and interpersonal resources (akin to Hobfoll's personal characteristics and social support). However, it differs from the conservation of resources model in that it does not link the induction of stress solely to the threat (i.e. the perception that an event is likely to occur which has negative consequences) of or loss (i.e. the perception of bad consequences that already exist) / harm (i.e. the discontinuation of a favoured state of affairs) to personal resources. Instead, the resources which characterize each person's reserve capacity are viewed as functional; allowing the person to manage stressful events that may result in threat of or harm / loss. The reserve capacity is considered important because by modifying the emotional and psychophysiological effects of stress it may enhance well-being and reduce the likelihood of illness.

Both intrapersonal and interpersonal resources (or psychosocial resources) may contribute to stress reactivity. For instance, psychosocial resources have been proposed to alter the likelihood of encountering potential threats by changing the perception of a threat or by assisting people in managing threatening events (Taylor & Stanton, 2007). Numerous studies have shown psychosocial resources to buffer the potential psychological and physiological effects of stress (e.g. Armitage & Conner, 2001; Helgeson, 1992; Rodin & Langer, 1977). Recent experimental studies have shown that intrapersonal resources such as self-esteem and optimism appear to buffer stress-induced psychophysiological changes (Taylor, Burklund, Eisenberger, Lehman, Hilmert, 2008). For instance, by protecting against heart rate variability decreases or increases in cytokines after a stressful exercise (e.g. interleukin-6, tumor-necrosis factor-α and interleukin-1 receptor antagonist) (O'Donnell, Brydon, Wright, & Steptoe, 2008; Brydon, Walker, Wawrzyniak, Chart, & Steptoe, 2009). Taylor et al. (2008) showed a composite measure of optimism, self-esteem, extraversion, and psychological health to be associated with

elevated right ventrolateral prefrontal cortex and reduced amygdala activity during the regulation of threat suggesting that such intrapersonal resources may assist in inhibiting the threat response to presented stressors. Amygdala activity mediated between coping resources and cortisol reactivity in this study indicating that intrapersonal resources may have health effects.

The reserve capacity model and prior stress-buffering models (e.g. Wheaton, 1985) explicate how psychosocial resources can both moderate and mediate the link between stress and health outcomes, primarily by affecting the emotional response to stressors. The mediation model implies that stressors, particularly throughout early development, can have a negative effect on ones reserve capacity. This resource deficit, in turn, impairs the management of stressors as illustrated in the moderation model. There may also be a direct relation between psychosocial resources and emotion. Whilst early stress-buffering models focused primarily on mental health, the reserve capacity model suggests that diminished intrapersonal, interpersonal, and tangible resources can have effects on both mental and physical health.

Several studies support this contention. For example, low self-esteem has been shown to lead to depression (Orth, Robins, & Roberts, 2008; Orth, Robins, & Meier, 2009), optimistic people have been shown to adapt well to stressful circumstances (Scheier, Weintraub, & Carver, 1986), extraversion underlies social engagement and has been linked to health benefits also, such as a decreased probability of contracting the common cold after exposure to the virus (Cohen, Doyle, Turner, Alper, & Skoner, 2003). Further, an analysis of a sample of 401 from the Health Women Study showed that a low level of reserve capacity as indexed by several measures of psychosocial resources was linked to a high level of negative emotion which mediated between the reserve capacity and a higher incidence of the metabolic syndrome (Matthews, Raikkonen, Gallo, &

Kuller, 2008). These findings indicate that psychosocial resources, which form the basis for the reserve capacity, may attenuate the emotional and health effects of stress. Within the context of the reserve capacity model it therefore essential to outline the link between emotion and health that is proposed to be influenced by psychosocial resources.

1.3.2. Emotion and health

Feelings of hopelessness, depression, anxiety, and hostility have all been shown to be predictive of later health outcomes. For instance, a review of the prospective association between depressive symptoms and cardiovascular disease identified a positive relation in 10 of 13 studies (Musselman, Evans, & Nemeroff, 1998). The authors of this study concluded that it was inaccurate to view depression as a response to cardiovascular disease; rather it is an independent risk factor that may determine the health trajectories of those with the disease. A 22-year follow-up study showed that depressive symptoms place people at 60 - 150% greater risk of stroke (Jonas & Mussolino, 2000). Several other prospective studies and meta-analytic reviews have indicated that there is a 'dose-response' relationship between depressive symptoms and subsequent cardiovascular health outcomes such as mortality, cardiovascular disease, and stroke (e.g. Pratt, Ford, Crum, Armenian, Gallo, & Eaton, 1996; Glassman & Shapiro, 1998; Penninx et al., 2001).

In addition to depression, research on other negative emotions has demonstrated a clear link to adverse health outcomes. For example, hopelessness has been shown to predict coronary risk factors such as the progression of atherosclerosis and mortality from ischemic heart disease (Everson et al., 1996). A recent study has shown that hopelessness predicts subclinical atherosclerosis, over and above the effect of depression (Whipple et al., 2009). A broad-range of studies support the role of hostility, a trait indicative of a negative attitude towards others and closely linked to aggressiveness and anger, as related to a high rate of mortality (Boyle, Mortensen, Gronbaek, & Barefoot, 2008). Recent meta-

analytic prospective evidence shows hostility and anger to predict a greater likelihood of the incidence and worsening progression of coronary heart disease (Chida & Steptoe, 2009a). Finally, high levels of anxiety appear to increase the risk of sudden coronary death (Fleet & Beitman, 1998) and other negative outcomes such as angina and re-infarction by between 250 and 490 percent (Gallo & Mattews, 2003). It is thought that negative emotion may contribute to coagulation, inflammation, and numerous other processes including platelet activation (Reid, Seidelin, Kop, Irvine, Strauss, & Nolan, 2009; Steptoe, 2009) which can lead to atherosclerosis.

Negative emotion has been shown to play a role in numerous illnesses in addition to cardiovascular conditions. For instance, mortality rates for depressed cancer patients have been shown to exceed those of the non-depressed by 25% (Satin, Linden, & Phillips, 2009). Similarly, psychological distress among cancer patients has been shown to predict elevated rates of mortality and fatigue predicts high rates of cancer recurrence (Groenvold, Petersen, Idler, Bjorner, Fayers, & Mouridsen, 2007). Reducing depressive symptoms through medication may promote survival by leading to greater adherence to cancer therapies (Musselman et al., 2001). Thus, it appears that negative emotion may influence the incidence, severity, and trajectories of health problems.

A growing evidence base also suggests that positive emotion and well-being may have an effect on health. For example, a recent study followed over 6,000 people for 15 years and examined the one-sixth of participants who developed coronary heart disease. Those who scored high on a measure of 'emotional vitality' at baseline had a markedly reduced risk of cardiovascular disease at follow-up after adjusting for demographic and health-related variables (Kubzhansky & Thurston, 2007). A general meta-analytic review of research in the area examined 26 studies of healthy people and 28 studies of groups with existing illness and showed that those with a high level of positive affect at baseline

had lower rates of mortality, particularly in studies of healthy people (Chida & Steptoe, 2008). It appears that whilst it is difficult to separate the temporal sequence between the experience of emotion and patterns of autonomic and other psychphysiological activity, there are clear associations between emotional traits and health outcomes. Thus, it may be the case that people who have a chronic tendency to experience certain types of emotion may react differently to stressors both behaviourally and psychophysiologically and that this may explain the relations identified between emotion and health. To evaluate this proposition requires a consideration of the nature of emotion and its constituent parts.

1.3.3. Stress, emotion and psychophysiology

Whilst there is currently no consensus regarding the theoretical conceptualization of emotion (e.g. Russell, 2009), it is broadly accepted that several components make up emotions. These include affective, cognitive, behavioural, and physiological changes that ready the body for action. Emotions are considered to be embodied in that they are characterized, at least partially, by bodily states of activation (e.g. increased heart rate, sweating) and movement (e.g. facial expression) (Niedenthal, Barsalou, Winkielman, Krauth-Gruber, & Ric, 2005). People often use bodily metaphors to describe emotional states (e.g. butterflies in my stomach, my heart skipped a beat). This lay-intuition often refers to a degree of awareness of the relation between peripheral autonomic and somatic activation and emotion.

Although people may be rarely aware of such causal connections there is a rich theoretical background detailing the links between autonomic activity and emotional states. As far back as 1884, William James proposed that specific patterns of autonomic activity may actually lead to different emotional states. Cannon (1927) contended that the reverse was true, emotions may elicit different patterns of somatovisceral activity. However, research has failed to consistently identify specific psychophysiological

signatures of discrete emotions (Larsen, Berntson, Poehlmann, Ito, & Cacioppo, 2008), although some research does support this contention (Ekman, Levenson, & Friesen, 1983). The lack of evidence for autonomic substrates for discrete emotions has prompted an emphasis on a small number of dimensions such as positivity and negativity which appear to better relate to the psychophysiological substrates of emotion (Cacioppo, Gardner, & Berntson,1999).

Several contemporary emotion theories conceptualize discrete emotions as an "emergent constructions" rather than latent entities (Russell, 2009). Essentially this view proposes that core affective feelings (e.g. feeling good or bad or energized) are combined with numerous factors such as ones appraisals of current situational and relational information and past experience to produce the construction of a discrete emotion (e.g. fear, anger) (Schachter & Singer, 1962; Barrett, 2006). Attentional processes are proposed to modulate the extent to which core affect is foregrounded in conscious awareness (Barrett, Mesquita, Ochsner, & Gross, 2007). When core affect captures attention this is thought of as a bottom-up form of attention to affect, whereas when processing goals take precedence top-down attention can foreground core affect. Specific emotional contents are then derived from how core affect is combined with the construal of the psychological situation. Within this account of emotion, core affect is proposed to be constituted by neurophysiological states and reflected in autonomic activity.

Thus, it appears that there are multiple potential causal pathways between emotion and psychophysiology. However, the tendency to experience certain emotions is reflected in psychophysiological reactivity in stressful situations, potentially suggesting that emotional traits indicate the probable activation of emotional states and the associated biological changes such as autonomic activation. This activation may then produce health consequences. In line with this idea, feelings of negative affect do appear to be a good

marker for the development of subsequent health problems (Larsen et al., 2008). For example, research by Sheldon Cohen, Janice Kiecolt-Glaser and others have clearly demonstrated that negative emotions are linked to elevated susceptibility to infection, slow wound healing and poor responses to vaccines (e.g. Herbert & Cohen, 1993; Kiecolt-Glaser, Marucha, Malarkey, Mercado, & Glaser, 1995). Recent findings suggest that positive affect may play a protective role in attenuating psychophysiological reactivity and potentially reducing mortality (Chida & Steptoe, 2008). Because the relation between emotion and health outcomes is central to the reserve capacity model, support for the psychobiological and behavioural factors that are likely to underlie the emotion-health link are outlined briefly below.

1.3.4. Mediators between emotion and health

A key issue addressed in the reserve capacity model is the core mediators between emotional states and health outcomes. Perhaps the most obvious potential mediators are lifestyle factors. Happy people may make more healthy choices than the distressed. For example, a large scale survey of over 17,000 people has shown that those with high levels of life-satisfaction typically smoke less and exercise more than their less satisfied counterparts (Grant, Wardle, & Steptoe, 2009). There may be a bidirectional relationship between health behaviours and affect. Studies have shown that depressed people exercise more after their depression lifts and that exercise promoting interventions can increase positive mood (Steptoe, 2006). Depressed people are also more likely to smoke and there is evidence that smoking can lead to depression, particularly in adolescence (Wu & Anthony, 1999; Steuber & Danner, 2006). Depression may interfere with daily activities causing people to skip meals and reduce physical activity (Allgoewer, Wardle, & Steptoe, 2001). For ill people, symptoms of depression like low energy and sleep disturbances could directly affect adherence to medication regimes and detrimentally influence disease

management (Detweiler-Bedell, Friedman, Leventhal, Miller, & Leventhal, 2008). Thus, it appears that lifestyle factors, whilst likely to have a bidirectional association with emotion, may partially account for relations between emotion and health.

In addition, the biological correlates of emotion have been studied in large-scale longitudinal epidemiological studies, smaller scale naturalistic monitoring studies, and detailed laboratory experiments. Experimental research in this area has been largely composed of laboratory stress testing. Such experiments often involve asking participants to engage in difficult mental activities or emotionally demanding social interactions. These kinds of manipulations are usually successful in invoking a biological stress response. For instance, a meta-analytic review showed enhanced cortisol reactivity to a laboratory stress paradigm if stress was induced using tasks that required motivated performance involving a lack of control or social threat to the participant (Dickerson & Kemeny, 2004). This methodology can be used to identify if psychosocial characteristics such as trait levels of emotion, appear to moderate the link between stress exposure and physiological reactivity.

A recent large-scale meta-analysis of 729 studies showed that hostility, aggression, and Type-A behaviour were linked to increased heart rate or blood pressure reactivity whereas neuroticism, negative affect, and anxiety were associated with lower cardiovascular reactivity but poorer recovery (Chida & Steptoe, 2009a). These findings suggest that physiological reactivity may be a key mechanism underlying the prospective association between negative emotions and detrimental cardiovascular health outcomes. They also indicate that delayed cardiovascular recovery after stressors may be an important characteristic of neuroticism and negative affect, rather than exaggerated reactivity. Those scoring highly on measures of positive state and traits showed reduced reactivity of the hypothalamic-pituitary-adrenal axis to stress, particularly in studies rated

as methodologically strong, supporting prospective research linking positive emotion to better health. Experimental research also suggests that emotion may be linked to immune functioning. Those high in negative affectivity have been shown to be vulnerable to catching a cold after administration of the rhino virus (Cohen, Doyle, Skoner, Fireman, Swaltney, & Newson, 1995). Positive affect has also been linked to more effective immune function as indexed by the antibody response following the administration of the vaccine for hepatitis B (Marsland, Cohen, Rabin, & Manuck, 2006).

1.3.5. Summary of research supporting the reserve capacity model

Taken together, there is evidence that the reserve capacity or psychosocial resources may modify the extent to which stressful circumstances impact on both emotional and health outcomes. Research also suggests that the link between emotion and health proposed in the reserve capacity model is justified and that the suggestion that psychophysiological activation and behaviour changes may mediate this relationship appears to be well-founded. However, these mediating processes may also explain, at least in part, how health can affect emotion. The reserve capacity model fails to address issues of reverse causality which are likely to be essential in developing an extensive understanding of the determinants of well-being and health. A central aim of this thesis is to address this problematic area in the reserve capacity model by outlining a rationale for including reciprocal causality pathways between emotion and health and providing empirical support for this suggestion. In addition, in this thesis I aim to show how genetic factors may be a central moderator of the stress-emotion-health pathway.

1.4 Mind the gap: addressing issues of reverse causation and the role of genetic factors which are neglected in the reserve capacity model

Persistent health conditions can often impair a person's ability to function normally and may require adherence to long-term pharmaceutical regimes as well as wide-scale changes across several life-domains. These challenges of adaption that accompany illness can be conceptualized much like other stressors (Kim, Stewart, Kim, Yang, Shin, & Yoon, 2009) with their emotional effect contingent on one's resilience and the presence of other stressors. However, alongside the psychological pressures that characterize extended illnesses, there are often biological changes that result from the treatment or the pathology of the condition. Health conditions can affect the neuroendocrine, cardiovascular, and immune systems, which may in turn induce changes in mood (e.g. Tops, van Peer, Wijers, & Korf, 2006; Porges, 2007; Miller, Maletic, & Raison, 2009).

In particular, illness is often closely linked to the activation of inflammatory processes which can cause a series of neurological changes that induce 'sickness behaviour': a constellation of symptoms such as weakness, fatigue, sleep disturbances, changes in appetite, and an inability to concentrate (Dantzer, O'Connor, Freund, Johnson, Kelley, 2008). The mediators in this process are pro-inflammatory cytokines and include interleukin-6, interleukin-1β, and tumour necrosis factor-α. Although these cytokines are produced peripherally, they can access the brain causing profound changes in mood and motivation. The administration of interleukin-1β or tumour necrosis factor-α to rodents leads to the aforementioned sickness behaviour (e.g. withdrawal, fatigue, appetite changes, impaired congition), suggesting that inflammation plays a causal role in producing the neurovegetative symptoms of depression which resemble sickness behaviour (Miller et al., 2009).

Depression has been shown to develop in a substantial portion of patients (30 – 50%) treated with cytokines (Musselman et al., 2001; Capuron, Ravaud, Dantzer, 2000). For example, interferon-α administration for the treatment of cancer can induce depressive symptoms due to its effect in producing proinflammatory cytokines including interleukin-1β, and tumour necrosis factor-α (Capuron et al., 2001). The behavioural symptoms induced by cytokines are thought to be adaptive in the short term as they allow the organism to conserve energy, to heal, and to prevent further injury. However, in chronic conditions the persistent, and in cases, progressively worsening, psychological response to inflammation can be maladaptive. For example, in rheumatoid arthritis, pain and lethargy can prevent physical activity, which is necessary to improve the condition (de Jong et al., 2003). Pharmacologically blocking the production of proinflammatory cytokines in rheumatoid arthritis patients then leads to a large (0.5 SD) increase in well-being, vitality and functionality (Heiberg et al., 2005; Quinn et al., 2005).

Numerous other conditions like cardiovascular disease, diabetes and obesity, are also associated with raised levels of inflammation which could contribute to sickness behaviour and potentially depression. This yields the question as to how best to model the dual-roles of illness as a stressor that can prevent/impair other activities and illness as a producer of inflammation, within a framework that aims to encompass the pathways from stress to health outcomes. Raison, Capuron, and Miller (2006) propose that stress, past experience, genetic factors, and inflammatory processes combine to predict mental health outcomes. Thus, the adverse effects of illness on mood are proposed to occur when a person has had a prior history of stress (e.g. poverty, job loss) or psychiatric disorder or potentially weakening immune challenges (e.g. surgery, chemotherapy), and has substantial stress currently in their lives (e.g. interpersonal conflict), as well as genetic vulnerabilities to the mental health effects of stress (e.g. serotonin transporter gene) or towards an exaggerated inflammatory response (e.g. variation in cytokine genes).

I see illness as a stressor in its own right due to its disruption across numerous life-domains and the changes in expectation and outlook it can invoke. As an extension of the reserve capacity model illness could thus be conceptualized an end-point in the stress process that also feeds back into that process as the stress of illness can have emotional effects which may be modified by psychosocial factors and other resource. However, the inflammation that often characterizes illness acts on neurochemical processes implicated in adverse mental health outcomes such as depression, has synergistic effects when combined with other stressors and can sensitize neurochemical processes to future insults (Anisman, Hayley, Turrin, & Merali, 2002). The impact of illness on well-being may thus be contingent on the impact of the disturbing influence of illness on different life-domains combined with the presence of inflammation and both of these processes are likely to be modified by the presence of prior and current stressors as well as resources and genetic factors.

The later process of causation from illness to psychobiology to emotion, may not be limited to inflammation and there is evidence that both neuroendocrine and cardiovascular functioning may also affect emotion. For example, cortisol can intensify dopaminergic activity and induce feelings of energy (Tops et al., 2006). Low levels of cortisol have been found in a constellation of syndromes that are characterized by fatigue, such as burnout, atypical depression, seasonal depression, fibromyalgia, and posttraumatic stress syndrome (Fries, Hesse, Hellhammer, & Hellhammer, 2005). Diminished levels of cortisol may disinhibit inflammatory activity and in this way lead to 'sickness behaviour' as indexed by lethargy, sleep disturbance, and impaired cognition (Boksem & Tops, 2009).

Glucocorticoids were once hailed as "wonder drugs" and prescribed for the treatment of inflammatory disease throughout the early 1950's. However, enthusiasm

waned for this use of glucocorticoids when a "vitalisation" effect was observed whereby a large portion of patients had difficulty sleeping and many felt tense and irritable, whilst some displayed elevated feeling of well-being (von Zerssen, 1976). Thus, appropriate levels of cortisol appear to enhance the motivation and ability to expend energy and to produce subjective feelings of vigour whereas excessive levels may induce tension and reduced volumes fatigue. Illness and disturbances in the amplitude and patterning of circadian rhythms may therefore have emotional effects.

In addition to psychobiological factors, it is feasible that gene polymorphisms implicated in neuronal functioning may influence emotion (e.g. Kaufman et al., 2004). In particular, a broad literature has recently emerged showing support for the role of gene polymorphisms in moderating the effect of stress exposure on subsequent mental health (e.g. Caspi et al., 2003). This moderating effect is similar to the role proposed for psychosocial resources in the reserve capacity model and may underlie these effects or act synergistically with them.

1.5. Gene-Environment interactions as an extension to the reserve capacity model

A common understanding of the causes of psychopathology and the effects of psychological factors on physical health rests on two assumptions. Firstly, that people differ in their genetic constitution and secondly that people experience varying degrees of stress throughout their lives. We therefore expect that those with a familial link to psychiatric illnesses like major depression or who are prone to the psychosomatic components of illness (i.e. the components of physical illness caused by mental processes) will show poor health trajectories over time. Similarly, people who endure substantial degrees of stress due to low socioeconomic status or other adverse conditions are likely to endure detrimental mental and physical health outcomes.

Diathesis-stress models (Monroe & Simons, 1991) insist that it is necessary to examine both a person's genetic makeup and the stressors they have experienced in order to understand health outcomes. Such models typically recognize that familial history is often discordant with the experience of subsequent family members. In addition, many people cope well even when exposed to major undesirable stressors for prolonged periods. One solution that has been proposed to account for these observations is that the diathesis may interact with stress exposure. In this case, the capacity of an environmental demand to bring about risks to health may be modified by the genotype. It is also possible that a genetic vulnerability may lead to negative outcomes in the absence of noticeably exacerbated environmental risks. Thus, the diathesis and stress interact synergistically to produce effects that go beyond the contribution of either factor alone. In particular, a person who encounters many life stressors and possesses a genetic vulnerability is thought to be likely to develop mental and physical health problems.

Exciting advances in the measurement of variation in the human genome have advanced the study of gene-environment interactions beyond theoretical propositions to precisely empirically identifiable effects. Gene-environment interaction studies in psychiatry have primarily examined the role of identifiable genetic variation in modifying the effect of a physical or environmental stressor on the incidence, trajectories, or expression of psychiatric disorders (e.g. Kim-Cohen & Gold, 2009). For instance, variation in a functional allele in the catechol-O-methyltransferase gene moderated the effect of cannabis smoking on the incidence of psychosis in adults (Caspi et al., 2005). A small meta-analysis of existing studies showed that a functional allele in the promoter region of the monoamine oxidase A gene moderated the effect of childhood physical abuse on later mental health (Kim-Cohen et al., 2006). The serotonin transporter gene (5-HTTLPR) has also been shown to moderate the effect of life-stress on the development of

depression (e.g. Caspi et al., 2003), although recent meta-analyses have questioned the robustness of this observation (Munafo, Durrant, Lewis, & Flint, 2009; Risch et al., 2009).

Based on this and other gene-environment research in psychiatry and psychology (reviewed in Moffitt, Caspi, & Rutter, 2006; Rutter, Caspi, & Moffitt, 2006; Brown & Harris, 2008) it appears likely that variation in theoretically relevant genotypes may moderate the effect of stress on emotion. Thus, genetic variation can confer elevated risk or resilience that modifies the degree to which people adapt successfully to different environments. Additional research suggests that the relationship between risk alleles and adverse emotional outcomes, may be further moderated by psychosocial factors. Kaufman et al. (2004) showed that maltreated children with the short s/s serotonin transporter genotype (linked to diminished bioavailability of serotonin) had higher depression ratings than non-maltreated children or maltreated children with other genotypes. Importantly, this interaction was further moderated by the child's degree of social support. Maltreated children with little social support and the risk s/s genotype had particularly high depression scores.

One issue in drawing causal inferences from such studies is that a person's environment is often unlikely to be independent to their genotype. A risk gene may cause depression in parents who may then create a rearing environment that reflects and transmits depressive traits (e.g. social withdrawal, inhibition) to their children. This is known as a passive gene × environment correlation as it is independent of the action of the child (Moffitt et al., 2006). In addition, a risk gene may induce social withdrawal in the child which could cause bullying and depression (known as an active gene × environment correlation). Such correlations can make it difficult to determine when true environmental causation exists. One solution to this issue is to examine the effect of stressors which are extremely unlikely to correlate with any specific risk genotype. For instance, Kilpatrick et

al. (2007) examined Florida residents exposed to severe hurricanes in 2004 and showed that the low-expression short 5-HTTLPR genotype in combination with low social support modified the likelihood of post-traumatic stress disorder and major depression. This study suggested that environmental stressors which are not correlated with genetic factors can interact with both genes and psychosocial factors to determine emotional outcomes. The prospect that identifiable genetic factors can modify the effects of stressors and that psychological factors can potentially attenuate the influence of genetic risk represents a promising direction in multi-disciplinary research in social science and medicine. This research has been enabled by the sequencing of the human genome and the increased availability of measurement techniques for identifying variation in single nucleotide proteins and other aspects of the genome. However, often this kind of revolutionary technological advancement is not matched with the same level of sophistication in the assessment of relevant psychological variables, a fundamental issue with important implications for the development of this literature.

1.6. Advances in the measurement of experience in context

The study of the experiences of daily life has become a rapidly expanding area of investigation across the social sciences driven by a desire for accurate and ecologically valid measurement in research. For example, areas of economics are beginning to focus on micro-level experiences of utility as a method for evaluating population well-being and the effects of policy changes (Diener & Seligman, 2004). Within psychology, models of psychopathology often assume dynamic interactions between processes but have until recently relied on cross-sectional static research designs and neglected temporal change and the response to situational factors. New areas of research, such as embodied accounts of emotional experience and cognition have begun to demonstrate strong effects of context. In addition, experimental researchers across many sub-disciplines of psychology

are increasingly seeking to demonstrate that findings derived from laboratory studies generalize to the settings of daily life.

In health psychology there is a growing awareness that powerful techniques for real-time assessment have substantial potential for the evaluation of treatments, and for furthering the understanding of underlying disease mechanisms and their expression (Myin-Germeys, Oorschot, Collip, Lataster, Delespaul, & van Os, 2009). This is not least because the assessment of patient well-being, health and symptomology within a narrow time-frame appears to be less prone to bias than retrospective accounts over longer periods. For instance, traditional self-report measures that elicit responses about experiences like pain and fatigue over extended periods, such as the last month, tend to yield upwardly biased reports of the intensity of negative experiences. Such reports contrast to those assessed within a shorter time frame, such as how one felt today or is feeling right now, which are typically rated as less intense (Stone, Broderick, Schwartz, & Schwarz, 2008). People are thought to rate their experiences over long periods as negative as they think about salient, infrequent 'peak' negative experiences which bias responses towards more intense or severe rating options. Mildly pleasant or unpleasant experiences fade quickly from memory and do not appear to be appropriately weighted in people's estimations of the intensity of their experiences in the recent past.

In addition to better understanding the phenomenology of illness, the study of experiences in naturalistic settings is of interest to health psychologists who wish to study the psychological response to everyday events. In general, pain and other symptoms of illness are likely to be influenced by certain situations, activities, interactions, and emotions. For instance, Krueger and Stone (2008) conducted a population-based diary study which showed that pain may be exacerbated by certain activities such as garden

work or sports. Comprehensive data detailing phenomenological experience in daily life is required to capture such variability and person-situation interactions.

1.7. The measurement of emotional experience

Prior to recent innovations in techniques for assessing momentary experience emotion research focused largely on between-person differences in retrospectively assessed feelings. However, it became apparent that people are not capable of accurately recalling and appropriately weighting experiences to provide estimates of affective experiences over extended durations such as weeks or months. Recall and memory biases are common when people try to cast their minds back over such time horizons. Principally, striking, unusual or recent intense experiences are likely to be remembered and are thus over-weighted in retrospective emotional reports where their brief duration is neglected.

Kahneman and Riis (2005) propose that an evaluating self uses cognitive heuristics to temporally integrate affective experiences to produce retrospective global judgments of well-being over extended periods. Robinson and Clore (2002) infer that in the absence of experiential information, semantic content such as memory for non-experiential episodic detail and beliefs about ones emotions combine to produce retrospective evaluations. Thus rather than systematically attending to and overweighting certain emotional experiences, Robinson and Clore (2002) propose that emotions are often reconstructed inaccurately and in line with how one usually feels in the type of situation referred to.

The perspectives of Kahneman and Riis (2005) and Robinson and Clore (2002) point to different ways in which retrospective evaluations could lead to inaccurate accounts of experience. However, potentially because the ratings produced by the evaluative self rely on heuristics and semantic knowledge, these judgments are thought to be rather stable and resistant to transient influences. In contrast, the experiencing self is in

a constant state of flux and is reflected in reports of momentary affective experience which are influenced by a wide-array of intrapersonal and environmental factors. One benefit of making the distinction between the remembered and the experiencing self is that both are proposed to have different consequences.

Important life-choices are thought to follow largely from the influence of the evaluative self. For instance, it is possible that a couple may experience an equal quantity of negative emotion over the course of their relationship but yet make different decisions regarding their future based on the intensity and timing of their experiences. If one person experiences several 'peak' negative emotional experiences whereas the other experiences a consistent marginally raised level of negative emotion, the salient peak emotions may overly influence the judgment of the first person's evaluative self so that s/he requests a divorce. Life-satisfaction and eudaimonic ratings of the purpose, meaning, and freedom one has in life all fall under the rubric of judgments made by the evaluative self. The reports of the experiencing self represent momentary affective conditions and are proposed to be a better predictor of health outcomes than reports from the evaluative self. The literature demonstrating links between affect levels and psychobiological factors has produced some evidence that momentary strategies for the measurement of affect predict simultaneously assessed biological factors within the flow of daily life better than retrospective accounts over longer periods that could be attributed to the evaluative self.

Steptoe, Wardle, and Marmot (2005) showed that well-being measured via momentary assessment experience sampling techniques linked closely to psychobiological functioning as indexed by cortisol output. Well-being measured with the General Health Questionnaire showed no such relation. Similarly, a recent study showed that Beck Depression Inventory scores showed no relation to the heart rate variability of patients with suspected coronary artery disease. However, when depressed mood was assessed

with the Day Reconstruction Method (DRM; Kahneman et al., 2004) it showed clear relations with high and low frequency elements of heart rate variability independently of covariates (Bhattacharyya, Whitehead, Rakhit, & Steptoe, 2008). Thus, evidence is emerging in support of the idea that the experiencing self has potentially greater consequences for health than the evaluative self.

The DRM is an alternative to 'in the moment' assessments provided by experience sampling techniques, which ask participants to complete reports at various intervals throughout the day. Such experience sampling methods represent the 'gold standard' for real-time subjective measurement. However, experience sampling requires costly equipment and participant compensation, and interrupts normal activities thus producing difficulties in participant recruitment and compliance. The DRM elicits retrospective information referring to the relatively short time-frame of the previous day. Specifically, participants divide their day into significant events, or episodes, and recall the activities, interactions, and affective experiences during each episode.

This method is designed to prime the accessibility of participants' memories by evoking the characteristics of each self-defined episode. The results from the DRM appear to resemble those of experience sampling whilst retaining the benefit of taking a relatively short time to administer and of being exogenous to the period under investigation. The DRM has been proposed as an efficient method for assessing experienced well-being and thus could link simultaneously measured aspects of emotion and psychobiology which may predict health change as proposed in Kahneman and Riis (2005) and demonstrated in Bhattacharyya et al. (2008). Such fine-grained measurements could allow researchers to examine how trait level factors, such as psychosocial resources, may moderate associations between stress, emotion and psychobiology, within the context of a dynamic framework such as the reserve capacity model.

1.8. Aims of the present study

As outlined earlier, the reserve capacity model proposes that psychobiological factors are key mediators between emotion and health (e.g. Matthews, Gallo, &Taylor, in press). However, research within the reserve capacity framework has not as yet examined these linkages. The empirical studies in this thesis are structured as follows: I aim, firstly, in chapter 2 to analyze primary data collected from a day reconstruction study of 204 students to show that it is possible to specify a relationship between emotion and psychobiology using sophisticated measurement techniques for the assessment of emotional and biological functioning in the context of daily life. In addition, I aim to demonstrate that these relations between emotion and psychobiology can be isolated by controlling for potentially confounding behavioural, personality, and health-based factors. Next, in chapter 3 I use this day reconstruction dataset to further extend research within the reserve capacity model. Specifically, I test to see if psychosocial resources may influence psychobiological functioning through their relationship with emotion. These two studies aim to demonstrate how detailed ecologically valid measurement techniques can be used to identify links between reserve capacity, emotional, and psychobiological factors.

I have suggested that causal pathways from health to psychobiology to emotion are neglected in the reserve capacity model. In two studies I aim to test, firstly, in chapter 4 if psychobiological functioning may affect emotion once again using the day reconstruction student sample. Then, in chapter 5, I examine if such biological factors may mediate between health conditions and emotional states using secondary data from two national samples of adults, one from the United States (The National Health and Nutrition Examination Survey) and one from England (The English Longitudinal Study of Ageing). I then utilize secondary data from the Social Environment and Biomarkers of Ageing

Study in chapters 6 and 7 to empirically evaluate the idea that genetic factors merit inclusion in the reserve capacity model. Specifically, in chapter 6 I propose that genes can enhance the extent to which people are vulnerable or resilient to the effects of stressful circumstances. Further, in chapter 7, I hypothesize that intrapersonal resources could modify the effect of the genetically determined vulnerability/resilience to stress.

In the General Discussion (Chapter 8) the main findings of the empirical chapters are considered collectively within the context of the reserve capacity model and contemporary research linking stress to health. In addition, the discussion focuses on the value of addressing three problematic areas within the reserve capacity model: (i) the testing of reverse causality feedback loops, (ii) the inclusion of a role of genetic factors, and (iii) the use of advanced assessment technologies for the measurement of experience and functioning in context. This chapter ends with a template of how best to model and empirically test the multi-component dynamic relation between stress and health and the key implications this research may have for basic and applied research.

Chapter 2

Naturalistic monitoring of the affect-heart rate relationship: A day reconstruction study

Chapter Aim

The current study used the day reconstruction method to examine the relation between emotion and psychophysiology, as indexed by heart rate. The day reconstruction survey requests participants to retrospectively assess the activities, interactions, and feelings of the previous day. Rather than requiring participants to scan their memory to report on extended time horizons of weeks or months, this approach aims to avoid memory biases by focusing on recent events and using memory priming techniques. Thus, if emotions are predictive of psychophysiological activity as suggested in the reserve capacity model, techniques like day reconstruction or experience sampling may be more likely to identify such associations than broad global measures. Further, this chapter aimed to show that it is possible to identify the specific relation between emotion and heart rate after adjusting for other potential mediators or modifiers of the emotion-psychobiology link such as behaviour, personality, measures of health and so forth. This chapter thus performs the dual-functions of testing a specific component of the reserve capacity model and showcasing recently developed techniques that can test components of the model and provide a promising avenue for future research.

Chapter Summary

Prospective studies have linked negative affect with hypertension, cardiovascular disease, and mortality. This study aims to identify if cardiovascular activity in day-to-day settings is related to affect levels as assessed using the Day Reconstruction Method (Kahneman et al., 2004). 186 people underwent baseline physiological testing and were monitored naturalistically for an entire day. Multilevel models were the principal analyses used. I utilized an online day reconstruction survey to produce a continuous account of affect, social interactions, and activity patterns during waking hours. Ambulatory heart rate (HR) was assessed during the same period. Personality, health behaviour, consumption, self-reported activity, and baseline physiological characteristics were assessed to isolate the relationships between affect and HR. Negative affect predicted an elevated ambulatory HR and tiredness predicted a lower HR. Associations between negative affectivity and increased cardiovascular activity were maintained after taking account of baseline physiological factors, health behaviour, and personality. Negative affect in everyday life is a reliable predictor of HR. Combining day reconstruction with psychophysiological and environmental monitoring is a minimally invasive method with promising interdisciplinary relevance.

2.1. Introduction

Longitudinal surveys have demonstrated that trait neuroticism and subjective distress levels increase the risk of the occurrence of stroke, cardiovascular disease, hypertension and mortality, independent of medical risk factors (Ockenfels, Porter, Smyth, Kirschbaum, Hellhammer, Stone, 1995; Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999; Hemingway et al., 2003). The cardiovascular response to life-events is one likely mechanism through which state and trait factors related to affect may modulate the effect of stressors on a range of cardiovascular health outcomes (Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005; Kulkarni, O'Farrell, Erasi, & Kochar, 1998). There is substantial experimental evidence that negative affect induced in laboratory settings can evoke adverse changes in physiological functioning such as decreased production of immunoglobulin A and increased cortisol secretion and cardiovasular activity (Tsuboi, Hamer, Tanaka, Takagi, Kinae, & Steptoe, 2008; Buchanan, al'Absi, & Lovallo, 1999; Kibler & Ma, 2004).

Previous research has also linked cardiovascular activity to fluctuations in affect in naturalistic settings (Shapiro, Jamner, Goldstein, & Delfino, 2001; Jacob et al., 1999; Johnston, Tuomisto, & Patching, 2008). However, the relationships observed typically do not separate the contribution of affect and personality or systematically specify the role of situational, consumption related, and stable biological characteristics. Carefully delineating the relative contribution of this set of factors will allow a clear specification of the role of affect and personality in cardiovascular activity outside of the laboratory. In particular, the recent literature has identified heart rate (HR) as a measure that may provide critical insight into cardiovascular activity in the field and potentially act as a prospective marker for both physical and mental health outcomes.

2.1.1. Heart rate as a metric of cardiac activity

There are numerous measures of cardiac activity (e.g. standard deviation of inter-beat intervals, mean of successive differences in inter-beat intervals), of which the most commonly endorsed have been found to correlate strongly with HR (r=-.56 to -.98) (Allen, Chambers, & Towers, 2007). HR is a highly responsive measure of autonomic nervous system functioning, reflecting the dual activation of the sympathetic and parasympathetic branches (Kohlish & Schaefer, 1996). Increases in HR are considered to derive from a pattern of increased sympathetic and decreased parasympathetic system activation, whereas HR decreases result from the inverse pattern (Ottaviani, Shapiro, Davydov, & Goldstein, 2008).

HR is also sensitive to both environmental and emotional changes (Carrillo, Moya-Albiol, Gonzalez-Bono, Salvador Ricarte, & Gomez-Amor, 2001). Large-scale prospective epidemiological studies have identified high HR as a risk factor for health outcomes such as increased cardiovascular mortality, morbidity, and myocardial infarction (Gillum, Makuc, & Feldman, 1991; Kannel, Kannel, Paffenbarger, & Cupples, 1987; Hsia et al., 2009) as well as acting as a marker for the development of hypertension in young people (Palatini et al., 2006; Selby, Friedman, & Quesenberry, 1990). Examining cardiovascular activity in naturalistic settings is of critical importance in order to identify the mechanisms through which cardiovascular activity may have cumulative effects which are protective or detrimental to cardiovascular health (Michaud, Matheson, Kelly, & Anisman, 2008).

For instance, excessive environmental demands such as chronic work stress have been shown to relate to higher HR both in and directly after work (Belkic, Landsbergis, Schnall, & Baker, 2004; Ritvanen, Louhevaara, Helin, Väisänen, & Hänninen, 2006). Momentary experiences of positive affect have been shown to attenuate ambulatory cardiovascular reactivity (Steptoe et al., 2005). Negative affect has been associated with a

rise in HR and fatigue and disengagement with a drop in HR (Kamarck, Schwartz, Shiffman, Muldoon, Sutton-Tyrrell, & Janicki, 2005; Shapiro et al., 2001**).**

Naturalistic monitoring paradigms are particularly useful considering the current lack of clarity as to whether stress reactivity in the laboratory is associated with diminished psychosocial resources or with increased resources, health and the ability to adapt dynamically to challenge**.** For instance, those with higher self-rated health have been shown to have a strong cortisol response to acute stress (Kristenson, Olsson & Kucinskiene, 2005), and depressed people have demonstrated less cardiac reactivity during stressful tasks than controls (Carroll, Phillips, Hunt, & Der, 2007**;** Salomon, Clift, Karlsdottir, & Rottenberg, 2009). The association observed between cardiovascular reactivity to acute laboratory stressors and cardiovascular activity in real life is often weak or inconsistent (Turner, Ward, Gellman, Johnston, Light & van Doornen, 1994). It may be the case that high negative affectivity is indicative of a tendency towards subtle but chronic patterns of raised heart rate in response to everyday events and an attenuated response to more exaggerated stressors such as those encountered in laboratory studies.

However, the relationship between affect and HR in ecologically valid continuous monitoring studies remains unclear, with some studies finding no relationship (Serrano, Moya-Albiol, & Salvador, 2008), and others finding correspondence in cases controlling for demographic and biological variables but not psychological factors such as personality (Carpeggiani et al., 2005). In several studies the assessment of affect has also been restricted, for instance through the use of measures requiring participants to endorse a single point on a circular dimension (circumplex) indicative of affective space (Jacob et al., 1999). Whereas in other cases the assessment of affect has been of sufficient detail to infer intensity and allow for mixed mood states, but the high-frequency of the diary assessments

(e.g. 40-50 per day) has involved a level of burden that would be likely to interfere with the flow of daily activities (e.g. Shapiro et al., 2001).

Other characteristics of previous studies such as choosing stressful days for ambulatory monitoring, or the use of heavy cardiovascular monitors or invasive blood pressure cuffs may diminish the ecological validity of the experience assessed (Johnston et al., 2008). In the current study, an explicit attempt was made to extend previous research by integrating minimally invasive cardiovascular monitoring and by utilizing a recently developed method for assessing experience in daily life that is exogenous to the assessment period in question.

2.1.2. The measurement of experience in naturalistic settings

Detailed accounts of everyday life have been generated by time sampling diaries, experience sampling, and ecological momentary assessment (Hektner, Schmidt, & Csikszentmihalyi, 2007). Such ambulatory psychological assessments have demonstrated their ecological validity but can be labour intensive for participants and expensive for researchers (Fahrenberg, Myrtek, Pawlik, & Perrez, 2007). The challenge of creating multi-method accounts of behaviour and experience in normal life settings involves adapting and integrating existing methods to produce measures which are non-invasive and minimally demanding (Bolger, Davis & Rafaeli, 2003).

Recently, researchers have begun to investigate the potential of retrospective alternatives to momentary assessment which are designed specifically to minimize erroneous reporting (Stone, Schwarz, Schkade, Schwartz, Krueger, & Kahneman, 2006). A key development in this literature is the Day Reconstruction Method (DRM), a survey which is structured to provide accurate and detailed retrieval of the experiences and objective circumstances of the previous day (Kahneman et al., 2004). The DRM elicits quantitative information about the frequency and timing of daily activities, social

interactions, and associated multidimensional affective reports which have been shown to satisfactorily approximate the results of ecological momentary assessment.

2.1.3. The present investigation

I sought to integrate methodological and technological innovations in naturalistic assessment to non-invasively examine the relationship between affect and HR. I utilized advancements in computer-aided survey design that exploit visualisation and memory priming techniques to minimize recall bias in the DRM. Findings from laboratory and epidemiological studies suggest that positive affect buffers the negative health effects of psychosocial stress and that negative affect increases susceptibility to such ill-effects (Pressman & Cohen, 2005; Smyth, Ockenfels, Porter, Kirschbaum, Hellhammer, & Stone, 1998; Ryff et al., 2006; Steptoe, O'Donnell, Badrick, Kumari, & Marmot, 2008).

Computer-assisted day reconstruction and both baseline and ambulatory biological measurements were used to examine how the intensity of positive and negative affect and tiredness as well as a range of physiological, behavioural and environmental factors relate to HR. I tested (i) the hypothesis that cardiovascular activity is associated with affect intensity in everyday settings and that this relationship is distinct from the role of personality, and (ii) that an association between affect and HR would remain having adjusted for a range of factors in addition to personality including activity engaged in, consumption, social interaction, time-of-day, and extraneous physiological and environmental factors. Specifically, I expected negative affect to increase HR, and positive affect, tiredness and the personality factor emotional stability to be linked to a lowered HR. To clarify the rationale for inclusion of the control variables in the second stage of the analysis: we recognise it is possible that the control variables (e.g. social interaction, consumption) may influence heart rate by determining patterns of affect. However, if this were the case then in the multilevel analysis when we adjust for the link between (i) the

control variables and affect and (ii) the link between affect and heart rate, any effect of the control variables that remains can be considered not to be mediated by affect. Thus, for instance, adjusting for situational factors (e.g. activity, social interaction) will show the link between these factors and heart rate that is not mediated by affect and the possibility remains that the potential relation between affect and heart rate may be determined by a range of variables including situational factors.

2.2. Method

2.2.1. Participants

Data were collected from 204 university students who volunteered to enroll in a diary study with medical testing and biological tracking. The students were compensated for taking part with either research credits towards their freshman psychology course or a cash incentive of €25. The drop-out rate was 3% (6 participants), which left a total of 198 participants in the study. An additional 12 students were eliminated from the analyses due to excessive artifactual measurement error identified during the analysis of their HR data (e.g. loss of signal, excessive number of outlier measurements). Of the 186 participants, 64 were men and 122 women aged 18 - 49 years (mean = 23.3, SD = 6.1), as shown in Table 2. Every participant received information relating what the study entailed and gave informed consent.

2.2.2. Procedure

One hundred and eighty six subjects with usable data took part in the study on all three consecutive days. Participants underwent a medical assessment on the first day during which a series of physiological parameters were examined by trained research nurses. During this consultation detailed written and verbal instruction on the operational procedures for physiological monitoring were provided to the participants (see Appendix

A). The next day, the students were fitted with ambulatory instruments to monitor HR throughout their normal day. On the third day of the study, participants completed an on-line questionnaire which included demographic information, psychometric measures and the computer-assisted DRM.

2.2.3. Measures

2.2.3.1. Computer-assisted online day reconstruction

The online version of the DRM included a time-diary of events followed by an assessment of objective details and affective experiences relating to the previous day. To reduce recall bias the survey follows a fixed format where participants initially separate the day into morning, afternoon, and evening stages based primarily on meal-times and subsequently break each stage down into a series of 'episodes'. Episodes are restricted to a time-period of between 20 minutes and 2 hours and are demarcated at the participants discretion based on any significant change (e.g. change of place, activity, mood, or the presence of others). The online DRM generates a 'flow chart' representation of the participant's day from diary responses to assist in the completion of items referring to specific events.

Participants then provide episode-by-episode information about the location, activities, interactions, and the subjective experiences associated with each episode as assessed by a series of 11 affect scales. The 11 items included in the affective assessment were parsed into the dimensions of positive affect (happy, calm, comfortable, affectionate, interested, confident), negative affect (impatient, depressed, stressed, irritated) and tiredness, a tripartite conceptualisation of affect that has been shown to satisfactorily represent the structure of diurnal patterns of affect (Stone et al., 2006). Participants were asked to what extent they felt a given emotion using response scales ranging from 0 (not at all) to 6 (very much). The adjectives are replicated from previous DRM research with

minor adjustment and are broadly similar to those used in other mood scales such as the Positive and Negative Affectivity Schedule or the Profile of Mood States (Krueger & Schkade, 2008).

2.2.3.2. Ambulatory heart rate monitoring

Heart rate was assessed using the Suunto Memory Belt. This is a lightweight (61g) heartbeat interval recorder and is worn around the chest and has a capacity to record 200,000 consecutive beat-to-beat intervals (Suunto memory belt, Suunto Oy, Vantaa, Finland). A comparison of recordings assessed from one of the researchers (MD) both as assessed by the Suunto device and HR information simultaneously captured from the 1000Hz 3-lead ECG BIOPAC MP35 data acquisition unit with BSL PRO software (Biopac Systems, Santa Barbara, CA) found no substantial difference between the heartbeat intervals recorded by both systems ($t(2206) = .34$, $p = .74$; $r = .995$, $p < .001$).

Briefly, accurate analysis of HR requires that every heartbeat is recorded and stored and that data are sampled with at least 1ms accuracy. Electrocardiogram (ECG) recording provides this level of precision and information on inner heartbeat dynamics (e.g. QT interval, P-wave duration). Where high resolution inner heartbeat information is not required, light-weight wearable heartbeat interval (or R-R) recorders have facilitated simple non-intrusive high frequency and accurate collection of HR data (Buchheit, Simon, Viola, Doutreleau, Piquard, & Gabrielle, 2004; Serrano et al., 2008). Agreement analyses for commercially available heartbeat interval recorders and traditional ECG recorders typically find a high degree of concordance between both types of systems across a range of samples and metrics of cardiac chronotropy (Gamelin, Berthoin, & Bosquet, 2006; Heilman & Porges, 2007; Nunan, Jakovljevic, Donovan, Hodges, Sandercock, & Brodie, 2008).

Participants received instructions from the research nurses on how to apply electrode gel to the heartbeat recorder and the optimal method of wearing and operating the device (see Appendix A). In accordance with previous research outliers and artifactual readings were removed from HR recordings preceding analysis (Jacob et al., 1999). Acceptable HR measurements were defined as those within the range 40 to 150 bpm (Shapiro et al., 2001). Outlier measurements accounted for less than 1% of the data. Ambulatory HR data was then fragmented into a series of ten minute segment averages for each individual. In order to accurately estimate the relationship between HR and affect, the episodes recorded in the DRM were matched to HR measurements by the start and mid-point of the episode duration. Thus, for each episode the level of HR utilized for analysis represented a combination of a ten minute average at the start of an episode and the ten-minute segment surrounding the half-way point of the self-reported episode.

2.2.3.3. Personality

A short-form measure of the Big Five domains of personality, the Ten Item Personality Inventory, was used to assess extraversion, agreeableness, conscientiousness, openness and emotional stability (Gosling, Rentfrow, & Swann, 2003). The factors that emerge from this measure have been shown to converge closely with those of widely-used Big Five measures (r = .65 - .87). Each factor is the sum of the scores on two of ten items, where each item is rated on a 7-point scale ranging from 1 (disagree strongly) to 7 (agree strongly).

2.2.3.4 Physiological parameters and consumption measures

As part of the medical assessment Body Mass Index (BMI), was calculated from height (m) and weight (kg), as measured by the research nurses using a Leicester portable stadiometer (Invicta Plastics Ltd, Leicester, United Kingdom) and Salter scales (Salter Weigh-Tronix, West Bromwich, United Kingdom). Lung capacity was assessed using the

Mini-Wright digital peak flow meter (Clement Clarke International Ltd, Harlow, UK), and percentage body fat was obtained using the validated Omron BF-306 body fat analyzer (Omron Corp., Kyoto, Japan) (Deurenberg & Deurenberg, 2002). The questionnaire component of the study also included questions related to current consumption of substances which may influence HR. Participants rated the number of alcoholic drinks they consumed on the reconstructed day, how many cigarettes they smoke per day, their exercise frequency, and whether or not they were currently on a diet.

2.2.4. Data analyses

The real-time nature of the HR and affect data in this study, particularly the associated uneven number of repeated assessments and autocorrelation amongst repeated measures, make it less amenable to traditional repeated measures analysis (Stone et al., 2006). Multilevel models have the advantage of allowing simultaneous estimation of between-person and within-person effects and can analyse multiple predictors in cases where there is an unbalanced number of cases per person (Reis & Gable, 2000). Multivariate multilevel random coefficient modelling considers two random components of HR data: one due to the sampling of participants and a second related to repeated samples within persons.

Multilevel modelling was therefore used to answer most of the study questions. First, each person's HR levels were predicted by the time of day of each episode reported. As change in heart rate over the day may not be linear (Degaute, van de Borne, Linkowski, & Van Cauter, 1991), a series of curvilinear models were examined. The best fit was achieved by the linear time of day term with neither the second degree polynomial function (including the linear and quadratic time of day terms) nor the third degree polynomial (adding the cubic time of day term to the second degree model) producing a significantly improved fit.

Following this, the influence of the within-person affect levels on HR was estimated. To do this, linear time of day was added at Level 1 and personality was added at Level 2. A simplified representation of the Level 1 and Level 2 model is detailed below (where *i* represents the individual, and *j* represents the repeated-measures instances, or episodes):

$$\text{Level 1: } HR_{ij} = \beta_{0i} + \beta_1 \times \text{Time}_{ij} + \beta_2 \times \text{Positive Affect}_{ij} + \beta_3 \times \text{Negative Affect}_{ij} + \beta_4 \times \text{Tiredness}_{ij} + e_{ij}$$

$$\text{Level 2: } \beta_{0i} = \gamma_{00} + \gamma_{01} \times \text{Personality}_i + r_{0i}$$

Next, to examine the robustness of the relationship between HR and affect I added location, social interaction, and self-reported activity ratings into the model at Level 1. The effects of stable individual differences factors were added to Level 2. These included demographic, consumption and baseline physiological factors, modelled at Level 2. The simplified model presented below was estimated using data from 1,720 episodes with usable data reported by the 186 participants.

$$\text{Level 1: } HR_{ij} = \beta_{0i} + \beta_1 \times \text{Time}_{ij} + \beta_2 \times \text{Positive Affect}_{ij} + \beta_3 \times \text{Negative Affect}_{ij} + \beta_4 \times \text{Tiredness}_{ij} + \beta_5 \times \text{Social interaction}_{ij} + \beta_6 \times \text{Location}_{ij} + \beta_7 \times \text{Activities}_{ij} + e_{ij}$$

$$\text{Level 2: } \beta_{0i} = \gamma_{00} + \gamma_{01} \times \text{Personality}_i + \gamma_{02} \times \text{Demographic}_i + \gamma_{03} \times \text{Health}_i + \gamma_{04} \times \text{Health Behaviour}_i + r_{0i}$$

2.3. Results

2.3.1. Descriptive analyses

2.3.1.1. Preliminary analyses

The mean length of episodes reported on by participants was 73.5 minutes (SD = 55). There were no problems with the functioning of the online DRM and feedback indicated that the modal appraisal of the survey was as 'interesting' (assessed on a scale ranging from 1 = very interesting to 5 = very uninteresting). The characteristics of the participants on the primary demographic, personality, health behaviours and baseline health variables are detailed in Table 2.

2.3.1.2. Behavioural and situational variables

The activities most commonly endorsed by the participants were conversing, eating, commuting and college work. In total these behaviours accounting for 51.4% of reported activities. In approximately 46% of episodes participants were alone and when they were with others they were most likely to be with more than one person (52.7%). The majority of episodes were reported to occur at either at home (46.6%) or in college (26.9%).

2.3.1.3. Heart rate data

The average ambulatory HR of participants as sampled from ten minute blocks at each day reconstruction episode start and mid-point was 85.2 (SD = 15.6), significantly higher than the average resting HR as assessed during the baseline medical assessment (M = 74.3, SD = 11.9), $t(185) = 14.8$, $p < .001$. Heart rate sampled from the episode was correlated with resting HR ($r = 0.57$, $p < .001$) and strongly correlated with ambulatory data for the entire day ($r = 0.93$, $p < .001$).

2.3.1.4. Descriptive examination of levels of affect and heart rate

Positive affect scores were substantially higher on average across adjective components (M = 3.56, SD = 1.1, alpha = .85) than the negative affect scores (M = 1.39, SD = 1.16, alpha = .76), (t (185) = 22.3, p < .001). Tiredness was measured by a single item and responses were anchored around the midpoint of the 0 - 6 scale (M = 2.95, SD = 1.83). The diurnal pattern of standardized aggregated HR and positive and negative affect variables as shown in Figure 2.

Table 2

Descriptive statistics for demographic characteristics, personality, health, and health behaviours of participants

	Mean / **%**	SD	Min	Max
Age	23.4	6.3	18	49
Gender (% female)	66%			
Conscientiousness	9.5	2.8	2	14
Openness	10.93	2.2	5	14
Agreeableness	9.9	2.1	2	14
Emotional Stability	9.0	2.9	2	14
Extraversion	9.49	2.7	3	14
Resting Pulse (bpm)	74.3	11.9	43	115
Body Fat (%)	28.8	8.6	10.9	47.3
BMI (kg/m2)	23.1	3.6	13.5	43.2
Peak flow (liters/minute)	406.5	116.1	178.3	771.7
Dieting (%)	10.1%			
Drank alcohol* (%)	27.3%			
Smoke (%)	12.8%			

* Drank alcohol on the day of the study.

The positive affect composite variable rises notably over the course of the day. Negative emotions demonstrate a decline throughout the day and also are characterised by a bimodal pattern spiking in between ten and eleven am and to a greater extent again at approximately three to five pm. Patterns of HR also indicate a decline from morning to evening and standardized levels appear to correspond with those previously identified in controlled environments where HR has been shown to peak in the early morning, decline to a 3pm afternoon nadir, then peak again between 6 and 8pm and decline sharply later in the evening (Degaute et al., 1991).

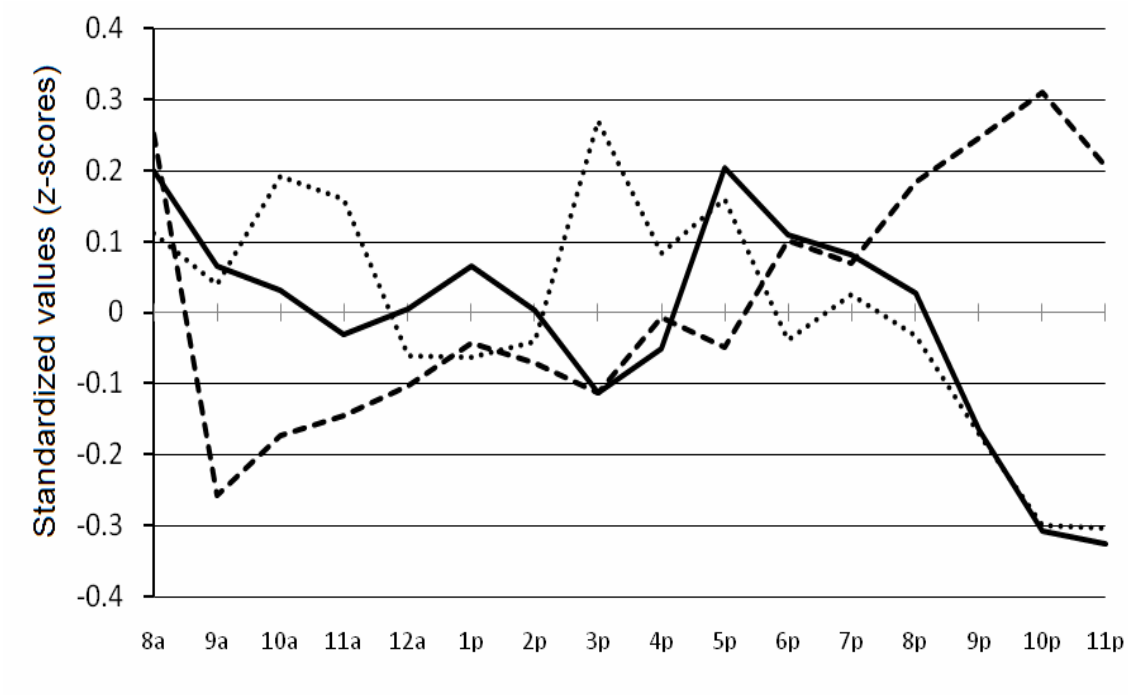


Figure 2. The trend in standardized state positive affect (broken line), negative affect (dotted line), and HR (solid line) as a function of time of day.

2.3.2. Affect and heart rate analysis

Multilevel modelling was used to estimate a series of models predicting HR at the episode mid-point using affect, personality traits and potentially confounding variables as predictors. Firstly, the best fitting unconditional model for HR was specified, and HR was found to have a significant random intercept ($p < .001$) and autoregressive covariance

structure ($p < .001$). This indicated that there was substantial variation in HR levels over the course of the day and that measures co-occurring closely in time demonstrated greater concordance than more temporally distant measures.

Next, I estimated a baseline model of the association between time of day, affect, personality and heart rate without adjusting for potential confounding variables. Time of day failed to significantly predict heart rate. Daily patterns of negative affect at the episode level were significantly related to elevated HR, $b = 0.3$, $SE = 0.09$, $t\ (1673) = 3.16$, $p < .005$, controlling for closely related factors such as tiredness and emotional stability. Average negative affect levels at the episode level were, however, unrelated to heart rate at rest as assessed during medical testing, $r = .04$, $p = .62$. Tiredness at the episode level was associated with a substantial decrease in HR, $b = -1.17$, $SE = 0.22$, $t\ (1674) = -5.25$, $p < .001$. The personality trait emotional stability was related to a low HR, $b = -0.65$, $SE = 0.27$, $t\ (181) = -2.39$, $p < .05$, whereas agreeableness predicted a raised HR, $b = 1.1$, $SE = 0.37$, $t\ (181) = 2.99$, $p < .01$.

I also conducted an analysis which utilized affect levels which were weighted by the episode duration. Ninety-nine percent of episodes were five hours or under and episodes with a greater duration were excluded from the analysis. The average length of the episodes which were included in the analyses was 1.23 hours (SD = .92). The product of the average affect per episode and the episode duration was log transformed to form a duration weighted positive (M = 1.28, SD = .37), negative (M = .69, SD = .49), and tiredness (M = .43, SD = .42) variables. Following this, heart rate was predicted as a function of hour of day, personality, and duration weighted affect. This showed that duration weighted negative affect predicted a raised heart rate, $b = 0.32$, $SE = 1.06$, $t\ (1215) = 2.96$, $p < .005$, and duration weighted tiredness predicted a low heart rate, $b = -6.27$, $SE = 1.54$, $t\ (1309) = -4.07$, $p < .005$.

2.3.3. Affect and heart rate adjusting for confounders

To address the robustness of the link between affect, personality and HR identified in the initial analysis I designed a multilevel model that considered a series of behavioural, demographic and health related variables. Heart rate was predicted on the basis of time of day, affect in an episode (positive, negative, tired), adjusting for simultaneously estimated effects of behavioural factors (nature of activity engaged in, social interactions), location (in college, at work, in a car), and time of day at Level 1 and demographic factors (age, gender), the big five personality traits, physiological (BMI, lung capacity, peak flow) and consumption variables (smoking, alcohol intake, dieting) at Level 2.

Having adjusted for the wide array of variables employed, negative affect measured at the episode level was found to predict a raised HR, $b = 0.2$, $SE = 0.1$, $t\ (1690) = 1.97$, $p < .05$. The association between tiredness at the episode level and lowered HR remained highly significant, $b = -0.76$, $SE = 0.23$, $t\ (1635) = -3.39$, $p < .01$, as shown in Table 3. The duration weighted affect analysis showed similar results with negative affect predicting an elevated HR, $b = 2.42$, $SE = 1.14$, $t\ (1128) = 2.12$, $p < .05$, and tiredness predicting a low HR, $b = -4.04$, $SE = 1.57$, $t\ (1269) = -2.58$, $p < .05$. The relationships identified were found to be robust to the inclusion of potentially confounding behavioural, environmental, physiological and psychological variables.

2.3.4. Within-person contextual level 1 variables and heart rate

Categorical effects of activity were analysed using 'relaxing' as the base category for comparison in the model. As detailed in Table 3, HR was found to be significantly higher during activities involving physical exertion such as exercising, walking, playing, and commuting, independent of time of day effects. Resting, attending lectures and to a lesser extent college work was associated with a decline in heart rate. In a small number of cases increases in cardiovascular activity were expected but not identified (e.g. caring for

children, making love). It is likely this was due to low power as each of such behaviours accounted for less than 1% of reported activities. These findings confirm that the recalled times when active behaviours were indicated to occur in the DRM survey aligned to objectively assessed changes in HR. To evaluate location specific effects on HR, being at home was used as the situational base category for the analysis. HR was raised in college, $b = 2.03$, $SE = .91$, $t\ (1577) = 2.24$, $p < .05$, at work $b = 8.53$, $SE = 3.22$, $t\ (1229) = 2.64$, $p < .01$, and during times where location was labelled as 'other', $b = 3.37$, $SE = .99$, $t\ (1461) = 3.38$, $p < .01$.

2.3.5. Between-person level 2 variables and heart rate

Age was unrelated to HR as were the health markers lung capacity, body fat, and body mass index. Agreeableness predicted a raised heart rate, $b = .76$, $SE = .36$, $t = 2.08$, $p < .05$, and emotional Stability was no longer significantly predictive of heart rate. Men had a lower HR over the course of the day, $b = -4.72$, $SE = 2.38$, $t = -1.99$, $p < .05$. Alcohol consumption and dieting were unrelated to HR whereas smoking predicted an elevated HR, $b = 2.62$, $SE = .99$, $t = 2.64$, $p < .01$.

2.4. Discussion

The primary aim of this study was to examine the correspondence of affect and heart rate within the context of a normal week day. Substantial concordance was evident between measures of affect and patterns of HR. Increased levels of psychological distress at the episode level, as indexed by a composite variable of depression, irritation, stress, and impatience scores were indicative of a high HR after adjusting for personality. Negative affect remained as a significant predictor of HR after a substantial number of potentially confounding within and between person variables (e.g. location, activity, social situation, physiological factors, time of day, consumption) were entered into the model.

Table 3

Results of multilevel model assessing the relationship between heart rate and affect, adjusting for location, activity patterns, demographic factors, personality, baseline health, and health behaviours

	B	*SE*		Beta	SE
Level 1 Variables			Preparing food	.25	2.63
Intercept	80.54**	8.59	Reading	-1.45	2.09
Time	-.11	.07	Rest/Sleep	-8.11**	2.57
Affect Variables			Shopping	3.46	2.42
Positive affect	.07	.07	Caring for children	-.46	4.25
Negative affect	.20*	.10	Conversation	-1.19	1.57
Tired	-.76**	.23	Walking	5.44*	2.41
Social interaction	1.10	.72	Television	-2.44	2.01
Location[a]			Paid work	-4.54	3.29
In college	2.03*	.91	Lectures	-4.05*	2.03
Work	8.53**	3.22	College work	-2.76†	1.62
Car	-3.52	2.32	Level 2 Variables		
Other location	3.37**	.99	Conscientiousness	-.20	.29
Activities[b]			Openness	.35	.34
Commute	4.86**	1.82	Agreeableness	.76*	.36
Housework	1.71	2.39	Emotional Stability	-.34	.28
Eating	-.21	1.56	Extraversion	-.10	.29
Exercising	11.50**	2.55	Age	-.21	.13
Grooming	1.89	2.01	Male	-4.72*	2.38
Home Computer	-1.44	2.15	BMI	.23	.24
Music	4.99	3.03	Body fat	.05	.13
Radio/News	-4.95	7.08	Lung capacity	-.004	.007
Making Love	-1.53	4.46	Smoke	2.62**	.99
Playing	7.63*	3.47	Alcohol	-1.11	.74
Praying	.15	5.79	Diet	-3.96	2.55

[a] 'Home' is base category for location analysis, [b] 'Relaxing' is base category for activity analysis. ** p<0.01, * p<0.05, † p<0.1.

Unlike ambulatory HR, resting HR was unrelated to negative affect in daily life. This discrepancy could indicate that a heightened cardiovascular activity linked to the stressors of everyday life may be a central way that affect-mediated dysfunctional autonomic control is expressed. Specifically, experiencing high levels of negative affect as part of a specific episode may lead to increased sympathetic activity and vagal withdrawal in reaction to stressors leading to a raised ambulatory HR. Those experiencing a substantial degree of negative affect may feel unable to cope with stressors and appraise stressors as threatening rather than a challenge, thus raising their HR (Salomon et al., 2009). It is also possible that negative affect may cause sympathetic activation without the presence of an immediate stressor though this activation could be construed as reflecting prior stressors that led to affect changes such as early adversity or a history of interpersonal conflict and social isolation.

Positive affect did not appear to be predictive of HR within the multilevel models specified. This may reflect the less prolonged impact of changes in positive as opposed to negative moods on HR (Brosschot, Gerin, & Thayer, 2006). Greater tiredness was linked to a decrease in HR. This result aligns closely to previous ambulatory monitoring studies where feelings of disengagement and sleepiness have demonstrated a robust relationship to declines in HR (e.g. Jacob et al., 1999). The relationship between tiredness and HR is likely to be indicative of diminished energetic arousal, potentially reflecting amygdala deactivation.

The association between personality and HR also warrants comment. In particular, the hypothesized relationship between emotional stability and lowered HR was identified. This finding lends some ecological validity to laboratory studies identifying greater cardiovascular activity in response to stressors amongst those with a higher level of neuroticism, the obverse of emotional stability (Schwebel & Suls, 1999; Riese, Rosmalen,

Ormel, Van Roon, Oldehinkel, & Rijsdijk, 2007). However, controlling for a substantial array of physiological, emotional, and behavioural variables eliminated the association between emotional stability and HR. This was potentially due to covariance between personality the wide array of control variables in the final model. For instance, emotional stability is likely to be expressed in situation-selection, interaction frequency, consumption, and emotional experience.

Taken together the results of the study indicate joint roles for state and trait factors closely related to negative affectivity in contributing to an elevated HR in day-to-day settings. One explanation of these results is that both personality and affect moderate the impact of everyday stressors on physiological responses in healthy young adults which may have cumulative effects on health over time. This interpretation corresponds well with the results of a recent large scale twenty one year prospective cohort study that found neuroticism to be a long-term risk factor for cardiovascular disease, a relationship that was partially mediated by psychological distress, after adjusting for health, consumption, and demographic factors (Shipley, Weiss, Der, Taylor, & Deary, 2007).

The present study also contributes to the accumulating evidence indicating that Type D personality (negative affectivity and social inhibition) is predictive of adverse cardiovascular health outcomes. The D-construct has been shown to be closely proxied by high neuroticism and low extraversion (De Fruyt & Denollet, 2002; Sher, 2005; Steptoe & Molloy, 2007). The results lend some credence to the possibility that negative affectivity, but not social inhibition may be reflected in cardiovascular activity in the daily life of healthy individuals, as neither extraversion nor engaging with others in social situations substantially affected HR.

In contrast to research on the D-construct I find that the personality trait agreeableness, typically characterised by approach rather than avoidance behaviour,

predicts an increase in heart rate. Recent research has linked agreeableness to a deficit in anger regulation upon receiving negative feedback which may indicate that agreeable individuals are more reactive to information that contrasts with their interpersonal orientation (Jensen-Campbell, Knack, Waldrip, & Campbell, 2007). It is also possible that agreeable people are more vigilant and receptive to information during interactions, a characteristic that has been shown to be associated with greater cardiovascular activity (Smith, Ruiz & Uchino, 2000). Further research is required to demonstrate if the increase in cardiovascular activity identified amongst agreeable people can be explained by greater cardiovascular and affective activity in interpersonal situations, particularly those closely linked to identity and self-construction (Lyons, Spicer, Tuffin, & Chamberlain, 2000).

The present study overcomes limitations of previous studies which lacked important methodological criteria, in particular concerning the measurement of affect, the representativeness of the assessment context, the consideration of personality factors, and the rigorous control of potentially confounding covariates. The utilisation of continuous scales incorporating multi-dimensional components of both positive and negative affect allows fine-grained inferences about the relative contribution of various affect dimensions to be made (Mauss & Robinson, 2009). Incorporating personality factors is advantageous as pervasive traits condition the situations individuals select themselves into, thus the assessment of personality allows a clearer specification of the contribution of the situation to experience. Also, a substantial degree of overlap has been identified between personality traits and affect variables (e.g. extraversion and happiness, agreeableness and affection) both in previous analyses and in the present study, and it is therefore preferable to delineate which component is contributing to HR at a given time-point.

It is also important to adjust for confounding factors such as consumption, biological factors, and environmental conditions which have been shown to correlate with

both affect and HR (e.g. body fat, peak flow, activity levels). In support of this rationale, in the present study smoking was indicative of a substantially raised HR, most likely due to the effect of nicotine on activating the sympathetic nervous system. In addition, the potentially busier college and work environments were related to a higher HR than the home setting. I also observed substantially raised levels of HR during times of self-reported active behaviours such as exercising, walking, commuting, and playing indicating that the DRM was effective in identifying periods of increased physical activity.

One major caveat of the study involved the method of control for physical activity. Previous studies have shown only modest relationships between objectively recorded activity levels and subjective reports (r = .24 - .61) (Welk, Wickel, Peterson, Heitzler, & Fulton, 2007). The recent development of multichannel ambulatory accelerometry devices has made possible the collection of high frequency objective behavioural data. These devices utilise enhanced computer processing and storage capacities, sophisticated algorithms, and sensitive piezoresistive and piezocapacitive sensors to perform complex analyses to separate movement and posture and to gauge the intensity of action (Bussmann, Ebner-Premier, & Fahrenberg, 2009). In particular, activity monitoring can operate as a method of separating the movement and posture related information out of HR analyses in order to produce an account of the psychophysiological response to emotion (Myrtek, 2004).

Incorporating ambulatory activity monitoring into the current study would have allowed for a stringent control of behaviour thus testing the possibility that movement during episodes associated with negative affect may explain the negative affect-heart rate relationship. However, several commentators have argued that care is warranted when controlling for factors such as posture or movement that may be causal antecedents of affect (Jacob et al., 1999; Shapiro et al., 2001). As specific movements are strongly linked

to affective states and their initiation may induce those states (Koch, Holland, Hengstler, & van Knippenberg, 2009; MacLachlan, 2004), controlling for affect-relevant behavioural information may diminish genuine mood effects on psychophysiology.

A caveat of the DRM protocol was that it requires that episodes be at least 20 minutes long and the average length of episodes reported was approximately 70 minutes meaning that briefer episodes are often missed. Encouraging episodes to be reported using a shorter time-frame would enhance the sampling frequency of the DRM and potentially improve the estimates identified. Running a more high-frequency moment-by-moment measurement of affect alongside the DRM, would allow the relative accuracy of the DRM in HR prediction to be gauged. Although the DRM is retrospective, it is structured to carefully evoke contextual information and has been shown to replicate affect patterns identified by experience sampling (Kahneman et al., 2004). As demonstrated in the current study, it is possible to administer the DRM to relatively large samples in an online setting in order to assess subtle intra-day variation in affect patterns. Combining real-time tracking of human functioning with online DRM is also achievable with relatively little additional participant burden. This integrative approach to the study of real-world behaviour was found to generate what I believe is an optimal level of sufficiently low respondent burden and high data reliability. Matching day reconstruction data to information derived from technological innovations such as psychophysiological and behavioural monitoring (e.g. Hasler, Mehl, Bootzin, & Vazire, 2008), ambulatory assessments of pollutants and crowding (e.g. Gold et al., 2000), and momentary changes in health markers (e.g. markers of inflammation and neuroendocrine functioning) is likely to facilitate collaboration between health psychology researchers and numerous other disciplines (Mehl, 2007).

In conclusion, this study demonstrates substantial correspondence between HR and state and to lesser extent trait affectivity in the context of daily life. This is evident

from the raw concordance of HR and psychological distress, and multilevel analysis of the relationship between HR, personality and affect. Most convincingly, controlling for a wide range of episodic and personality variables, negative affect in an episode predicts higher heart rate. In the context of the reserve capacity model, the increased level of heart rate associated with negative emotion suggests support for sympathetic adrenal medullary axis activation as a psychobiological process linking emotion to health. Furthermore, the greater consistency of the relation between heart rate and state rather than trait negative affect (i.e. neuroticism) suggests that the naturalistic monitoring paradigm employed in the current study for the analysis of episodic human emotion and psychophysiology may be a useful tool for the evaluation of hypotheses drawn from the reserve capacity model.

Chapter 3

Willpower and cardiovascular and neuroendocrine processes: Emotional stability mediates the health benefits of trait self-control

Chapter Aim

Although the reserve capacity model has proposed several intrapersonal traits that may influence emotion (e.g. self-esteem, optimism, extraversion) these traits often overlap conceptually with emotion. This chapter aims to identify how self-control, a trait proposed to be conceptually distinct from emotion, relates to changes in emotion. In addition, this chapter aims to build on chapter 2 which showed a link between emotion and psychobiological functioning in daily life. It does this by testing if self-control is linked to two aspects of psychobiological functioning and if this relation is mediated by patterns of emotion, as suggested in the reserve capacity model.

Chapter Summary

This study aimed to ascertain whether self-control predicts adaptive cardiovascular and neuroendocrine processes and to determine whether favorable health behaviours and affect patterns mediate these relationships. A sample of 198 healthy young adults completed baseline medical testing, a day of ambulatory psychophysiological monitoring, and a battery of questionnaires. Self-control was measured using the Self-Control Scale, smoking and exercise were self-reported, and affect intensity and variability were assessed using the Day Reconstruction Method. Salivary cortisol samples were provided at morning and evening and cardiovascular activity was monitored for a full day. Body mass index, body fat, and lung capacity were assessed at baseline. High trait self-control predicted low ambulatory heart rate, high heart rate variability, and a steep cortisol slope, even after controlling for age, gender, parental education, and baseline health measures. High self-control led to stable emotional patterns, which mediated cortisol and a low heart rate. Participants with high self-control had low rates of smoking which predicted a low heart rate. Trait levels of self-control were indicative of favorable patterns of cardiovascular and neuroendocrine functioning. The capacity to sustain stable patterns of affect across diverse contexts may be an important pathway through which self-control relates to health.

3.1. Introduction

There is now considerable evidence that personality is associated with longevity. In particular, the conscientious appear to outlive the irresponsible and the emotionally stable live substantially longer than the emotionally volatile (Friedman, 2008; Terracciano, Löckenhoff, Zonderman, Ferrucci, & Costa, 2008). However, the psychological mechanisms and biological intermediaries that explain these links remain largely unknown (Smith, 2006). To address this issue, in this study I examined the relationships among self-control, affect patterns and health behaviour, and psychobiological processes. Measures of cardiovascular (i.e., heart rate and heart rate variability) and neuroendocrine (i.e., the diurnal cortisol slope) functioning were included because they have established links to health outcomes (Schoorlemmer, Peeters, & van Schoor, 2009).

The superior longevity of people high in conscientiousness suggests that in some way good self-control confers biological benefits (Friedman, 2008). Self-control is one central, even defining aspect of conscientiousness (Bogg & Roberts, 2004). Accordingly, I predicted that high levels of self-control would be associated with favorable patterns of psychobiological functioning. Moreover, I predicted that more self-controlled persons would behave in healthier ways than other persons, specifically exercising more and being less likely to smoke. I hypothesized that these healthy behaviours would help explain a link between self-control and physiological functioning.

Cross-sectional and experimental intervention studies have demonstrated that high self-control is associated with emotional control and stability (e.g. Tangney, Baumeister, & Boone, 2004; Oaten & Cheng, 2006). I therefore tested the hypothesis that high levels of trait self-control would reduce variability in emotional state. I expected that a trend toward stable patterns of affect may help explain the relationship between high self-control and adaptive patterns of cardiovascular and neuroendocrine functioning.

3.1.1. Personality and health

Personality traits are relatively stable dimensions of individual differences in cognitive, socioemotional and behavioural patterns. Early research aiming to evaluate the link between personality and health demonstrated that personality traits predict future health behaviours, well-being, and self-rated health (Hampson, Goldberg, Vogt, & Dubanoski, 2006). However, relying on self-reported subjective and behavioural outcome measures to establish a link between personality and health can be problematic. This is largely because such self-report measures may reflect the same methodological variance as personality. Thus, people who say good things about their personality may also say good things about their health, but that could indicate simply a positive style of answering questions rather than objective differences in health.

However, recent evidence has begun to link personality with objective physiological measures of cardiovascular disease and longevity (Friedman, 2008). Longevity has been shown to be predicted by high levels of conscientiousness (Martin, Friedman, & Schwartz, 2007) and emotional stability (Terracciano et al., 2008). For instance, robust evidence for a link between personality and mortality has accumulated in data from the Terman study of cognitively gifted children. Those who were rated as having high levels of "conscientiousness-social dependability" and "permanency of mood" as children had low levels of mortality over the seven decades of the study (Martin et al., 2007). Likewise, a 55-year longitudinal study in Scotland found a link between childhood personality measures and lifetime mortality (Deary, Batty, Pattie, & Gale, 2008). Specifically, a variable that the authors labelled dependability emerged from factor analysis combining measures of childhood perseverance, stability of moods, and conscientiousness. Children who scored high on dependability went on to live longer than other children.

High self-control has also been prospectively related to low rates of mortality, in particular from coronary heart disease and cancer (Grossarth-Maticek & Eysenck, 1995). One might assume that high self control and, by extension, conscientiousness and dependability can lengthen life by virtue of minimizing unhealthy behaviours. However, Grossarth-Maticek and Eysenck found that the association between self-control and mortality could not be accounted for by physical risk factors such as smoking, excessive drinking, caffeine intake, and exercise. Similarly, the link between conscientiousness and mortality in the seven decade follow-up of the Terman study participants could not be attributed to adult behaviour or risk taking. At least some of the health benefits of self-control must lie elsewhere.

These findings suggest that the relationship between personality and mortality risk is complex and involves multiple psychological and biological pathways (Smith, 2006). The literature has produced remarkable advances in identifying *which* personality traits are predictive of mortality. I argue that to further develop this literature we need to open the 'black box' of the personality-longevity relationship and systematically analyze *how* personality traits lead to different approaches and responses to situations and the resulting psychobiological reactions. Self-regulation theory details how people can resist temptation, cope with stress, and implement strategies to regulate affect (Baumeister, Vohs, & Tice, 2007). An understanding of how self-control operates can therefore contribute to delineating the relationship between personality and health outcomes.

3.1.2. Self-control, health behaviour and emotion

Self-control is a remarkably adaptive personality process by which people manage their own thoughts, impulses, emotional states, and behaviours. Evidence suggests that the operation of self-control relies on a limited resource, similar to an energy or strength, thus reaffirming the folk notion of willpower (Baumeister et al., 2007). When sufficient

resources are not available, self-control can no longer function at optimal capacity. Inadequate self-control stemming from depleted resources can contribute to impulsive, disinhibited behaviour and can reduce the ability to stay on task, pursue goals, delay gratification, and follow socially prescribed rules (e.g., Baumeister et al., 2007).

Poor self-control has been linked to smoking, substance abuse, problem drinking, overeating patterns, and sexually impulsive behaviour. Brief periods of exercises have successfully enhanced the self-control levels of participants (Oaten & Cheng, 2007). These self-control interventions have shown a variety of positive side effects including marked reductions in caffeine, alcohol, and cigarette consumption (Oaten & Cheng, 2006).

Unfavorable health behaviours are one clear way that a lack of self-control could lead to negative health effects. Another possible route is suggested by evidence that high levels of self-control are linked to high emotional stability ($r = .5$; Tangney et al., 2004). Improvements in self-control brought about by targeted exercises have sometimes led to an increase in emotional control (Oaten & Cheng, 2006). Insofar as a lack of emotional control is predictive of negative health outcomes (e.g. Suls & Bunde, 2005), it is feasible that self-control may promote health by regulating and stabilizing emotion.

There are several ways that high self-control could reduce fluctuations in negative affect and possibly stabilize positive affect (e.g. Larsen, 2000). The regulation of affect has been compared to the operation of a thermostat where discrepancies from one's favoured 'set-point' are monitored and noted. Strategies (e.g. selecting certain situations, distracting oneself or concentrating on an alternative) can then be implemented to ensure that feelings fall back in line with one's ideal levels (Chow, Ram, Boker, Fujita, & Clore, 2005; Gross & Thompson, 2007). People with high self-control may also be less likely than others to experience wide fluctuations in affect. This is because the highly self-controlled are competent in organizing and planning activities, problem solving, self-presentation, active

coping, controlling thoughts, and persistently engaging with tasks, as well as being able to regulate their own emotional states (Baumeister et al. 2007).

People high in self-control are therefore less likely than others to experience both task and self-control failure and the associated surges in negative affect (Tice, Bratslavsky, & Baumeister, 2001). Negative emotional feedback is functional in that it serves as a deterrent for future self-defeating behaviour and to stimulate reflection on the details of what went wrong. However, those with poor self-control may seek out transient immediate pleasures that bring long-term risk and costs as a way to avoid negative emotion (Tice et al., 2001; Oaten & Cheng, 2005).

Taken together, the evidence suggests that low levels of self-control and poor management of self-regulatory resources can lead to more changeable patterns of affect. Intraindividual variability in affect has been demonstrated to be a stable trait (Eid & Diener, 1999). Low variability (high stability) of emotion is thought to reflect successful maintenance of psychological functioning, good personality integration, and high ego strength. Several studies have linked diminished psychosocial resources to greater variability in affect (Kuppens, Van Mechelen, Nezlek, Dossche, & Timmermans, 2007). Diminished intrapsychic resources such as self-control can have a substantial effect on patterns of emotion which may in turn influence psychophysiological processes (e.g. neuroendocrine, immune, inflammatory, and cardiovascular functioning) and health (Gallo, Bogart, Vranceanu, & Matthews, 2005).

3.1.3. Self-control and psychobiological functioning

A psychobiological approach can help specify the mechanisms that connect self-control, affect, and health (Martin et al., 2007). For example, recent research has identified heart rate variability, a reliable predictor of cardiovascular morbidity and mortality, as

varying with self-regulatory effort, particularly efforts directed at emotional control (Segerstrom & Nes, 2007; Appelhans & Luecken, 2006).

Self-regulatory capacities are possibly linked to heart rate variability as a result of the colocalization of autonomic and self-control systems in the brain. The neural activity that corresponds with self-regulatory activity has thus been linked to simultaneous high levels of heart rate variability (Segerstrom & Nes, 2007). Specifically, the central autonomic network overlaps considerably with neural areas involved in self-control such as the medial prefrontal cortex, anterior cingulate, and amygdala (Appelhans & Luecken, 2006). This network activates the vagal nerve which influences cardiovascular activity. It is hypothesized that self-control may engage the vagal nerve, putting a brake on heart rate and increasing heart rate variability. High levels of heart rate variability are adaptive as they are thought to conserve energy resources and can induce a calm emotional state (Segerstrom & Nes, 2007). In other words, self-control may produce health benefits by increasing heart rate variability.

As in the case of heart rate variability, low heart rate may reflect the activity of the cortical control centres through which self-control operates. Prospective studies have linked high (fast) heart rate to hypertension in young people and to myocardial infarction and mortality in older adults (e.g. Palatini et al., 2006). Hence it is plausible that the health benefits of good self-control could be mediated by low heart rate.

In addition to linkages between self-control and cardiovascular activity, recent research suggests that high levels of self-control may be related to adaptive patterns of neuroendocrine functioning (Taylor et al., 2008; Urry et al., 2006). In terms of neuroendocrine biomarkers, there is particularly strong evidence linking flat diurnal cortisol slopes with adverse health outcomes (e.g. cancer, fatigue, post-traumatic distress) and early mortality (e.g. Saxbe, 2008). Cortisol levels peak approximately 30 minutes after

waking and then decline across the day, but they decline faster for some people than for others. People with flat cortisol slopes (i.e., the unhealthy pattern) show a slow rate of decline in cortisol output over the day with evening levels not differing substantially from morning levels. For people with the more typical steep cortisol slope, cortisol output declines quickly from elevated morning levels to substantially reduced evening levels.

High levels of activity in the prefrontal cortex, a brain area heavily implicated in self-control and executive functioning, during an emotion regulation task have been shown to predict a steep cortisol slope over the course of a day (Urry et al., 2006; Cunningham-Bussel et al., 2009; Taylor et al., 2008). The activity of the prefrontal cortex may dampen amygdala reactivity to emotive stimuli and in this way prevent high levels of emotional reactivity and the associated cortisol output. Hence another prediction would be that those low in self-control may have difficulty controlling their emotions which may in turn lead to a flat cortisol slope. In this way, the rate of decline in cortisol could help mediate some of the health impact of self-control.

3.1.4. Present investigation

The present investigation had several goals. First, I sought to show that self-control would have positive relationships to health markers, specifically cardiovascular and neuroendocrine functioning as assessed in everyday life. More precisely, I tested the hypotheses that high self-control would predict high variability of heart rate, low heart rate, and a steep cortisol slope. Second, I looked for mediation by health behaviours and emotional patterns. Multiple measures of positive and negative emotions were included and smoking and exercise were assessed.

The variability and intensity of people's affective lives was assessed for an entire day using the day reconstruction method (Kahneman et al., 2004), a survey that incorporates memory priming techniques to assist in the recall of experiences of the

previous day. I hypothesized that a link between self-control and the psychobiological process examined would be mediated by health behaviours and the affect patterns observed.

3.2. Method

3.2.1. Study participants

Data were collected from 204 participants, recruited via email from the student population. Participants were requested to take part in the study on three consecutive days: to complete baseline physiological tests and receive instruction on the study procedures (day 1), to provide ambulatory monitoring data during a normal working day (day 2), and to complete an online questionnaire which included health behaviours and a day reconstruction affect measure (day 3). Those who took part were compensated with either research credits or a cash incentive of 25 euro. The drop-out rate was 3% (6 participants), leaving 198 participants: 68 men and 130 women aged 18 - 49 years (mean = 23.4, SD = 6.3). Twenty participants were removed from the heart rate analyses and 24 participants were removed from the heart rate variability analyses due to the presence of excessive artifactual measurement error (e.g. excessive number of outlier measurements, repeated loss of signal) leaving 178 participants with usable data for the heart rate analyses and 174 participants in the heart rate variability analyses. Fully usable cortisol data was provided by 181 participants, with the remaining participants removed due to incomplete, inappropriately timed, or unanalyzable samples. Each participant received verbal and written information detailing what the study entailed and gave informed consent.

3.2.2. Procedures

3.2.2.1. Ambulatory measurement of heart rate

Heart rate was measured with the Suunto Memory Belt, a lightweight (61g) heartbeat interval recorder, worn around the chest, with a capacity to record 200,000 beat-to-beat intervals (Suunto memory belt, Suunto Oy, Vantaa, Finland). This commercially available heartbeat interval recorder has been shown to align closely with measurements from the 1000Hz 3-lead ECG BIOPAC MP35 data acquisition unit (Biopac Systems, Santa Barbara, CA) (Daly, Delaney, Doran, Harmon, & MacLachlan, in press). The research nurses instructed participants on how to apply electrode gel to the heartbeat recorder and the operating procedures for the device (see Appendix A). Outlier and artifactual readings were isolated and removed from HR recordings prior to analysis. Outlier measurements were defined as measurements of heart rate outside the range of 40 to 150 beats per minute (Daly et al., in press) and accounted for less than 1% of the data. The Kubios heart rate variability analysis package (Biosignal Analysis and Medical Imaging Research Group, Kuopio, Finland) was used to produce indices of heart rate (M = 87, SD = 10.6) and heart rate variability (standard deviation for the mean value of the time between heart beats), (M = 136.52, SD = 41.3). Resting heart rate was also assessed using a professional blood pressure monitor when the participant was sitting quietly on a chair during the initial day of medical testing.

3.2.2.2. Salivary cortisol measurement

On the first day of the study the research nurses provided each participant with detailed verbal and written instructions on the saliva collection procedures (see Appendix A). Salivary cortisol samples were collected using the Salivette sampling device (Sarstedt, 51582 Numbrecht, Germany); a centrifuge tube containing a sterile cylindrical cotton wool swab. For this study participants provided four saliva samples: two samples at 30 minutes

post waking, and two 12 hours after waking up. Participants were requested not to brush/floss their teeth, smoke, eat, or drink beverages (water was permitted up to 5 minutes prior to sampling), during the 30 minutes prior to saliva collection. They were asked to place each cotton swab in their mouth for at least 45 seconds and then place it in the centrifuge tube. Samples were returned to the laboratory the next day and stored at room temperature for a maximum of 1 week before being centrifuged and frozen at - 80 ^{o}C. The average cortisol level of the saliva samples at 30 minutes after waking and at 12 hours post-waking was computed for each participant. The average values were log-transformed cortisol as is typical (e.g. Saxbe, 2008) and cortisol slope from morning to evening was calculated (M = -.078, SD = .026).

3.2.2.3. Self-control measure

The brief *Self-Control Scale* (Tangney, et al., 2004) consists of 13 statements, rated on a 5 point scale from 1 (not at all like me) to 5 (very much like me). The 13 items are worded such that endorsement of 8 items is indicative of a reduced capacity for self-control (e.g. "I am lazy") and an enhanced capacity for the other 5 items (e.g. "I refuse things that are bad for me"). The range of possible scores on the scale is 13 – 65 with higher scores signifying better self-control. The average score on the Self-Control Scale was 39.56 (SD = 8.44) with scores ranging from 18 – 59 (Cronbach's alpha = .834). In support of the validity and reliability of the Self-Control Scale, the scale has been shown to be predictive of more appropriate emotional responding, superior interpersonal skills and adjustment, better grade point averages, and has demonstrated high internal consistency and test-retest reliability (Tangney et al., 2004).

3.2.2.4. Health related variables

Participants underwent a baseline medical testing session on the initial day of the study, during which each individual's body mass index, body fat, and lung capacity were

assessed. The average body mass index of the participants was 23.2 (SD = 3.9) and the mean percentage body fat was 28.7 (SD = 8.7). The mean peak flow indicator of lung capacity was 406.5 liters/minute (SD = 116.1). As part of an online questionnaire completed on the third day of the study participants endorsed items relating to their health behaviour. Participants rated how often they engage in exercise (from 0 = Never to 4 = 4 or more times per week) and whether or not they currently smoke cigarettes. Fourteen percent of the sample was current smokers and participants exercised on average 2-4 times per month (M = 3.29, SD = 1.32).

3.2.2.5. Assessment of affect

On the day after completion of the ambulatory procedures, participants returned the relevant data collection devices and completed a computer-assisted day reconstruction survey. The online survey follows a fixed format where participants recall an entire day from waking to sleep. The day is separated into morning, afternoon, and evening stages and subsequently each stage is broken down into a series of 'episodes'. Episodes are between 20 minutes and 2 hours and participants provide episode-by-episode information about the subjective experiences linked with each episode as measured by 10 affect scales. The 10 scales were parsed into six items measuring positive affect (happy, calm, comfortable, affectionate, interested, confident), and four items assessing negative affect (impatient, depressed, stressed, irritated). Participants rated each emotion using response scales ranging from 0 (not at all) to 6 (very much). The adjectives used are similar to those used in other mood scales such as the Positive and Negative Affectivity Schedule or the Profile of Mood States (Kahneman et al., 2004).

On average participants provided affect ratings for approximately 11 episodes recounted from their day. Collated averages of each person's positive affect (M = 3.53, SD = 1.2, alpha = .85) and negative affect scores (M = 1.39, SD = 1.16, alpha = .76) were

calculated based on mean affect intensity levels across all reported episodes. The mean within-person standard deviation in each affect item over the reported episodes was computed. Variability in positive affect items (M = 1, SD = .38) was found to be closely related to variability in negative affect (M = 1.07, SD = .42), ($r = .53$, $p < .0005$). The deviation scores for positive and negative affect variability were therefore summed to produce an overall index of total affect variability (M = 2.07, SD = .7).

3.2.3. Data analysis

Correspondence between self-control and heart rate, heart rate variability, and the cortisol slope were firstly assessed using multiple linear regression adjusting for the inclusion of demographic variables (e.g. age, gender, father's education) and baseline health information (e.g. body mass, body fat, lung capacity) . Following this, I tested the role of health behaviours and affect patterns in mediating between self-control and the psychobiological variables examined.

Mediation was defined by the guidelines outlined by Baron and Kenny (1986) whereby three essential conditions are required: (i) Levels of the independent variable must predict the mediator variable (e.g. self-regulation must be related to smoking), (ii) the mediator must account for substantial variation in the dependent variable (e.g. smoking must predict heart rate), (iii) controlling for the associations between the independent and mediating variable and the mediating variable and the dependent variable, the independent variable must no longer significantly predict the dependent variable and the mediating variable must remain as a predictor of the dependent variable (e.g. adjusting for covariance in self-control and smoking and the association between smoking and heart rate, self-control must no longer significantly predict heart rate, whilst smoking must be retained as a predictor of heart rate).

Partial mediation occurs when the relationship between the independent and dependent variables is diminished but not reduced to non-significance in the third step of the analysis. Both unstandardized and standardized regression coefficients are presented for all multiple regression analyses.

3.3. Results

3.3.1. Self-control and psychobiology

I used multiple linear regression analyses to test the association between trait self-control and the psychobiological measures. The demographic factors age, gender and father's education were included in the regression, as were these baseline physiological measures: body mass index, body fat, and lung capacity.

In line with predictions, self-control was inversely related to heart rate (B = -.224, $SE\ B$ =.097, $p < .05$, Cohen's d = .33), and positively related to heart rate variability (B = .791, $SE\ B$ =.385, $p < .05$, Cohen's d = .29). The average heart rate of those with self-control scores in the upper half of the distribution was 85.1 (SD = 9.8) compared with a heart rate of 89.1 (SD = 11) among those with self-control scores in the lower half. Participants in the top half of the distribution of self-control scores had heart rate variability scores (M = 143.2, SD = 43) which were higher than those with lower self-control (M = 131.7, SD = 40). High trait self-control was also predictive of a steeper cortisol slope (B = -.001, $SE\ B$ =.0005, $p < .05$, Cohen's d = .28), as can be seen in Table 4. For those with high self-control cortisol levels declined at a rate of .066 ug/dL each hour, a faster rate than amongst participants with lower self-control (.052 ug/dL per hour).

Further analyses revealed that high self-control predicted a low resting heart rate, even after adjusting for potentially confounding demographic and baseline health variables (B = -.259, $SE\ B$ = .103, $p < .05$, Cohen's d = .37). Participants in the top half of self-

control scores had a resting heart rate of 72.5 (SD = 11.4), which was significantly slower than the heart rate of 76.3 (SD = 12.2.) among those with lower self-control, ($t = 2.23$, $p < .05$). Thus, trait self-control successfully predicted several biological measures relevant to health.

Table 4

Summary of multiple regression analysis assessing self-control as a predictor of psychobiological variables

Variable	Heart rate		SDNN		Cortisol slope	
	B (*SE B)*	β	*B* (*SE B)*	β	*B* (*SE B)*	β
Intercept	98.11 (10.36)		92.84 (41.01)		.023 (.31)	
Self-control	-.224 (.097)	-.18*	.791 (.384)	.16*	-.001 (.0005)	-.16*

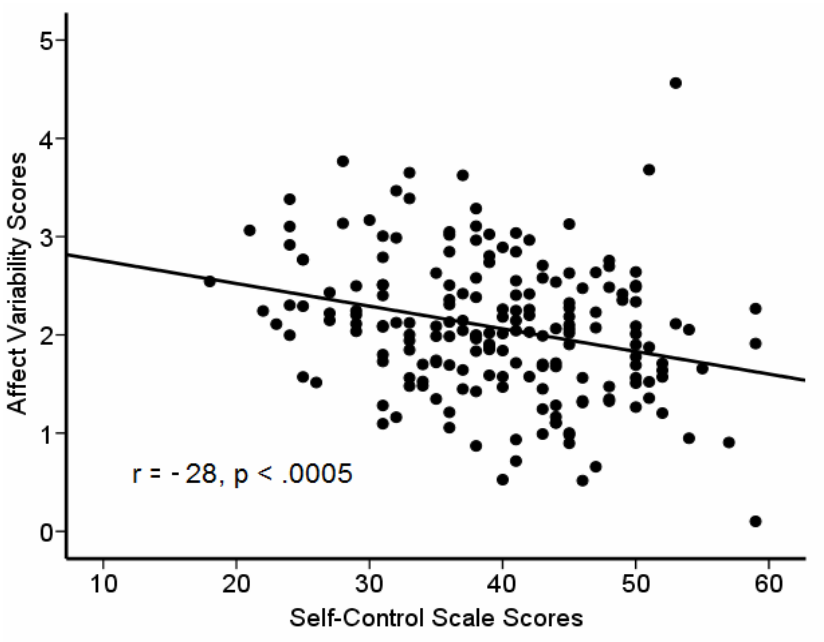
Note. SDNN = Standard deviation of interbeat intervals. *SE* = standard error. All analyses are adjusted for demographic variables (age, gender, father's education) and baseline health (body mass index, body fat, peak flow). * $p < .05$.

3.3.2. Emotion and mediation

Mediation involves identifying a relation whereby an independent variable causes a mediating variable which in turn causes a dependent variable (Baron & Kenny, 1986). Therefore I next aimed to determine whether affect patterns and health behaviours were associated with both self-control and psychobiological functioning.

The first phase of the mediation analysis examined the relation between trait self-control and the intensity or variability in affect. High levels of self-control predicted both high levels of positive affect ($B = .015$, $SE\ B = 0.007$, $p < .05$, Cohen's $d = .3$) and low

(a)



(b)

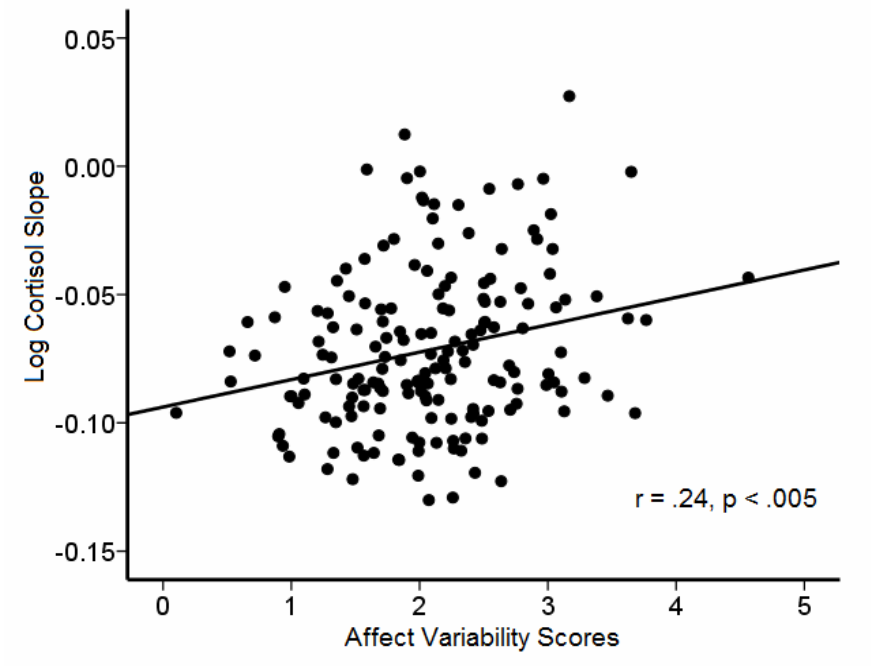


Figure 3. Affect variability scores as a function of Self-Control Scale scores (a), and log cortisol slope (slope from morning to evening levels) as a function of affect variability scores (b).

variability in affect (B = -.022, $SE\ B$ = 0.006, p < .01, Cohen's d = .52), as illustrated in Figure 3a. Multiple regression analyses showed that positive affect was not related to heart rate, heart rate variability or to the cortisol slope, however, which effectively rules out positive affect as a mediator of the benefits of self-control.

Affect variability, on the other hand, was positively associated with heart rate (B = 2.99, $SE\ B$ = 1.14, p < .01, Cohen's d = .37) and predicted a trend towards flat cortisol slopes (B = .011 $SE\ B$ = 0.003, p < .01, Cohen's d = .52), as can be seen in step 1 of Table 5 and as illustrated in Figure 3b. Affect variability failed to significantly predict heart rate variability. Entering self-control into the regression failed to substantially diminish the relationship between affect variability and heart rate and the cortisol slope. Self-control was also no longer a significant predictor of either heart rate or the cortisol slope, thus satisfying the third criterion for successful mediation.

Taken together, the findings in combination with additional Sobel tests (i.e. the ratio of the product of the regression coefficients of (i) the relationship between the independent variable and the mediator and (ii) the mediator and the dependent variable, to the standard error terms of these relations or z-value = $a*b/SQRT(b^2*s_a^2 + a^2*s_b^2)$) indicate support for affect variability as a mediator between self-control and the diurnal cortisol slope (Z = 2.32, p < .05), and as a partial mediator between self-control and heart rate (Z = 1.87, p = .06). Affect variability did not mediate heart rate variability, and positive affect levels did not mediate any of the effects. The analyses testing affect variability as a mediator between self-control and psychobiological functioning are summarised in Figure 4.

Table 5

Summary of two-step multiple regression analysis assessing the ability of affect variability to predict psychobiological variables

Variable	Heart rate		SDNN		Cortisol slope	
	B (*SE B)*	β	*B* (*SE B)*	β	*B* (*SE B)*	β
Step 1						
Affect variability	2.99 (1.14)	.2**	-8.66 (4.67)	-.15†	.011 (.003)	.24**
Step 2						
Affect variability	2.58 (1.19)	.18*	-6.64 (4.86)	-.01	.009 (.003)	.22**
Self-control	-.13 (.1)	-.1	.6 (.41	.12	-.004 (.004)	-.1

Note. SDNN = Standard deviation of interbeat intervals. *SE* = standard error. All analyses are adjusted for demographic variables (age, gender, father's education) and baseline health (BMI, body fat, peak flow).

** p<0.01, * p<0.05, † p<0.1.

Next, I tested whether engaging in more or less favorable health behaviours may help mediate association between self-control and psychobiological functioning. Regression analysis, again controlling for demographic and baseline health variables, showed that high self-control scores were associated with a reduced likelihood of being a current cigarette smoker (B = -.02, $SE\ B$ =.007, p < .005, Cohen's d = .42). Exercise was unrelated to self-control levels (B = .003, $SE\ B$ =.011, p = .82), and for this reason exercise was not considered for further mediation analyses.

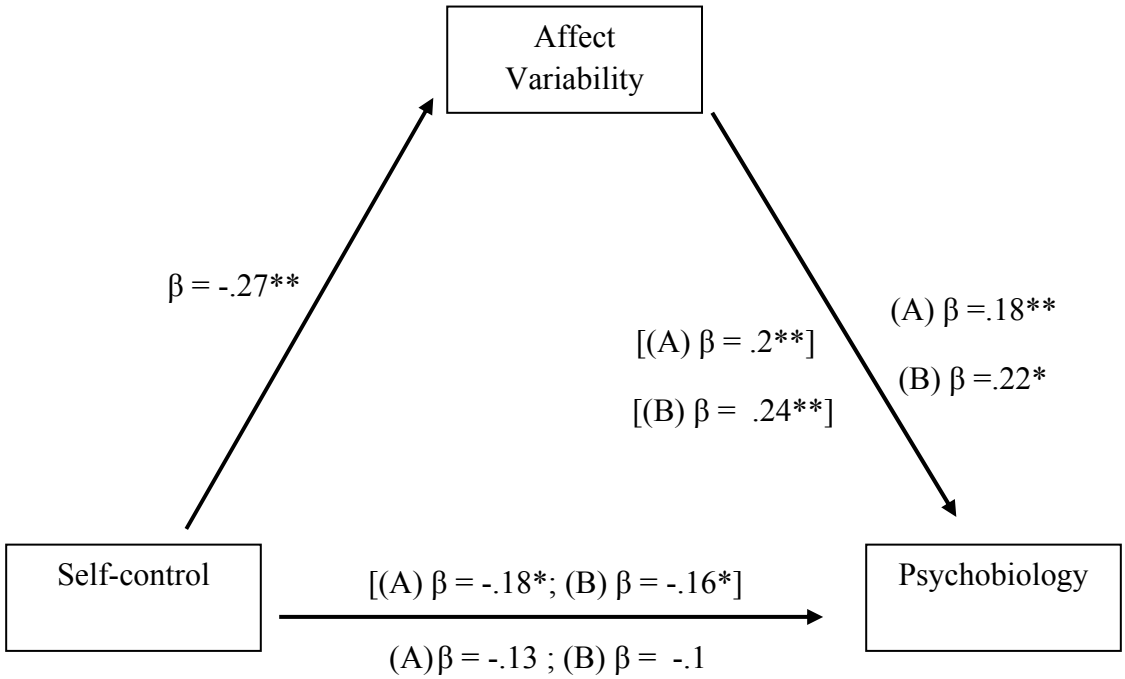


Figure 4. Summary of mediation analyses testing if variability in affect explains the association between self-control and psychobiological functioning. Mediation analyses are shown for heart rate (Pathway A) and the cortisol slope (Pathway B). Results are controlled for demographic variables (age, gender, father's education) and baseline health (BMI, body fat, peak flow). Regression coefficients are reported in brackets. ** $p < 0.01$, * $p < 0.05$, † $p < 0.1$.

Being a current smoker was related to a high heart rate *(B* = 2.88, *SE B* = .98, p < .005, Cohen's *d* = .42) but was not associated with heart rate variability (*B* = -14.51, *SE B* = 9.27, p = .12) or the diurnal cortisol slope (*B* = .01, *SE B* =.007, p = 1.7). I therefore tested smoking as a mediator between self-control and heart rate. Entering smoking into the regression model marginally diminished the association between self-control and heart rate (*B* = -.23, *SE B* = 0.1, p < .05, to *B* = -.17, *SE B* = .09, p = .07), and smoking remained as a significant predictor of heart rate (*B* = 5.67, *SE B* = 2.41, p < .05). Sobel tests were used to confirm that smoking could be considered a partial mediator between self-control and heart rate (Z = 1.81, p = .07).

3.4. Discussion

The present results indicate that the benefits of self-control are more than skin deep or, more precisely, more than merely enhancing subjective health and self-reports. Self-control was associated with several biological markers of cardiovascular and endocrine health. Specifically, high trait self-control predicted having a slower heart rate, higher variability in heart rate, and a steeper rate of decline in cortisol across the day.

The present study also found some effects of self-control that are not directly biological. People with high self-control were less likely than others to be regular smokers. They had more positive affect and pleasant emotions overall. They showed greater emotional stability in the sense of less variation in affect across the day.

Perhaps most important, self-control, cardiovascular and neuroendocrine patterns, and emotional and behavioural patterns were found to be interlinked. The strongest mediation effect was the pathway running from high self-control through low affective variability to the cortisol slope. Apparently, one important consequence of having high trait self-control is that it stabilizes the person's emotional state, which in turn contributes to a relatively fast and thorough reduction in cortisol levels over the day. The emotional stability associated with high self-control was also found to contribute to a slower average heart rate (i.e., partial mediation).

I also examined two health behaviours, smoking and exercise. For these, I relied on self-reports, which may not be entirely reliable, especially insofar as people may seek to give socially desirable answers. I found self-reported exercise had no significant associations with self-control. In contrast, being a smoker was associated with low self-control, and smoking partly mediated the link between self-control and heart rate.

3.4.1. Self-control and psychobiology

These data are quite compatible with several strands of recent research examining personality and health. Conscientious and self-controlled people have been shown to live longer than other people, and the activation of brain areas implicated in self-regulation has been linked to adaptive patterns of cardiovascular and neuroendocrine function (e.g. Deary et al., 2008; Segerstrom & Nes, 2007; Cunningham-Bussel et al., 2009). In a similar vein, and in keeping with my predictions, I found that highly self-controlled people had low and variable heart rates and a substantial decline in cortisol from morning to evening.

It could be that self-control predicts ambulatory cardiovascular activity because diligent people have patterns of physical activity that differ from those of less disciplined persons. It should be noted, however, the relationship between high self-control and low heart rate was found even when participants were monitored sitting quietly in a chair during baseline medical testing. Hence the biological effects of self-control were at least partly independent of current physical activity. Self-control was also unrelated to self-reported exercise frequency in this sample. The adaptive patterns of cardiovascular activity amongst those high in self-control are therefore unlikely to be due to individual differences in patterns of activity or fitness levels.

A flat overall cortisol slope could result, at least in part, from the numerous stressors people encounter on a daily basis (Adam, Hawkley, Kudielka, & Cacioppo, 2006). That is, the normal and healthy pattern is for cortisol to decline across the day from its morning high, but among some people, stressful events may reduce or prevent this decline. Animal and human studies suggest that the stress of forming new groups, meeting new people, or even being alone can cause elevations in cortisol (Levine, Wiener, & Coe, 1993). Even the thought of an upcoming exam or a dreaded visit to the dentist could

trigger the activation of fear and defensive systems in the brain (e.g. amygdala), flooding one's system with cortisol (Schulkin, McEwen, & Gold, 1994).

Self-control facilitates effective self-presentation, thereby making encounters with strangers less formidable (Vohs, Baumeister, & Ciarocco, 2005). The highly self-controlled are also more competent managers of their thoughts and less prone to rumination over anxiety provoking thoughts than more impulsive people (Gailliot, Schmeichel, & Baumeister, 2006; Segerstrom, Tsao, Alden, & Craske, 2000). Self-controlled people may be able to deal with the various common stressors thrown their way during their everyday activities and to cope with internal stressors such as disturbing thoughts. These advantages may help explain why, in the present sample, people with high self-control did not seem to succumb to stress-related upsurges in cortisol throughout the day but instead showed a rapid decline in cortisol from morning to evening. In particular, the present study indicated that the affective stability was decisive in enabling people with high self-control to benefit from the steep decline in cortisol. Apparently, in response to whatever stressors arise, good self-control enables the person to avoid the worst emotional turmoil, which thereby facilitates adaptive neuroendocrine functioning.

3.4.2. Emotional stability and psychobiology

Rapid fluctuations in affect are a reliable feature of psychopathology and may be due, at least in part, to a poor ability to repair negative feelings or sustain positive emotion (Kuppens et al., 2007). People with sufficiently high levels of self-control have a good deal of self-regulatory resources at their disposal and may use these resources to implement strategies to maintain optimal levels of affect. As well as assisting in self-presentation and the control of emotion provoking thoughts, self-control allows people to persist successfully on tasks, pursue goals, and to invoke adaptive strategies to maintain

desired affect levels. In these ways, self-control can enable people to get through life with less emotional volatility, less stress, and as a result better health.

The finding that emotional stability accounted for some of the benefits of high self-control represents a new direction in the research literature. Whilst previous reports have linked affect variability to psychological disorder, depression, and neuroticism, researchers have not given much consideration to the consequences of emotional stability stemming from high self-control. There is some evidence that impulsivity predicts high levels of mood variability (r = .39) (McConville & Cooper, 1999) and that practicing self-control exercises can enhance the ability to control emotion (Oaten & Cheng, 2006). But most writings about the benefits of self-control have focused on cognitive and performance effects.

The results suggest that the improvement in self-control may have health benefits (e.g., Tang et al., 2007). In particular, cortisol levels might be reduced quickly among those with high self-control. In terms of a mechanism, an improvement in self-control may make daily stressors less frequent and improve the ability to deal effectively with stressors, thus reducing emotional reactivity and in turn cortisol output. I inferred that heart rate may also be reduced if stressors fail to induce sympathetic nervous system activation amongst the highly self-controlled. The results support this hypothesis as stable patterns of emotion partially explained a link between high levels of self-control and low heart rate. To be sure, I also found that refraining from smoking was another important pathway by which high self-control can produce biological benefits, in this case low heart rate.

3.4.3. Health behaviour mediation

In line with previous studies, I showed a link between low self-control and an elevated risk of current smoking (Bogg & Roberts, 2004). Smokers had substantially elevated heart rate levels, presumably due to sympathetic nervous system activation

induced by nicotine. In line with health behaviour models that map the link between personality and health (Smith, 2006), the mediation analysis suggested that the raised likelihood of smoking among those with poor self-control partially explains the link between self-control and heart rate. Thus, it appears that self-control may stabilise emotion and reduce one's likelihood of smoking, which can both in turn reduce heart rate. Examining the influence of self-control on emotional factors and health behaviours and how these affect biological intermediaries will help further the understanding of the complex pathways between important personality traits like self-control and health.

3.4.4. Self-control and emotional stability

The link I identified between high self-control and stable patterns of affect warrants further comment. In contrast to this pattern, readers might infer that affect variability should be more likely to be observed among those high in self-control rather than those with poor self-control. This is because changeability in affect might reflect a dynamic ability of the psychological system to react flexibly to different situations. I argue that high levels of self-control allow rapid adjustment of affect levels from moment to moment, not unlike a thermostat that can adjust quickly to changing environments (Chow et al., 2005). Thus, a continuous record of someone's emotions might show fast minor fluctuations from a 'set-point' among those with high self-control, indicating the robustness of regulative mechanisms that quickly return emotional state to its set point. In contrast, people with low self-control may respond to events with changes in affect that persist for several minutes or longer before reverting to optimal levels. This would explain why people with poor self-control reported quite different levels of intensity of emotion from one (approximately hourly) episode to the next in the current study.

In addition, it could be argued that decreases in negative affect are likely to be characteristic of poor self-control but increases in positive affect are unlikely to reflect

impulsivity. However, in the current study and several previous reports, there was a strong positive correlation between the variabilities of negative and positive emotions, sharing approximately 25% of variance (e.g. McConville & Cooper, 1999). People with highly variable negative feelings appear to typically experience large fluctuations in positive affect. Researchers in the area of emotion regulation have continually emphasized that people need to be able to manage positive as well as negative emotions (Gross & Thompson, 2007). There are numerous contexts in daily life where restricting the expression of positive affect is necessary. For students this could be during lectures, in the library, or in the context of 'serious' conversation topics where expressing positive emotions would be inappropriate. Self-control may help sustain stable levels of positive as well as stabilizing negative affect.

3.4.5. Limitations and issues for further research

The present research showed self-control to predict adaptive patterns of psychobiology, which appeared to result from engaging in positive health behaviour (i.e., not smoking) and having stable patterns of emotion. A large body of research supports a model whereby personality produces a risk to health through inducing or preventing adverse health behaviours and dysfunction in physiological systems such as the hypothalamic – pituitary – adrenal – axis or the autonomic nervous system (e.g. O'Donnell et al., 2008; Martin et al., 2007). I followed this model of interpretation throughout the current research. However, the cross-sectional nature of the data precludes a causal examination of the direction of the relationships between self-control, emotion and behaviour, and psychobiology. For example, it is possible that those with a constitutional disposition toward low levels of physiological responsiveness may tend to develop stable patterns of affect which in turn can facilitate self-regulation (Smith, 2006; Tice et al., 2001). Future prospective studies, possibly including experimental manipulations and

multi-wave real-time diary studies examining both the trait and state operation of self-control, may help establish the direction of causality.

Future studies would also benefit from testing a broader sample of participants. It is likely that by restricting the current study to university students I observed a narrow range of health behaviours and self-control levels. However, this restriction of range may imply that the relationships observed in the current study are likely to hold in a representative sample of the same cohort where ceiling effects are less of an issue. A greater concern is that the results identified in this sample of mostly healthy young people may not generalize to more vulnerable populations such as the elderly or those with chronic illnesses. Then again, old age and illness could be considered as contexts where possessing high levels of self-control may be all the more valuable. For example, managing chronic illness requires people to persist with invasive regimes of medication and various therapies as well as consistently regulating anxiety-provoking thoughts about the trajectory of one's illness (Segerstrom, Schipper, & Greenberg, 2007).

Future research could combine detailed trait measures of self-control with functional imaging and real-time tracking of emotion and biological processes to answer numerous questions arising from the current research: Do people with high levels of trait self-control engage key brain areas such as the ventromedial and medial prefrontal cortex to a greater extent during tasks where the regulation of affect is required? Are the people who successfully control their emotions in the laboratory capable of doing so just as well in everyday life? And do the neural activation patterns observed in the laboratory explain individual differences in daily levels of cardiovascular and neuroendocrine function? As numerous forms of psychopathology have been shown to be characterised by both variability in affect and detrimental patterns of psychobiological functioning (e.g. Peeters, Berkhof, Delespaul, Rottenberg, & Nickolson, 2006) future studies could also examine the

role of self-control in explaining such associations. Within the reserve capacity framework, the extent to which various aspects of affect including intensity, duration, and variability mediate between psychosocial resources such as self-control and psychobiological functioning remains an important avenue for future studies. In addition, specifying how such relations may lead to changes in objective markers of health and the progression of disease processes will help further test the tenets of the reserve capacity model (e.g. mortality, CD4 count in HIV).

3.4.6. Conclusions

The present findings suggest several answers as to how self-control produces health benefits. People with good self-control have lower heart rates than others, partly due to their emotional stability and partly due to the healthy behaviour of not smoking. Low self-control contributes to good endocrine function in the sense of a steep decline in stress (cortisol) hormones across the day, in this case mainly because of the emotional stability. The link between self-control and high heart rate variability was not explained by any of the mediation tests and remains for further research to investigate. All these effects were found even after controlling for age, gender, parental education, and baseline health measures. Thus, the benefits of self-control were independent of several factors that have been known to contribute to health-related outcomes. Future studies will assist in determining whether poor self-control heralds a trajectory of ill-health and early mortality. Such research will also delineate the impact of self-control on health behaviour and patterns of emotion in order to explain changes in psychobiological functioning and the associated downstream health-effects.

Chapter 4

Awakening cortisol and diurnal rhythms of affect in psychological distress

Chapter Aim

This chapter proposes that psychobiological factors may influence emotion, a tenet that is problematic for the reserve capacity model. However, evidence suggests that the relationship between emotion and neuroendocrine, cardiovascular, and immune functioning is bidirectional. This chapter therefore aims to examine the likelihood that one aspect of psychobiological functioning, morning cortisol levels, may influence emotion. It does this by summarizing research suggesting that cortisol can lead to changes in affect and by testing the link between morning cortisol and subsequent affect levels in the context of daily life. In addition, this chapter specifies how the relation between cortisol and affect may be conditional on contextual factors like time of day and individual differences like psychological distress, thus demonstrating the complexity of the links between psychobiology and emotion.

Chapter Summary

People often feel unhappy in the morning but better later in the day, and this pattern may be amplified in the distressed. Past work suggests that one function of cortisol is to energize people in the mornings. In a study of 174 people I tested to see if daily affect patterns, psychological distress, and awakening cortisol levels were interlinked. Affect levels were assessed using the Day Reconstruction Method (Kahneman et al., 2004) and psychological distress was measured using the Depression Anxiety Stress Scales (Antony, Bieling, Cox, Enns, & Swinson, 1998). On average positive affect increased markedly across the day whilst negative affect decreased. For the highly distressed this pattern was stronger for positive affect. Low morning cortisol, as assessed by two saliva samples at waking and two samples 30 minutes after waking, predicted a clear pattern of low positive affect in the mornings. When I examined the interlinkages between affect patterns, distress, and cortisol the results showed that a pronounced increase in positive affect from morning through to evening occurred chiefly among distressed people with low cortisol levels upon awakening. Psychological distress, whilst not strongly associated with morning cortisol levels, does appear to interact with cortisol levels to profoundly influence affect, and this has important therapeutic implications.

4.1. Introduction

For some people the morning alarm clock invokes feelings of enthusiasm, but for others feelings of apathy in the morning are commonplace. Distressed people and people with an evening preference often experience low positive affect in the morning relative to the evening (Peeters et al., 2006; Jankowski & Ciarkowska, 2008). Prior research suggests a potential biological basis for morning affect patterns in cortisol, a major glucocorticoid hormone, which amongst other things functions to mobilize energy resources (Boksem & Tops, 2009). In this study, I predicted that deficient morning cortisol levels may, at least partially, explain low mood in the morning and why distressed people often do not feel energized towards a positive start to the day.

4.1.1. Diurnal variation of emotion

Numerous studies have found positive affect to rise substantially throughout the day whereas negative emotions have not yet demonstrated a robust diurnal pattern (Clark, Watson, & Leeka, 1989; Egloff, Tausch, Kohlmann, & Krohne, 1995; Hall, Spear, & Stirland, 1964; Murray, 2007; Stone et al., 2006). Whilst environmental factors appear to impact substantially on diurnal affect patterns (e.g. Stone et al., 2006), certain groups consistently experience more pronounced trends in affect than others and this may have a biological basis. For example, there is particularly strong evidence that the mood of people with depression improves substantially throughout the day (e.g. von Zerssen et al., 1985; Peeters et al., 2006). Not only do depressed people often feel worse in the morning they also tend to dislike the early hours of the day regarding themselves as evening-types (Hirata, Lima, de Bruin, Nobrega, Wenceslau, & de Bruin, 2007; Hidalgo, Caumo, Posser, Coccaro, Camozzato, & Chaves, 2009). This “morning worse” pattern is characterized by poor concentration and low positive affect in the morning that transitions into greater

alertness and more intense positive feelings later in the day (e.g. after 5pm) (Jankowski & Ciarkowska, 2008).

It has been suggested that circadian rhythms are blunted in various forms of distress and in evening types, particularly rhythms which are regulated by brainstem and hypothalamic areas (Schulz & Lund, 1983; Wirz-Justice, 2008). Early morning hypoactivity of the hypothalamus–pituitary–adrenal– axis as indexed by the diminished release of cortisol may therefore be an important candidate for explaining a morning worsening pattern of affect (Fries et al., 2005). More precisely, if cortisol can cause an increase in feelings of energy and reinvigourate fatigued people (e.g. Tops et al., 2006; Tops, Riese, Oldehinkel, Rijsdijk, & Ormel, 2008), then large individual differences in the volume of cortisol upon awakening may invoke divergent patterns of affect in the initial part of the day.

4.1.2. Cortisol and emotional rhythms

The diurnal pattern of cortisol release is a well-documented biological process with an established circadian component. Cortisol declines steeply throughout the day and is then regenerated during sleep so waking levels are raised substantially and then increase further, by 40-75% in the next half hour, to a daily peak (de Weerth & Buitelaar, 2005). The cortisol awakening response is assessed primarily with two metrics: the increase in cortisol from waking and the total integrated volume of cortisol released in the period immediately after waking (e.g. Chida & Steptoe, 2009b; Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). The former, cortisol increase from waking, has been well-examined and is thought to be a distinct component of the cortisol cycle (e.g. Clow, Thorn, Evans, & Hucklebridge, 2004). The awakening increase is responsive to psychosocial factors potentially signaling the effect of anticipation of the upcoming challenges of the day (e.g. Fries, Dettenborn, & Kirschbaum, 2009).

The less studied overall volume of awakening cortisol is closely linked to the circadian cortisol cycle and does not appear to be strongly related to psychosocial factors (e.g. Chida & Steptoe, 2009b). The total volume of cortisol released over the waking period may be relatively unaffected by psychosocial factors but yet interact with such factors to explain morning mood. For instance, for some people, with high levels of psychosocial resources, low levels of morning cortisol may have little impact on their morning mood. In contrast, low morning cortisol levels may act to compound the already lethargic and negative state of the distressed.

Several recent studies show partial support for this idea. Healthy people with a morning preference have been shown to have higher cortisol levels in the first hour after waking than evening types (Kudielka, Federenko, Hellhammer, & Wust, 2006; Kudielka, Bellingrath, & Hellhammer, 2007; Bailey & Heitkemper, 1991). In other research, people with low wakeup cortisol levels have been shown to experience high levels of fatigue at waking and later in the day (Dahlgren, Kecklund, Theorell, & Akerstedt, 2009; Adam et al., 2006). Further evidence suggests that the effects of low morning cortisol may be most pronounced in the distressed. For instance, cortisol levels over the wakeup period have been shown to be reduced in people with chronic fatigue and burnout (Fries et al., 2005), those with post-traumatic stress disorders (Chida & Steptoe, 2009b; Rohleder, Joksimovic, Wolf, & Kirschbaum, 2004), people with mild to moderate depression (Bhattacharyya et al., 2008), major depression (e.g. Posener, DeBattista, Williams, Kraemer, Kalehzan, & Schatzberg, 2000) and in anxious people and suicide attemptors (Sjogren, Leanderson, & Kristenson, 2006; Lindqvist, Isaksson, Traskman-Bendz, & Brundin, 2008).

4.1.3. The present investigation

In the present study, I therefore assessed people's psychological distress using the Depression Anxiety Stress Scales (DASS-21) (Brown, Chorpita, Korotitsch, & Barlow, 1997; Antony et al., 1998). I also measured the total volume of cortisol each person released over the waking period and the increase in cortisol from waking to thirty minutes post-waking (e.g Pruessner et al., 2003), and I comprehensively assessed affect and activity patterns throughout a single day using the Day Reconstruction Method (Kahneman et al., 2004). Using multilevel regression I firstly sought to investigate whether people with high psychological distress would experience a "morning worse" pattern of decreased positive affect early in the day. Although existing research does not appear to generate robust predictions regarding diurnal patterns of negative affect, I tested to see if it followed the opposite trend to positive affect (i.e. increased in the morning among the distressed). I next examined the possibility that low cortisol levels in the first half hour after waking as indexed by the total integrated volume of cortisol and the cortisol awakening response may be linked to low positive and perhaps high negative affect early in the day. Finally, I sought to link the two initial hypotheses by testing to see if people with high psychological distress and low morning cortisol levels are particularly likely to experience low positive affect and high negative affect in the morning and subsequent improvement in mood later in the day.

4.2. Method

4.2.1. Participants

One hundred and seventy four (59 males, 115 females) students participated in the study for course credit or 25 euro. The participants ranged in age from 18 to 49 (M = 23, SD = 5.7) and 62% of the sample's fathers had attended third level education. On the first day of the study participants received verbal and written instructions detailing what the

study entailed and provided informed consent. They then completed baseline physiological tests administered by trained research nurses and received instruction detailing the cortisol sampling procedure (see Appendix A). The next day participants provided cortisol samples at waking and thirty minutes after waking. On the third day of the study the participants completed a day reconstruction survey (Kahneman et al., 2004), a measure of psychological distress, and a series of questions about their health behaviour.

4.2.2. Measures

4.2.2.1. Online day reconstruction

On the day after providing the saliva samples participants completed a computer-assisted reconstruction of the objective details and affective experiences of the previous day. As in the original pen-and-paper day reconstruction survey (Kahneman et al., 2004) the online survey follows a fixed format in order to reduce recall bias. Participants firstly complete a diary by breaking their day into morning, afternoon, and evening stages and then recalling and labeling each 'episode' from their day. Participants are instructed to consider their day as a film and episodes as 'scenes' from that film, with the transition to each new episode representing a significant change (e.g. change of place, activity), and with each episode typically lasting between 20 minutes and 2 hours (Stone et al., 2006).

Participants provided specific information about the location (e.g. home, at work), activities (e.g. commuting, exercising), interactions, and feelings that occurred during the episode. For each episode participants rated how much they felt positive affect (happy, calm, comfortable, affectionate, interested, confident) and negative affect (impatient, depressed, stressed, irritated), on a scale from 0 (not at all) to 6 (very much). Average levels of positive ($M = 3.6$, $SD = 1.1$) and negative ($M = 1.4$, $SD = 1.2$) affect were then converted to standardized Z-scores for the subsequent regression analyses. As is typical,

positive and negative affect scores were found to be related ($r = -.5$, $p < .001$) but sufficiently separable to be considered distinguishable.

4.2.2.2. Psychological distress

Psychological distress was assessed using the short-form version of the Depression Anxiety Stress Scales (DASS-21) (Brown et al., 1997; Antony et al., 1998), a brief 21-item instrument which has been shown to yield a general factor dimension representing psychological distress (Henry & Crawford, 2005). The DASS-21 is composed of three 7-item self-report scales from the extended version of the DASS. Each item refers to a particular symptom and participants rate the extent to which the symptoms applied to them in the past week on a scale from 0 (did not apply) to 3 (applied to me very much or most of the time). Possible scores on the DASS-21 range from 0 to 63. In the present study two participants were found to score over 50 and were considered statistical outliers and not included in subsequent analyses. The DASS-21 scores of the remaining participants ranged from 0 to 42, ($M = 14.4$, $SD = 9.6$) and the Cronbach's alpha for the scale was .912.

4.2.2.3. Health and health behaviour

An array of baseline health variables and health behaviours were assessed primarily to ensure that the cortisol analyses were not confounded by individual differences in health, behaviour or consumption. During the baseline medical assessment trained research nurses measured each participant's body mass index ($M = 23$ kg/m^2, $SD = 3.3$), body fat ($M = 29\%$, $SD = 8.2$), lung capacity ($M = 402.1$ liters/minute, $SD = 117$), and systolic ($M = 121$mmHg, $SD = 13.5$) and diastolic ($M = 68.2$ mmHg, $SD = 9.4$) blood pressure. As part of the questionnaire component of the study the participants responded to items related to their health behaviour. Participants rated how often they exercise (from 0 = Never to 4 = 4 or more times a month), ($M = 3.31$, $SD = 1.29$). Twelve percent of the sample indicated they were current smokers and 9% were currently on a diet. The frequency of alcohol

consumption was rated on scale from 0 (Never) to 5 (Four or more times a week), (M = 3.31, SD = 1.29). Finally, I inquired as to if the participants consumed alcohol on the day prior to the monitoring day (25% drank) and also if they drank during the monitoring day (24% drank).

4.2.2.4. Salivary cortisol sampling

A total of four salivary cortisol samples were collected from each participant using a Salivette collection device (Sarstedt, 51582 Numbrecht, Germany). Two samples were taken immediately at waking and then two samples 30 minutes after waking. Six participants indicated that they collected one or more of their samples at times greater than 10 minutes from when scheduled and were thus not included in the final sample for this study (i.e. N = 174). The participants received detailed verbal instruction as well as a written protocol relating how to collect the saliva samples. Participants were requested not to eat, drink beverages, smoke, or brush/floss their teeth during the 30 minute period from waking to when they had collected their fourth sample. The samples were returned to the laboratory the day after collection and frozen at -80 ^{o}C and subsequently assayed. The two samples at waking yielded highly consistent results ($r = .93$, $p < .001$) as did the later two samples at 30 minutes post-waking ($r = .91$, $p < .001$). I converted awakening and 30-minute cortisol levels using log transformation to reduce skewness as is typical. The total integrated volume of cortisol over the awakening period was calculated as the total area under the curve (relative to zero) between the waking and 30-minutes post-waking samples (e.g. Pruessner et al., 2003; Chida & Steptoe, 2009b). The cortisol awakening response was calculated as the area under the curve between waking levels and cortisol levels at 30-minutes post-waking. For those with lower levels of cortisol at 30 minutes than at waking a negative awakening response was documented. Due to the multiple comparisons made in this paper an adjusted alpha level of .004 (for 12 central analyses) was utilized.

4.3. Results

There were 1,886 episodes reported in total (on average 10.77 per person) in the day reconstruction survey. To assist in the interpretation and presentation of the results I focused on episodes for which the temporal mid-point of their duration was between 8am and 12pm (1,821 episodes). I applied multilevel analyses to examine most of the study questions. Multilevel analysis was particularly suited to the nested structure of the data in the current study and the uneven number of repeated assessments at the episode-level (Stone et al., 2006). In this study, I had two levels of nesting: the episode level at which the

Table 6

Summary of multilevel models testing the association of demographic factors and core study variables with positive and negative affect adjusting for covariates

	Dependent Variable			
	Positive Affect		Negative Affect	
Variable	B (SE)	t	B (SE)	t
Intercept	-.056 (.8)	-.07	.54 (.66)	.83
Age	.006 (.01)	.62	-.013 (.008)	-1.63
Male [b]	-.55(.2)	-2.28**	.072 (.167)	.43
Fathers Education	-.048 (.06)	-.82	-.076 (.05)	-1.56
Time of Day	.02 (.004)	5.47***	-.011 (.004)	-2.61**
Psychological Distress	-.028 (.005)	-5.38***	.39 (.004)	9.1***
Morning Cortisol Volume	.128 (.056)	2.27*	-.038 (.046)	-.83
Awakening Cortisol Volume	.131 (.126)	1.04	-.061 (.103)	-.59

[a] Analyses are adjusted for activity patterns, social interaction, and location at Level 1 and health (body mass index, body fat, lung capacity, blood pressure), and health behaviour (exercise, smoking, alcohol consumption, dieting) at Level 2. [b] Base category is 'female'. ** $p < .01$, *** $p < .001$.

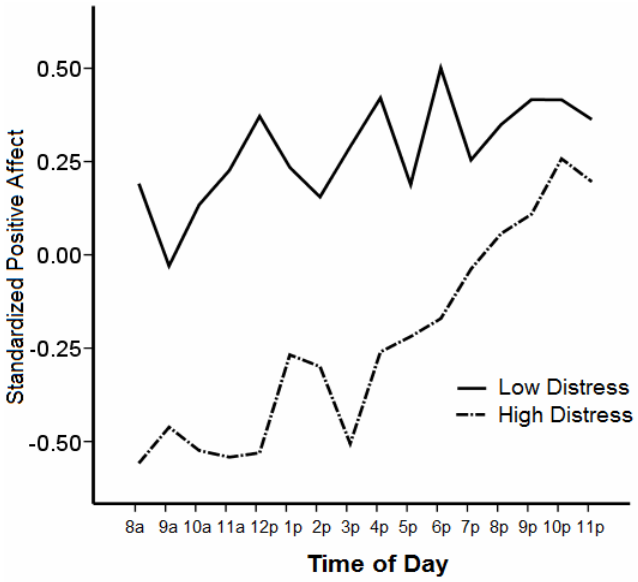
affect and other day reconstruction data (e.g. location & activity information) was recorded (Level 1), and the person level at which the demographic, health and health behaviour, psychological distress and cortisol metrics were assessed (Level 2).

I firstly tested to see if demographic factors and the core study variables: *time of day, psychological distress, and morning cortisol levels,* predicted positive and negative affect after adjusting for control variables. The control variables included were activity patterns, social interactions and location details at the episode level as well as demographic factors, baseline health and health behaviour variables at the level of the individual. Age and father's education were largely unrelated to affect. Females were found to experience more positive affect than males, as shown in Table 6. In line with previous studies (e.g. Stone et al., 2006), positive affect increased across the course of the day whilst negative affect declined markedly. Distressed participants experienced high levels of negative affect and low positive affect as anticipated. The morning volume of cortisol was somewhat predictive of positive affect (B = .128, SE = .056, t = 2.27, $p < .05$), as illustrated in Table 6. In additional analyses, I showed that morning cortisol levels and psychological distress were unrelated (r = -.04, p = .56).

Following this, I tested the three interaction effects that corresponded to the main hypotheses of the study. Demographic variables and control variables were included in each analysis as were lower order interaction effects for the three-way interaction analyses. Firstly, I examined the relation between psychological distress and diurnal rhythms of affect. Participants with high levels of psychological distress were found to demonstrate a clear pattern of lower positive affect in the morning than the evening (i.e. *psychological distress* × *time of day* interaction), as shown in Table 7 and illustrated in Figure 5a. Tests for the simple slopes suggested that the relationship between time of day and an increase in positive affect was stronger for those with above average psychological distress, (B = .031,

SE = .006, t = 5.31, p < .0005), than for those with below average levels of distress, (*B* = .011, SE = .004, t = 2.54, p < .05). Confidence intervals for the simple slopes confirmed that the slope for positive affect across the course of the day was steeper for those with high psychological distress (.031 ± .012) than for participants with low psychological distress (.011 ± .008).

(a)



(b)

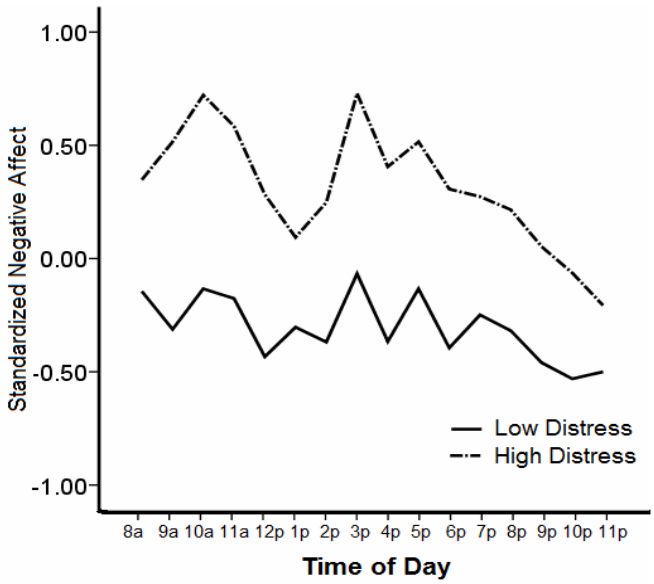


Figure 5. Standardized (a) positive and (b) negative affect as a function of time of day for participants with low and high psychological distress.

The large differences in positive affect between distressed and non-distressed participants early in the day diminished as the day progressed and converged in the evening, as anticipated. Distress also interacted with time of day to predict negative affect. Distressed people showed a significant decrease in negative affect from morning through evening (B = -.014, SE = .007, t = -2.15, p < .05) whereas the non-distressed did not (B = -.007, SE = .005, t = -1.43, p = .15). However, the confidence intervals for the simple slopes of those with high distress (-.014 ± .013) and those with low distress (-.007 ± .01) overlapped substantially indicated that the two slopes were not different.

Table 7

Summary of multilevel models testing study hypotheses relating to the association of time of day, psychological distress, and cortisol levels with positive and negative affect adjusting for covariates

	Dependent Variable			
	Positive Affect		Negative Affect	
Hypothesized Interaction [a]	B (SE)	t	B (SE)	t
1. Psychological Distress × Time of Day	.002 (.0004)	4.56***	-.001(.0004)	-2.79**
2. Morning Cortisol × Time of Day	-.0205 (.005)	-3.74***	.02 (.006)	3.29***
3. Psychological Distress × Morning Cortisol × Time of Day [b]	-.002 (.0006)	-3.42***	.001 (.001)	1.15

[a] Analyses are adjusted for time of episode, social interaction, activity patterns and location at Level 1 and psychological distress, morning cortisol levels, demographic factors (age, gender, fathers education), health (body mass index, body fat, lung capacity, blood pressure), and health behaviour (exercise, smoking, alcohol consumption, dieting) at Level 2.

[b] Analyses are adjusted for two-way interaction effects in addition to control variables.

** p < .01, *** < .005

I then tested to see if people with low morning cortisol levels experience low positive and high negative affect levels in the morning relative to the evening (i.e. *cortisol × time of day* interaction). As expected those with a low volume of morning cortisol experienced low levels of positive affect in the morning and then subsequently converged with people with high morning cortisol in the evening, as shown in Table 7 and illustrated in Figure 6a. More precisely, the relationship between time of day and positive affect was positive and highly significant for those with a below average volume of morning cortisol (B = .039, SE = .006, t = 6.03, p < .0005) but non-significant for those with an above average volume of morning cortisol (B = .006, SE = .004, t = 1.48, p = .14). Morning volume of cortisol also interacted significantly with time of day to predict negative affect. Whilst the interaction effect identified was not easily interpretable from an inspection of the graph (Figure 6b), those with a low volume of morning cortisol did appear to experience a decrease in negative affect throughout the day (B = -.021, SE = .0066, t = -3.12, p < .005) whereas those with a high volume of morning cortisol did not (B = -.005, SE = .005, t = -.94, p = .35).

Next, I examined the link between the cortisol awakening response and levels of affect in the morning and evening. There was no interaction between the waking response and time of day in predicting either positive (B = -.011, SE = .009, t = -1.3, p = .19) or negative affect (B = -.007, SE = .01, t = -.7, p = .48). Similarly, the cortisol awakening response did not interact with psychological distress and time of day to predict either positive (B = -.005, SE = .005, t = -.94, p = .35) or negative affect (B = .0006, SE = .001, t = .67, p = .51).

To summarize, the initial interaction analyses clearly showed that low levels of positive affect in the morning were predicted by both high psychological distress and a low volume of cortisol upon waking. There was also some tentative evidence that those with

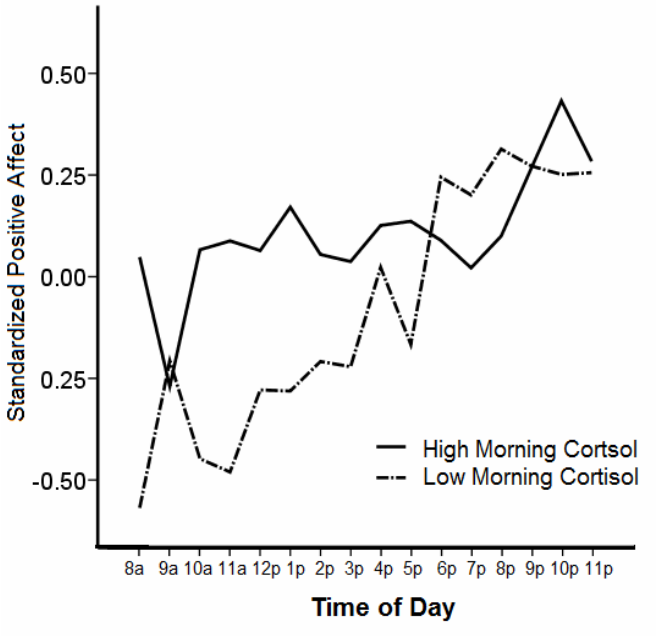
high psychological distress and a low volume of morning cortisol experience high negative affect in the mornings that diminishes as the day progresses.

Finally, I sought to show that the results from the initial two interaction effects were interlinked. More precisely, I expected that people with high psychological distress and low morning cortisol would experience the steepest increase in positive affect and potentially decrease in negative affect during the day (i.e. *psychological distress* × *cortisol* × *time of day* interaction). As expected, participants with above average levels of distress and with lower than average morning cortisol levels were the only group to experience a highly significant trend towards low positive affect early in the day followed by a sharp rise in positive affect (B = .052, SE = .009, t = 5.4 p < .0005), as shown in Table 8 and illustrated in Figure 7a.

Those with both lower than average psychological distress and morning cortisol were the only other group for which there was a statistically significant relationship between time of day and positive affect (B = .018, SE = .008, t = 2.1p < .05), as illustrated in Figure 7b. However, the slope of the relationship between time of day and positive affect was stronger for those with low morning cortisol and high psychological distress (.052 ± .018) than for those with both low psychological distress and morning cortisol (.018 ± .016) as hypothesized.

Apparently low awakening cortisol levels predict a particularly low trough in positive affect levels for the distressed in the mornings. But this group subsequently goes on to feel more positive as the day progresses and may even surpass the positive affect levels of distressed people with high morning cortisol by the evening. Non-distressed people with low morning cortisol did experience a rise in positive affect during the day, but this increase was smaller in magnitude than the increase in the low cortisol - high distress group. No such three-way interaction was identified for negative affect.

(a)



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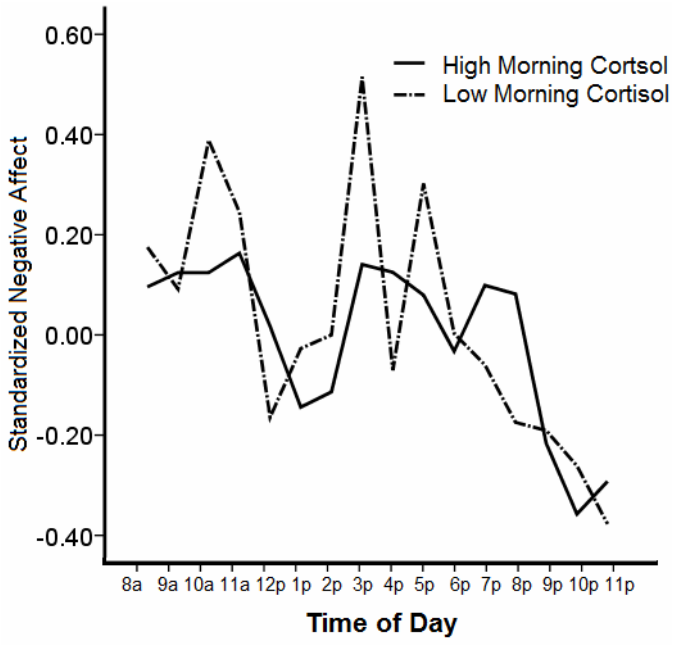


Figure 6. Standardized (a) positive and (b) negative affect as a function of time of day for participants with high and low morning cortisol levels.

Next, I examined the relation between awakening cortisol levels and affect as a function of time of day and psychological distress. These analyses revealed that awakening cortisol levels did not interact with psychological distress or time of day to predict positive or negative affect. There was some evidence for a three way interaction between

awakening cortisol levels, time of day and psychological distress, B = .002, SE = .009, t = 2.1, $p < .05$. However, this result failed to meet the Bonferronni corrected alpha level and was therefore not examined further.

4.4. Discussion

The present results indicate that there are predictable patterns in diurnal rhythms of affect that can be explained by both psychological and biological factors. Consistent with previous reports, in the sample as a whole I found that positive affect increased considerably throughout the day and negative affect declined slightly (Stone et al., 2006). However, although these patterns have been identified in healthy people and appear to be robust, average changes in affect may mask the effect of important individual differences that could explain emotional fluctuations.

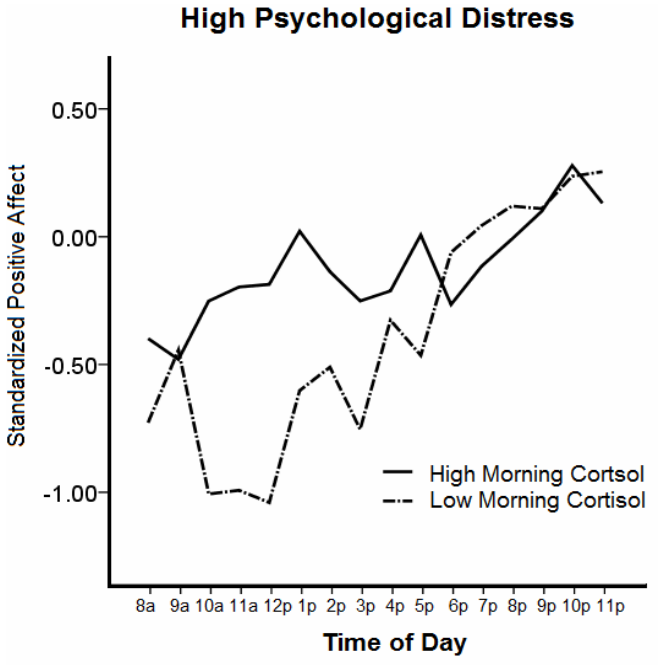
Although it is clear that distressed people are likely to feel worse than the non-distressed throughout a given day, the diurnal trend in the emotions of the distressed is perhaps less obvious. Using a measure of psychological distress composed of depression, anxiety, and stress scores I showed that distressed people experienced a clear trend towards low positive affect and to some extent high negative affect in the mornings relative to the evenings. It could therefore be the case that the diurnal changes in affect observed across numerous studies (e.g. Murray, 2007; Stone et al., 2006; Peeters et al., 2006), may be a direct result of large fluctuations in affect among certain groups such as the distressed. Moreover, prior observations of "morning worst" mood in distressed people have lead commentators to suggest that such changes in affect may have a physiological basis grounded in systems that follow a circadian rhythm (Axelsson, Akerstedt, Kecklund, Lindqvist, & Atterfors, 2003).

4.4.1. Cortisol, affect patterns, and distress

The present study also found that morning cortisol was of crucial importance to affect levels early in the day. I analyzed the total cortisol output in the first half hour after waking and the increase in cortisol from waking to half hour post-waking, two important marker sof neuroendocrine functioning (e.g. Chida & Steptoe, 2009b; Pruessner et al., 2003). People with a low total cortisol output in the first half hour after waking had low positive affect and high negative affect in the morning which improved substantially over the course of the day, converging with those with a high volume of morning cortisol late in the evening. This result links well with earlier findings showing that people with a morning personality have higher wakeup cortisol levels than evening types (Bailey & Heitkemper, 1991; Kudielka et al., 2007). In particular, the strong association between low levels of awakening cortisol and diminished positive affect is also consistent with research showing that groups with generally diminished energy (e.g. chronic fatigue, burnout) and low mood levels (e.g. post-traumatic stress, melancholic depression) tend to have reduced cortisol levels in the period after waking (Boksem & Tops, 2009). In addition, the analyses suggest that the divergent diurnal patterns of affect predicted by morning cortisol levels were unlikely to be explained by activity patterns, environmental changes or baseline health or health behaviour differences.

Perhaps most important, I found that psychological distress, morning cortisol, and affect patterns were interlinked. I showed a clear shift in positive affect from low morning levels to a much improved evening state among distressed people with low cortisol levels in the period after waking. This finding draws together two parallel streams of research: the first demonstrating that distressed people are likely to experience a steeply increasing pattern of positive affect during the day (Murray, 2007; Peeters et al., 2006) and the second

(a)



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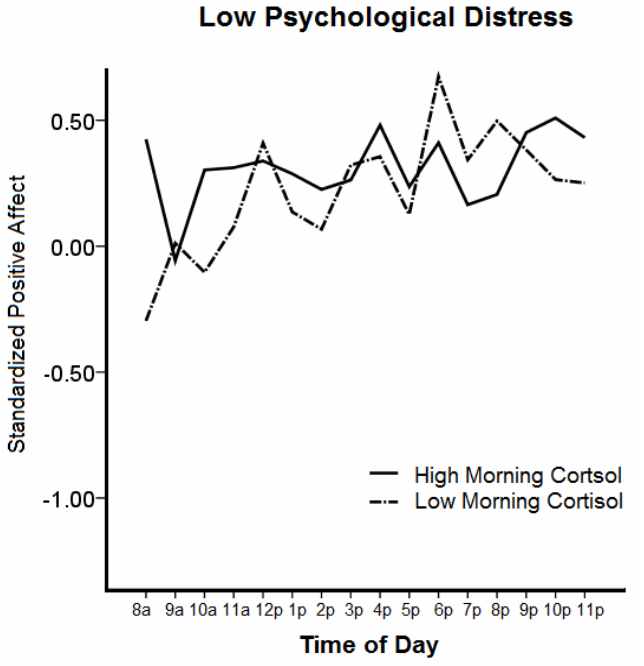


Figure 7. Standardized positive affect as a function of time of day and morning cortisol levels for those with (a) high and (b) low psychological distress.

showing that reduced volumes of morning cortisol are linked to a dislike of the initial hours of the day (e.g. Kudielka et al., 2006) and to clinical conditions characterized by low energy and mood (e.g. Fries et al., 2005). I suggested that psychological distress whilst not

strongly associated with morning cortisol levels may interact with cortisol levels to profoundly influence affect. In line with this idea, the distressed appear to be particularly vulnerable to a physiological susceptibility to low positive affect early in the day, as indexed by diminished awakening cortisol levels.

If the finding from the current study can be replicated they will have several implications. For instance, distressed people who experience a marked evening improvement in mood on a daily basis may postpone seeking treatment, as has been noted to occur in "morning worse" depression (Carpenter, Kupfer, & Ellen, 1986). It is possible that the exogenous administration of cortisol to those with deficient morning levels, may stimulate an enhancement of positive mood (e.g. Tops et al., 2006) and reduce negative affect (e.g. Putman, Hermans, Koppeschaar, van Schijndel, & van Honk, 2007). The growing field of chronotherapeutics is likely to offer numerous non-pharmaceutical treatments for disruptions in affective rhythms such as light therapy and manipulations of the sleep-wake cycle (e.g. sleep deprivation) which may assist in normalizing the cortisol cycle (Wirz-Justice, 2006).

4.4.2. Limitations

Three limitations of this study should be noted. Firstly, experience sampling techniques for the ambulatory assessment of mood may yield more high frequency and potentially more accurate results than the day reconstruction survey. However, as the day reconstruction survey is designed to be exogenous to the period under scrutiny and to minimize recall bias I felt it represented the optimal trade-off between respondent burden and ecological validity. Secondly, whilst I took measures to ensure compliance with the saliva sampling protocol, I relied on the accuracy of the participants self-reports which may overestimate actual compliance (Broderick, Arnold, Kudielka, & Kirschbaum, 2004). Finally, it is unclear that the present data can be generalized to representative samples or

indeed clinical conditions. For example, as the activity of the hypothalamus–pituitary–adrenal– axis varies substantially over the life-course (e.g. Saxbe, 2008), the results of this study may not be generalizable to older adults. Also, groups such as hospitalized psychiatric patients may differ qualitatively in their affective and psychobiological functioning from healthy people with mild or moderate affective disturbances (Stetler & Miller, 2005).

4.4.3. Conclusions

Despite these limitations, the current data provide strong evidence linking several existing strands of research. I replicated research showing a "morning worse" affect pattern to be a prominent feature of psychological distress. In addition, I found that people with high morning cortisol tend to be happier in the morning. I extended this finding to psychological distress, showing that distress did not influence morning cortisol levels but instead interacted with cortisol output to predict positive affect. More precisely, distressed people with low morning cortisol started off the day with especially low positive affect that subsequently increased quickly. In the context of the reserve capacity model, this finding suggests that it could be the case that the model may need to be extended to incorporate the possibility that changes in psychobiological functioning may contribute substantially to diurnal patterns of mood. The naturalistic monitoring measurement protocol and multilevel modeling analytic approach utilized allowed the conditions of the relation between cortisol and positive affect to be specified (i.e. primarily in the initial hours of the day among distressed people). The design used in the current study could be extended to examine the dual associations of psychosocial resources and psychobiological functioning to affect in order to specify the main factors that contribute to affect patterns on a given day. Future research could also examine the role of genetic factors in predicting how people respond to interventions which aim to modify the psychological and biological factors underlying

diurnal rhythms of affect. This research will yield potentially critical insights into the understanding everyday rhythms of affect in both healthy people and those with affective disorders.

Chapter 5

C-reactive protein links obesity and the neurovegetative symptoms of depression in two samples

Chapter Aims

Chapter 4 showed that morning cortisol appears to predict patterns of emotion in the first half of the day, particularly among the distressed. This study provided some support for the idea that psychobiological factors may not only be influenced by emotion but may also influence emotion, a possibility that is omitted from the current reserve capacity model. Furthermore, I have suggested that the relationship between health conditions and emotion may be bidirectional and that psychobiological factors may be key mediators of this relation. In this chapter I sought to provide evidence that the unfavorable psychobiological profiles of those with health conditions may lead to emotional changes. More precisely, I aimed to identify if it is feasible that obesity may give rise to high levels of inflammation which could explain the elevated levels of depressive symptoms that have been shown to characterize obesity.

Chapter Summary

This study tested to see if obesity was associated with depressive symptoms and if inflammation plays a role in explaining this association. Participants were 8190 obese and non-obese people drawn from the English Longitudinal Study of Ageing (ELSA) (46.1% male, aged 65.5 years) and the National Health and Nutrition Examination Survey (NHANES) (50.7% male, aged 48.1 years) of the American population. Obesity and C-reactive protein were positively related to total depressive symptoms and the neurovegetative symptoms of depression in both samples. Adding C-reactive protein to the regression models reduced the association between obesity and neurovegetative depressive symptoms in the ELSA sample (B = .14, SE = .03, t = 4.55, p <.001, to B = .11, SE = .03, t = 3.56, p <.001) and the NHANES sample (B = .25, SE = .065, t = 3.82, p <.001, to B = .17, SE = .068, t = 2.54, p <.05). Inflammation may partially account for the relation between obesity and the neurovegetative features of depression.

5.1. Introduction

Mounting data indicate that depressive symptoms are strongly associated with obesity (Simon et al., 2006). A recent meta-analysis identified four prospective studies examining the obesity-depression link all of which showed obesity to increase the likelihood of future depression (Atlantis & Baker, 2008). However, the key mechanisms underlying the development of depression in obese people have yet to be explicated. A central feature of obesity is low level, chronic inflammation as indexed by elevated plasma concentrations of C-reactive protein (CRP) and cytokines such as interleukin-6, and tumor necrosis factor-α (Mohamed-Ali et al., 1997; Capuron et al., 2008). Adipocytes and progressive macrophage infiltration into adipose tissue following the development of obesity contribute considerably to low-grade inflammation (Xu et al., 2003).

Cytokines typically function to orchestrate biological changes to remove pathogens and assist in tissue repair. However, peripheral proinflammatory cytokines can access the brain and influence neuroendocrine functioning and mood-relevant neurotransmitters producing mental health effects (Miller et al., 2009). For instance, CRP, a marker of systemic inflammation, has been shown to prospectively predict the development of depression (Diehl, 2002; Gimeno et al., 2008). In addition, studies examining the effect of cytokine administration in both healthy individuals and cancer patients have shown the activity of brain pro-inflammatory cytokine signaling to lead to sickness behaviour (Dantzer et al., 2008). Sickness behaviour is characterized by fatigue, changes in appetite, and sleep disturbances and closely resembles the neurovegetative symptoms of depression.

A recent study has shown elevated CRP and interleukin-6 levels to partially mediate between the metabolic syndrome, a condition directly linked to abdominal obesity, and the neurovegetative symptoms of depression (Capuron et al., 2008). A previous study found no evidence supporting inflammation as a mediator between adiposity and

depression but this may have been due to a failure to separately analyse the neurovegetative symptoms of depression (Miller, Freedland, Carney, Stetler, & Banks, 2003). This study aims to identify if raised levels of the inflammatory marker CRP may potentially result in the behavioural symptoms of depression in obese participants, with neurovegetative symptoms predominating over mood-based or cognitive symptoms.

5.2. Methods

5.2.1. Participants

The first group of participants was drawn from wave 2 of the English Longitudinal Study of Ageing (ELSA) carried out in 2004. This sample is representative of English citizens aged 52 and over. There were 9433 interviews in wave 2 of ELSA, of which 7666 people completed a nurse visit and a questionnaire. A further 2733 participants were excluded because of incomplete anthropometric or blood data, or elevated CRP levels (>10mg/L) which may be indicative of acute infection (e.g. Myers et al., 2004). This left a final sample size of 4933 people (46.1% male, aged 65.5 ± 9.3 years). Participants with complete data were younger (65.5 vs 69 years) and more likely to be male (46.1% vs 43.1%), but did not differ on socioeconomic status compared to those excluded.

The second set of participants was a national probability sample of adult Americans drawn from the National Health and Nutrition Examination Survey (NHANES) conducted between 2005 and 2006 (CDC, 2009). There were 5563 adult participants interviewed for the initial survey and a subsample of 4836 completed a depression screener. A further 1579 of these participants were lost due to incomplete data or elevated CRP levels leaving a final sample of 3257 participants (50.7% male, aged 48.1 ± 18.7 years). Those with complete data did not differ in age from those with incomplete data but were more likely to be male (50.7% vs 41.7%) and better educated.

5.2.2. Assessment of symptoms of depression

In the ELSA sample depressive symptoms were assessed by an abbreviated version of the Centre for Epidemiological Studies Depression (CES-D) scale (M = 1.4, SD = 1.84, Cronbach's alpha = .789), an 8-item self-report symptom questionnaire 0 to 8 (Radloff, 1977). The three items of the CED-S which relate to the neurovegetative symptoms of depression (two items relating to fatigue and one to sleep disturbances) were combined. Confirmatory principal components factor analysis with varimax rotation showed that these three items loaded heavily onto a single factor which explained 12.5% of the variance in depression scores (Cronbach's alpha = .602). The remaining five items loaded onto a mood-based factor describing feelings of loneliness, sadness, depression, happiness, and enjoyment of life. The mood factor explained 42.6% of the variability in depression scores and the five-item scale had a reliability level of $\alpha = .77$.

The nine item version of the Patient Health Questionnaire (PHQ-9) was used to assess depressive symptoms in the NHANES sample (Kroenke, Spitzer, & Williams, 2001). Scores on the PHQ-9 scale ranged from 0 to 27 (M = 2.6, SD = 3.62, $\alpha = .822$). In previous studies the CES-D and the PHQ-9 have been shown to produce comparable results (e.g. r = 0.72; Dbouk, Arguedas, & Sheikh, 2008). Confirmatory factor analysis showed that three items from the PHQ-9 formed a factor which referred to the neurovegetative symptoms of depression (fatigue, sleep disturbances, appetite changes). These three items explained 10.8% of the variance in depression scores and were combined to form a scale ($\alpha = .68$). The remaining six items loaded onto a second factor composed of mood/cognition-based items. These items explained 43.5% of the variability in depressive symptoms and were combined to form a mood/cognition scale ($\alpha = .78$). All depression indices were analyzed as continuous variables.

5.2.3 Clinical, health, and laboratory assessment

Those with a body mass index of 30kg/m^2 or above were defined as obese. Blood samples were collected for the analysis of CRP. Systolic and diastolic blood pressures were assessed by trained research nurses. Alcohol consumption, smoking, and the presence of a recent illness were self-reported. The analysis of blood data in ELSA was undertaken by the Royal Victoria Infirmary (Newcastle-upon-Tyne, UK). The quality control guidelines followed by the laboratory are specified in the Health Survey of England technical report (Graig, Deverill, & Pickering, 2006). NHANES specimens were analysed in 28 centers across the United States. Details of the blood sample processing protocols can be retrieved from the NHANES Laboratory/Medical Technologists Procedures Manual (CDC, 2001).

5.3. Results

5.3.1. Sample characteristics

See Table 8. Approximately twenty-six percent (n = 1291) of participants from the ELSA sample and 31% of the NHANES sample (n = 1014) were classified as obese. Obese participants were younger, more likely to be female, of lower socioeconomic status and education level and had higher blood pressure than the non-obese.

5.3.2 Mediation Analyses

Obese participants reported significantly higher levels of depressive symptoms and neurovegetative symptoms in both ELSA and NHANES samples and higher levels of mood-based/cognitive symptoms in the NHANES sample (B = 1.93, SE = .062, t = 3.13, p <.005), after adjusting for control variables, as shown in Table 9. Stepwise linear regression analyses indicated that obesity was strongly associated with CRP in both samples after adjusting for potentially confounding variables including the

Table 8

Characteristics of ELSA and NHANES samples divided by the presence of obesity

	ELSA (n = 4933)			NHANES (n = 3257)		
Variables	Obese (n = 1291)	No Obesity (n = 3642)	*p* Value	Obese (n = 1014)	No Obesity (n = 2243)	*p* Value
Core study variables:						
Depressive symptom rating:						
Total	9.56	9.33	<.001	2.98	2.42	<.001
Mood-based / Cognitive	5.68	5.61	<.1	1.28	0.99	<.001
Neurovegetative	3.88	3.72	<.001	1.67	1.40	<.001
C-reactive protein (mg/dL)	3.37	2.14	<.001	3.72	2.12	<.001
Demographic:						
Age (mean years)	64.55	65.8	<.001	48.7	47.8	n.s.
Male (%)	44	47	<.001	51	49	<.001
Socio-Economic Status [a]	8.4	7.8	<.001	-	-	
Education [b]	-	-		3.23	3.37	<.01
Health behaviour:						
Alcohol Consumption (%)	63.4	71	<.001	67	72	<.05
Cigarette Smoking (%)	11.6	14.2	<.01	19.3	23.5	<.01
Clinical factors:						
Systolic BP (mmHg) [c]	139.4	134.4	<.001	126.9	121.1	<.001
Diasystolic BP (mmHg) [c]	78.9	74.8	<.001	70.9	68.2	<.001
Recent illness [d, e]	8.1	7.7	n.s.	0.34	0.29	<.01

n.s. non-significant. [a] Social Class and Socio-economic Group linked to Operational Categories of the NS-SEC, where "1 = Employers in large scale organizations" (see Appendix C). [b] Education assessed on a five-point scale, where "5 = College graduate or above". [c] Systolic and diasystolic blood pressure were measured on the right arm. [d] In the ELSA sample recent illness was defined as any respiratory infections such as influenza, pneumonia, bronchitis or a severe cold in the last three weeks. [e] In the NHANES sample recent illness was a composite score indicating if the participant had (i) a head cold or chest cold, (ii) a stomach or intestinal illness with vomiting or diarrhea, or (iii) flu, pneumonia, or ear infections in the last 30 days.

presence of a recent illness (ELSA: (B = 1.17, SE = .066, t = 17.7, p < .001, Cohen's d = .5; NHANES: B = .15, SE = .008, t = 18.85, p <.001, Cohen's d = .66). CRP was positively associated with total depressive symptoms and only neurovegetative depressive symptoms in the ELSA and NHANES samples, as shown in Table 9.

Table 9

Summary of regression models of the association of C-reactive protein and obesity with depressive symptoms in ELSA and NHANES samples

	Dependent Variable							
	ELSA Depression				NHANES Depression			
	Total score		Neurovegetative symptoms		Total score		Neurovegetative symptoms	
	β	t	β	t	β	t	β	t
Obesity Analysis [a]:								
Step 1								
Obese	.037	2.65**	.064	4.54***	.065	3.96***	.063	3.82***
Step 2								
Obese	.031	2.14*	.052	3.56***	.051	2.98***	.044	2.54*
CRP	.026	1.76†	.051	3.47***	.046	2.69**	.065	3.75***
CRP Analysis [a]:								
Step 1								
CRP	.035	2.49*	.063	4.53***	.065	3.97***	.08	4.89***

CRP, C-reactive protein. [a] The models are adjusted for age, gender, education (NHANES only), socio-economic status (ELSA only), alcohol consumption, smoking, blood pressure, and recent illness. *** p<.005, ** p<0.01, * p<0.05, † p<0.1

Finally, the relationship between obesity and the neurovegetative symptoms of depression were reduced somewhat in the ELSA (B = .14, SE = .03, t = 4.55, p < .001, to B = .11, SE = .03, t = 3.56, p < .001; Cohen's d reduced from .13 to .1) and the NHANES (B = .25, SE = .065, t = 3.82, p < .001, to B = .17, SE = .068, t = 2.54, p < .05; Cohen's d reduced from .13 to .09) samples following the inclusion of CRP in the model. The results indicated support for CRP as a mediator between obesity and the neurovegetative symptoms of depression in the ELSA sample (Sobel z = 3.41, p < .001), and in the NHANES sample (Sobel z = 3.68, p < .001).

5.4. Discussion

The findings from the present study demonstrate that obesity is associated with depression and specifically with the neurovegetative symptoms of depression. Consistent with previous data (Trayhurn & Wood, 2004) this study found obese participants to exhibit raised levels of the acute phase reactant CRP. Obesity and CRP exhibited a consistent relationship with total depressive symptoms and the neurovegetative symptoms of depression in both samples. Crucially, adding CRP to the model of the relationship between obesity and neurovegetative depressive symptoms attenuated that relationship. The inflammatory response, as indexed by CRP, thus met the criteria for mediation between obesity and neurovegetative depressive symptoms.

CRP may be indicative of the causal effect of its primary inducer interleukin-6 that can access the brain causing sleepiness and fatigue (Mohamed-Ali et al., 1997). Examining a broad set of inflammatory biomarkers in future studies will assist in specifying the inflammatory pathways from obesity to depression (Capuron et al., 2008). A large portion of variance in levels of inflammatory biomarkers, such as interleukin-6, is explained by anthropometric measurements (e.g. waist to hip ratio, body mass index) (Yudkin, Kumari, Humphries, & Mohamed-Ali, 2000). This robust observation combined with studies

showing the administration of cytokines to lead to a dose-dependent elevation in neurovegetative depressive symptoms, instantiates a causal role of inflammation in the development of depression amongst obese individuals (Dantzer et al., 2008). Future multi-wave prospective studies will be needed to delineate the association between excessive activation of innate immunity in obesity and depressive symptoms. In conclusion, data from two large-scale population-based samples indicated that a marker of inflammation, C-reactive protein, mediates between obesity and the neurovegetative symptoms of depression. This research provides tentative support for integrating the proposed bidirectional relationship between health conditions and emotion into an extended version of the reserve capacity model. Specifically, it is possible that the inflammation caused by illness could directly induce affect changes which may potentially fuel further stress-related declines in health. Further research is required to specify how psychosocial resources and genetic factors interact with the presence of inflammation to determine affect patterns and reactivity to the presence of stressors.

Chapter 6

Heredity links natural hazards and human health: Apolipoprotein E gene moderates self-rated health in earthquake survivors

Chapter Aims

In addition to incorporating causal pathways from health to emotion in the reserve capacity model I have suggested that genetic factors warrant consideration. This is because recent evidence in psychiatry and psychology suggests that identifiable genetic variation may modify the link between stressors and mental health outcomes. Within the context of the reserve capacity model it is feasible that, like psychosocial factors, individual differences in genotype could modify the relation between stressors and emotion and thus lead to changes in health. In this chapter I aim to broadly test this possibility by examining the link between an extremely stressful life event and subsequent health and identifying if genetic variation modifies this relation.

Chapter Summary

Prior research has shown that genetic factors interact with life-stressors to produce decrements in physical and mental health. Evidence from clinical, neuroimaging, and transgenic mice studies suggests that carriers of the apolipoprotein ε4 allele show detrimental physiological and psychological responses to stress. As genetic variation could conceivably cause the stressors previously examined, this study aimed to investigate the role of genetic factors in moderating the influence of an exogenous stressor, an earthquake, on self-rated health. A single gene was assessed and examined using a 'natural experiment' design where the interaction between the presence of the apolipoprotein E ε4 allele and the level of objective and subjective exposure to a devastating earthquake was specified in a population-based cohort of elderly Taiwanese (N = 755). Non-comparative Self-rated Health was the main outcome measure assessed (Eriksson, Unden, & Elofsson, 2001). Those who experienced damage to their property or were forced to move from their homes (high objective exposure) had low self-rated health a year later, only if they were ε4 carriers. Similarly, those who found the earthquake severely distressing (high subjective exposure) were shown to have low self-rated health a year later only if they possessed the apolipoprotein ε4 allele. These findings suggest that genetic variation in the apolipoprotein gene may modify the health effects of the exogenous stress of natural disaster exposure. The results support a model of health where one's genetic endowment and environmental stressors interact to produce resilience and thriving or distress and illness.

6.1. Introduction

The concept of gene × environment interaction is central to diathesis-stress models which aim to explain people's varied responses to stress. However, until recently such models have tended to be descriptive rather than explanatory. Developments in the assessment of variation in highly specific aspects of the human genome have made it possible for researchers in behavioural science to show gene × environment interactions using measured genes (Moffitt et al., 2006). This study was based on the idea that genetic factors may not necessarily directly affect health; rather our genetic endowment can interact with environmental stressors to change physical and mental health. The study examined the effects of exposure to a devastating earthquake that occurred in Taiwan in 1999 using data from the 2000 Social Environment and Biomarkers of Aging Study (SEBAS). The 'Chi-Chi' earthquake, measuring 7.3 on the Richter scale, caused more than 2300 deaths, collapsed over 100,000 homes and sent thousands of aftershocks through the region in the weeks that followed (Lin, Huang, Huang, Hwang, Tsai, & Chiu, 2002). I sought to show that a high level of exposure to the earthquake had a detrimental effect on self-rated health (SRH) of elderly Taiwanese people, conditional on the apolipoprotein E genotype (APOE).

6.1.1. Self-rated health

SRH implies a global subjective assessment of one's health and is thought to be a broad index of biopsychosocial functioning (Fayers & Sprangers, 2002). Low SRH is indicative of a range of unfavorable influences including symptoms of illness, stressful life events, depression, and social isolation (Kaplan & Camaeho, 1983). SRH is a key outcome measure as it robustly predicts mortality in elderly people, with some studies finding SRH to be a more powerful predictor of mortality than medical diagnoses (Idler & Benyamini, 1997). SRH has been shown to be detrimentally affected by traumatic experiences such as

the death of a child, sexual abuse and war exposure (Kendall-Tackett, 2002). For example, a meta-analytic review showed that those who have been sexually victimized as children or adults are more likely than non-abused controls to have a low level of SRH (Golding, Cooper, & George, 1997). Similarly, SRH over a 20-year period was negatively affected by war exposure in a sample of veterans (Benyamini, Ein-Dor, Ginzburg, & Solomon, 2009).

Findings from twin studies have shown a substantial genetic component to SRH, particularly within the age bracket of 45-74 (Harris, Pedersen, McClearn, Plomin, & Nesselroade, 1992; Svedberg, Lichtenstein, & Pedersen, 2001). Prior analysis of the SEBAS data has shown that both the apolipoprotein (APOE) gene and life stress appear to directly influence SRH (Zhang, Chen, & Chen, 2008). However, recent evidence suggests that genes often do not operate in isolation, but instead interact with environmental 'pathogens' to produce their effects (e.g. Caspi et al., 2003). Thus, the APOE gene and significant life stressors may not be directly linked with SRH. Rather, they may interact to produce health effects.

The SEBAS dataset is unique in that it contains genetic information and detailed self-reported data specifying the personal impact of the 'Chi-Chi' earthquake that occurred a year previously. It thus provides unique data for a natural experiment to test to see if the APOE gene does in fact interact with stressors to produce its effect on health. I hypothesized that exposure to the exogenous environmental stress of a severe earthquake is therefore likely to have a detrimental effect on SRH, conditional on one's APOE genotype (Moffitt et al., 2006).

6.1.2. Apolipoprotein E, stress, and health

The three alleles of the APOE gene (ε2, ε3, ε4) code for protein isoforms involved in lipid metabolism and neuronal maintenance. The ε4 allele has been shown to interact

with physical injury to produce detrimental effects. For instance, the head trauma experienced by boxers, American football players, and those with a traumatic brain injury hastens cognitive decline in those with the ε4 allele (Teasdale, Nicoll, Murray, & Fiddes, 1997; Savitz, van der Merwe, Stein, Solms, & Ramesar, 2007). Evidence from human subjects and mouse models suggests that in addition to regulation of the response to physical trauma, the APOE gene is implicated in the regulation of emotional trauma (Raber, 2007; Lee et al., 2008). For instance, healthy carriers of the ε4 allele have been shown to be at elevated risk of depression following psychological stress, in comparison with those possessing the ε3/ ε3 genotype (Gallagher-Thompson, O'Hara, Simmons, Kraemer, & Murphy, 2001)

The elevated reactivity to stressors associated with the ε4 allele may be partially explained by neuropathological changes induced in the amygdala, as demonstrated in mice expressing the APOE ε4 allele and human brain atrophy studies of ε4 carriers (Raber, 2007; Cherbuin, Leach, Christensen, & Anstey, 2007; Basso et al., 2006). Augmentations in amygdala functioning are likely to disrupt the regulation of the hypothalamic-pituitary-adrenal axis, resulting in the prolonged release of glucocorticoids in response to stress which can have detrimental health effects (e.g. decreased bone density, immune suppression) (Peavy, 2008). In support of this idea elevated concentrations of the hormone cortisol have been found in stressed older human carriers of the ε4 allele (Peavy et al., 2007). Transgenic mice expressing the ε4 allele were shown to have an impaired capacity to suppress cortisol levels after receiving dexamthasone, indicating potential hypercorticolism (Robertson, Curley, Kaye, Quinn, Pfankuch, & Raber, 2005). There is thus sufficient biological and psychological evidence from neuroimaging, transgenic mice, and clinical studies to suggest that carriers of the APOE ε4 allele react adversely to stress. In summary, it appears likely that when carriers of the APOE ε4 allele encounter stressors

the 'fight or flight' physiological stress response is subsequently over-engaged which may lead to adverse physical and mental health outcomes.

6.1.3. Natural experiments and gene × environment research

Whilst current evidence indicates that identifiable gene polymorphisms may cause a dysfunctional response to stressors it is also possible that the same genes may confer an elevated likelihood of encountering stressors (Moffitt et al., 2006). Previous gene × environment interaction (hereafter G × E) studies have incorporated novel methods to statistically control for potential correlations between genetic vulnerability and environmental risk, such as adjusting for parental disposition (e.g. Caspi et al., 2003). However, as yet few G × E studies have examined the effect of exposure to an exogenous shock (e.g. earthquake, tsunami, terror attack) which cannot be attributed to one's genetic makeup.

As exogenous shocks are difficult to predict, longitudinal data that allows G × E effects to be studied are often unavailable. In the absence of longitudinal data, the natural variation in the level of exposure to an exogenous stressor is an important determinant of post-disaster health which can act as a substitute method of testing G × E effects (Norris, Friedman, Watson, Byrne, Diaz, & Kaniasty, 2002). Those highly exposed to the stressor can be contrasted with the less severely exposed. For instance, Kilpatrick and colleagues (2007) showed that when combined with low social support the low-expression serotonin transporter genotype (5-HTTLPR) predicted an increased risk of post-traumatic stress disorder among those highly exposed to a hurricane in Florida. Similarly, in two studies, polymorphisms in the RGS2 gene were shown to modify the risk of post-traumatic stress disorder (Amstadter et al., 2009) and generalized anxiety disorder (Koenen et al., 2009) contingent on participants level of exposure to a severe hurricane in Florida. In the context of a G × E study, as both the level of exposure to an exogenous stressor and the assortment

of alleles at the time of gamete formation are likely to be randomly assigned and uncorrelated with each other or with health prior to the earthquake, this design can be considered a 'natural experiment' (Moffitt et al., 2006; Giltay et al., 2009).

6.1.4. The current study

The present investigation sought to show a G × E effect where high levels of subjective and objective exposure to the Chi Chi earthquake decreased SRH among those possessing the APOE ε4 allele but not among those without this allele. Adults aged from 54 to 74 were examined as this group, with available data, fell within the age bracket where genetic factors have been deemed to have their greatest effect on SRH (Svedberg et al., 2001). To test the G × E hypothesis, an objective measure of earthquake exposure (i.e. experiencing damage to one's home, having to move from one's home) which is likely to be uncorrelated with SRH prior to the earthquake was utilized as a test of a G × E effect. Subjective ratings of the immediate fear in response to the earthquake were also used as an index of the severity of exposure and to test for a potential G × E effect. As the ε4 allele has been shown to predict a number of salient health-relevant factors, these potentially confounding variables (e.g. cognition, smoking, chronic illnesses) were included as covariates in the analyses.

6.2. Method

6.2.1. Participants

The sample (N = 755) consisted of a cohort of older adults aged 54 to 74 drawn from the 2000 SEBAS survey (for detailed information on sampling and recruitment strategy see Goldman et al., 2003). The 'Chi-Chi' earthquake occurred on September 21st, 1999 and the self-reported information on SRH and earthquake experiences was collected as part of the SEBAS survey between June and December the following year. The average

duration from the earthquake to participant assessment was approximately 12 months. In total 27 participants were lost from analyses due to incomplete questionnaire, anthropometric, or blood data, leaving 718 participants (57.2% male, mean age = 64.5 (SD = 6.2)) in the final sample. Approximately 29% of participants had no formal education, whilst a further 45.2% had primary education only. Twenty percent had completed secondary school education and 6.5% had attended college. Ethnicity was classified into Fukien (73.3%), Mainlander (13%) and Hakka (13.7%).

6.2.2. Core Study Variables

Self-rated health was assessed using a non-comparative measure (comparative measures request participants to contrast current health with previous health or the health of others) that asked participants to rate their current health on a scale from 1 = poor to 5 = excellent (Eriksson et al., 2001). The single-item measure has been shown to be a reasonable alternative to multi-item measures, producing comparable predictions of mortality, hospitalization, and other aspects of healthcare utilization (De Salvo, Bloser, Reynolds, He, & Muntner, 2006; De Salvo, Fan, McDonell, & Fihn, 2005). The scores on the SRH measure were found to be normally distributed and centered close to the scale mid-point (M = 3.14, SD = .97).

Participants were categorized as having experienced a high level of objective exposure to the earthquake if they reported that their home had been damaged by the earthquake and/or if they had reported that they had to move from their home as a result of the earthquake (18.1% of the sample). A dummy variable was produced for regression analyses with those highly exposed to the earthquake coded as 1 and with the remaining portion coded as 0.

Participants indicated their subjective level of exposure to the earthquake by rating their level of fear in response to the event on a scale from 1 = not scared to 5 = extremely scared. Subjective exposure scores were normally distributed around the scale mid-point (M = 2.93, SD = 1.27). 37.5 % of participants indicated they were 'very' or 'extremely' scared by the earthquake and were classified as highly exposed to earthquake trauma for inclusion in a 'total exposure' composite variable. In total 44.3% of participants were classified as experiencing a high level of objective and/or subjective exposure in the 'total exposure' metric. These participants were coded as 1 with the remaining 55.7% classified as experiencing a low level of exposure and coded as 0.

6.2.3. Genotyping

In the SEBAS survey one gene, the APOE gene, was genotyped using the polymerase chain reaction amplification refractory mutation system (PCR-ARMS) and polymerase chain reaction restriction fragment length polymorphism (PCR-RFLP) techniques (see Goldman et al., 2003). Dummy coding was used with those possessing at least one copy of the APOE ε4 allele coded as 1 (14.4% of the sample) contrasted with the remaining base group of non-carriers (85.6%) who were coded as 0.

6.2.4. Potentially confounding variables

6.2.4.1. Cognitive functioning

Each participant's average score on the Rey Auditory Verbal Learning Test (Lezak, 1983) and the Digits Backward test (Wechsler, 1981) were standardized and combined to produce a normally distributed metric of cognitive functioning.

6.2.4.2. Chronic Illness and Smoking

The number of chronic illnesses experienced (M = 1.62, SD = 1.46) and the use of long-term medication (59.7% currently taking medication) were detailed. As the number of chronic illnesses experienced was positively skewed this variable was transformed via a square-root transformation. The degree to which each chronic condition interfered with functioning was rated on a 3-point scale from 0 = No inconvenience to 2 = Much inconvenience (M = 1.12, SD = 1.56). Illness interference scores were also positively skewed and were transformed by a square-root transformation. Participants also indicated their smoking status (24.4% current smokers).

6.2.5. Statistical Analysis

Prior to analyses data were inspected for accuracy, and all study variables were examined for normalcy. An unadjusted regression framework was firstly used to test if genotype moderated the association between level of exposure to the earthquake and SRH. The main effects of genotype and the level of exposure to the earthquake were firstly explored and then the G × E interaction term was entered into the model. For the binary objective exposure and total exposure variables, the product of the genotype and exposure variables formed the interaction terms for analyses. As the subjective exposure measure was continuous it was converted into a standardized z-score and the product of this score and the genotype dummy variable was then used for the analysis of the genotype × subjective exposure interaction. Following this, the moderation regression analyses were repeated to test the robustness of the results to the inclusion of demographic factors (age, gender, education and ethnicity) and potentially confounding variables (cognitive function, smoking, and chronic illness) (Frazier, Tix, & Barron, 2004). Covariates were introduced in the first step of the analyses followed by the genotype, exposure variables and the interaction term in the following step.

6.3. Results

6.3.1. Preliminary analyses

T-tests were used to test if carriers of the APOE ε4 allele possessed characteristics that differed from non-carriers. There were no significant differences between APOE genotype groups on any of the demographic characteristics or potentially confounding variables assessed. Importantly, possession of the APOE ε4 allele was unrelated to the chances of being highly exposed to the earthquake on the three exposure metrics assessed (objective, subjective, total).

Demographic and control variables were used to predict exposure to the earthquake in regression analyses in order to identify non-random variation in earthquake exposure. I found that younger adults were more likely than older adults to experience objective effects, indicating a non-random component to exposure. Females were more likely than males to report experiencing high levels of fear in response to the earthquake. In the case of lower age and female gender the increased likelihood of earthquake exposure did not differ between genotypes. No other demographic or control factors predicted measures of exposure to the earthquake.

6.3.2. Gene × Environment Moderation analyses

The non-conditional main effects of the APOE ε4 allele and objective, subjective, and total levels of exposure to the Chi-Chi earthquake and SRH were firstly specified in regression analyses. Those possessing the APOE ε4 allele had marginally lower SRH (b = -.17, SE = .099, t = -1.72, p <.1) and those highly exposed to the earthquake had significantly lower levels of SRH one year later than those who were less exposed for objective (b = -.23, SE = .092, t = -2.47, p <.05), subjective (b = -.09, SE = .035, t = -2.54, p <.05), and combined exposure metrics (b = -.197, SE = .071, t = -2.76, p <.01).

Table 10

Moderation analyses of the association of objective, subjective, and combined composite earthquake exposure and APOE genotype with self-rated health

	Self-rated Health					
	Objective Exposure		Subjective Exposure		Combined Exposure	
Step and predictor	B (SE)	t	B (SE)	t	B (SE)	t
Intercept	3.143 (.043)	73.69**	3.123 (.039)	80.67**	3.162 (.052)	61.39**
Level of Exposure	-.115 (.102)	-1.13	-.054 (.039)	-1.39	-.089 (.078)	-1.14
APOE ε4 genotype	-.054 (.111)	-.48	-.165 (.099)	-1.67	.133 (.133)	1.00
Exposure × APOE ε4	-.496 (.247)	-2.01*	-.225 (.103)	-2.20*	-.638 (.197)	-3.23**

** $p < .01$, * $p < .05$

The next analysis tested if genetic vulnerability to stress moderated the association between the objective level of exposure to the earthquake and current levels of SRH. The hypothesized interaction was identified (b = -.497, SE = .247, t = -2.01, p <.05, Cohen's d = .15) and added significantly more explanatory power to the model as indexed by an F_{Change} value of 4.83 (p <.05). The interaction clearly showed that high objective exposure to the earthquake predicted a substantial decline in SRH among carriers of the APOE ε4 allele (b = -.612, SE = .215, t = -2.84, p <.01), but there was little evidence for such a decline in SRH among non-carriers (b = -.115, SE = .103, t = -1.12, p = .26), as shown in Figure 8a.

The potential G × E interaction identified in the objective exposure analysis was replicated in the subjective exposure analysis (b = -.225, SE = .103, t = -2.2, p <.05, Cohen's d = .16) as shown in Table 10. Once again, the interaction contributed significantly to variance in SRH explained by the regression model (F_{Change} = 4.03, p <.05).

The interaction showed that high levels of subjective exposure to the earthquake was linked to a marked decrease in SRH among carriers of the APOE ε4 allele (b = -.279, SE = .09, t = -3.09, p <.005). Subjective exposure, as indexed by having experienced high levels of fear as a result of the earthquake, did not appear to affect SRH in those without the ε4 allele (b = -.054, SE = .039, t = -1.38, p = .17), as illustrated in Figure 8b.

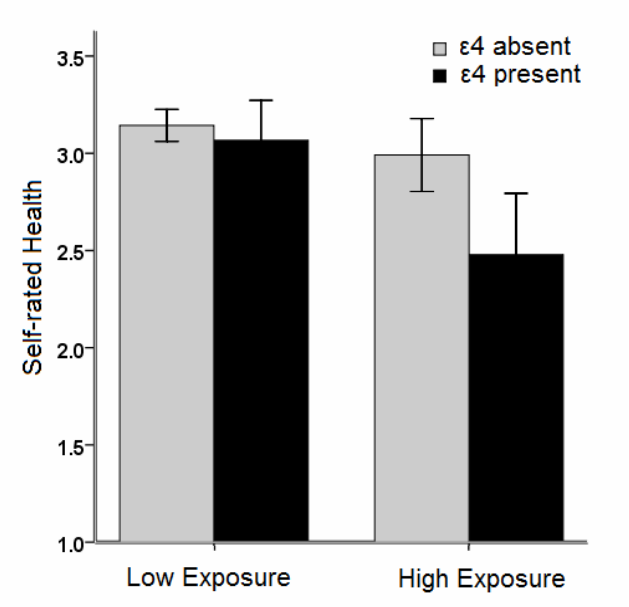
Objective and subjective exposure variables were then combined into a single metric, which classified 43.2% of participants as highly exposed to the earthquake. The hypothesized G × E was highly significant (b = -.638, SE = .197, t = -3.23, p <.005, Cohen's d = .24) and added significantly to the model (F_{Change} = 10.44, p <.005). As in the subjective and objective analyses, this interaction showed that high levels of exposure to the earthquake appeared to have a detrimental influence on SRH in APOE ε4 carriers (b = -.726, SE = .167, t = -4.35, p = <.001) but not non-carriers (b = -0.089, SE = 0.079, t = -1.13, p = .26), as illustrated in Figure 8c. Neither the APOE ε4 genotype nor the level of exposure to the earthquake remained as a significant predictor of SRH in any of the moderation models, as shown in Table 10 and Table 11.

6.3.3. Robustness Analyses

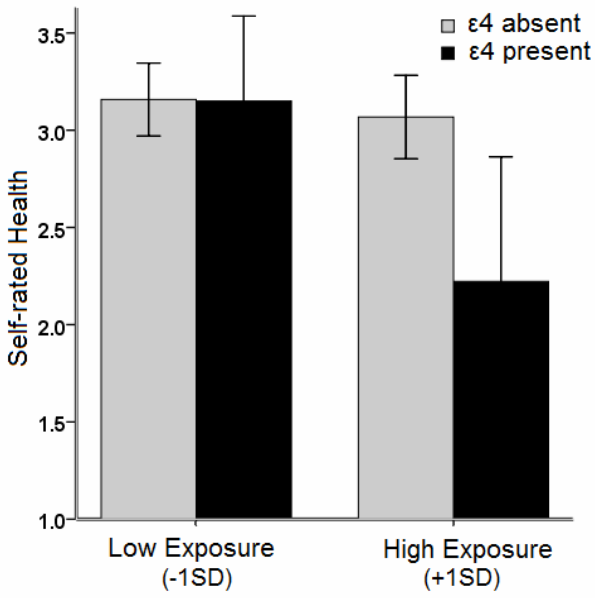
In the three moderation analyses conducted, a high level of exposure to the earthquake was shown to predict low SRH only among ε4 carriers. The addition of demographic and potentially confounding factors to the moderation analyses failed to substantially reduce the association between the exposure × APOE genotype interactions and SRH. As can be seen in Table 10 and Table 11, the subjective exposure × APOE genotype interaction remained effectively unchanged as a predictor of SRH after the inclusion of a wide array of covariates (unadjusted: b = -.225, SE = .103, t = -2.2, p <.05 to adjusted: b = -.185, SE = 0.091, t = -2.03, p <.05). The link between the objective exposure × APOE genotype interaction and SRH was marginally diminished (unadjusted: b = -.497,

SE = .247, t = -2.01, p <.05 to adjusted: b = -.375, SE = 0.217, t = -1.72, p <.1). The relationship between the total exposure × APOE genotype interaction and SRH remained highly significant (unadjusted: b = -.630, SE = 0.198, t = -3.18, p <.005 to adjusted: b = -.496, SE = 0.175, t = -2.84, p <.01).

(a) Objective Exposure



(b) Subjective Exposure



(c) Composite Measure of Exposure

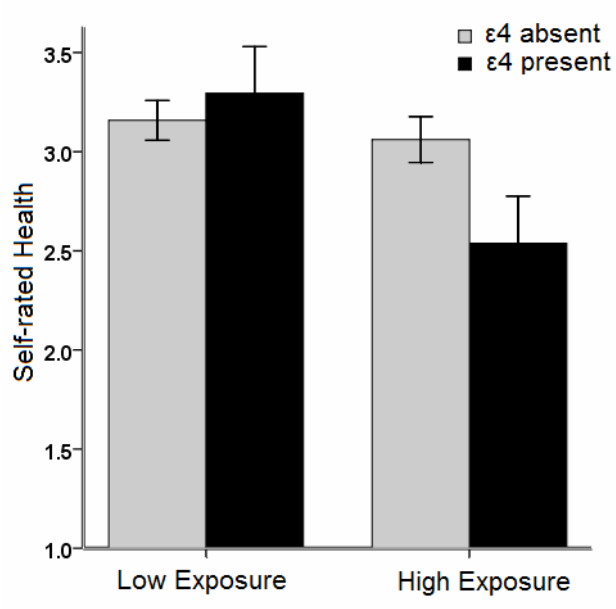


Figure 8. Self-rated health as a function of level of exposure to the earthquake by APOE genotype for (a) objective, (b) subjective, and (c) combined composite exposure variables.

6.4. Discussion

Although previous research has shown that unfavorable health outcomes can result from a combination of genetic vulnerability and early maltreatment or physical injury (e.g. Teasdale et al., 1997), little is known about the role of genetic factors in moderating how people respond to natural disaster situations. The exogenous shock of a disaster situation is by definition unrelated to a person's genetic makeup and thus offers a suitable opportunity to examine G × E interactions. The present investigation assessed the level of exposure to the 1999 Chi-Chi earthquake in a cohort of elderly Taiwanese people using both objective and subjective measures.

Table 11.

Summary of regression analysis of the association of objective, subjective, and combined composite earthquake exposure and APOE genotype with self-rated health adjusting for demographics and potentially confounding variables

	Self-Rated Health					
	Objective Exposure		Subjective Exposure		Combined Exposure	
Step and predictor	B (SE)	t	B (SE)	t	B (SE)	t
Intercept	3.19 (.402)	7.93**	3.084 (.402)	7.67**	3.120 (.404)	7.72**
Step 1[a]						
Age	.001 (.006)	.19	.001 (.006)	.19	.001 (.006)	.19
Male	-.006 (.077)	.08	.006 (.077)	.08	.006 (.077)	.08
Education	.124 (.045)	2.73**	.124 (.045)	2.73**	.124 (.045)	2.73**
Mainland[b]	.164 (.093)	1.76†	.164 (.093)	1.76†	.164 (.093)	1.76†
Hakka[b]	-.018 (.105)	-.17	-.018 (.105)	-.17	-.018 (.105)	-.17
Chronic Illnesses	-.123 (.043)	-2.85**	-.123 (.043)	-2.85**	-.123 (.043)	-2.85**
Illness Inconvenience	-.274 (.041)	-6.62**	-.274 (.041)	-6.62**	-.274 (.041)	-6.62**
Cognitive functioning	.079 (.043)	1.83†	.079 (.043)	1.83†	.079 (.043)	1.83†
Medication	-.178 (.083)	-2.16*	-.178 (.083)	-2.16*	-.178 (.083)	-2.16*
Smoking	.066 (.082)	.80	.066 (.082)	.80	.066 (.082)	.80
Step 2						
Level of Exposure	-.144 (.091)	-1.59	-.022(.036)	-.61	-.031 (.072)	-.43
APOE ε4 genotype	-.053 (.097)	-.54	-.139 (.087)	-1.59	.091 (.118)	.77
Exposure × APOE ε4	**-.375 (.217)**	**-1.72†**	**-.185 (.091)**	**-2.03***	**-.496 (.175)**	**-2.84****

[a]This preliminary step does not differ between exposure groups (subjective, objective, total), [b]Base category for ethnicity analysis refers to those of Fukien origin. ** $p < .01$, * $p < .05$, † $p<0.1$.

Self-rated health was markedly reduced in APOE ε4 carriers for whom the event had personal objective consequences and for those who subjectively perceived the earthquake as severely disturbing. On average the SRH of ε4 carriers who were highly exposed to the earthquake was half a standard deviation lower than the SRH of highly exposed non ε4 carriers. This difference was equivalent to more than a half-point difference in SRH which meant the earthquake appeared to shift the highly exposed ε4 group from 'average' health, towards 'not so good' health. Importantly, in the absence of considerable exposure to the earthquake the ε4 allele appeared to contribute little to a decline in SRH. The effect of exposure to the earthquake indicates that a traumatic event in itself or genetic susceptibility alone may not be enough to cause negative effects. Instead, the analysis suggests a complex relation between SRH and genetic factors where a person's genotype *only in combination* with the severity of their exposure to stressors may act to modify the risk of detrimental health outcomes.

The subjective exposure measure assessed the degree to which participants were afraid during the aftermath of the earthquake. Interestingly, carriers of the ε4 allele were equally as fearful as non-carriers. However, feeling afraid at the time of the earthquake predicted low SRH a year later among ε4 carriers but not non-carriers. Several studies have shown that the effects of trauma unfold over many years (Ben-Zur & Zeidner, 2009). For instance, a separate study of survivors of the Chi-Chi earthquake showed suicidality to increase from 4.2% at six months after the earthquake to 6% at three years (Chou et al., 2007). It may be the case that a restricted capacity to respond to challenges to the central nervous system could lead to a worsening health status among APOE ε4 carriers (Gallagher-Thompson et al., 2001; Peavy, 2008). Consistent with this idea experiencing earthquake damage or having to move home predicted substantial decrements in SRH one

year later only among carriers of the ε4 allele. Once again, this G × E result suggests that it is the exposure to trauma that has a detrimental effect on ε4 carriers.

Several limitations to the current study warrant comment. The retrospective assessment of participants' response to the earthquake may have been influenced by memory distortions, bias, and normal forgetting (Moffitt et al., 2006). Nonetheless, these potential confounds would impact on the results only if they produced estimates of exposure that varied by genotype which did not appear to be the case. The design of the present investigation was based on the idea that longitudinal data, while preferable, is not essential to examine a G × E effect on physical or mental health. This is because a natural experiment can be constructed if genetic variation (e.g. possession of the ε4 allele) and the level of exposure to an exogenous stressor (e.g. an earthquake, terror attack) are likely to be randomly assigned and uncorrelated with each other and with pre-earthquake health. In the absence of longitudinal data the possibility remains that SRH may have been associated with the APOE ε4 allele prior to the earthquake. However, the existence of such a relationship would not explain why low levels of SRH were only observed among the group who possessed the risk ε4 allele and were highly exposed to the earthquake.

Whilst the results suggest that the APOE genotype could be considered a quasi-experimental independent variable (Giltay et al., 2009), there was some evidence of non-randomness in exposure to the earthquake. Younger adults were more likely to experience objective exposure to the earthquake, potentially reflecting non-random factors other than pre-earthquake health, such as age-related trends in home occupancy. The possibility that less healthy female ε4 carriers experienced an elevated subjective impact of the earthquake is potentially more problematic for the results. However, as the increased likelihood of high subjective exposure found among females did not differ by genotype this explanation is unlikely. Nonetheless, future multi-wave studies will assist in providing essential tests

for replication of the identified results and in demonstrating the within-person change in the health of ε4 carriers resulting from stress exposure (e.g. Munafo et al., 2009; Risch et al., 2009).

Future studies should also investigate pathways through which the identified trauma exposure × APOE genotype interaction may influence SRH. APOE ε4 carriers have demonstrated elevated levels of anxiety, a trait characterized by a propensity towards rumination and persistent physical hyperarousal (Raber, 2007; Grootendorst, de Kloet, Vossen, Dalm, & Oitzl, 2001). It is feasible that trauma could induce these psychological states which in turn may enhance sensitivity to physical symptoms and promote illness behaviour (Schnurr & Jankowski, 1999). A diminished sense of mastery and personal control may also follow trauma and lead to poor health (Krause, Shaw, & Cairney, 2004). It is possible that the threat to life that is often inherent in traumatic experiences could cause people to engage in riskier health behaviours such as excessive drinking (Ben-Zur & Zeidner, 2009). Trauma may also have effects on biological function and health, altering the body's response to stress by heightening noradrenergic function, influencing the hypothalamic-pituitary-adrenal axis and activating immune-mediated inflammation (Peavy et al., 2007; Schnurr & Jankowski, 1999).

Rather than supporting a model where genetic factors lead to poor health, the findings from this study suggest that exogenous stressors can interact with genetic vulnerability to explain trajectories of physical and mental health. During an individual's lifetime they are likely to experience at least one potentially traumatizing event and carriers of high risk polymorphisms, such as the APOE ε4 allele, may be susceptible to subsequent negative health outcomes (Bonanno, Galea, Bucciarelli, & Vlahov, 2006). It is therefore important to examine the interaction between genetic and psychological factors in coping with traumatic exposure to disaster situations.

The results suggest that while both carriers and non-carriers of the APOE ε4 allele experienced the Chi Chi earthquake as equally disrupting and fear provoking, those with the APOE ε4 allele appraised and/or reacted to the event in a way that produced lower SRH. A better understanding of whether the APOE ε4 allele is involved in psychological appraisal mechanisms and/or emotional and health-related reaction mechanisms would be helpful. This knowledge could be used to design randomized treatment experiments to both prevent post-exposure health decline in those at genetic risk and help extend the frontier of our understanding of the determinants of outcomes following adversity.

The findings from this study provide support for the idea that, like psychosocial resources, genetic factors may be considered to buffer the health effects of stressors within the reserve capacity model. However, a number of questions remain such as: (i) whether the vulnerability associated with a risk genotype may convert into decrements in objective measures of physical health following stress, (ii) what mediating factors (e.g. emotion, behaviour, psychobiological functioning) explain how risk genes lead to health outcomes in the presence of stressors, and (iii) do psychosocial resources further interact with gene-environment interactions to produce mental and physical health outcomes. I now turn to the later two questions which are the focus of the final empirical chapter of this thesis.

Chapter 7

Sickness without sorrow: the role of perceived control and the apolipoprotein E gene in psychological adjustment to chronic illness in a population-based cohort of elderly Taiwanese

Chapter Aims

This chapter aimed to examine several core tenets of the reserve capacity model and to test the idea that identifiable genetic variation may modify the relation between stressful circumstances and cognitive/emotional factors, thus warranting consideration as an additional component in the reserve capacity model. Specifically, this study evaluated the likelihood that variation in the APOE gene may modify psychological adjustment to the stress of chronic illness. Similarly, the intrapersonal psychosocial resource, perceived control, was predicted to attenuate the link between illness and psychological adjustment, as suggested in the reserve capacity model. These two predictions were linked by the idea that the vulnerability of poor adjustment to comorbid illnesses conferred by the APOE gene may be modified by perceived control. Thus, perceived control, a resource largely derived from one's history of success in influencing outcomes in one's environment, was conceptualized as an important resource that may condition both one's sensitivity to stressors and one's sensitivity to genetic vulnerability of adverse stress responses. Further, in this chapter several processes that may explain the link between stress and health are explicated. More precisely, this chapter aims to test the idea that stressful circumstances (i.e. number of illnesses), genetic factors (i.e. variation in the APOE gene), and psychosocial resources (i.e. perceived control) can have independent and synergistic effects of on health (i.e. self-rated health) that are mediated by cognitive/emotional factors and behaviour (i.e. psychological adjustment).

Chapter Summary

This study aimed to determine if perceptions of control and an identifiable genetic factor, the apolipoprotein E gene, predict psychological adjustment to chronic illness thus potentially leading to changes in self-rated health. Data was utilized from a cross-sectional population-based cohort study consisting of home and local hospital-based assessments in multiple centres across Taiwan in 2000. 698 older persons aged between 54 and 75 (M = 64.5, SD = 6.2) were drawn from the 2000 Social Environment and Biomarkers of Aging Study. Psychological adjustment to illness was assessed using a composite scale comprised of measures of: perceived stress, depression, mobility, and instrumental activities of daily living, and the perceived inconvenience of illness. Non-comparative self-rated health was measured using a single item (Eriksson et al., 2001). Participants had been diagnosed with 1.52 (SD = 1.3) of the 14 chronic illness assessed. Having comorbid chronic illnesses and low perceptions of control were both independently associated with poor self-rated health and both of these relations were mediated by psychological adjustment. The number of illnesses a participant had been diagnosed with interacted with perceptions of control ($t = -4$, $p < .005$) and the apolipoprotein genotype ($t = -3.29$, $p < .005$) to significantly predict psychological adjustment scores. There was also evidence that the genetic risk of poor psychological adjustment associated with many illnesses was attenuated among those with high perceived control ($t = -.98$, $p > .05$) relative to those with low perceived control ($t = -3.47$, $p < .005$). Risk for poor adjustment to chronic illness is likely to be modified by genetic vulnerability to stress and perceived control, with elderly people with certain genotypes and low personal control having a high risk of poor adjustment to illness which may lead to unfavourable self-perceptions of health.

7.1. Introduction

Chronic illnesses develop and unfold over many years and are often prolonged and incurable. People with persistent medical conditions have been shown to experience elevated levels of depression (Katon & Sullivan, 1990) and anxiety (Wells, Golding, & Burnham, 1988) and decrements in physical and social functioning (Michael, Kawachi, Berkman, Holmes, & Colditz, 2000). People with persistent medical conditions have been shown to experience elevated levels of depression (Katon & Sullivan, 1990) and anxiety (Wells, Golding, & Burnham, 1988) and decrements in physical and social functioning (Michael, Kawachi, Berkman, Holmes, & Colditz, 2000). Stanton, Collins, & Sworowski (2001) identified five key components that characterize those who adjust well to their condition. Such people (i) master illness related tasks, (ii) maintain their functional status, and (iii) well-being in numerous life-domains, and have (iv) low levels of negative affect, and (v) psychological disorder.

The present inquiry examines psychological adjustment to chronic illness within a framework derived from the reserve capacity model (Gallow & Matthews, 2003). Within this perspective, it is proposed that for many people the stress of chronic illness leads to negative feelings, stress across several life-domains, perceptions that illness is intrusive, and difficulties in carrying out common activities and functions. Psychosocial resources, as indexed by perceptions of control, are hypothesized to buffer the effect of illness on these aspects of psychological adjustment (e.g. Affleck, Tennen, Pfeiffer, & Fitfield, 1987). In addition, I propose that genetic factors may interact with the presence of illness to explain patterns of adjustment. Specifically, I sought to show that for elderly people psychological adjustment is contingent on the independent and synergistic effects of three factors: (i) the number of chronic illnesses they have been diagnosed with, (ii) perceptions of personal control, and (iii) the apolipoprotein E (APOE) genotype. Furthermore, I expected that these

three factors may be associated with self-rated health through their relationship with psychological adjustment.

7.1.1. Perceived control and adjustment to illness in the reserve capacity model

Those with chronic health conditions are faced with variable illness trajectories, disruption to everyday activities, difficult illness-management regimes, and permanent changes across multiple life-domains. For many, these changes can be threatening and the associated stress can prompt deterioration in mental health (Wells et al., 1988). Those with comorbid illnesses may be particularly vulnerable to poor adjustment (Stommel, Kurtz, Kurtz, Given, & Given, 2004). There may even by a 'dose-response' relationship between the number of conditions a person has been diagnosed with and the likelihood of poor adjustment (e.g. Mui, 1993; Husaini & Moore, 1990; Mills, 2001). However, detrimental psychological consequences are by no means inevitable after a diagnosis of chronic illness.

For example, Helgeson, Snyder, and Seltman (2004) tracked the adjustment trajectories of women diagnosed with breast cancer. Although twelve percent experienced a substantial decline in well-being and functioning, for 38% the decline was small and 43% showed stable psychological functioning. Indeed, heterogeneity in adjustment appears to characterize chronic illness, with studies of heart disease (Hemingway & Marmot, 1999), arthritis (Walker, Jackson, & Littlejohn, 2004), stroke (Ahmed, Mayo, Corbiere, Wood-Dauphinee, Hanley, & Cohen, 2005), and diabetes (Peyrot & Rubin, 1997) demonstrating diverse profiles of adjustment.

Psychosocial resources have been shown to explain a substantial degree of the heterogeneity in psychological adjustment to chronic illness (Roesch & Weiner, 2001). There is particularly strong evidence that people with high perceived control sustain high levels of well-being and have low rates of mortality late in life (Rodin & Langer, 1977), suggesting that control beliefs may play a role in adjustment to illness. The construct of

perceived control has been defined in numerous ways within different theoretical frameworks (e.g. see Jacelon, 2007). Perceived control is conceptually similar and may encompass constructs such as mastery and locus of control (Haidt & Rodin, 1999; Wallston, Wallston, Smith, & Dobbins, 1987; Pearlin & Schooler, 1978; Rotter, 1954).

Perceptions of control over valued aspects of life ultimately derive from a track-record of attaining desired goals and avoiding unfavorable outcomes (Bullers, 2000). People who feel that their actions correspond well with rewarding future outcomes are characterized by a sense of personal agency and a belief that the chances of life can be controlled. This belief may buffer against feelings of helplessness when people are confronted with the uncertain course and unpredictable symptoms of chronic illness (Helgeson, 1992). Perceived control may protect against fatalism in the context of life-threatening illness and promote active engagement with disease management (Taylor, Kemeny, Reed, Bower, & Gruenewald, 2000). Furthermore, those with strong perceptions of control may make positive changes in health behaviours that could improve functioning and health (Armitage & Conner, 2001). Indeed, among people with chronic illnesses, perceptions of control tend to correlate positively with ratings of mental and physical health, reduced functional problems and a greater likelihood that protective action will be taken (Tennen, Affleck, Urrows, Higgins, & Mendola, 1992; Taylor, Helgeson, Reed, & Skokan, 1991). However, many studies lack a well-supported a priori theoretical framework that outlines precisely how psychosocial resources may relate to illness.

The reserve capacity model was designed to specify how a small set of factors link the stress associated with low socioeconomic status to increased vulnerability to disease (Gallo, 2009). If illness is considered a stressor (e.g. Wells et al., 1988) the simplified version of the reserve capacity model shown in Figure 9 illustrates how chronic health conditions could have cognitive-emotional and behavioural effects (arrow A) and how

these difficulties in adjustment could lead to a worsening trajectory of illness (arrow B). The model uses an economic metaphor, proposing that intrapersonal, interpersonal, and tangible resources could attenuate the negative emotional and behavioural effects of illness (arrow C).

Although the reserve capacity encompasses a broad-array of potential psychosocial resources (e.g. extraversion, optimism, social support) in this paper I focus on personal control as its role in adjustment to illness is well-supported and it is one of the most important intrapersonal resources that is conceptually readily distinguishable from affect (e.g. Andersson & Conley, 2008). The effect of a range of chronic illnesses on depressive symptoms has been shown to be modified by feelings of personal control (Penninx, van Tilburg, Boeke, Deeg, Kriegsman, & van Eijk, 1998). It is also possible that the continuous strain of illness could diminish one's existing reserve capacity/perceived control and deter the accumulation of further resources (arrow G). For example, a recent study showed that the impact of multiple sclerosis on subjective quality of life was mediated in part by perceived control (Bishop, Frain, & Tschopp, 2008). Taken together, the stress of persistent medical conditions coupled with low levels of resources could generate pervasive negative feelings (e.g. Hobfoll, 1989).

The effect of low levels of resources like perceived control on emotion and potentially functioning could lead to health changes (e.g. lower rates of hospitalization and mortality) (arrows F, C, and B) (Menec & Chipperfield, 1997). For example, perceived control has been linked to good health, low levels of somatic symptoms, quick recovery from illness, and enhanced longevity (Rodin, 1986; Krause & Shaw, 2000). Several studies have shown that those with a high level of perceived control appear to be buffered against the detrimental influence of socioeconomic status on self-rated health (Lachman & Weaver, 1998; Bailis, Segall, Mahon, Chipperfield, & Dunn, 2001). Self-rated health is a

robust predictor of mortality in elderly people indicating that perceived control may be a substantive protective factor against adversity (Mackenbach, Simon, Looman, & Joung, 2002; Idler & Benyamini, 1997). A further study confirmed the importance of perceived control for health showing it to moderate the increased risk of mortality associated with low socioeconomic status (Bosma, Schrijvers, & Mackenbach, 1999). In this study over half of the increased risk of mortality associated with having a low socioeconomic status was explained by perceived control.

To summarize, it is feasible that psychological adjustment to chronic illness may be affected by the number of illnesses one has experienced and by one's level of reserve capacity as indexed by perceived control. The reserve capacity model suggests that the stress of chronic illness and a diminished reserve capacity (e.g. low perceived control) may act both independently and synergistically to influence psychological adjustment and potentially health, as outlined in Figure 9. Recent research suggests a potential similar role for identifiable genetic variation in modifying the impact of stressors on well-being and behaviour that may have implications for psychological adjustment to chronic illness (e.g. Rutter et al., 2006; Moffitt et al., 2006; Kim et al., 2009).

7.1.2. The role of genetic factors in adjustment to illness

Whilst not currently part of the reserve capacity model, it is very likely that predisposing genetic factors influence the psychological effects of stressors with potential downstream health consequences (e.g. Taylor & Stanton, 2007). Diathesis-stress accounts (e.g. Monroe & Simons, 1991) propose that people's sensitivity to stressors depends on their genetic makeup. This gene-environment interplay takes several forms (Moffitt et al., 2006). Firstly, environmental effects can induce stable changes in gene expression through epigenetic programming processes such as DNA methylation and histone modification (Masterpasqua, 2009). The effect of environmental stress can thus be mediated through

gene expression, explaining one way how people with the same genotype can have different health outcomes (Mill & Petronis, 2007; Stahl, 2009). Conversely, the effect of genetic factors can be mediated through the environment. For instance, a parent with the same risk genotype as the child may mistreat the child thus enhancing the child's level of environmental risk. A genetic risk could cause the child to provoke hostility from others. Thus a risk genotype may elicit stressful environments in a variety of ways.

In the present study, I was concerned with the possibility that a specific identified variation in the DNA sequence could interact with a measurable stressor to induce mental and physical health effects (Moffitt et al., 2006). Prior research suggests that stressors such as the turmoil of a conflict-ridden and chaotic environment can amplify the effects of genetically-based risks (Repetti, Taylor, & Seeman, 2002). For instance, a meta-analytic review of the effect of childhood physical abuse on later mental health showed that outcomes were contingent on a functional allele in the promoter region of the monoamine oxidase A gene (Kim-Cohen et al., 2006). Additional research has shown the serotonin transporter gene (5-HTTLPR) modifies the relation between stress and both amygdala reactivity (e.g. Munafo, Brown, & Hariri, 2008) and the incidence of depression (e.g. Caspi et al., 2003). Although it must be noted that recent meta-analytic findings question the robustness of the later findings from community studies (Munafo et al., 2009; Risch et al., 2009).

While it is recognized that genetic factors may influence people's reactions to the stress of chronic illness (Stanton, Revenson, & Tennen, 2007), few studies have yet examined this possibility. A small number of studies have shown that candidate genes enhance the likelihood of depression among patients with medical conditions such as coronary artery disease (Otte, McCaffery, Ali, & Whooley, 2007) or those who have suffered a hip fracture (Lenze et al., 2005). To my knowledge only one study has examined

the role of identifiable genetic factors in modifying the psychological response to illness in a community sample (Kim et al., 2009). In this study somatic health disorders were treated as stressors and those with a many somatic disorders were shown to be at risk of developing depression if they were carriers of the 5-HTTLPR s/s genotype or the MTHFR T/T genotype.

The present investigation applies a similar design to the treatment of the relation between chronic illness and psychological adjustment, proposing that this link varies as a function of the number of illnesses a person has been diagnosed with and their APOE genotype (composed of pair combinations of the ε2, ε3, and ε4 alleles). The APOE gene is implicated in cholesterol transport and neuronal maintenance and repair after injury. Carriers of the ε4 allele have been shown to suffer a rapid decline in cognitive functioning following physical insult in sports such as American football and boxing and in cases of traumatic brain injury (Teasdale et al., 1997). In addition, caregivers who carry the ε4 allele have been shown to have an elevated risk of depression suggesting that this allele may enhance human vulnerability to psychological stress (Savitz et al., 2007; Gallagher-Thompson et al., 2001).

Evidence from genetically modified mice expressing the ε4 allele and human brain atrophy studies suggests that the adverse reactivity to stress observed in ε4 carriers may be due to neuropathological changes in the amygdala (Basso et al., 2006; Cherbuin et al., 2007; Raber, 2007). Alterations in the functioning of the amygdala could disrupt the hypothalamic-pitruitary-adrenal axis, leading to a prolonged release of glucocorticoids. This idea is supported in studies of older people where elevated cortisol levels have been observed among carriers of the ε4 allele and in studies of transgenic mice where evidence of hypercorticolism has been found among ε4 carriers (Peavy, 2008; Robertson, Curley, Kaye, Quinn, Pfankuch, & Raber, 2005). Taken together, there is clear evidence from

clinical, neuroimaging and transgenic mice studies to suggest that the APOE ε4 allele confers an elevated risk of poor adjustment to stress.

A recent meta-analytic review examined the genetic basis of major depression by analyzing 183 studies and 393 candidate gene polymorphisms (Lopez-Leon et al., 2008). This review found that the APOE ε2 allele predicted a reduced likelihood of depression (OR = 0.51). In addition, carriers of the ε2 allele have demonstrated low levels of numerous risk factors for poor mental health including total cholesterol, low-density lipoprotein, and Alzheimer's disease (Giltay et al., 2009, Martins et al., 2006). Thus, it is possible that carriers of the ε2 allele may be buffered from the ill effects of stressors.

Through incorporating genetic factors in the simplified reserve capacity model outlined in Figure 9, I propose that like psychosocial resources such as perceived control, genetic variation may attenuate or exaggerate emotional and behavioural reactivity to stressors. Prior research has demonstrated that perceived control can augment the potential detrimental effect of genetic diathesis on health (Johnson & Krueger, 2005). Several studies have shown three-way gene × stressor × psychosocial resources interaction effects to exist. For example, Kaufman et al. (2004) demonstrated that maltreated children with the short s/s serotonin transporter genotype (linked to diminished bioavailability of serotonin) had particularly high depression ratings when combined with little social support. Similarly, Kilpatrick et al. (2007) showed that the Florida residents who were highly exposed to severe hurricanes in 2004 and who had both the low-expression s/s serotonin transporter genotype and low social support were at the greatest risk of major depression and post-traumatic stress disorder. Thus, consistent with prior research, it may be the case that those with several chronic illnesses and a risky genotype (e.g. APOE ε3ε4) may be successful in adjusting to illness if they have high levels of perceived control.

7.1.3. The current study

In the context of the current study, I aimed to expand the literature on psychological adjustment to illness by assessing several factors thought to broadly reflect a generalized construct of adjustment. Consistent with recommendations by Stanton et al. (2001) the participants, a nationally representative group of elderly Taiwanese people aged between 54 and 75, completed a range of questionnaires utilized to measure good adjustment as indexed by: (i) low negative affect, assessed using a measure of *depressive symptoms*, (ii) sustained well-being in several domains, measured using *domain-specific ratings of perceived stress*, (iii) preserved functional status, evaluated using measures of *the instrumental activities of daily living* and of *mobility*, and (iv) mastery of disease-related tasks which is assessed indirectly by asking participants to indicate to what extent they find their *illness to intrude* on their lives. These measures of adjustment were standardized and aggregated to comprehensively gauge the underlying construct of psychological adjustment.

On the basis of prior research and as denoted in the simplified reserve capacity model outlined in Figure 9, I hypothesized that a composite index of psychological adjustment would relate inversely to the number of chronic illnesses a person had been diagnosed with (Hypothesis 1; arrow A). The measure of reserve capacity, *perceived control*, was predicted to have a direct effect on psychological adjustment (Hypothesis 2; arrow C), with those who have strong perceptions of personal control showing good adjustment. In addition, I sought to clarify if perceived control was independently linked to psychological adjustment or if it mediates the potential effect of illness on adjustment (arrow G, C). I further hypothesized that perceived control would modify the relationship between the number of illnesses and adjustment (Hypothesis 3; arrows A, F). I expected

that those who score highly on perceived control would adjust well to comorbid illnesses unlike those who feel they lack control.

The current study measured variation in the APOE gene in order to test if, like perceived control, this genetic variation might moderate the association between the number of illnesses and psychological adjustment (Hypothesis 4; arrows A, E). As in prior research of this kind (Kim et al., 2009) I assumed that the relation between the number of previous diagnoses of chronic illness and psychological adjustment would be stronger for those with a risk genotype (i.e. APOE ε3ε4) than for those with a potentially protective genotype (i.e. ε3ε2). I expected little difference between genotype groups for those who had not encountered the stress of a chronic illness and large genotype-based differences between those with several illnesses. Further, I hypothesized that the APOE genotype could have a direct effect on psychological adjustment with those with a risk genotype experiencing worse adjustment than those with a protective genotype (Hypothesis 5, arrow D). Next, I sought to show that having a greater number of illnesses was chiefly problematic if one has a risk genotype (i.e. APOE ε3ε4) and low perceived control. Thus, I predicted a three-way interaction between the number of illness, APOE genotype, and perceived control in their relation to psychological adjustment (Hypothesis 6, Arrows A, E, F).

Consistent with the model depicted in Figure 9, I expected that the relations between the key study variables and psychological adjustment (as outlined in hypotheses 1 through 6) may lead to changes in self-rated health. Thus, I hypothesized that psychological adjustment would mediate the association between the number of illnesses and health (Hypothesis 7, arrows A, B), perceived control and health (Hypothesis 8, Arrows E, B), APOE genotype and health (Hypothesis 9, arrows D, B), the number of illnesses × perceived control interaction and health (Hypothesis 10, arrows A, F, B), the

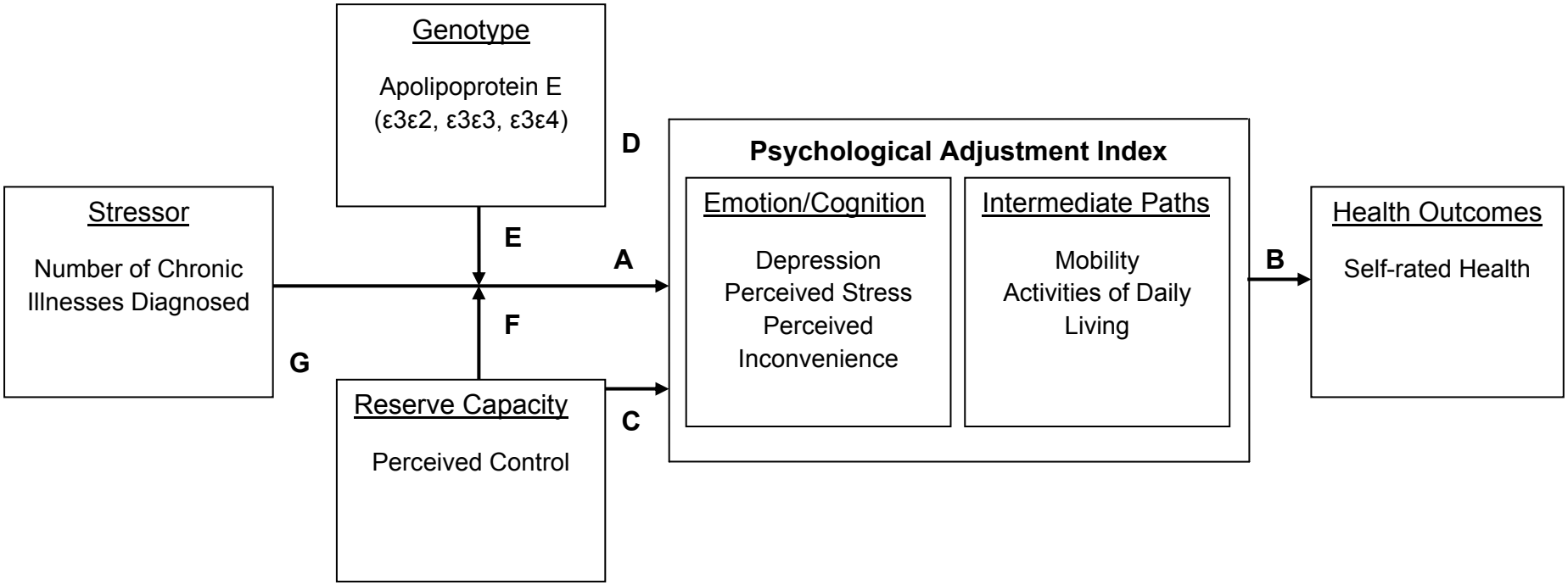


Figure 9. The adapted reserve capacity model utilized in the current research. Black lines indicate pathways that were supported in the current research, whereas grey lines signal unsupported relationship.

number of illnesses × APOE genotype interaction (Hypothesis 11, Arrows A, E, B), and finally the three-way number of illnesses × perceived control × APOE genotype interaction (Hypothesis 12, Arrows A, F, E, B).

7.2. Methods

7.2.1 Participants

Participants (N = 698 with complete data) were adults aged between 54 and 75 drawn from the 2000 Social Environment and Biomarkers of Aging Study (SEBAS) (Weinstein & Goldman, 2007). Participants in SEBAS were a random sub-sample of the nationally representative Taiwan Survey of Health and Living Status of the Elderly. Potential participants in the original survey were drawn from a probability sample of Taiwanese townships and 92% agreed to participate. SEBAS was composed of two parts. Firstly, an in-home interview lasting on average an hour (median = 57.5 minutes) was conducted by a local public health nurse. The interview component included all self-report questions and took place between July and December, 2000. This section was completed by 92% of those contacted (N = 1497).

The second component of SEBAS consisted of a local hospital medical examination with biospecimen collection. This took place several weeks after the interview and was completed by a subsample (68.3 %, N = 1023) of those interviewed. Of the 31.7% who did not participate, 7.4% were not eligible for the health examination (e.g. had a health condition that precluded the drawing of blood) and 24.2% refused to participate, chiefly because these participants were reluctant to provide blood or urine. Sex, level of education, and employment status did not predict participation in the health examination. There was a lower rate of participation among those over 70 and amongst the very healthy. However, on average the participants in the medical examination were not found to differ

in their self-rated health status (M = 2.93) from those who did not complete this component (M = 2.94) (Goldman et al., 2003).

The young-old are examined in this study as this is a crucial age-bracket within which chronic illnesses first emerge and where psychological effects may be most evident (Krause et al., 2004). In addition, the established relationship between the APOE ε4 allele and impaired cognition and Alzheimer's disease appears to be most evident in those over 75 potentially confounding the proposed relations if these individuals were included (Riley, Snowdon, Saunders, Roses, Mortimer, & Nanayakkara, 2000). Written informed consent was obtained for participation in both the interview and the hospital medical examination. The final sample consisted of 408 men (58.1%) and 294 women (41.9%) aged between 54 and 75 (M = 64.5, SD = 6.2). Twenty-eight percent of participants had no formal education, whilst a further 45.7% had primary education only. Twenty percent had completed secondary school education and 6.4% had attended third level college. Ethnicity was classified into Fukien, (73.5%), Mainlander (13%) and Hakka (13.5%).

7.2.2. *Instruments*

7.2.2.1. Chronic conditions

Participants were asked "has a doctor diagnosed you with" each of fourteen specific chronic conditions: high blood pressure (29% diagnosed), diabetes (13.1%), heart disease (13.1%), stroke (2.8%), cancer (2.1%), lower respiratory tract disease (10.3%), arthritis or rheumatism (12%), gastric ulcer or stomach ailment (17.5%), liver or gall bladder disease (7.7%), hip-fracture (0.4%), cataracts (19.2), kidney disease (7.5%), gout (6%), and spinal or vertebral spurs (11.6%). The interviewers gave due consideration to individual differences in health literacy. If a participant did not understand what was meant by a certain medical condition the interviewer read a standard description of the condition. For example, gout would be described as follows: "*it is caused by the build-up of "body*

waste"-uric acid in joints, mucous membranes, cartilage and auricles. Typical symptoms include joint inflammation, redness and swelling, especially on the largest toe." The total number of illnesses each participant had been diagnosed with was then summed. Sixteen people, or approximately 2% of the eligible sample, had been diagnosed with greater than five chronic illnesses. This group were considered statistical outliers and removed from subsequent analyses. The mean number of illnesses among the remaining participants was 1.52 (Min = 0, Max = 5, SD = 1.3).

7.2.2.2. Perceived control

The Pearlin-Schooler (1978) mastery scale was utilized to assess participant's perceived control over important life events. The scale is composed of seven-items worded to reflect either perceptions of personal control (e.g. "What happens to me in the future mostly depends on me") or lack of control (e.g. "There is little I can do to change many of the important things in my life."). Participants rated each item using a five-point scale from 1 = Strongly agree to 5 = Strongly disagree. The scores ranged from 8 to 35 with a mean of 22.67 (SD = 4.38), with higher scores indicating greater perceived control. The Cronbach's alpha score for the scale was .61.

7.2.2.3. Apolipoprotein E genotype

The APOE gene has six expected genotypes formed from pairs of its three alleles (ε2, ε3, ε4). The genotyping of APOE was carried out using the polymerase chain reaction amplification refractory mutation system (PCR-ARMS) and polymerase chain reaction restriction fragment length polymorphism (PCR-RFLP) techniques (see Goldman et al., 2003). Fourteen participants or 1.9% of the sample possessed rare genotypes (i.e. ε4ε4, ε2ε2, or ε2ε4) and were not included in the analyses due to insufficient statistical power. A genetic risk variable was constructed with those possessing ε3ε2 (13.8% of remaining participants) coded as '0' indicating low risk of poor psychological adjustment, those with

the ε3ε3 (71.8%) genotype rated as '1' and ranked as medium risk, and those with the ε3ε4 (14.4 %) genotype were coded as '2' and ranked as high risk of poor adjustment.

7.2.2.4. Psychological adjustment

The conceptualization of psychological adjustment outlined by Stanton et al. (2001) guided the choice of key variables used to measure adjustment in the current study. The set of conceptualizations proposed and the measurement techniques utilized were as follows: (i) mastery of chronic illnesses-related tasks was gauged by assessing the degree to participants found their illness to be inconvenient, (ii) the preservation of functional status was identified using standard a measure of mobility and a measure of the instrumental activities of daily living (e.g. Lawton & Brody, 1969), (iii) participant's perceived quality of life in several domains was identified using a measure of stress/anxiety in seven life domains (i.e. finances, health, family relations, family health, family finances, family work, and the family's marital situation), and finally, (iv) low levels of negative affect were assessed using a measure of depressive symptoms. Due to data limitations we did not assess the presence of psychological disorder.

Depressive symptoms. Depressive symptoms were assessed with a 10-item version of Centre for Epidemiological Studies Depression Scale (CES-D) (Radloff, 1977). The 10-item CES-D demonstrated satisfactory internal consistency and construct and concurrent validity in a validation study of Chinese people (Boey, 1999). Participants respond to the CES-D by rating their experience of depressive symptoms in the past week on a four-point scale (0 = No, 4 = Often (> 4 days)). The CES-D produces an index ranging from 0 to 30, with scores of 10 and over deemed indicative of depression (Andresen, Malmgren, Carter, & Patrick, 1994). The mean score was 5.16 (Min = 0, Max = 28, SD = 4.92) and the Cronbach's alpha score was .766.

Stress/anxiety in multiple life-domains. Participants indicated whether or not they experience stress/anxiety in relation to seven different aspects of their lives. Specifically, participants responded 'Yes' or 'No' to whether they experience stress/anxiety about: finances, health, family relations, family health, family finances, family work, and the family's marital situation. On average participants indicated they experienced stress/anxiety relating to 1.69 life-domains (Min = 0, Max = 7, SD = 1.81, α = .751).

Inconvenience of illness. The degree of inconvenience perceived for each illness was rated on a 3-point scale from '0 = No inconvenience' to '2 = Much inconvenience'. Participants with no illness were automatically scored as '0' on this scale. A total illness inconvenience score was then tabulated for each participant (M = 0.74, Min = 0, Max = 6, SD = 1.23). The inconvenience of chronic illness variable was positively skewed and was therefore transformed via square root transformation.

Functioning. Participants self-reported their ability to perform nine different physical movements on a four-point scale (0 = No difficulty, 1 = Some difficulty, 2 = Great difficulty, 3 = Unable to do it). More precisely, participants indicated the difficulty they had standing for 15 minutes, standing for 2 hours, squatting, raising both hands over their head, grasping objects with their fingers, lifting 11-12 kilograms, running 20-30 metres, walking 200-300 metres, and walking up 2-3 flights of stairs. Most participants had few mobility restriction and could complete most of these tasks without difficulty (M = 2.67, Min = 0, Max = 23, SD = 4.16, α = .86). However, as some participants had severe mobility difficulties this variable was positively skewed and was therefore normalized via square root transformation.

Six Instrumental Activities of Daily Living (IADL) were also measured (i.e. ability to buy personal items, manage money, ride public transport along, do light housework or physical work at home, make phone calls) and rated on the same four-point scale as mobility restrictions (0 = No difficulty, 1 = Some difficulty, 2 = Great difficulty, 3 = Unable to do it) (Lawton & Brody, 1969). The scale showed satisfactory reliability (Cronbach's alpha = .754). However, the majority of the sample indicated they were capable of completing all these activities with no difficulty. I therefore constructed a binary variable with those not experiencing difficulty (78.3%) coded as 0 and people experiencing any functional problems (21.7%) coded as 1.

Composite index of psychological adjustment. The five measures of adjustment (i.e. depressive symptoms, stress/anxiety in multiple life-domains, inconvenience of illness, mobility limitations, and instrumental activities of daily living) were standardized and summed to form an overall adjustment to chronic illness index (e.g. Kim et al., 2009).

7.2.2.5. Self-rated health

To gauge self-rated health, participants rated their current health on a five-point scale (1 = poor, 2 = not so good, 3 = average, 4 = good, 5 = excellent). This single-item measure has consistently been shown to be closely linked to important health outcomes including mortality (Mackenbach et al., 2002). Scores were distributed around the mid-point of the rating scale (M = 2.88, SD = .96).

7.2.2.6. Possible confounders

A broad-set of control variables were assessed. Age, sex, and educational level were included as they have been found to be linked to chronic illness and measures of psychological adjustment (e.g. Katon & Sullivan, 1990; Katon, 2003). Further, to ensure

that the potential effects of the key study variables were not due to individual differences in cognitive ability two tests were included to measure cognitive function. In particular, I was concerned that a link between the APOE gene and psychological adjustment could be confounded by genetically mediated changes in cognition (e.g. Small, Rosnick, Fratiglioni, & Backman, 2004). Modified versions of the Rey Auditory Verbal Learning Test (Lezack, 1983) and the Digits Backward test (Wechsler, 1981) were administered. The scores on both tests were converted to z-scores and summed to produce an index of cognitive function. To try to ensure individual differences in adjustment were not due to the side-effects of medication I included a control variable for those taking long-term medication (58.5%). As it is possible that people who engage in unfavorable health behaviours may have high rates of illness and poor psychological adjustment I wished to control for this variable. The prevalence of smoking (24.4%) was self-reported. Participants also reported their frequency of drinking on a scale from 0 = No to 3 = Everyday, (M = .41, SD = .9). The frequency of exercising each week was assessed on a scale from 0 = None to 4 = 6+ times, (M = 2.14, SD = 1.78).

7.2.3. Statistical Analysis

Prior to analysis all of the study outcome measures were tested for normalcy. The dependent variables were psychological adjustment and self-rated health and the independent variables were the number of chronic illnesses, perceived control, and APOE genotype. Preliminary analyses were conducted to test the correlations between the key study variables. In particular, I wanted to test if the APOE gene appeared to contribute to the prevalence of chronic illness as this correlation would potentially limit inferences regarding a gene-environment interaction. Next I estimated a series of regression models to examine the first six study hypotheses. Firstly, I examined the relations between the demographic and potentially confounding variables and psychological adjustment.

Following this, to test the first three hypotheses I tested if (a) the number of illnesses, (b) perceived control, and (c) the APOE genotype predicted psychological adjustment after adjusting for control variables.

Hypothesis 4 proposes that perceived control will moderate the relation between the number of illnesses and psychological adjustment (i.e. an interaction effect). To test this prediction a model including the standardized main effects of control perceptions and illness and the product of both standardized terms gauged the interaction effect. All interaction terms were produced in this manner, by converting the independent variables into z-scores and calculating the product of the relevant variables (Aiken & West, 1991). Hypothesis 4 would be supported if the interaction effect was shown to be significant and post-hoc tests demonstrate that the inverse relationship between the number of illnesses diagnosed and adjustment is stronger for those with low rather than high perceptions of control.

A moderation regression analysis was also used to test hypothesis 5 which suggested that the number of illnesses a person had been diagnosed with would predict psychological adjustment more strongly for those with a risk APOE genotype than those with a less risky APOE genotype. I examined the main effects of the standardized number of illnesses and APOE genotype variables and the interaction term composed of the product of these z-scores. The buffer hypothesis was supported if the interaction term was significant and if illness showed a graded relation to adjustment as a function of the APOE genotype with those with the ε3ε2 genotype showing good adjustment, ε3ε3 carriers demonstrating moderate adjustment, and ε3ε4 carriers showing poor adjustment.

Next, I sought to show that the number of illnesses could interact with both the APOE gene and perceptions of control (Hypothesis 6). In this analysis I entered the three main effects of the independent variables and their higher-order interaction terms including

their three-way interaction. Hypothesis 6, was deemed to be supported if the interaction term was significant and if post-hoc tests revealed that the hypothesized APOE gene × number of illnesses interaction exists principally for those with low perceived control. This would suggest that perceived control attenuates the effect of genetic risk on emotional reactivity and the likelihood of poor psychological adjustment.

The final set of analyses sought to assess if psychological adjustment mediated between the independent variables (number of illnesses, perceived control, APOE gene), their interaction terms (perceived control × number of illnesses, APOE gene × number of illnesses, perceived control × APOE gene × number of illnesses) and self-rated health. I therefore conducted six separate mediation analyses, testing hypotheses 7 – 12, using the guidelines detailed in Baron and Kenny (1986). Successful mediation was deemed to require that three essential conditions were satisfied. The first condition replicates hypotheses by requiring that the independent variable or independent variable interaction term must predict the mediator variable, psychological adjustment. Next, the mediating variable is required to account for a substantial level of variance in the dependent variable (i.e. psychological adjustment must predict self-rated health). Finally, mediation requires that the after controlling for both the link between the independent variable and the mediating variable, the mediating variable remains as a predictor of the dependent variable and the relationship between the independent variable and the dependent variable is significantly attenuated.

For displaying associations between chronic illness and adjustment as a function of perceived control and genotype, the number of chronic conditions variable was divided into four groups (0, 1, 2, 3+). As this study includes a substantial number of hypotheses I utilized a Bonferronni corrected alpha value of 0.004 (for 12 analyses) to reduce the likelihood of false positive results.

Table 12. *Correlation matrix detailing relationships between key study variables*

Variable	Self-rated Health	Psychological Adjustment	APOE Genotype	Perceived Control
Number of Illnesses	r = -.37***	r = -.49***	r = .07	r = -.09*
Perceived Control	r = .22***	r = .41***	r = -.02	-
APOE genotype	r = -.06	r = -.06	-	-
Psychological Adjustment	r = .5***	-	-	-

*p < .05, ***p<.005

7.3. Results

7.3.1. Preliminary analyses

Principal components analysis with varimax rotation was performed on the individual psychological adjustment items. This revealed a single factor (eigenvalue = 2.52) which explained 50.3% of the variance. Reliability tests confirmed that the five adjustment scores form a sufficiently reliable scale (Cronbach's alpha = .75) and removing any of the items did not improve the reliability of the scale. These results suggest that the five components measured appear to assess a single underlying construct of psychological adjustment.

The correlations between the main study variables are shown in Table 12. There were no significant differences in the number of illnesses experienced as a function of genotype (r = .07, p = .07). Additional analyses confirmed that the APOE gene was unrelated to the prevalence of any specific illness. Further, the demographic characteristics age, gender, and level of education were not significantly related to the genotype variable. These analyses indicated that different genotypes of the APOE gene were not overly

represented among any particular group, and that it was unlikely that the APOE gene contributes to the development of illness.

7.3.2. Demographic factors, potentially confounding variables and psychological adjustment

Psychological adjustment was lower among females, older people, and the less well educated, as shown in Table 13. Those taking long-term medication were particularly likely to have low levels of adjustment and those who exercise frequently were found to be well adjusted. Ethnicity, smoking, drinking, and cognitive ability appeared to have little impact on psychological adjustment scores.

7.3.3. Illness, perceived control, APOE genotype and psychological adjustment

The number of chronic illnesses experienced predicted substantially worse psychological adjustment (B = -.266, SE = .025, t = -10.56, $p < .004$, Cohen's d = .8) confirming the first hypothesis. Those with higher levels of perceived control had better psychological adjustment than those with low control (B = .195, SE = .021, t = 9.21, $p <$ .004, Cohen's d = .7) supporting hypothesis two. Further analysis showed that perceived control appeared to be unaffected by the number of illnesses a participant had been diagnosed with (B = -.07, SE = .045, t = -1.55, p = .12). This indicated that perceived control was directly related to psychological adjustment rather than a mediator of the relation between the number of chronic illnesses diagnosed and psychological adjustment. The third hypothesized main effect of the APOE gene was non-significant suggesting that genotype did not impact directly on psychological adjustment (B = -.016, SE = .02, t = -.84 p = .4). The results indicate that both having comorbid illnesses and lacking a sense of control over important life events may be detrimental to adjustment.

I then sought to test if the link between the number of illnesses and psychological adjustment was modified by perceived control. The interaction term entered was found to be significant (B = -.158, SE = .038, t = -4, p < .001, Cohen's d = .3), and indicated that for those with below average levels of perceived control the decline in psychological adjustment associated with an increase in the number of chronic illnesses (simple slope and confidence interval = -.33 ± .078) was steeper than for those with above average perceived control (slope = -.21 ± .062). Thus, as expected those with lower perceptions of control show more difficulties in adjusting to comorbid illnesses than do those with strong perceptions of control, as shown in Figure 10a. It appears that whilst the number of illnesses a person has been diagnosed with is unrelated to perceived control, illness does interact with control perceptions to predict psychological adjustment.

As predicted, the number of chronic illnesses experienced interacted with APOE genotype to predict psychological adjustment (B = -.072, SE = .022, t = -3.29, p < .004, Cohen's d = .25). The number of chronic illnesses experienced was unrelated to psychological adjustment among carriers of the APOE ε3ε2 genotype (B = -.027, SE = .092, t = -.29, p = .77) but was strongly predictive of lower adjustment for APOE ε3ε3 carriers (B = -.269, SE = .028, t = -9.52, p < .0005) and for those with the APOE ε3ε4 genotype (B = -.425, SE = .072, t = -5.89, p < .0005), as illustrated in Figure 10b. Examination of the simple slopes showed that the decline in adjustment as a function of the number of chronic illnesses was strongest for ε3ε4 carriers (-.425 ± .144), followed by ε3ε3 carriers (-.269 ± .056), and then ε3ε2 carriers (-.027 ± .184) as hypothesized. The two-way interaction results suggest that perceived control and the APOE gene act as dual-buffers modifying the link between how many illnesses a person and their level of psychological adjustment.

Next, I aimed to test hypothesis 6 which proposed that poor psychological adjustment would be particularly likely among those with many illnesses, a risk APOE genotype and low levels of perceived control. Thus, I entered a three way interaction between the number of illnesses, perceived control scores, and the APOE genotype into the a regression model after adjusting for control variables, main effects of independent variables, and two-way interaction effects. This showed a marginally significant three-way interaction effect (B = -.079, SE = .045, t = -1.76, p = .08, Cohen's d = .13). Although this interaction failed to reach the appropriate alpha-level for significance I conducted further analyses to identify if there was partial support for this hypothesis. I found that the interaction between the APOE gene and the number of illnesses predicted psychological adjustment for those with below average perceived control (B = -.111, SE = .032, t = -3.47, p < .004, Cohen's d = .26) but not for those with above average perceived control (B = -.03, SE = .03, t = -.98, p = .33), as illustrated in Figure 11. For those with low control having more chronic illnesses was most closely related to diminished psychological adjustment for ε3ε4 carriers (-.539 ± .102), followed by ε3ε3 carriers (-.326 ± .047), and then ε3ε2 carriers (-.025 ± .268) for whom there was no relation between illness and adjustment. This analysis replicated the pattern of results found in the APOE gene × number of illnesses analysis. It thus appears that the genetic risk of poor psychological adjustment in the presence of many illnesses is conditional on one's level of perceived control.

In the final set of analysis I sought to show that psychological adjustment mediated the relations between the independent variables and their interactions and self-rated health. The preliminary analysis showed that self-rated health was substantially lower among those who exercise little and those taking long-term medication, as shown in Table 13. Further, there was some evidence that older, better educated participants with high levels of cognitive ability were in better health than others.

Table 13.

Parameter estimates for the relationship of demographic factors, cognitive ability, and health behaviours to psychological adjustment and self-rated health

	Psychological Adjustment			Self-rated Health		
	B (*SE*)		t	*B* (*SE*)		t
Intercept	1.295	(.289)	4.49***	.787	(.437)	1.8
Age	-.017	(.004)	-4.09***	-.012	(.006)	-2.01*
Gender	-.199	(.056)	-3.55***	-.031	(.085)	-.37
Level of Education	.175	(.033)	5.3***	.102	(.050)	2.04*
Ethnicity Dummy 1	.027	(.066)	.43	.169	(.100)	1.69
Ethnicity Dummy 2	.062	(.075)	.82	.073	(.114)	.64
Cognitive Ability	.044	(.031)	1.41	.093	(.047)	1.98*
Smoker	.029	(.062)	.48	.144	(.093)	1.55
Alcohol Intake	.001	(.027)	.003	.028	(.042)	.67
Exercise Frequency	.052	(.013)	3.92***	.102	(.020)	5.04***
Long-term Medication	-.404	(.047)	-8.59***	-.566	(.071)	-7.96***

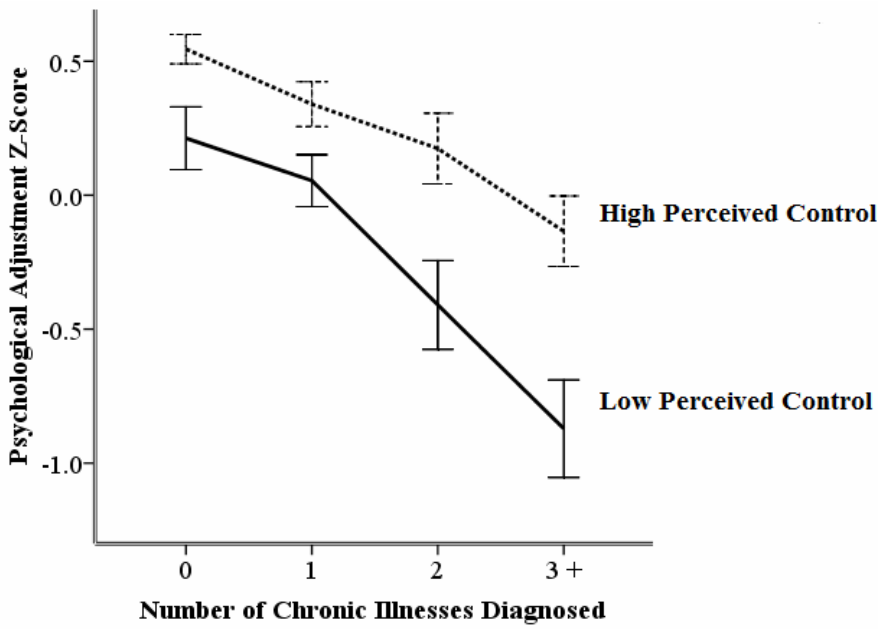
*p < .05, ** p < .01, *** p < .005

7.3.4. Indirect effects of independent variables on self-rated health through psychological adjustment

The first mediation test examined if psychological adjustment explains the relationship between the number of illnesses and how participants perceive their health. Those with more illnesses had far lower rating of self-rated health as would be expected (B = -.239, SE = .042, t = -5.66, p < .0005, Cohen's d = .43). I have already established that the number of diagnosed illnesses is a strong predictor of psychological adjustment. To test for mediation I regressed the number of illnesses and psychological adjustment on self-rated health. The results showed an attenuation of the relation between the number of illnesses and self-rated health (B = -.107, SE = .044, t = -2.47, p < .05). An examination of the simple slopes suggested that this attenuation was significant (-.239 ± .084 reduced to -.107 ± .088). Psychological adjustment remained as a strongly significant predictor of self-rated health (B = .495, SE = .061, t = 8.07, p < .0005). Furthermore, additional Sobel tests suggested that psychological adjustment contributes to the association between the number of illnesses diagnosed and self-rated health (z = 6.41, p <.004).

The initial analyses showed that perceived control was positively related to psychological adjustment. Next, I sought to extend the analysis by examining if psychological adjustment mediates between perceived control and self-rated health. Perceived control predicted better self-rated health (B = .138, SE = .035, t = 3.7, p < .0005, Cohen's d = .28) and statistical control for psychological adjustment reduced this relationship to non-significance (B = .041, SE = .036, t = 1.15, p = .25). Psychological adjustment remained as a significant predictor of self-rated health (B = .495, SE = .061, t = 8.07, p < .0005, Cohen's d = .61). These results coupled with the Sobel equation (z = 3.36, p < .004) strongly suggest the link between perceived control and self-rated health is explained by psychological adjustment.

(a)



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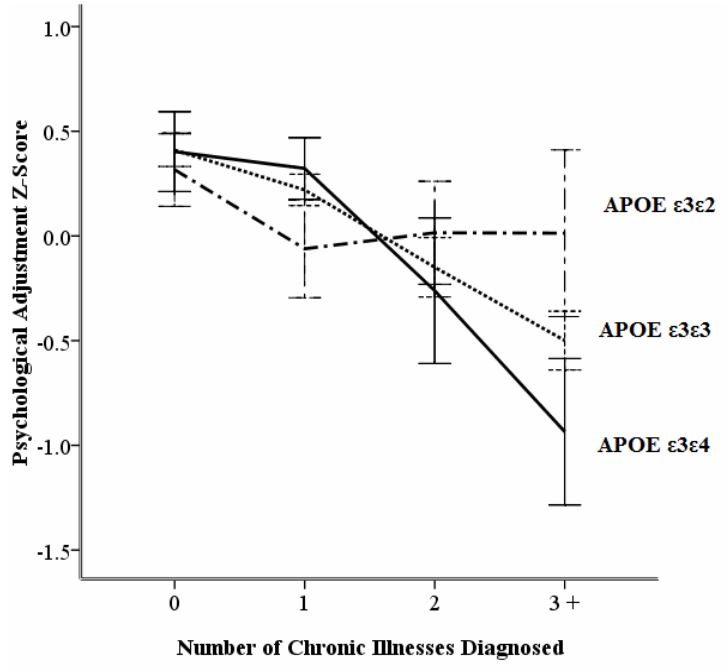


Figure 10. The relationship between the number of illnesses diagnosed and standardized psychological adjustment scores as a function of: (a) level of perceived control (high = above average, low = below average), and (b) APOE genotype.

As the APOE gene was unrelated to psychological adjustment I did not test for mediation in this instance. I did, however, examine the possibility that psychological adjustment may mediate between the interaction between the APOE gene and the number of illnesses and self-rated health. I found that the APOE genotype interacted with the number of illness a participant has been diagnosed with to predict self-rated health (B = -.08, SE = .037, t = -2.16, $p < .05$, Cohen's d = .16). As anticipated, for those with the APOE ε3ε2 genotype there was little relation between the number of illnesses diagnosed and psychological adjustment (B = -.107, SE = .158, t = -.67, p = .5). Whereas those with a greater number of illnesses had lower self-rated health if they were ε3ε3 carriers (B = -.236, SE = .048, t = -4.88, $p < .0005$) or ε3ε4 carriers (B = -.347, SE = .109, t = 3.19, $p <$ .004). Mediation analysis showed that the relation between the APOE gene × number of illnesses interaction and self-rated health was reduced to non-significant upon entering psychological adjustment into the regression model (B = .04, SE = .036, t = 1.1, p = .27). An additional Sobel analysis provided some support for psychological adjustment as a mediator of this relation (z = 2.08, p < .05). However, this result is tentative as the confidence intervals for the slopes of the relations between the number of illnesses and self-rated health for different APOE genotypes overlapped substantially. Secondly, the Sobel test did not reach the Bonferroni corrected level of significance. In aggregate, I infer that these results suggest partial support for the hypothesis that psychological adjustment explains the link between the APOE gene × number of illnesses interaction and self-rated health.

Next, I tested if those with many illnesses had worse self-rated health particularly if they had low levels of perceived control. I failed to find an interaction between the number of illnesses and perceived control in predicting self-rated health (B = -.67, SE = .613, t = -1.09, p = .47). Finally, a regression analysis testing the link between the APOE gene × perceived control × number of illnesses interaction and self-rated health found no evidence

of this relation (B = .026, SE = .077, t = .343, p = .73). Although there was little support for four of the study hypotheses, the results indicated full support for six of the twelve proposed hypotheses at the Bonferroni corrected alpha level and partial support for a further two hypotheses, as summarized in Table 14.

7.4. Discussion

To my knowledge, this study is the first to examine how perceived control combines with genetic factors to predict psychological adjustment to illness. This investigation, among a population-based cohort of elderly Taiwanese, tested several tenets of the reserve capacity model and aimed to establish if and how genetic factors may fit into this analytic framework. This motivation for this study was to examine the conditions whereby individual differences in the number of chronic illnesses diagnosed account for variation in psychological adjustment and perceptions of health. As a precautionary note, the causal linkages implied by the theoretical model guiding this research (see Figure 9) should be heeded with care due to the cross-sectional design of the current research. In particular, theories supporting the reverse sequence of causation than those specified in the model cannot be ruled out in the absence of longitudinal data. However, this caveat considered, the results of this analysis do provide important insights for how psychosocial resources may combine with identifiable genetic variation to explain patterns of adjustment to illness and the health trajectories that follow.

7.4.1. Illness, perceived control, and psychological adjustment

On the basis of prior research and theory (e.g. Stanton et al., 2001) psychological adjustment was gauged using several measures assessing depressive symptoms, stress or anxiety in multiple life-domains, the inconvenience generated by each illness, as well as mobility limitations and the ability to carry out everyday tasks. Poor adjustment to chronic illness is common in most long-term medical conditions (e.g. diabetes, stroke, heart

disease, lung disease, arthritis) (Polsky et al., 2004, Michael et al., 2000). In particular, the presence of multiple comorbid conditions is especially likely to cause the onset or exacerbation of depression for many people (Cole & Dendukuri, 2003; Detweiler-Bedell et al., 2008; Noel et al., 2004).

Consistent with this contention, in this study those with more illnesses scored far lower on the measure of psychological adjustment. However, whilst this strong relation is of modest interest in itself, I sought to demonstrate that there are important psychological and biological factors that may explain the heterogeneity that has been observed in psychological adjustment to illness (Stanton et al., 2007). The simplified version of the reserve capacity model used in the current study envisions a protective role for the psychosocial resource, perceived control, as enhancing the likelihood of successful adjustment to illness. Prior research has shown perceived control to mediate between illness and quality of life (Bishop et al., 2008). In the current research perceived control was largely unrelated to illness but strongly predicted better psychological adjustment. This result suggests that perceptions of control may be unaffected by illness but yet may diminish negative affect and enhance positive functioning.

Further, perceived control moderated the relation between illness and psychological adjustment. Those with low levels of perceived control and comorbid illnesses were particularly vulnerable to poor adjustment. As identified in prior research, perceived control appears to buffer the effect of stressful circumstances (e.g. low socioeconomic status, low education) on mental health outcomes and functional status (e.g. Lachman & Weaver, 1998; Turner & Noh, 1983). Although several studies have tested the reserve capacity model, there has been little support in these studies for the core tenet that psychosocial resources modify the relation between stressors and cognitive/emotional factors (e.g. Gallo et al., 2005; Matthews et al., 2008). It should be noted that the index of

psychological adjustment utilized in the current study included behavioural measures (e.g. activities of daily living) in addition to emotional/cognitive factors (e.g. depression, perceptions of inconvenience). However, the results do show a clear difference in the illness-related decline in psychological adjustment for those with low perceived control and those with high control. Amongst those with high perceived control adjustment declined approximately half a standard deviation from the group without a chronic illness to those with three or more illnesses. For participants with low control this decrease in adjustment exceeded a full standard deviation.

This result is likely to indicate, at least in part, a modification of emotional/cognitive factors by perceived control, thus providing partial support for a role of psychosocial resources in attenuating the emotional response to stressful circumstances as suggested in the reserve capacity model. More broadly, this finding invokes empirical support for the insight of the Greek Stoic philosopher, Epictetus, "[people]..are disturbed not by things, but by the view which they take of them". Although the development of illness is often outside of one's control those who believe they can control important events in life, may not appraise illness as completely uncontrollable. Their non-fatalistic perspective may shift their attention to the controllable aspects of their illness such as treatment regimes and symptom management and thus dampen feelings of helplessness and the adverse mental health effects that follow. However, the results suggest that how people react to the stress of illness depends not only on their subjective perceptions of control but also on objective and uncontrollable variation in their genetic make-up which can now be precisely identified.

7.4.2. Illness and genotype interaction

While diathesis-stress accounts of disease often relate to their cause, until now there has been little evidence of a genetic role in their consequence, or in how people react

to them, at least as regarding psychological aspects of chronic illness conditions. The findings support the general principal that genetic endowment can act as a protective factor ameliorating susceptibility to poor adjustment to chronic illness. Other research has found genetic variation to moderate psychological adjustment to non-health stressors (e.g. childhood maltreatment, natural hazards) producing resilience and thriving, or distress and maladjustment (Caspi et al., 2003, Kaufman et al., 2004). Furthermore, a recent study showed that the link between the number of somatic conditions people report and their level of depression was modified by the serotonin transporter and the MTHFR genes (Kim et al., 2009).

Similarly, I suggest that the APOE gene may be only weakly directly related to psychological adjustment. Instead the APOE gene may interact with the presence of illness to produce different profiles of adjustment (Monroe & Reid, 2008). This study provides evidence that the APOE ε3ε2 genotype confers substantial protection from poor adjustment to illness. For APOE ε3ε2 carriers with three or more illnesses adjustment scores were less than a half a standard deviation lower than those with no illness. Among ε3ε3 carriers this gap was closer to one standard deviation and for ε3ε4 carriers the difference in adjustment between those with the least and most illnesses approached 1.5 standard deviations.

Thus, these results suggest that the modifying effect of the APOE gene may follow the pattern suggested by the clinical, neuroimaging and transgenic mouse model literature, whereby the ε2 provides a protective mental health effect (e.g. Lopez-Leon et al., 2008) relative to the ε3 allele and the ε4 allele confers a relative risk of adverse stress reactivity (e.g. Raber, 2007; Gallagher-Thompson et al., 2001). Although the current sample size did not include sufficient number of ε2ε2 or ε4ε4 carriers to allow analyses of these groups I expect that the protective effect of the ε2 allele may be amplified in those with two copies of this allele who could be relatively resilient to illness. Conversely, ε4ε4 carriers suffering

from multiple illnesses may be particularly vulnerable to poor psychological adjustment. In summary, the results suggest that both perceived control and genetic risk can attenuate or exaggerate the link between the number of illnesses and levels of psychological adjustment.

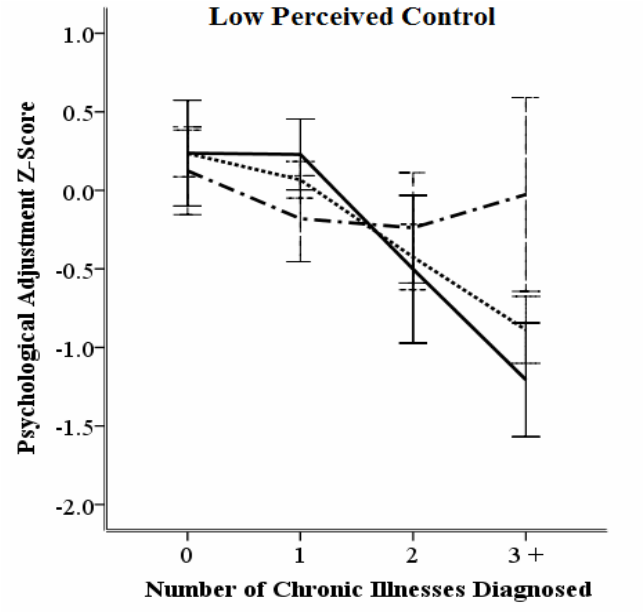
Next, I sought to test if high levels of perceived control and genetic risk might be interlinked and specifically if perceived control could diminish the potential adverse effect of genetic risk on psychological adjustment to illness.

7.4.3. Synergistic effects of illness, perceived control, and genotype

The results showed that the modifying effect of the APOE gene on the illness-adjustment link was highly significant for those with below average perceived control and non-significant for participants with high control. This analysis suggests that perceiving that a high level of control over life events may act to buffer a genetic diathesis towards poor adjustment. Amongst those with low perceptions of personal control carriers of the ε3ε4 genotype showed a marked steep decline in adjustment as a function of illness, as did ε3ε3 carriers, whereas adjustment was unaffected by the number of illnesses for ε3ε2 carriers. However, although the hypothesized pattern of results was identified, as shown in Figure 11, the three-way interaction was marginally significant, potentially due to power limitations in the identification of such higher-order interaction effects (Gauderman, 2002).

In aggregate, I can conclude that as noted in prior research (Johnson & Krueger, 2005) there is partial support for the proposition that genetic factors can modify a stressor-outcome link (in this study the illness-adjustment link) depending on a person's level of perceived control.

(a)



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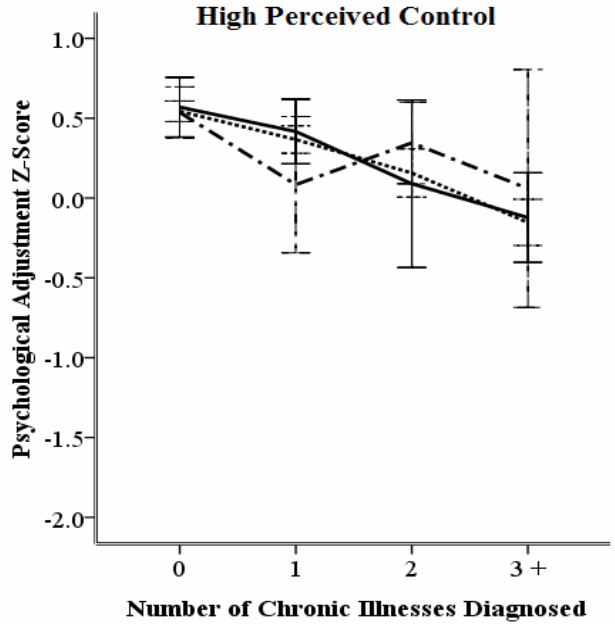

Figure 11. The relationship between the number of illnesses diagnosed and standardized psychological adjustment scores as a function of APOE genotype (continuous line = ε3ε4, dotted line = ε3ε3, broken line = ε3ε2) for those with low perceived control (a) and high perceived control (b).

This finding extends prior research demonstrating that stressors, psychosocial resources, and genetic factors can interact to predict mental health outcomes (e.g. Kaufman et al., 2004; Kilpatrick et al., 2007). In the context of the reserve capacity model it is worth noting that in the current study there was little evidence that either perceived control or variation in the APOE gene were directly related to the prevalence of illness. Thus, it appears likely that these factors are not causally related to illness but may play a vital role in modifying the psychological effects of illness. In particular, the results suggest that perceived control may condition both sensitivity to the psychological effects of illness and the genetic control of sensitivity to illness. To paraphrase Epictetus, one's perception of one's circumstances may override the likely effects of those circumstances and even prevail in overriding genetic vulnerability to adverse reactions to stressful circumstances.

If levels of perceived control do not decline with an increase in illnesses, as suggested in this study, modifying control perceptions (e.g. Craig, Hancock, Chang, & Dickson, 1998) represents one strong avenue for interventions aimed to improve adjustment to illness. An improvement in control beliefs is likely to be accompanied by greater engagement in self-care and better treatment management alongside emotional and functioning benefits. The potential ameliorative effect of an enhancement of control perceptions may be greatest among those at genetic risk of poor adjustment and may even extend beyond adjustment to benefit health.

7.4.4. Psychological adjustment as a mediator between illness and perceptions of health

Self-rated health has been conceptualized as a broad biopsychosocial index of both physical and mental health (Kaplan & Camaeho, 1983). Perceptions of health, rather than reflecting the presence of illness or the objective medical severity of illness, are instead a generalized evaluation of a person's health state. For those attempting to adjust to

Table 14

Result for each hypothesis testing the relationship between the independent variables (number of illnesses, perceived control, APOE genotype) and psychological adjustment, and psychological adjustment as a mediator between the independent variables and health

	Psychological Adjustment			Health
Independent variable	H[a]	*B* (*SE*)	t	H	Mediation[b] (Sobel z)
Number of Illnesses	1.	-.266 (.025)	-10.56***	7.	6.41***
Control[c]	2.	.195 (.021)	9.21***	8.	3.36***
Genotype[d]	3.	ns	ns	9.	ns
Number of Illnesses × Control	4.	-.158 (.038)	-4.00***	10.	ns
Number of Illnesses × Genotype	5.	-.072 (.022)	-3.29***	11.	2.08*
Number of Illnesses × Control × Genotype	6.	.079 (.045)	-1.76†	12.	ns

[b] H = Hypothesis number.

[b] Health Mediation refers to the result of the Sobel equation testing if psychological adjustment mediates between the independent variable or independent variable interaction term and self-rated health.

[c] Control refers to participant levels of perceived control as assessed via the Pearlin-Schooler (1978) mastery scale.

[d] Genotype refers to APOE gene variable (ε3ε2 = low risk, ε3ε3 = medium risk, ε3ε4= high risk)

† < .1, *p < .05, *** p < .004 (Bonferroni corrected alpha level).

the changing expectations, needs, and disease-specific constraints associated with chronic illness, feelings of negativity may develop which can hasten functional decline causing illness to worsen (Friedman, 2008). Thus, at any particular time, self-rated health may partially follow from one's success in adjusting to illness. Although secondary to the primary focus of examining psychological adjustment to illness, this study aimed to identify if psychological adjustment explained the variation in self-rated health accounted for by the number of illnesses, perceived control, the APOE genotype and their interactive effects.

Psychological adjustment was found to mediate the relationship between the number of illnesses and self-rated health (pathways A and B in Figure 9). In the present study, having low levels of negative affect, a reasonable level of functionality, and perceiving one's illness as not posing a great inconvenience, appeared to explain why some elderly people with several illnesses rated their health as good. Further, prior research has shown that perceptions of control are linked to good functional and mental health outcomes among those living with chronic illnesses (e.g. Tennen et al., 1992; Taylor et al., 1991). Perceived control was positively related to perceptions of health in the current sample and mediation analysis showed that psychological adjustment explained this association. Thus, it appears that either having many illnesses or low perceptions of control could lead to changes in psychological adjustment which in turn may impact detrimentally on health perceptions.

Little support was obtained in the analysis examining the idea that perceptions of control would moderate the link between the number of illnesses and self-rated health. It may be the case that high perceptions of control can attenuate the psychological effects of numerous illnesses, promoting psychological adjustment by enhancing well-being and functioning. However, whilst people with high perceptions of control may tend to rate their

health positively in general, this tendency does not appear to increase when multiple illnesses are present. Perceived control could therefore have enhanced beneficial psychological effects in the presence of numerous illnesses, but may not necessarily induce overly misrepresentative positive perceptions of health that may have dysfunctional effects (e.g. failing to maintain vigilant adherence to treatment management regimes).

Although the APOE gene was not directly related to self-rated health, consistent with my initial predictions, the current study did identify a trend towards moderation of the illness-self-rated health link by the APOE gene. In line with predictions, carriers of the ε3ε4 genotype showed a rapid decline in self-rated health associated with numerous illnesses and those with the the ε3ε3 genotype showed a decrease of almost the same magnitude. Those with the ε3ε2 genotype demonstrated little change in self-rated health as a function of illness. This pattern broadly replicated that found in the analysis testing the degree to which variations in the APOE gene modify changes in psychological adjustment as a function of illness. Additional regression analyses and Sobel tests supported the hypothesis that those with many illnesses are likely to adjust poorly to illness, particularly if they are carriers of a risky APOE genotype, and that this failure to adjust may lead to changes in self-rated health. This finding supports the potential existence of a genetic moderation pathway from stress to health in the reserve capacity model, as the APOE gene consistently modified the relations between the stress component (number of illnesses), cognitive/emotional factors (proxied by psychological adjustment), and health (self-rated health).

However, there was no support for a three-way interaction between perceived control, illness, and the APOE genotype in predicting self-rated health. This may indicate that perceived control functions to create positive illusions that can overcome genetic vulnerabilities to improve well-being and the ability to engage in adaptive tasks. But

perceived control may not promote overly unrealistic and potentially maladaptive health perceptions for those with comorbid illnesses and thus fails to buffer the tendency of those with certain APOE genotypes to see their health as poor.

7.4.5. Limitations of the study

One important limitation of the present study is that the cross-sectional nature of the data precludes causal analysis of the relationship between the chronic illness × genotype interaction and psychological adjustment and the role of adjustment in leading to changes in health. Future multi-wave prospective studies could assist in examining the predictive power of chronic illness × genotype interactions in determining psychological adjustment controlling for baseline psychological distress and other adjustment metrics. In addition, it is likely that self-perceptions of health share methodological and conceptual variance with subjective measures of psychological adjustment to illness. For example, those who are prone to experiencing negative emotions often experience an elevated level of psychosomatic symptoms potentially biasing inferred linkages between psychological adjustment and health (e.g. Watson & Pennebaker, 1989). Thus, future studies could measure how health, as proxied by objective endpoints such as mortality or measured indicators of disease (e.g. viral load in HIV), is affected by psychological adjustment and its antecedents and modifiers. Further, studies would benefit from the assessment of multiple facets of psychological adjustment and their psychobiological correlates in context using sophisticated measurement techniques like experience sampling (Hektner et al., 2007) or day reconstruction (e.g. Kahneman et al., 2004) coupled with continuous monitoring of biological functioning. This kind of measurement would allow the effects of psychosocial resources and genetic factors in modifying adjustment and the subsequent pathways to health to be gauged in real-time. Time lagged multilevel regression analyses

would assist in further delineating the causal mechanisms suggested within the augmented reserve capacity framework utilized in this research.

Nonetheless, these limitations considered, the data do indicate that a robust linear association between diagnoses of chronic illness and psychological adjustment exists chiefly amongst those without the protective APOE ε3ε2 genotype. Reverse causation is also unlikely, as this would imply that psychological difficulties increase the incidence of chronic illness only amongst certain genotype groups. However, there were no differences in the prevalence of chronic conditions between the genotype groups implying this explanation is improbable.

It will be important for future studies to delineate pathways explaining how adjustment to chronic illness varies by APOE genotype. For instance, consistent with the 'vascular depression' hypothesis, APOE ε3ε2 may protect against vascular risk affecting prefrontal functioning, enhancing the ability to regulate the emotional response to stressors (Bennet et al., 2007; Holley, Murrell, & Mast, 2006; Kalix, Meynet, Garin, & James, 2001). Alternatively, the APOE gene may be a marker for a psychological or social process such as differences in appraisal or social support, which may moderate the link between chronic illness and psychological adjustment. Future studies, of this possible 'Epictetus gene', should attempt to integrate psychological theory, functional imaging, and momentary assessment techniques for phenomenological, psychobiological and contextual sampling. Such studies will assist in explicating the cascade of biological events underlying reactivity to the stress of chronic illness and the factors that may prevent this. This knowledge could be used to develop novel treatment programs to protect those at genetic risk of poor adaptation to chronic illness in order to ensure they master the necessary adaptive tasks, and their health impairments do not develop into disabling

mental and physical conditions. (Brody, Beach, Philibert, Chen, & Murry, 2009; De Silva et al., 2009).

7.4.6. Conclusions

Over eighty percent of people aged 65 can expect to have at least one chronic condition, with one in four people having greater than three illnesses (Taylor & Stanton, 2007). The current study examined heterogeneity in psychological adjustment to chronic illness and showed that those with numerous chronic illnesses and low levels of perceived control had poor self-rated health, largely as a result of unfavorable patterns of psychological adjustment. The relation between illness and psychological adjustment was buffered substantially by perceived control and the APOE genotype. In the context of the reserve capacity model it is interesting that people with strong perceptions of personal control, appear to be capable of, at least partially, overriding the effects of uncontrollable variation in their genetic make-up on psychological adjustment. Thus, it may be the case that psychosocial resources can compensate for deficits in genetic resources, as assessed by the possession of risk genotypes, in determining mental and physical reactions to stress. Overall, six of the twelve study hypotheses were supported at the Bonferronni corrected alpha level and two further hypotheses were supported at less stringent significance levels. This indicates substantial support for key proposed extensions to the reserve capacity model, as shown in Figure 9. In particular, this study provides evidence to suggest that the emotional and functional processes that characterize psychological adjustment to illness may be derived from a complex process that is contingent on both psychosocial resources and genetic modifiers that act both independently and synergistically to affect adjustment and potentially even the health changes that follow.

Chapter 8

General Discussion

8.1. Summary of the general discussion

This thesis outlined a set of proposed relations specifying how psychosocial resources and genetic factors may influence health by determining the emotional and physiological response to stress. It also addressed issues of reciprocal causation that are problematic for the reserve capacity model (shown in Figure 1; Gallo & Matthews, 2003). In the discussion which follows I firstly summarize the main findings of this thesis and then consider the support garnered for the reserve capacity model in its current format. Next, I describe how the collective set of relations outlined in the six empirical chapters of this thesis provide support for an augmented version of the reserve capacity model, shown in Figure 12. I pay close attention to two potentially problematic areas for the reserve capacity model: the testing of reverse causality feedback loops and the role of genetic factors. I end this general discussion chapter by suggesting ways to empirically test the proposed multi-component interacting biological and psychological relations linking stress and health.

8.2. Summary of main findings

In Chapter 2 an explicit attempt was made to show that a specific relation between emotion and heart rate (an index of sympathetic adrenal medullary axis activation) could be identified in everyday life after adjusting for the role of health behaviours, baseline health factors, and several other potentially confounding contemporaneous factors. High levels of negative affect reported during a weekday day were found to be associated with a raised heart rate. Chapter 3 built on this analysis by showing a specific theoretically important aspect of the reserve capacity, self-control, to be linked to favourable patterns of

psychophysiological functioning, potentially by altering levels of emotion. More precisely, those with high levels of self-control had low heart rates, high levels of heart rate variability, and a steep cortisol slope. Self-control was associated with low levels of emotional variability which partially explained the links between self-control and heart rate and self-control and the cortisol slope.

Chapters 4 and 5 introduced the first proposed extension to the reserve capacity model by evaluating the idea that health states and psychobiological factors may influence emotion. The proposed pattern of theoretically and empirically supported relations was in the opposite direction to that proposed in the reserve capacity model. Chapter 4 showed that high levels of morning cortisol were closely linked to positive affect in the first half of the day, particularly amongst distressed people. Chapter 5 extended the analysis of emotion and psychobiological functioning by demonstrating that obesity-related inflammation predicted the neurovegetative symptoms of depression.

Chapters 6 and 7 focused on the second extension this thesis makes to the reserve capacity model by outlining how genetic factors may modify the effects of stressors on emotion and health. Chapter 6 identified that variation in the apolipoprotein E gene interacts with the level of exposure to the exogenous stress of a major earthquake to predict self-rated health one year later, thus spanning the proposed reserve capacity model from stress to health outcome. Elderly Taiwanese people who were highly exposed to the 'Chi-Chi' earthquake experienced low levels of self-rated health subsequently only if they were APOE ε4 carriers.

Chapter 7 integrated many of the insights from the preceding chapters and tested several tenets of the proposed theoretical model. It did this centrally by showing that the stress of chronic illness interacts with both genetic factors (the APOE gene) and a person's reserve capacity (perceived control) to predict psychological adjustment to illness (a

composite of emotional and behavioural/functional capacity variables) which partially explains individual differences in self-rated health. The central findings from this study were that those with high levels of perceived control adjusted well to comorbid illnesses in contrast to those with low personal control. Also, people with the APOE ε3ε2 genotype adjusted well to multiple illnesses, whereas ε3ε3 carriers suffered a decline in adjustment and ε3ε4 carriers experienced a major decrease in adjustment as a function of the number of illnesses diagnosed. Perhaps most importantly, high levels of perceived control appeared to buffer the genetic sensitivity to poor adjustment to illness conferred by the variation in the APOE gene.

8.3. Components of the reserve capacity model tested in the current research

In the augmented version of the reserve capacity model used in this research socioeconomic status was not included as a component. This was because socioeconomic status is considered to influence health primarily by increasing the frequency and intensity of stress exposure (e.g Lantz, House, Mero, & Williams, 2005). If socioeconomic status or race/ethnic or minority grouping are conceptualized as psychosocial adversity stress vulnerability factors they can thus be studied as antecedents to stress, as discussed in detail in section 8.7. below (e.g. Myers, 2009). However, for the purposes of this thesis such precursors to stress were not of specific interest. Rather, stress exposure was considered as a starting point and I aimed to specify and provide empirical support for the interweaving psychological and biological pathways that link stress to health in the reserve capacity model.

Firstly, the reserve capacity model suggests that those exposed to more stressors may experience an elevated level of negative affect and diminished positive affect (arrow A in Figure 12). The current research identified some support for this contention. Those who had been diagnosed with several comorbid illnesses (Chapter 7) were found to show

poor psychological adjustment, a composite index which chiefly contained measures of cognitive/emotional factors. The relation between stressors and detrimental emotional effects is of limited interest in itself. However, factors which explain (i) how these emotional changes may induce health effects or (ii) how heterogeneity in emotional reactions to stressors emerges are potentially of greater interest. I now turn to each of these points and detail the support provided by the current research.

Prior research suggests that emotion may contribute to the relation between stress and health as negative states such as depression, hostility, and anxiety have been closely linked to ill-health, whereas positive emotions are associated with health and longevity (Chida & Steptoe, 2009a; Chida & Steptoe, 2008). There are a broad array of intermediate factors that could explain the relation between emotion and health, the foremost of which are health behaviours and psychophysiological reactivity and recovery which could lead to detrimental health changes. In the first empirical chapter of this thesis I sought to outline a relation between emotion and heart rate, thus focusing on arrow B in Figure 12. An elevated heart rate is a risk factor for an array of detrimental cardiovascular health outcomes from the development of hypertension (Palatini et al., 2006) to heart attack and cardiovascular mortality (Gillum et al., 1991; Hsia et al., 2009).

In Chapter 2 no link was found between negative affect in daily life and heart rate measured at rest in the laboratory. Other studies have shown psychobiological functioning measured in context to be unrelated to a general trait measure of well-being but to be predicted by state levels of affect assessed with the DRM (Bhattacharyya et al., 2008). Similarly, in the current research a DRM measure of negative affect in context (composed of depression, irritation, stress, and impatience scores), was positively related to contiguously measured heart rate. As the link between emotion and health could be

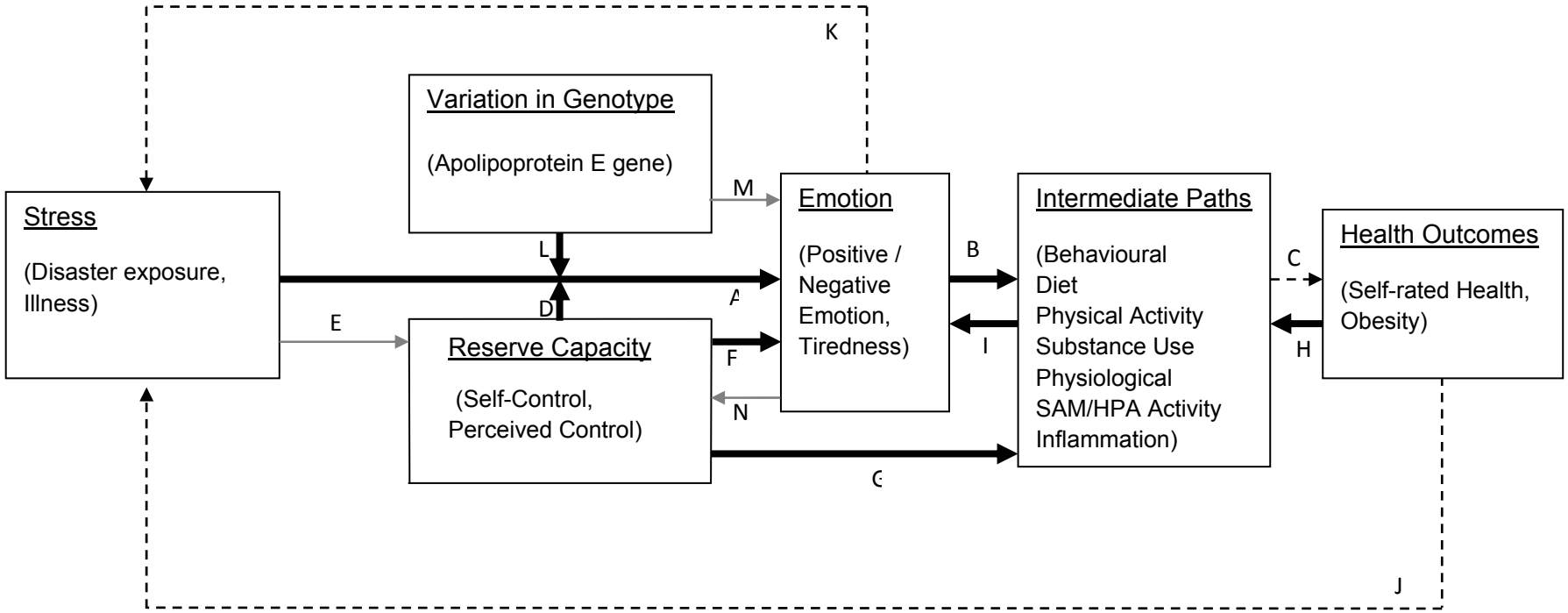

Figure 12. Bidirectional resource model outlining the relations between key variables utilized in the current research. Continuous lines indicate relationships which were supported by the current research, grey lines indicate unsupported relationships, and broken lines refer to relationships which are inferred to exist but were not explicitly tested in this research.

mediated by health behaviours these factors were controlled for as were numerous situational factors, thus illustrating the robustness of the negative affect-heart rate link.

The aforementioned study utilized computer-assisted online day reconstruction to help participants to accurately recall the activities, interactions, and experiences of the previous day. Heart rate was assessed using a lightweight non-invasive heart monitor worn on the chest. Although this approach can yield a large quantity of data that needs to be refined and carefully scanned for outliers and periods of missing data, both self-report and objective physiological measures can then be combined and analysed with relative ease using multilevel statistical software. At present, the availability of sufficiently non-invasive technology is the central factor that constrains the rolling out of many studies of this type. For instance, a key limitation of this study was that devices for monitoring ambulatory activity were not accessible to the study. However, already options are becoming available for the assessment of ambulatory activity using sensitive piezoresistive and piezocapacitive sensors that can separate the degree of movement, its direction, change in posture, and acceleration (e.g. see Bussmann et al., 2009).

This thesis also addressed factors that may explain heterogeneity in emotional reactions to stressors. Psychosocial resources are the main factors currently thought to contribute to emotional reactivity and psychobiological functioning in the reserve capacity model (Gallo & Matthews, 2003). Chapter 3 aimed to show that an important psychosocial resource, self-control, may lead to changes in patterns of emotion (arrow F) that could at least partially explain an association between self-control and measures of cardiovascular and neuroendocrine functioning (arrow G). In this study I found, firstly, that those with good self-control had stable patterns of emotion (arrow F). This is a novel finding and there are several reasons why self-control may be linked to the changeability of emotions that I will outline briefly.

Self-control is a highly adaptive and potentially uniquely human capacity that determines the ability to suppress certain behavioural tendencies, feelings, and impulses. Through using self-control people can optimize the fit between their traits and their current environment thus promoting happiness and health (Tangney et al., 2004; Baumeister & Alquist, 2009; DeWall, Baumeister, Stillman, & Gailliot, 2007). One key idea behind this stream of research is that in normal circumstances when regulatory resources are plentiful the person behaves in line with his or her behavioural standards. However, when resources are depleted the person may resort to underlying impulsive, behavioural, and emotional tendencies (Friese & Hofmann, 2009).

This is a dual-process explanation whereby control is exerted to inhibit or manipulate inappropriate, inaccurate, or unwanted responses. For instance, Hofmann, Rauch, and Gawronski (2007) showed that when self-control resources were high eating behaviour was consistent with personal standards for dietary restraint. In contrast, when regulatory resources were low eating was predicted by automatic or implicit attitudes to the tempting candy used. Self-control can modify the expression of existing traits, as in the case of attitudes towards tempting candy. Self-control could also modify the relation between psychosocial resources like extraversion and emotional and physiological reactivity. Thus, if an introverted person has a natural tendency to withdraw from social interaction they could utilize their regulatory resources to override the expression of this avoidant trait and attempt to engage with others (Baumeister, Gailliot, DeWall, & Oaten, 2006). Thus, a person's ability to exert self-control could be utilized to achieve optimal emotional outcomes by modifying the expression of traits, selecting appropriate situations, controlling attention where needed, and influencing behaviour favourably.

Self-control may contribute further to the reserve capacity model by acting as a special psychosocial resource, in that it may modify the effects of other such resources.

Studies of psychosocial resources often imply that such resources are universally beneficial. However, it is easy to imagine situations whereby, for example, acting in an overly outgoing fashion (e.g. at a funeral, in a library) could be inappropriate. Moreover, there may be cultural differences in the extent to which certain traits are greeted with favorable feedback from others. Traits such as extraversion and outgoing sociability may be more valued in Western society than in the East (Ahadi, Rothbart, & Ye, 1993). It might be the case that the ability to tailor ones actions and expressions to match the situational environment is a capacity that is universally important. Self-control is the ability to override prepotent responses, in line with one's standards, to accomplish important goals. Thus, in situations when, for example, optimism or extraversion are not the optimal response, self-control may modify the effects of these psychosocial resources in order to invoke a more adaptive response.

I thus propose that self-control is likely to be an important psychosocial resource that merits examination within the reserve capacity model. The self-control literature suggests that self-regulatory resources are often used to adaptively diminish or enhance underlying tendencies towards avoidance or approach behaviours in order to achieve goals and sustain well-being (e.g. Carver & Connor-Smith, 2010). This two-layered conceptualization of self-control suggests that traits like aggression or depression which are central to stress reactivity, may be indicative of a bidirectional relationship between reward-sensitivity and control and between punishment-sensitivity and control respectively (Carver, Johnson, & Joorman, 2009). Carver, Johnson, and Joormann (2008) summarize research linking low serotonergic functioning to a lack of constraint. They suggest that the common link between avoidance traits like depression and anxiety and approach traits like impulsivity and aggression is a lack of constraint as indexed by the contribution of low serotonergic functioning to both types of conditions. When serotonin is low underlying traits may be amplified as is the case in experiments showing tendencies towards

aggression and social withdrawal to be displayed when participants self-regulatory resources are temporarily 'depleted'.

Indeed, several studies have shown that decreasing tryptophan (a precursor to serotonin) can promote depression or impulsivity whereas increasing serotonin through tryptophan administration can have the reverse effect (Cools, Blackwell, Clark, Menzies, Cox, & Robbins, 2005; Young, aan het Rot, Pinard, & Moskowitz, 2007). There is also evidence to suggest that the modulating serotonin only has mood effects when underlying traits like depression or aggression exist (Ruhe, Mason, & Schene, 2007). For instance, aggressive people have been shown to demonstrate less hostile responses to provocation in the form of electric shocks whereas no such modulation was observed in non-aggressive people (Berman, McCloskey, Fanning, Schumacher, Coccaro, 2009).

Whether or not serotonin underlies the ability of self-control to dampen emotional reactivity it is likely to remain the case that the changeability in affect may place physiological demands on emotion-activated stress systems. Thus, the process of returning such systems to an optimal set-point may occur more frequently and be more difficult among those who show a high degree of variability in the intensity of their emotions. In line with this contention I showed that those with high levels of self-control had low heart rate levels and a steep decline in cortisol over the course of the day and both of these relations were mediated, in part, by how variable participants affect levels were (arrow F and B). It thus appeared that the tendency of self-control to stabilize one's emotional state had positive implications for biological functioning. Self-control also was linked to higher heart rate variability, a finding that was not mediated by emotion suggesting support for an independent link between this psychosocial resource and patterns of psychobiological functioning (arrow G). Future studies will assist in identifying if this pattern of results can

be replicated and the extent to which changes in physiological functioning that follow from self-control could lead to beneficial health effects.

As suggested in the case of self-control, psychosocial resources may attenuate emotional reactivity to stressors. Although this moderation pathway was not tested in the case of self-control in Chapter 3, in Chapter 7 I tested the extent to which another psychosocial resource, perceived control, attenuated the link between the stress of illness and psychological adjustment (arrow D). The reserve capacity model suggests that those who have encountered frequent stressors throughout their lives may not have developed the same level of reserve capacity as indexed by intrapersonal, interpersonal, and tangible resources, and for this reason may be less able to manage contemporary stressors (arrows E and D). In addition, independent of stress, psychosocial resources may beneficially influence positive and negative feelings and lead to favorable health behaviours and patterns of psychophysiological reactivity (arrows F, G) as shown for self-control in Chapter 3.

In chapter 7, perceived control was found to be unrelated to the stressor examined (i.e. the number of illnesses a person had been diagnosed with) thus failing to support the stressor-resource pathway in the reserve capacity model (arrow E). As a trait perceived control appeared to be somewhat robust to adversity though no causal inferences could be made due to the cross-sectional nature of the data in this study. This finding contradicts a recent review of studies testing the reserve capacity model which proposed that psychosocial resources were more likely to mediate the effect of stressors on emotion than to moderate this relation (Matthews et al., in press). It may be the case that the effects of illness among a group of relatively young elderly people (aged 54 to 75) have not persisted long enough to erode psychosocical resources which have been accumulated over a lifetime. The findings reviewed by Matthews and colleagues focused largely on the effects

of socioeconomic status on psychosocial resources. It is possible that the stress associated with low socioeconomic status occurs at critical periods early in life and thus restricts the development of psychosocial resources. However, when such resources are established they may be resilient to later stressors such as illness.

Rather than identifying support for psychosocial resources as a mediator between stress and emotion I found support for resources as a moderator of this link. Among vulnerable people who had been diagnosed with multiple illnesses those with a strong sense of personal control appeared to adjust well to illness (arrow D). This finding is consistent with prior research that has identified a similar protective role for perceived control in attenuating the mental and physical health effects of low education or low socioeconomic status (e.g. Lachman & Weaver, 1998; Turner & Noh, 1983). In the context of illness, those with strong perceptions of personal control may anticipate successful management or recovery from their condition and thus feel relatively positive about their illness. They may focus on adhering to treatment regimes and controlling aspects of their self-care management and thus achieve personal and rehabilitation goals and a sense of success in the illness-domain. Those with a more fatalistic perspective may fail to perceive contingencies between their actions and success in improving the trajectory of their illness (e.g. Alloy & Abramson, 1982). These people may not invest sufficient effort in managing their disease and could go on to develop a generalized sense of helplessness which could lead to depression.

Further, the results of this study support a mediation model where high levels of perceived control positively predict psychological adjustment to illness which in turn predicts perceptions of good health (pathway linking arrows F, B, C). However, from this research it is currently unclear how improvements in adjustment to illness potentially induced by perceived control might alter one's health state. An examination of changes in

disease-management practices or psychobiological functioning would help specify the mediating factors linking the presence to illness to one's level of health (arrows B, C). In addition, assessing both perceptions of health and objective disease markers would identify if perceived control alters health appraisals or objective end-points as suggested by prior research (e.g. Rodin & Langer, 1977).

To summarize, in this thesis several pathways within the reserve capacity model were tested. There was evidence that stress can predict health and substantial support for mediating and moderating pathways. For example, stressors were shown to be linked to emotional changes (arrow A) and emotional intensity and variability was related to the activity of the sympathetic adrenomedullary system and the HPA (arrow B). Further, the reserve capacity, as indexed by the psychosocial resources self-control and perceived control, predicted emotional stability and raised levels of psychological adjustment (an index consisting mainly of measures of well-being) respectively (arrow F). There was little evidence that stressors can induce change in psychosocial resources (arrow E). However, there was clear support for a moderating role of perceived control in promoting psychological adjustment to illness (arrow D). As well as bolstering the existing reserve capacity model this thesis research utilized sophisticated diary methods for the measurement of experience and non-invasive technology for real-time psychophysiological monitoring. In addition to incorporating novel methodologies for the assessment of experience and functioning in context, this thesis aimed to test the presence of relationships that are not given due attention in the current reserve capacity model.

To address these shortcomings I pay close attention to two problematic areas for the reserve capacity model: the testing of reverse causality feedback loops from health conditions to emotion and the inclusion of a role for genetic factors in stress reactivity. In both cases this research extends the reserve capacity model by explicating the bidirectional

nature of the relationship between biological and psychological factors. In addition, I consider the extent to which variation in genotype could be considered a resource that becomes increasingly important in times of stress. Thus, I have labeled the extended version of the reserve capacity model utilized in this thesis as the 'bidirectional resource model', as shown in Figures 12 and 13.

Specifically, this model replicates the relations within the reserve capacity model but differs in three important ways. Firstly, it does not limit itself to examining socioeconomic disparities in health and instead considers multiple potential stress antecedents as discussed below (e.g. organizational justice, racism, health conditions). Secondly, the bidirectional resource model proposes that genetic as well as psychosocial factors can modify stress reactivity. Thirdly, the bidirectional resource model extends the reserve capacity model to consider the possibility that in some cases health conditions may lead to changes in emotion by increasing stress (arrow J in Figure 12) and influencing intermediate psychobiological pathways (arrows H, I) rather than vice versa as proposed in the reserve capacity model (arrows B, C).

8.4. Reverse causation in the bidirectional resource model

The reserve capacity model aims to account for the psychological and psychobiological factors that lead to poor health among those exposed to adverse conditions. However, it is clear that persistent health conditions are a large part of life for many people, particularly the elderly (e.g. Stanton et al., 2006). These conditions can adversely affect human welfare and functioning and this decline may hasten the progression of illness (Chida & Steptoe, 2008). One goal of proposing the bidirectional resource model is to account for the effect of illness.

A simple way to do this is to conceptualize illness as a stressor and take health conditions as the starting point in pathway from stress to health (as illustrated by pathway

1 in Figure 13). This approach assumes that the challenges of illness can be thought of in much the same way as external environmental stressors, as has been proposed in prior research (e.g. Kim et al., 2009). For example, treatment regimes can place constraints on numerous life domains and illness is often characterized by aggravating symptoms like coughing or internal discomfort. This potential intrusiveness is akin to a stressor in that people are likely to differ in their cognitive/emotional sensitivity to its effects depending on certain factors like their level of psychosocial resources.

However, upon closer examination it is evident that there are certain health conditions that may affect well-being directly through their impact on psychobiological processes like inflammation, cortisol production, and heart rate variability (Miller et al., 2009; Tops et al., 2006; Porges et al., 2007). For instance, the neurochemical action of proinflammatory cytokines released in arthritis may generate the symptoms of depression (Heiberg et al., 2005; Quinn et al., 2005). These initially imperceptible biological changes may induce changes in mood that are unrelated to the intrusiveness of illness and occur outside of the realm of symptom perception. Such effects are modelled as pathway 2 in Figure 13. When a substantial change in well-being is noticed by the person the detrimental effects of psychobiologically induced mood changes may become a source of stress and alter the perception of stressors, thus feeding back into the model through pathway 3 in Figure 13. To summarize Figure 13: pathway 1 suggests that illness can be viewed as a stressor as outlined in detail in Chapter 7, pathway 2 invokes the idea that health conditions can alter well-being directly through biological pathways, and pathway 3 reiterates the existing tenet of the reserve capacity model that emotional changes can lead to stress or kindle the potential detrimental influence of stressors (Gallo et al., 2009). Thus, pathway 2, the link from health to emotion, remains the key set of relations that remains unaddressed.

In two studies I aimed to show, firstly, that it was likely that psychobiological functioning could potentially generate trends in affect. I then aimed to demonstrate that a health condition could feasibly predict low levels of well-being and that this link could be explained by patterns of psychobiological functioning that were largely attributable to the illness. More precisely, the first study showed that low morning cortisol levels in the first half hour after waking were associated with low levels of positive affect in the first half of the day. This finding links well with prior descriptions of cortisol administration as producing a "vitalisation" effect where people experience raised levels of energy and vigour (e.g. von Zerssen, 1976).

Following this, I showed that the inflammatory marker C-reactive protein partially explained the presence of the neurovegetative symptoms of depression among obese people. As low-grade inflammation is proposed to be derived centrally from adipose tissue in obesity (Xu et al., 2003) this mediation analysis suggests that inflammation may be one way that obesity could lead to depression. These two studies provide tentative support for the idea that a pathway exists from health conditions to psychobiological functioning and subsequent cognitive/emotional factors as proposed in the bidirectional resource model. However, longitudinal studies multiple objective measurements of health states and psychobiological functioning, coupled with detailed assessments of subjective variables (e.g. symptom perception, affect) are needed to delineate the relative contributions of the different pathways outlined in Figure 13. Such studies will help specify how illness can lead to changes in mental health and will assist in identifying the psychological factors that determine health trajectories following the diagnosis of illness. Recent research in psychology and psychiatry suggests that in order to adequately understand such health outcomes it is important to consider the role of genetic factors alongside details of stressors and subjective and psychobiological measures (e.g. Kim et al., 2009).

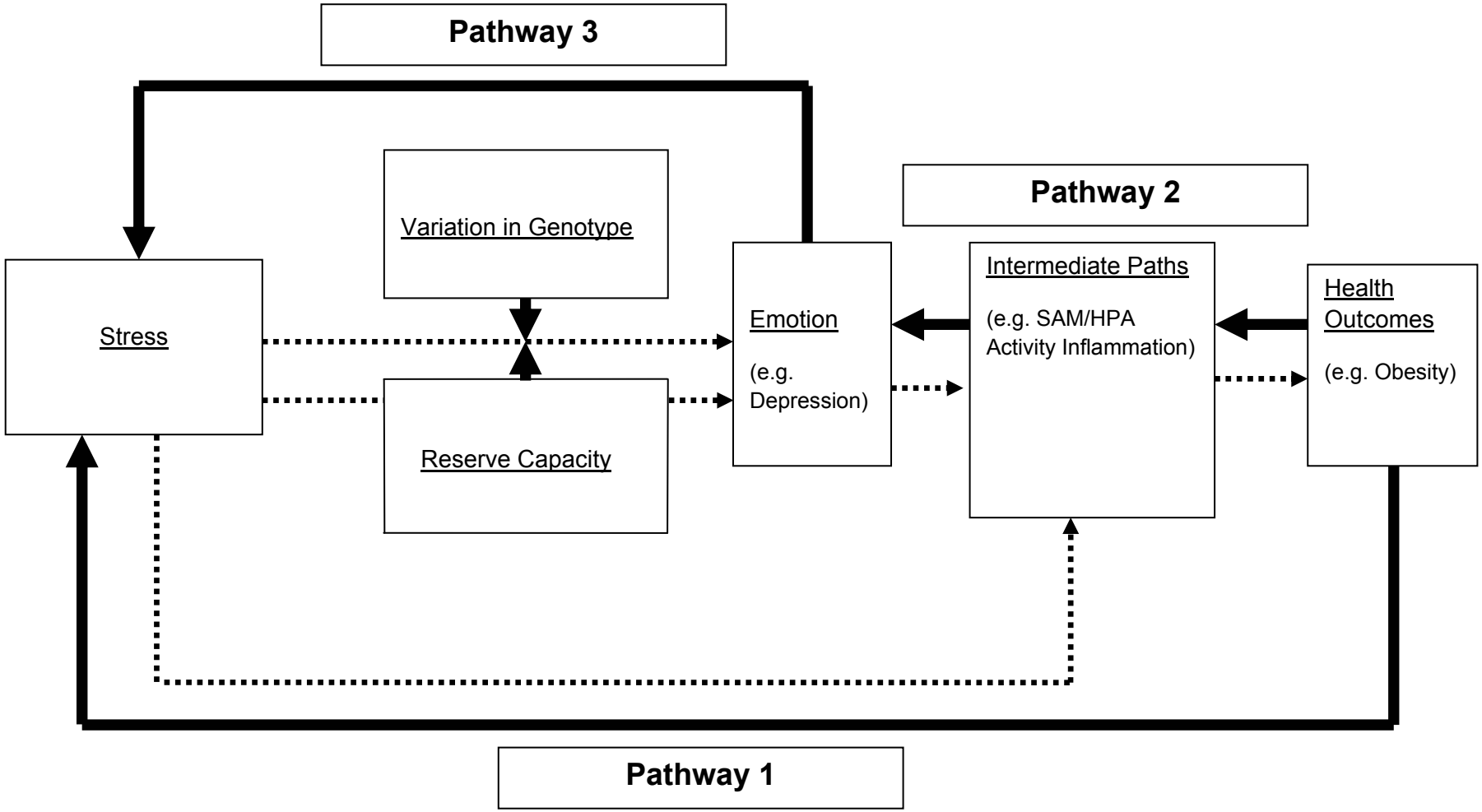

Figure 13. Feedback processes in the bidirectional resource model (continuous lines) illustrating how health outcomes can become stressors (pathway 1) and how health outcomes may have psychobiological and emotional effects (pathway 2) which can lead to elevated levels of stress (pathway 3).

8.5. Genetic factors in the bidirectional resource model

A key goal of this thesis was to extend the reserve capacity model to include genetic factors and to empirically test the set of relations proposed. The gene-environment interaction literature in psychiatry is founded on the diathesis-stress model (Monroe & Simons, 1991) which proposes that mental health outcomes are contingent on the presence of stressors and genetic vulnerability to the adverse effects of such stressors (Moffitt et al., 2006; Rutter et al., 2006). In the bidirectional resource model identifiable genetic variation is proposed to modify the relation between stressors and cognitive/emotional factors, potentially influencing intermediate variables and thus leading to health effects (arrows L, B, C in Figure 12).

In an initial test of these relations I examined the link between exposure to an earthquake and self-rated health one year later in a population-based sample of elderly Taiwanese people. One interesting aspect of this study was that earthquake exposure was likely to be broadly randomly distributed in the population of Taiwanese people. Unlike the vast majority of gene-environment interaction studies this design largely ruled out the possibility of a gene-environment correlation where risky genes may attract risky environments. The analysis showed that those who scored highly on measures of either subjective or objective exposure to the earthquake had low self-rated health a year later only if they were carriers of the APOE ε4 allele. The ε4 allele contributes to dysfunctional lipid transport and has been shown to predict adverse stress reactivity in clinical, neuroimaging, and transgenic mouse studies (e.g. Peavy, 2008; Robertson et al., 2005; Gallagher-Thompson et al., 2001). The link identified between objective earthquake exposure (i.e. having property damage or having to move from one's home as a result of the earthquake) and low self-rated health among ε4 allele carriers was particularly compelling. This finding suggested that it is possible for genetic factors to modify the

relationship between stressors and health outcomes. In a second study I aimed to verify that genetic factors can modify the extent to which stressful circumstances are linked to adverse mental and physical health outcomes in a study that considered several of the intermediate pathways in the bidirectional resource model.

This second study examined if the APOE gene can modify links between illness, psychological adjustment, and perceptions of health. Adjustment was gauged using three measures of cognitive/emotional factors and two measures of current functioning (e.g. ability to carry out common instrumental tasks of daily living). Consistent with the hypotheses psychological adjustment declined in line with the number of illnesses diagnosed and this decrease was strongest for ε3ε4 carriers, less so for ε3ε3 carriers, and almost absent for those with the ε3ε2 genotype. These findings reiterate the potential detrimental effect of the ε4 allele in adaptation to adverse circumstances. In addition, they propose a protective role for the ε2 allele as has been identified in a recent meta-analytic investigation of gene polymorphisms linked to depression (Lopez-Leon et al., 2008). This pattern of moderation was also observed in the analysis of the relation between illness and self-rated health with carriers of the ε3ε4 genotype with multiple illnesses rating their health as particularly bad and those with several illnesses and the ε3ε2 genotype rating their health as particularly good. Crucially psychological adjustment mediated the link between the illness × APOE genotype interaction and self-rated health, suggesting that the genetic modification of the link between illness and adjustment may have health consequences.

Thus, the contention that identifiable variation in a candidate gene may modify the link between stressors and cognitive/emotional factors (arrow L in Figure 12) which could invoke health changes was supported by two studies. However, it must be noted that meta-analytic support for the modifying effect of the APOE gene on stress reactivity is

warranted as is further research delineating the biological processes underlying the role of the APOE gene in producing health outcomes. Further, the independent and interacting roles of numerous other promising candidate genes such as the brain-derived neurotrophic factor gene (e.g. Savitz, Solms, & Ramesar, 2006; Savitz et al., 2007), the catechol-O-methyl transferase gene (e.g. Egan et al., 2001), and the serotonin transporter gene SLC6A4 (e.g. Canli & Lesch, 2007) require attention within the bidirectional resource framework. Integrating gene polymorphisms into the bidirectional resource model is useful as this model can generate numerous predictions regarding the causal mechanisms linking stress and health. This integration would extend current research on gene-environment interactions that focuses largely on single interactive effects and ignores higher-order interactions and multi-step links between theoretically meaningful psychological and biological pathways (e.g. Moffitt et al., 2006; Matthews et al., in press).

For example, in chapter 7 I showed that the psychosocial resource, perceived control, interacts with the APOE gene to predict how people adjust to chronic illness. This finding showed that those with high levels of perceived control adjusted well to illness and did not appear to be affected by genetic vulnerability. On the other hand, those with low levels of personal control suffered poor adjustment to comorbid illnesses, particular if they were carriers of the APOE ε3ε4 or ε3ε3 genotypes. This finding suggests that perceiving that one is in control of life's events could enable people to avoid the adverse experiences typically associated with illness and even to override a genetic vulnerability towards maladaptive psychological reactions to illness.

In terms of the bidirectional resource model, this result indicates that the genetic modification of cognitive/emotional reactions to stressors may be differentially expressed for those with low rather than high levels of psychosocial resources. If this interaction between psychosocial resources and genetic variation can be replicated in larger samples,

across diverse age-groups, with a range of stressors and outcomes, it is likely to raise interesting possibilities for public policy. For instance, it may be more worthwhile to invest resources in psychosocial interventions which aim to enhance how people view their objective circumstances (e.g. Craig et al., 1998). Furthermore, such psychosocial interventions may be most beneficial for those with risk genotypes (e.g. Brody et al., 2009). Thus, it could be possible to intervene early among those who appear at genetic risk of suffering adverse health consequences following a major stressor.

Several further challenges remain in integrating genetic factors into the bidirectional resource model. In the research in this thesis there was little evidence that genetic variation could directly influence cognitive/emotional factors and augment health outcomes (arrow M in Figure 12). However, this remains a distinct possibility for future research. Further, genome-wide gene-environment interaction studies guided by complex algorithms could provide exploratory findings supporting the role of important but yet previously unexamined gene polymorphisms in stress reactivity. In addition, the explication of the role of epigenetics is one key area that is just beginning to be explored (Mill & Petronis, 2007; Sweatt, 2009). It is likely that the expression of certain genes is influenced directly by certain stressors (e.g. chemical exposure) and emotional reactions to psychosocial stressors through epigenetic programming (e.g. DNA methylation, histone modification) (Stahl, 2009). Epigenetic processes have the potential to introduce new relations into the bidirectional resource model and to further specify how the mental and physical health effects of stressors unfold. A final challenge in fully explicating the role of genetic factors in stress reactivity is the development of more efficient and accurate measurement techniques for the assessment of stressors and their psychological and physical effects (Monroe, 2008; Monroe & Reid, 2008). Such advancements would facilitate a coherent integration of the gene-environment literature with the existing literature on stress and stress reactions through facilitating the measurement of the

important relations proposed in analytic frameworks such as the bidirectional resource model.

8.6. Central limitations and suggestions for future research

Several limitations of the present research have been mentioned throughout the thesis, the most prominent of which is the problem of making inferences based on correlational and cross-sectional data. This limitation is particularly important considering that the reserve capacity model and its extended form, the bidirectional resource model, both propose a temporal sequence of events (see Figure 1). Although some of the studies examined variation in the study variables over time (e.g. the relation between morning cortisol and affect as a function of time) these relations provide only suggestive evidence of the existence of theoretically and empirically supported causal pathways. There are several ways that this limitation can be addressed.

For instance, in the case of the relationship between heart rate and negative affect in everyday life it is possible to conduct time-lagged analyses that contrast the predictive value of a model of negative affect in an episode as a predictor of subsequent heart rate and vice versa. This would provide further support for the likely direction of causation between the two variables. Further, in the case of variables that are less likely to change over brief time periods such as psychosocial resources and health, it may be worthwhile to collect longitudinal data over extended periods. Such multi-wave data would assist in identifying the potential causal direction of relations between resources like self-control and perceived control, patterns of affect, and health outcomes.

A second major caveat of the findings in this thesis concerns the possibility of Type 1 error. Whilst I corrected for multiple comparisons in Chapter 7 due to the large number of contrasts being made, there was no correction made in other studies or across all studies. Thus, the results should be interpreted in light of the possibility of significant results

arising due to chance. In terms of the composition of the study samples, it must be noted that the number of participants in the study upon which the first three empirical chapters were based was small. In addition, it is likely that this group of college students was relatively homogenous in terms of socioeconomic status and cultural and ethnic backgrounds. Caution is warranted in generalizing the findings from this group to more diverse groups.

However, it is also possible that in some cases relationships may have been attenuated due to restriction of range in the sample. For example, college students are likely to have high levels of psychosocial resources such as self-control as they have shown persistence in pursuing long-term goals (e.g. Duckworth, Peterson, Matthews, & Kelly, 2007). In a more diverse sample the greater variation in self-control may have strengthened the hypothesized relations. Another key limitation relating to the sample composition is the diversity of the samples examined. Whilst it is useful to test theoretical frameworks across widely varied samples, it is currently unclear if the findings identified in the group of students in the initial chapters generalize to the elderly samples in the later chapters and vice versa. A key issue for future research will be to outline the strength and stability of the relations within the reserve capacity model, and its extended version, the bidirectional resource model, across the lifespan.

To accurately do this requires that a further limitation of the current research be addressed: the issue of non-adherence to the study protocol (see Appendix A). As the heart rate monitor utilized as part of the research for chapters 2 and 3 automatically logged the time of each measurement it was clear when participants began and completed data collection. However, compliance to the cortisol assessment protocol was self-assessed. I removed participants from the analysis if they reported providing cortisol samples at times that differed substantially from the allotted time (i.e. greater than 10 minutes).

Unfortunately, it is likely that self-reported compliance differed from actual compliance (e.g. Stone, Shiffman, Schwartz, Broderick, & Hufford, 2002). For example, in an intensive study that assessed salivary cortisol at 50 random times over the course of 5 days self-reported compliance was 96.4% whereas electronically verified compliance was just 81% (Jacobs, Nicolson, Derom, Delespaul, van Os, & Myin-Germeys, 2005). Another study found a self-reported compliance rate of 93% though just 71% of the samples were collected at the reported time (Broderick et al., 2004).

These studies illustrate that there is a clear motivation on the part of participants to present themselves as adhering to the study protocol even when this may not be the case. Misreporting of compliance is unlikely to be orthogonal to the study variables in most psychological studies, especially those involving variables such as emotion and self-control. For instance, those with high self-control could adhere well to the study protocol and report their adherence honestly. Alternatively, because people with high self-control also score highly on measures of social desirability (e.g. Tangney et al., 2004) they could be more motivated than impulsive people to present themselves well when they fail to adhere to the study protocol. In the absence of objectively verified compliance data that identifies misreporters it is not possible to construct a profile of these participants and gauge their influence on the results of the current research.

Thus, future studies would benefit from verifying compliance electronically. Similar issues have been identified in compliance with paper diaries (e.g. Stone, Shiffman, Schwartz, Broderick, & Hufford, 2003). Although there are no difficulties in identifying if participants completed the online DRM on the correct day due to electronic data stamping of responses, the correspondence between reported activities , emotions, and interactions and the actual activities engaged in and feelings experienced is currently unknown. New technologies for ambulatory assessment may go some way towards verifying the kinds of

activities engaged in (Bussmann et al., 2009) and mobile technology could even verify social interactions (e.g. Eagle, Pentland, & Lazer, 2009). One pertinent challenge for the DRM is the verification of subjective experience data. Asking participants to report on their mood at different points during a specific day may affect the mood ratings they give subsequently in the DRM when reflecting on that day, potentially biasing correspondence estimates between the DRM and experience sampling. Although heart rate was found to be linked to emotion in the current research, it is unlikely that currently available measures of physiological functioning could be used to objectively verify the presence or intensity of a particular emotion (e.g. Larsen et al., 2008). However, these difficulties noted, research which aims to gain insight into the concordance between activities, interactions, and emotions reported in the DRM and those actually engaged in/experienced would be invaluable in gauging the validity of the DRM survey.

8.7. A template for future tests of the bidirectional resource model

A primary limitation of the existing literature on the reserve capacity model is the failure of any single study to address the model in its entirety. Within the context of the bidirectional resource model, much headway can be made in testing the tenets of the model using available secondary datasets especially those with nationally representative samples. However, it is unlikely that such databases will contain the relevant information to test all aspects of the model, and it is almost certain that the design of these studies will not be optimal for identifying the proposed relations. For example, epidemiological surveys typically rely on aggregate retrospective self-assessments of affect that are prone to heuristic recall strategies that may bias reporting (e.g. beliefs about prototypical experiences, influence of peak-end salient experiences) (Kahneman & Riis, 2005).

Although in the current research no one study contained all the relevant measures to fully test proposed bidirectional resource model, collectively the studies contribute

insights which together can produce such a design. I envisage the collection of primary data sources that consider the mediational and moderational pathways within the bidirectional resource model as optimal. The design I outline could be applied to the study of many major social and organizational factors implicated in mental and physical health disparities. In this section I will briefly summarize some of the key questions that the bidirectional resource model could be used to address. I will then outline a format for future studies of the bidirectional resource model which considers the theoretical and methodological insights acquired throughout this thesis.

8.7.1. Questions that the bidirectional resource model can address

The reserve capacity model was designed to specify the contribution of psychosocial factors to the relationship between socioeconomic status and health outcomes. The model is founded on the idea that people from a low socioeconomic status background encounter stressors more frequently and perceive stressors as more threatening than do those from wealthier backgrounds (e.g. McLeod & Kessler, 1990; Chen & Matthews, 2001). Understanding how psychosocial factors contribute to status related disparities in health outcomes represents a broad and vastly worthwhile research stream. However, there is no reason that the reserve capacity model should be restricted to the explication of status related health changes. Thus, in the bidirectional resource model there are many important stress antecedents, other than socioeconomic status, that could be considered as primary causes of stress-related declines in mental and physical health.

For instance, recent reviews have shown that there is robust support for the idea that ethnic/racial health disparities exist as evidenced by the raised levels of numerous health conditions (e.g. cardiovascular disease, stroke, diabetes) among African Americans, Native Americans, and Hispanic Americans relative to Caucasian Americans (e.g. Myers, 2009; Mays, Cochran, & Barnes, 2007). Interpersonal racism can be conceptualized as a

stressor that has been shown to partially explain ethnic/racial differences in mental and physical health outcomes (Brondolo, ver Halen, Pencille, Beatty, & Contrada, 2009). Race can enhance the likelihood that a person will encounter stressors such as interpersonal conflict and neighbourhood segregation (Massey, 2004).

The perception of discrimination has been linked with psychological distress, numerous health behaviours (e.g. smoking, alcohol consumption) (Landrine & Klonoff, 2000), psychophysiological reactivity (Armstead, Lawler, Gordon, Cross, & Gibbons, 1989), and health outcomes (Taylor, Repetti, & Seeman, 1997). Psychosocial resources have also been shown to buffer the adverse effects of discrimination (e.g. Fischer & Shaw, 1999; Lewis-Coles & Constantine, 2006). Thus racial/ethnic health disparities appear to be linked to each aspect of the bidirectional model, and utilizing this framework may be a beneficial way of outlining and organizing the mediating and moderating processes that explain how race differences can lead to divergent health trajectories. Although I am not aware of any studies that have examined how psychosocial resources interact with specific genetic polymorphisms to predict reactivity to interpersonal racism, this represents one promising avenue enabled by the inclusion of genetic factors in the bidirectional resource model.

Organizational justice, or the perception that people are treated equally in a way that is fair and just, is another important determinant of health outcomes that could be studied within the framework of the bidirectional resource model. There are three principal facets of organizational justice all of which could feasibly generate stress and influence health outcomes. Distributive justice refers to perceptions of fairness regarding outcomes such as pay. Those who exert effort that they perceive to exceed the rewards provided in return have been shown to be at high risk of health conditions such as coronary heart disease and myocardial infarction (e.g. Kuper, Singh-Manoux, Siegrist, & Marmot, 2002).

Procedural justice broadly gauges the extent to which people feel they have a voice within decision-making systems and emphasizes the importance of considering the views of those that will be affected by the decisions made. Relational justice focuses on the fairness of interpersonal treatment (e.g. polite, considerate, respectful of human dignity) (Bies & Moag, 1986).

Both procedural and relational injustice have been shown to predict low self-rated health, a high incidence of psychiatric disorder and an enhanced likelihood of absenteeism (Elovainio, Kivimaki, & Vahtera, 2002; Kivimaki, Vahtera, Pentti, & Ferrie, 2000; Kivimaki, Elovainio, Vahtera, Ferrie. 2003). Thus, injustice in the workplace may act as a stressor that has important implications for mental and physical health that could be organized within the bidirectional resource model. A central motivation for organizations to embrace principles of organizational justice is that a positive organizational environment is likely to improve workers job satisfaction, commitment to the organization, productivity, absenteeism, and worker health (Colquitt, Conlon, Wesson, Porter, & Ng, 2001). The case of healthcare workers is particularly interesting as improving perceptions of fairness within the organizational system could have the triple advantage of enhancing positive outcomes for the organization, improving health outcomes for the staff and even enhancing outcomes for patients (Laschinger, 2004).

Staff treatment is just one of a complex of factors that can influence patient health outcomes. Others important factors include the perceived trajectory of one's illness, the presence of additional life stressors, the degree of symptom severity, and the presence of disrupted psychobiological processes (e.g. changes in neuroendocrine, cardiovascular, or immune functioning), all of which may influence well-being. Health conditions are a particularly complex form of stressor that can influence mental and physical health through numerous pathways within the bidirectional resource model as discussed earlier and

illustrated in Figure 13. Taken together, it is feasible that future tests of the bidirectional resource model could examine the role of psychosocial and genetic factors in modulating the effect of stress antecedents such as socioeconomic status, race/ethnicity, organizational justice, and the presence of health conditions, on well-being and subsequent health outcomes. As stress is proposed to be the main mediator between antecedents like socioeconomic status and subsequent health I now turn to potential directions for research measuring stress within the bidirectional resource model.

8.7.2. Assessing stress

Stress measures can be classified on a continuum between environmental stimuli and person-based subjective or psychobiological responses. In the current thesis, different aspects of stress were gauged. Exogenous exposure to an earthquake (chapter 6) could be considered to be very much an environmental account of the stressful stimulus. In a second study, stress was measured using a life-domain checklist (e.g. are you experiencing stress about your family finances?) (chapter 7), an intermediary measure between environmental and person-based assessments. Taking a person-based approach, the intensity of subjectively perceived stress was measured at different points of the day (chapter 2). Whilst it is advantageous to take multiple measurements of stress using varied techniques, an integrative assessment model is preferable. Such a model should consider the stress measurement literature and its limitations which are outlined briefly below.

Measures of the environmental component of stress are often self-reported and this is problematic because such measurements are difficult to objectively verify. In particular, self-reported measures of stressful events can be confounded by variability in reporting on the part of the participant. For example, a neurotic participant may report a family reunion as an extremely stressful event whereas an extravert may not view this event as stressful. Similarly, if stress is measured from the response perspective (e.g. heart rate

reactivity/recovery, subjectively reported stress) the same stress response may correspond to vastly different stressors. For one person a heart rate increase of 10 beats per minute could follow the mental strain of a puzzle solving task, whereas for another person the same elevation in heart rate would only follow an awkward social evaluation through a difficult public speaking task.

When attempting to gauge human stress exposure most studies examine important life events (Monroe, 2008). This approach relies on the assumption that on average people respond in a certain way to specific stressors and that deviation from this response can largely be ignored. The Social Readjustment Rating Scale (Holmes & Rahe, 1967) is one popular checklist measure that assesses the occurrence of stressful life events. Importantly, both the number of events that have occurred and the stress weighting of each event are calculated (e.g. two events could be: minor violation of the law = 11, death of a spouse = 100, total = 111). As proposed in the bidirectional resource model, the occurrence of stressful life events predicts the development of both emotional and health problems (e.g. Kessler, 1997; Kilpelainen, Koskenvuo, & Terho, 2002).

A central problem with self-report checklists is that participant perceptions of life events are often at odds with the type of life event researchers imagine to have occurred, potentially over half of the time (Monroe, 2008). The Life Events and Difficulties Schedule (Brown & Harris, 1978) is an interview-based measure that includes a comprehensive manual with specific examples in an attempt to address individual differences in thresholds for endorsing certain events (e.g. sexual difficulties, major illness). Such structured interview techniques allow clarifications to be made by the interviewer and increase the likelihood that the participant and the interviewer will converge upon a common understanding of what a particular event involved. Interview techniques thus appear to assist people in reporting stressful life events relatively

accurately for recall periods of up to 10 years (Neilson, Brown, & Marmot, 1989) and may be better predictors of depressive symptoms than life event checklists (e.g. McQuaid, Monroe, Roberts, Kupfer, & Frank, 2000).

However, even when semi-structured interview-based approaches accurately define each discrete event in line with the researcher's criteria, the occurence of stressful events may not account for the magnitude of stress experienced or the emotional response to the stressor (McQuaid et al., 2000). Measures of subjectively perceived stress can be used to gauge the extent to which people feel their lives are overloaded or uncontrollable (Cohen, Kamarck, & Mermelstein, 1983). Such measures of perceived stress prospectively predict the development of emotional problems such as depression (e.g. Shahar, Cohen, Grogan, Barile, & Henrich, 2009). Assessments of perceived stress are also good predictors of psychobiological functioning and health problems even after adjusting for psychological symptoms which overlap considerably with subjective stress ratings (e.g. Cohen, Tyrrell, & Smith, 1993). However, like global assessments of well-being (Kahneman & Riis, 2005), retrospective measures of subjective stress may be biased by heuristic influences (e.g. how one usually feels, the presence of contemporaneous stressors, peak-end characteristics).

To summarize, whilst measures of stressful events and perceived stress predict emotional and health changes, such instruments are often beset with recall biases and challenging issues of differential item functioning. In addition, the often static nature of measures of stressful events or subjective ratings of stress, neglects the dynamic nature of stress. Rather than being restricted to a stimulus or a response, stress is a complex interactive process that incorporates transactions between the organism and the environment (Lazarus & Folkman, 1984). It thus appears that to accurately assess the stress process researchers must assess (i) the stimulus and (ii) response aspects of stress, and

crucially (iii) the psychosocial and potentiallybiological factors that moderate the stimulus-response link. These aspects should be measured simultaneously, on numerous occasions, and in multiple contexts in order to assess the full gamut of the stress process.

8.7.3. Suggestions for future bidirectional resource model studies

This proposed dynamic interplay between stressors, psychosocial resources and stress reactivity, raises the question as to how best to empirically assess such relations. As mentioned, this is difficult to achieve with brief measures in cross-sectional samples or large-scale epidemiological surveys as such data is temporally restricted and people may misreport events and feelings experienced over distant time horizons. It is therefore important to gauge variability in stress exposure and emotional reactivity variables in context where possible (Myin-Germeys et al., 2009). In particular, a potentially fruitful approach to testing the bidirectional resource model is to assess stress sensitivity in daily life.

Rather than focusing on broad effects of rare life events, such studies could investigate the relation between daily hassles or minor stressors and emotional reactions (e.g. Lataster et al., 2008). Within the bidirectional resource model stress sensitivity in the flow of daily life may be an affective pathway to mental and potentially physical health problems (Myin-Germeys et al., 2009). When confronted with a stressor people vary substantially in numerous aspects of their emotional response including its duration, variability, and intensity (Kuppins et al., 2007; Verduyn, Delvaux, Van Coillie, Tuerlinckx, & VanMechelen, 2009). For example, following a dispute with a work colleague some people may feel briefly frustrated and these feelings may vanish quickly, yet for others frustration may linger, disappear and return, or even fester and escalate over several hours.

Such individual differences in emotional reactivity are likely to have implications for psychobiological functioning (Chida & Steptoe, 2008). In the current investigation of

the link between self-control and psychobiological functioning, emotional variability rather than intensity was the key mediator. Further studies are needed to identify the extent to which self-control moderates or mediates the emotional response to stressors. Such studies could use experience sampling method or recently developed techniques like day reconstruction (Kahneman et al., 2004) to assess when people are exposed to minor stressors, the context stressors occur in, and the feelings which follow. This approach would allow the role of psychosocial resources in explaining the emotional response to stressors to be examined in detail.

Psychosocial resources could play several potential roles in the stress process. Centrally, they may (i) reduce the likelihood that stressors are encountered, (ii) diminish the intensity, duration, or recurrence of emotional responses when stressors occur, or (iii) they may be influenced by stressors and mediate the effect of stressors on emotional responses. To clearly delineate these different possibilities requires that a wide-array of psychosocial resources (e.g. optimism, self-esteem, social support) are measured in different ways. For instance, taking the example of self-control, this resource could be measured using a self-report trait-level questionnaire. Such trait measures are useful in specifying if psychosocial resources condition the frequency that stressors are experienced or the extent to which resources modify the emotional effects of stress. However, psychometric instruments that assess psychosocial resources may share methodological variance with reports of the emotional response to stress and this may conflate estimates of the degree of their relation. It thus could be beneficial to gauge psychosocial resources via behavioural, neural, or psychobiological responses to standardized tasks administered in the laboratory. In support of this idea prior research has shown that neural and behavioural measures of performance (e.g. error related negativity, prefrontal activity) on tasks which require participants to exert self-control (e.g. ability to regulate feelings, thoughts, or behaviour) can predict stress levels in daily life (e.g. Compton, Robinson, Ode, Quandt,

Fineman, & Carp, 2008**;** Urry et al., 2006). The recent explosion in usage of mobile devices could even facilitate the administration of standardized tasks in naturalistic settings thus allowing psychosocial resources to be measured behaviourally in context (Eagle et al., 2009).

A potentially less invasive approach is to assess psychosocial resources at the state level using momentary assessment self-report techniques. Within the bidirectional resource model measuring state levels of psychosocial resources is particularly important in order to identify if resources mediate the emotional effects of stressors. For example, stressors are suggested to induce low self-esteem leading to feelings of negative affect though it is feasible that negative emotions may follow directly from stress and even lead to decreases in self-esteem (e.g. Orth et al., 2008; Orth et al., 2009).

It is an open question whether other theoretically interesting stable individual differences can be conceptualized as intermediaries between stress and emotion within the bidirectional resource model. In this thesis I proposed that identifiable genetic characteristics which have been implicated in stress reactivity could have a role in the model similarly to that of psychosocial resources. Like those with low levels of psychosocial resources those with risk genotypes may be more likely to encounter stressors (Moffitt et al., 2006). Similarly, akin to resources, genetic variation could lead directly to emotional changes in the absence of stressors, or exaggerate the emotional effects of stressors. With the advent of the study of epigenetics, it is even possible that the effects of stressors may modify gene expression through DNA methylation and histone modification thus genes could mediate the relation between stressors and emotional changes (Masterpasqua, 2009).

Taken together, genetic factors overlap substantially with psychosocial resources in their proposed role in the bidirectional resource model. What is unclear at present is the

degree to which identifiable genetic factors underlie the effects of psychosocial resources. The current research and prior studies (e.g. Kaufman et al., 2004; Kilpatrick et al., 2007) have shown that identifiable gene polymorphisms can act synergistically with individual differences in psychosocial resources to predict well-being. In these studies the genetic risk of adverse emotional reactivity to stressors was largely unrelated to psychosocial resources, yet this genetic risk appeared to be attenuated by the presence of psychosocial resources. Thus, rather than underlying psychosocial resources the expression of risk genotypes may be suppressed by such resources. Future research could potentially uncover psychological processes which are modified by genetic variation and psychosocial resources. This would assist in providing an integrative explanation of the pattern of results observed. For example, individual differences in the appraisal of stressors could be detrimentally influenced by the presence of genes such as the short version of the serotonin transporter (Munafo et al., 2008) but this effect may be overruled by the role of psychosocial resources in explaining patterns of appraisal (e.g. Taylor & Stanton, 2007; Taylor et al., 2008).

The momentary assessment methods that I proposed as suitable for the study of how the emotional reaction to everyday stressors is modified by psychosocial resources could also be used to examine the role of identifiable genes in the stress process within the bidirectional resource model. Momentary assessment techniques have already provided support for genetic modifiers of the emotional response to stress. For instance, a recent diary study found carriers of the catechol-*O*-methltransferase Val/Val genotype to show a larger increase in paranoia following stressors than did those with other genotypes (Simons et al., 2008). A second momentary assessment study of patients with psychosis reported Val carriers to display a greater level of hallucinations following cannabis use (Henquet et al., 2008). Experience sampling data has also provided evidence that positive affect can buffer a genetic risk (associated with variation in the gene encoding brain-derived

neurotrophic factor) of depression following social stress (Wichers et al., 2007). Within the bidirectional resource model this research on experience in context can be extended to examine the synergistic effects of gene polymorphisms and psychosocial resources on emotional reactivity to stress induced by important antecedents such as racial discrimination and organizational injustice.

Further, the likely health effects of stress can be gauged through an analysis of key psychobiological factors associated with stress responsivity. Psychobiological factors have been neglected in research testing the reserve capacity model even though the activity of the sympathetic adrenomedullary system and the HPA are considered to be key mediators between emotion and health (Matthews et al., in press). This relation may not have been given sufficient attention due to the technological and methodological constraints that can make such studies difficult to conduct and demanding for participants. In traditional large scale epidemiological studies or cross-sectional surveys of convenience samples variables are often assessed at one time-point, usually in a laboratory setting. This approach may not identify links between emotion and physiological functioning that can be uncovered through more detailed methodologies.

For example, ecologically valid measurement of experience and psychobiological functioning in context yields multiple data-points allowing variation over time to be investigated (Myin-Germeys et al., 2009). This longitudinal nature of the design means that prospective analyses can suggest support for causal interpretations of the data. In addition, sophisticated multi-level statistical models allow interactions with contemporaneous circumstances to be considered and the conditions under which emotion-psychobiology relations exist to be specified.

To summarize, I have outlined, in several steps, a template for future studies testing the bidirectional resource model. In the context of the insights garnered from the studies in

this thesis, it appears possible and beneficial to measure each component of the stressor-emotion-psychobiology link simultaneously and over multiple adjacent time-periods. This approach holds promise in (i) predicting and understanding the long-term health effects of emotional reactions to measurable stressors and (ii) in demonstrating how such effects are modified by psychosocial resources and identifiable genetic factors.

8.8. Conclusions

The research in this thesis demonstrates ample support for multiple components of the reserve capacity model and for newly proposed relations within the bidirectional resource model. The stress of illness was shown to predict adverse patterns of emotion and functioning that statistically explained the association between illness and perceptions of poor health. These findings broadly linked the sequential relations between the stressor, emotion, and health components of the model. Although it is currently unclear from research on the reserve capacity model how emotional factors may lead to health conditions two of the studies within this thesis suggested potential mechanisms. Firstly, I measured the experience of 186 students in daily life using the day reconstruction survey. This study showed that negative emotion in naturalistic settings predicts an elevated heart rate suggesting that cardiovascular reactivity/diminished recovery may be one pathway between emotion and health. Next, I aimed to extend the coverage of the reserve capacity model by empirically evaluating the suggestion that psychosocial resources predict adaptive patterns of emotion and health. I showed that self-control was linked to stable patterns of affect and that this emotional stability mediated between self-control and favourable psychobiological functioning as indexed by a low heart rate and a steep cortisol slope. In addition, I showed a second psychosocial resource, perceived control, to predict enhanced well-being and functioning, which mediated between perceived control and perceptions of good health. Taken together these studies provided support for the relations

proposed in the reserve capacity model. Furthermore, this support was achieved primarily by utilizing advancements in the surveying of experience in context and the monitoring of psychophysiological functioning in real-time.

This research proposed the bidirectional resource model primarily to address two gaps in the reserve capacity model: the lack of coverage regarding reverse causality and the neglect of consideration of genetic factors. Although the reserve capacity model proposes that emotional changes augment patterns of psychobiological functioning which can impact on health, I demonstrated the existence of two theoretically and empirically supported pathways from health conditions to psychobiological functioning to emotion. Firstly, low levels of morning cortisol were demonstrated to link to diminished levels of positive affect in the first half of the day, particularly amongst the distressed. The next demonstrated that obesity-related inflammation partially explains the neurovegetative symptoms of depression which are often associated with obesity. Thus these two studies suggest that it is possible that health conditions may invoke emotional changes through their impact on psychobiological functioning as gauged by neuroendocrine and inflammatory markers. Although further prospective testing of these relations is warranted, these studies highlight the importance of considering reverse causality pathways as proposed in the bidirectional resource model.

The second, and major extension of the reserve capacity model included in the bidirectional resource model, focused on the incorporation of genetic factors. I suggested that identifiable genetic variation may act much in the same way as psychosocial resources in that they may modify the emotional effects of stressors with potential implications for health. This contention was supported in two studies. The first showed that the apolipoprotein E gene modified the likelihood that a high level of exposure to the stress of an earthquake may be linked to a low level of self-rated health. Following this I

demonstrated that the apolipoprotein E gene moderated the link between chronic illness and psychological adjustment. In addition, the apolipoprotein E gene also explained divergent patterns of self-rated health as a function of illness. Furthermore, psychological adjustment mediated between the illness × gene interaction and self-rated health. This study thus spanned the bidirectional resource model suggesting support for a specific aspect of genetic variation as a modifier of the emotional and functional response to stress which then may lead to augmented health perceptions. Furthermore, and potentially most interestingly, this study identified tentative evidence that perceived control may buffer the portion of the effect of the stress of illness on psychological adjustment which is conditional on the genetic vulnerability to stress reactivity conferred by the apolipoprotein E gene. These results support a model of health where one's genetic endowment, exposure to stressors, and psychological resources interact synergistically to produce patterns of emotion which may lead to or exacerbate health conditions.

Within the context of the bidirectional resource model, new developments in the real-time monitoring of biological functioning hold promise chiefly in further outlining the links between stress, emotion, psychobiology, and health. Stressors could be gauged using devices that monitor environmental conditions such as noise pollution, crowding, and potentially even the content of human interactions (Mehl & Holleran, 2007; Mehl, 2007; Hasler et al., 2008). Health outcomes could be assessed frequently also (e.g. viral load in HIV) and combined with advances in environmental, experiential, and psychophysiological monitoring thus providing rich contextual linkages through from stress to health. Further, additional genetic and psychosocial components of the model could be drawn into the analysis to tie together the entire bidirectional resource framework ranging from phenomenology to the underlying biological mechanisms that link stress and health.

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Appendix A

Standard operating procedures for medical testing and naturalistic monitoring for the day reconstruction method study

Section (A) Pre-testing period

1. **Participant Information sheet**
2. **Consent form**
3. **Debriefing sheet**
4. **Participant code and physical test recording sheet**

Section (B) Protocols for testing period

1. Body weight and height

2. Body fat

3. Peak flow and FEV1

4. Blood pressure

5. Blood glucose levels

6. Saliva sampling (overview)

7. Heart monitoring (overview)

8. Participant instructions

Section (A) Pre-testing period

Information sheet, consent form, and debriefing sheet

The participants will have received an information sheet via e-mail. However, it is preferable to give participants the option of reading the information sheet at the initial medical testing.

They will also have to complete a consent form at the medical testing which is shown below. It is important that the participant knows that there are two sections which s/he may sign, the consent section, and the consent to re-contact section.

The participant is given the debriefing sheet at the end of the study when the online questionnaire is completed.

1. Participant Information Sheet

The purpose of this study is to examine Irish student health and daily experience. This study is coordinated by Mr. Michael Daly, a postgraduate student in the School of Psychology, Trinity College Dublin, under the supervision of Professor Malcolm MacLachlan.

If you agree to take part in the study you will be asked to do the following:

Day 1. Come to the School of Psychology for a half-hour to complete a brief set of simple health tests (e.g. blood pressure, height and weight, lung capacity, glucose levels) and receive some instructions about the study.

Day 2(a). Wear a non-intrusive heart rate monitor during your normal activities and give four sets of two saliva samples at specified intervals. These saliva samples are taken to assess your daily levels of biological markers of functioning (e.g. cortisol levels which indicate stress). All samples will be disposed of after analysis is completed.

Day 2(b). If you are amongst a subgroup randomly selected, you will be asked to respond to questions on a palmtop computer which takes a total of 10 minutes throughout the day.

Day 3. Complete an online questionnaire which asks you to recall the previous day. This takes up to an hour and a half to complete.

The health assessment is non-intrusive with the exception of a self-administered blood sugar pin-prick test. Those taking the test will be talked through the simple step-by-step procedures. The glucose testing equipment used needs a tiny amount of blood (less than one hundredth of a millilitre). The blood sample is analyzed on the spot and immediately disposed of.

You can contact Michael Daly about participation in the study by e-mail at dalym7@tcd.ie , by phone at 018963912, or at the following postal address: School of Psychology, Áras an Phiarsigh, Trinity College, Dublin 2. This project is supervised by Professor Malcolm MacLachlan who can be contacted at the same postal address or by e-mail: malcolm.maclachlan@tcd.ie, or phone 01-8961453.

2. Consent Form

Your participation in this study is entirely voluntary and you may withdraw from the study at any point or refuse to answer any questions with which you are uncomfortable. A participant ID number will be used for identification purposes and your name will never be connected to your results. No information that would make it possible to identify you will ever be included in any sort of report. The data will be accessible only to those working on the project. You may be provided with a summary of your health assessment results if you wish.

If you have any questions at any stage please ask one of the instructors or contact Michael Daly by e-mail at dalym7@tcd.ie , by phone at 018963912, or at the following postal address: School of Psychology, Áras an Phiarsigh, Trinity College, Dublin 2. This project is supervised by Professor Malcolm MacLachlan who can be contacted at the same postal address or by e-mail: malcolm.maclachlan@tcd.ie, or phone 01-8961453.

I have read the above information. I have asked any questions I had regarding the procedures of the study and they have been answered to my satisfaction. I understand that I will not be requested to engage in any task which may be detrimental to my health or well-being. This study has received prior approval from the Ethics Committee of the School of Psychology, Trinity College Dublin. I consent to participate in this study.

Name of Participant_____Date: _____

(please print)

Signature of Participant _____

Age: _____**(Note: You must be 18 years of age or older to participate in this study. Let the experimenter know if you are under 18 years old.)**

I consent to be re-contacted in relation to further research carried out by the coordinator of this study. I understand that I do not have to consent to be re-contacted to take part in this study. I am aware I have no obligation to participate in any additional research and that I may request to disengage from any further contact at any time.

Signature of Participant _____

3. Debriefing Sheet

Thank you for your participation in the study. The information which you have provided will be used by our research team to address a number of important questions about how social factors and health risk behaviours impact on health, daily experience and time-use. Both the heart rate monitor that you wore and the salivary cortisol samples you provided will be used as biological markers of fluctuations in stress and mood over the course of the day. We hope that the research which you have helped us to develop can soon be used as a tool at the population level for wide-scale examination of psychosocial factors influencing health.

Please note that all the information which you have provided will be stored anonymously and will be in no way traceable to you. The saliva samples you have provided will be used solely for salivary cortisol analysis and will be disposed of immediately after analysis. The blood sample you provided was disposed of immediately after analysis and will not be stored.

If you have any further questions about the research or concerns about the information you have provided please contact Michael Daly by e-mail at dalym7@tcd.ie , by phone at 018963912, or at the following postal address: School of Psychology, Áras an Phiarsigh, Trinity College, Dublin 2. This project is supervised by Professor Malcolm MacLachlan who can be contacted at the same postal address or by e-mail: malcolm.maclachlan@tcd.ie, or phone 01-8961453.

4. Participant code and physical test recording sheet

Each participant will be provided with a code via e-mail. It is important that the participant knows his/her code and this information is recorded on the 'Physical Tests Sheet' (shown below). CO is the code for the control group.

Day Reconstruction Study: Physical Tests Sheet

Testers_____

Participant code: ___CO_____

Participant AGE and GENDER _____ _____

When did participant last eat? _____

Test	Test 1	Test 2	Other
Body height (cm)		NA	
Body weight (kg)			
Body fat (%)			
Blood pressure (average)			
Systolic		NA	
Diasystolic		NA	
Pulse (BMP)		NA	
Peak flow			
FEV1			
Blood glucose (mmol)		NA	
Comments:			

Participant Instruction:

Received instruction on how to
set up and use heart monitor _____

Received instruction on when and
how to take saliva samples and
what to avoid beforehand _____

Received instruction on how to
use experience sampling device _____

Section (B) Protocols for testing period

1. Standard Operating Procedure for Body Weight and Height Measures

The devices used will be the Leicester height measure and the Salter heavy duty glass electronic scales. The participant will be asked to remove his/her shoes for both tests. The weighing scales will be out of view of other participants. In the case where a participant is not wearing socks s/he will be not be asked to remove footwear for these tests. Height (cm) is recorded once and weight twice (kg).

2. Standard Operating Procedure for Body Fat

Body fat will be assessed via bioelectrical impedance analysis using the Omron HBF-306C Body Fat Analyzer. This device is accurate only for men aged 18-70 and women between 18 and the menopause.

1. Participant height, weight, and age information are entered into the device.
2. For this test it is recommended that hands should be moist. An alcohol wipe will be used to disinfect and moistening hands prior to testing.
3. The test involves gripping the device in a specified manner with both hands and arms straight out at a 90 degree angle to the body. An unnoticeable microelectrical current then passes through the body to produce the percentage body fat measure.
4. A repeat measure is taken and both values are recorded.
5. The device is wiped down with an alcohol wipe before the next use.

3. Standard Operating Procedure for Peak Flow and FEV1

Peak flow and forced expiratory volume will be assessed using the Mini-Wright meter.

1. A disposable mouthpiece will be provided for each participant.
2. The participant will be instructed to take a deep breath and blow into the device “as hard and as fast as you can”.
3. This will be repeated twice unless there is an error message, in which case the process will continue until there have been three valid measurements.
4. Peak flow and FEV1 measurements will be recorded for both trials.
5. The device will be cleaned with an alcohol wipe after each participant.

4. Standard Operating Procedure for Blood Pressure

1. The participant is requested to remove his/her jumper to facilitate this procedure.
2. The participant's lower arm is supported at the elbow by a towel and the hand is facing palm upwards.
3. The arm cuff is then wrapped around the skin of the right upper arm at the level of the heart with the 'art' arrow lined up with the brachial artery.
4. The device is set to read the average of two readings. The cuff inflates and deflates twice on pressing the automatic operation button. Average systolic and diasystolic blood pressure, and heart rate in BPM are recorded.

5. Standard Operating Procedure for Accu-Chek Advantage Blood Glucose Testing

1. Test sample taking

1.1. Test sample taking summary

- Sample to be taken at one time point.

Samples to be obtained:

- 1 x 3.5 microlitres blood sample

1.2. Materials

Blood sampling and testing kit needed for each participant

- 1 x Accu-Chek Soft-T Pro Disposable Finger Pricker
- 1 x Accu-Chek Advantage Test Strips
- 1 x Accu-Chek Advantage Meter
- Alcohol wipes
- Cotton wool
- Small plasters
- 1 x Sharps bin for used finger prickers and test strips (multiple use)

1.3. Taking the glucose test

1.3.1. Requirements prior to collection

- The glucose test should be taken at the end of the physiological testing session.

1.3.2. Details to be recorded:

- The level of glucose in the blood in mg/dL
- Date and time of the test.
- Last time the subject consumed food/drink.

1.4. Protocol for taking blood

1.4.1. Guidelines for capillary sampling

Preparation

- Ensure subject is positioned comfortably in a chair.
- Explain the procedure to the participant.
- Select the appropriate lancet and test strip. Ensure the sharps disposal bin, alcohol wipes, cotton wool, and plasters are readily available.

Note: The following instructions are adapted from the 'Accu-Chek Advantage Blood Glucose Management Ward Manual'.

- Wash hands thoroughly. Put gloves on.
- Clean the side of the participants' finger using gauze swabs. Ensure that the finger is thoroughly dry. Promote blood flow to the site by allowing the arm to hang down by the side for a few seconds and then flex arm and fingers.
- Insert a test strip into the test strip slot. The test strip symbol stops flashing and a blood drop will appear on the display.
- Ask the participant to remove the safety cap from the Safe-T-Pro and firmly place the Safe-T-Pro against the side of the finger and press the firing button.
- Once used, the lancet will permanently retract into its protective case. Safe-T-Pro is a single use device; dispose whole device into a sharps bin.
- Allow 5 seconds to elapse after pricking the finger and then rather than squeeze at the site of puncture, ask the participant to milk the blood down the hand towards the finger.
- Touch the droplet of blood to the curved side of the strip and keep it in place until the yellow target area is covered entirely.
- An hourglass symbol flashes on the display until measurement is completed. Result appears.
- The result is recorded immediately using appropriate documentation.
- The test strip and any soiled materials are removed and disposed of.
- A plaster is applied over the puncture site.

6. Standard Operating Procedure for Cortisol Sampling

1. Upon waking the participant removes the swab from the Salivette collection device, places it in the mouth and chews for 45 seconds.

2. S/he then returns the swab to the collection device and replaces the stopper. This is repeated immediately.

3. This procedure is repeated 30 minutes after waking, 6 hours after waking, and 12 hours after waking.

4. The participant is requested to take note of his/her waking time and the time s/he provided saliva samples throughout the day.

7. Standard Operating Procedure for Heart Monitoring

1. The participant is asked to adjust the chest belt strap in order for the device to be tight against the skin but not uncomfortably so.

2. The participant is then asked to place electrode gel on his/her chest and also on the electrodes on the back of the monitor.

3. The heart monitor is then put on and should pick up the heart beat automatically within 1-2 minutes. When this occurs the device will beep and display a green light. The participant can then go about his/her normal activities.

4. The device is returned to the experimenters (Michael) the next day who will wipe down the plastic component of the device with an alcohol wipe and run the elastic material straps through the washing machine.

8. Participant Instructions

Day Reconstruction Study: Instructions for your day of participation

Thank you for taking part in our study!

In the pack you received after your physical tests you will find **a heart rate monitor, 8 saliva collection devices**, and also a Palm computer if you were randomly selected for this group.

These devices are easy to use and the instructions for using them are included below. We would ask you to **please read these instructions carefully.**

For the monitoring day we ask you to do the following in the stated order:

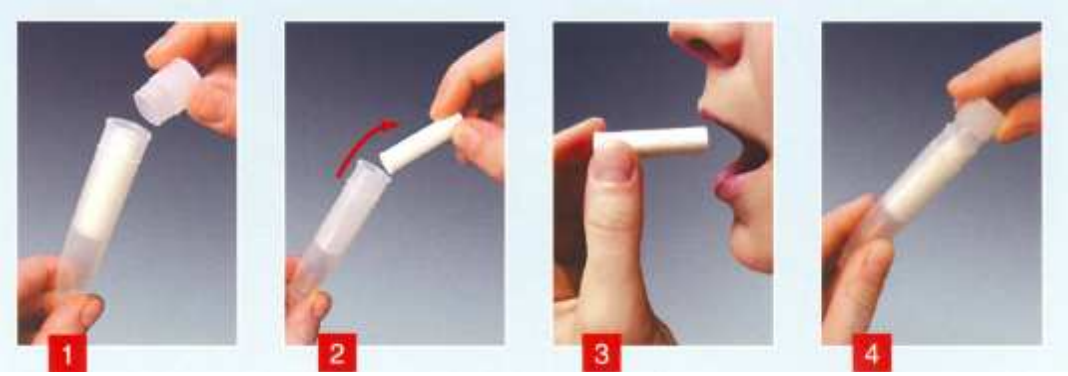
(1) Provide two saliva samples immediately when you wake up
(2) Put on the heart monitor after you wake up
(3) Activate the Palm computer on waking if applicable
(4) Provide two saliva samples 30 minutes after waking
(5) Provide two saliva samples 6 hours after waking)
(6) Provide two saliva samples 12 hours after waking

The instructions follow: Section 1- Saliva samples

Section 2- Heart monitor

Section 1. Saliva collection instructions for use

To use, remove the cotton swab from the collection device (picture 1 and 2). Place the swab in your mouth and **chew it for approximately 45 seconds** or until it becomes saturated (picture 3). Return the swab to the device and replace the stopper (picture 4).



It is essential that you refrain from eating, drinking (water can be drunk up to 5 minutes prior to sampling), smoking, or brushing/flossing your teeth for 30 minutes before all saliva sampling or during the saliva collection. These activities will contaminate the sample.

Saliva Expected Collection Times and Time Actually Taken

1. Follow the collection instructions with the first two samples labelled ‘at wakening (1) & at awakening (2)’providing these two samples **immediately when you wake up and your eyes open and you are ready to get up.**
2. Please fill in the table below after you have taken the first two samples.

Cortisol collection times

Today I woke up at:_____(enter time for samples 1&2) time completed_____

30 minutes after wakening is_____(enter time for samples 3&4) time completed_____

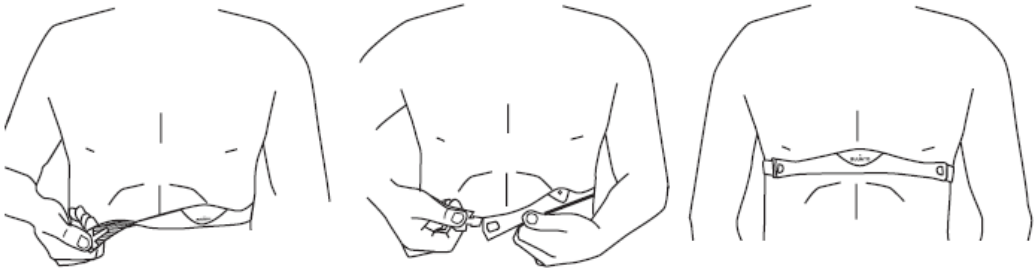
6 hours after wakening is_____(enter time for samples 5&6) time completed_____

12 hours after wakening is_____(enter time for samples 7&8) time completed_____

3. Take the second two samples 30 minutes after waking up. The third set of two samples is to be taken 6 hours after wakening up, and the fourth 12 hours after waking. To assist you in this we have included a table where you can check off the samples you have provided.

Section 2. Heart monitor instructions for use

1. Adjust the heart monitor strap so as that it fits snugly around your chest (see picture below). It will need to be tightened or loosened for the best fit for you. The heart monitor should be worn tightly (enough so that it feels like it will not slip down) but not uncomfortably so. It should be worn against the skin around your chest.



2. When you have adjusted the heart monitor strap to the best fit for you remove the heart monitor and open the electrode gel. Squeeze out enough gel to coat the electrodes on the back of the heart monitor (see picture).

3. Place the heart monitor back around your chest as in step 1. The heart monitor

should beep within 1-2 minutes to indicate that it is picking up your heart beat. A green light will also flash every 5 seconds to indicate that it is operational. This light is not very bright so may only be seen in a mirror or if you cup your hands over the device.

Note: If the heart monitor fails to start within twenty minutes please apply water instead of electrode gel (as this works when the battery is low). If the device still does not start working please call or text Michael on 018963912 or 0851453812 and he will call you back to assist in making the device operational.

Appendix B

Standardized Scales Utilized in the Current Research

(i) Ten Item Personality Inventory (Gosling et al., 2003).

(ii) Self-control Scale (Tangney et al., 2004).

(iii) Depression Anxiety Stress Scales (DAS-21) (Brown et al., 1997).

(iv) Personal Health Questionnaire Depression Scale (PHQ-9) (Kroenke, Spitzer, & Williams, 2001).

(v) Center for Epidemiologic Studies Depression Scale (CES-D) 8-item (Radloff, 1977) (utilized in Chapter 5).

(vi) Center for Epidemiologic Studies Depression Scale (CES-D) 10-item (Radloff, 1977) (utilized in Chapter 7).

(vii) Pearlin Mastery Scale (Pearlin & Schooler, 1978).

(i) Ten Item Personality Inventory (Gosling et al., 2003).

Here are a number of personality traits that may or may not apply to you. Please write a number next to each statement to indicate the extent to which you agree or disagree with that statement. You should rate the extent to which the pair of traits applies to you, even if one characteristic applies more strongly than the other.

1 = Disagree strongly, 2 = Disagree moderately, 3 = Disagree a little, 4 = Neither agree nor disagree, 5 = Agree a little, 6 = Agree moderately, 7 = Agree strongly

I see myself as:

1. _____ Extraverted, enthusiastic.

2. _____ Critical, quarrelsome.

3. _____ Dependable, self-disciplined.

4. _____ Anxious, easily upset.

5. _____ Open to new experiences, complex.

6. _____ Reserved, quiet.

7. _____ Sympathetic, warm.

8. _____ Disorganized, careless.

9. _____ Calm, emotionally stable.

10. _____ Conventional, uncreative.

(ii) Self-control Scale (Tangney et al., 2004).

Using the scale provided, please indicate how much each of the following statements reflects how you typically are (1 = Not at all, 5 = Very much).

1. **I am good at resisting temptation.**
2. **I have a hard time breaking bad habits.**
3. **I am lazy.**
4. **I say inappropriate things.**
5. **I do certain things that are bad for me, if they are fun.**
6. **I refuse things that are bad for me.**
7. **I wish I had more self-discipline.**
8. **People would say that I have iron self-discipline.**
9. **Pleasure and fun sometimes keep me from getting work done.**
10. **I have trouble concentrating.**
11. **I am able to work effectively toward long-term goals.**
12. **Sometimes I can't stop myself from doing something, even if I know it is wrong.**
13. **I often act without thinking through all the alternatives.**

(iii) Depression Anxiety Stress Scales (DAS-21) (Brown et al., 1997).

Please read each statement and enter a number 0, 1, 2, or 3 which indicates how much the statement applied to you over the past week. The rating scale is as follows: 0 Did not apply to me at all 1 Applied to me to some degree, or some of the time. 2 Applied to me a considerable degree, or a good part of the time. 3 Applied to me very much, or most of the time.

1. **I found it hard to wind down.**
2. **I was aware of dryness of my mouth.**
3. **I couldn't seem to experience any positive feeling at all.**
4. **I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion).**
5. **I found it difficult to work up the initiative to do things.**
6. **I tended to overreact to situations.**
7. **I experienced trembling (e.g., in the hands).**
8. **I felt that I was using a lot of nervous energy.**
9. **I was worried about situations in which I might panic and make a fool of myself.**
10. **I felt that I had nothing to look forward to.**
11. **I found myself getting agitated.**
12. **I found it difficult to relax.**

13. I felt down-hearted and blue.

14. I was intolerant of anything that kept me from getting on with what I was doing.

15. I felt I was close to panic.

16. I was unable to become enthusiastic about anything.

17. I felt I wasn't worth much as a person.

18. I felt that I was rather touchy.

19. I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat).

20. I felt scared without any good reason.

21. I felt that life was meaningless.

(iv) Personal Health Questionnaire Depression Scale (PHQ-9) (Kroenke, Spitzer, & Williams, 2001).

How often during the past 2 weeks were you bothered by any of the following problems (0 = Not at all, 1 = Several days, 2 = More than half the days, 4 = Nearly every day).

1. Little interest or pleasure in doing things.

2. Feeling down, depressed, or hopeless.

3. Trouble falling or staying asleep, or sleeping too much.

4. Feeling tired or having little energy.

5. Poor appetite or overeating.

6. Feeling bad about yourself, or that you are a failure, or have let yourself or your family down.

7. Trouble concentrating on things, such as reading the newspaper or watching television.

8. Moving or speaking so slowly that other people could have noticed. Or the opposite –being so fidgety or restless that you have been moving around a lot more than usual.

9. Thoughts that you would be better off dead, or of hurting yourself in some way.

(v) Center for Epidemiologic Studies Depression Scale (CES-D) 8-item (Radloff, 1977).

Now think about the past week and the feelings you have experienced. Please tell me if each of the following was true for you much of the time during the past week (Yes/No).

1. **(Much of the time during the past week), you felt depressed?**
2. **(Much of the time during the past week), you felt that everything you did was an effort?**
3. **(Much of the time during the past week), your sleep was restless?**
4. **(Much of the time during the past week), you were happy?**
5. **(Much of the time during the past week), you felt lonely?**
6. **(Much of the time during the past week), you enjoyed life?**
7. **(Much of the time during the past week), you felt sad?**
8. **(Much of the time during the past week), you could not get going?**

(vi) Center for Epidemiologic Studies Depression Scale (CES-D) 10-item (Radloff, 1977).

Below is a list of some of the ways you may have felt or behaved. Please indicate how often you have felt this way during the past week: (circle one number on each line) (0 = Rarely or none of the time (less than 1 day), 1 = Some or little of the time (1-2 days), 2 = Occasionally or a moderate amount of time (3-4 days), 3 = All of the time (5-7 days)).

1. **Past week: Not interested in eating/poor appetite.**
2. **Past week: doing anything was exhausting.**
3. **Past week: slept poorly.**
4. **Past week: felt in a terrible mood.**
5. **Past week: felt lonely.**
6. **Past week: people weren't nice to you.**
7. **Past week: felt anguished.**
8. **Past week: no will/energy to do things.**
9. **Past week: felt joyful.**
10. **Past week: felt life going well.**

(vii) Pearlin Mastery Scale (Pearlin & Schooler, 1978).

How strong do you agree or disagree that (1 = strongly agree, 2 = agree, Don't know/don't understand/can't tell, 4 = disagree, 5 = strongly disagree):

1. I have little control over the things that happen to me.

2. There is really no way I can solve some the problems that I have.

3. There is little I can do to change many of the important things in my life.

4. I often feel helpless in dealing with the problems of life.

5. Sometimes I feel that I'm being "pushed around" in life.

6. What happens to me in the future mostly depends on me.

7. I can do just about anything I really set my mind to do.

Appendix C

Single-item Questions Utilized in the Current Research

Chapter 2:

(a) Are you currently on a diet?

Response categories: Yes / No

(b) How many cigarettes per day do you smoke?

Response categories: 1- None; 2- 5 or less; 3- 6 to 10; 4- 11 to 20; 5- 21 to 30; 6- 31 or more.

(c) How many drinks containing alcohol did you have yesterday?

Response categories: 1- None; 2- 1 or 2; 3- 3 or 4; 4- 5 or 6; 5- 7 to 9; 6- 10 or more.

Chapter 3:

(a) What is the highest level of education your father attained?

Response categories: 1- primary; 2- lower secondary; 3- upper secondary; 4- higher education/university.

(b) How often do you engage in more than 20 minutes of vigorous exercise (e.g. running, cycling, swimming, vigorous team sports) on one occasion?

Response categories: 1- Never; 2- Monthly; 3- 2 to 4 times per month; 4 2 to 3 times per week; 5- 4 or more times per week.

(c) How many cigarettes per day do you smoke?

Response categories: 1- None; 2- 5 or less; 3- 6 to 10; 4- 11 to 20; 5- 21 to 30; 6- 31 or more.

Chapter 4:

(a) What is the highest level of education your father attained?

Response categories: 1- primary; 2- lower secondary; 3- upper secondary; 4- higher education/university.

(b) Are you currently on a diet?

Response categories: Yes / No

(c) How many cigarettes per day do you smoke?

Response categories: 1- None; 2- 5 or less; 3- 6 to 10; 4- 11 to 20; 5- 21 to 30; 6- 31 or more.

(d) How often do you engage in more than 20 minutes of vigorous exercise (e.g. running, cycling, swimming, vigorous team sports) on one occasion?

Response categories: 1- Never; 2- Monthly; 3- 2 to 4 times per month; 4 2 to 3 times per week; 5- 4 or more times per week.

(e) How often do you have a drink containing alcohol?

Response categories: 1- Never, 2- Monthly or less, 3- 2 to 4 times a month; 4- 2 to 3 times a week; 5- 4 or more times a week.

Chapter 5:

Variables from the English Longitudinal Study of Ageing

(a) Social Class and Socio-economic Group linked to Operational Categories of the NS-SEC:

1- Employers in large organizations; 2- Higher managerial; 3- Higher professionals (traditional) – employees; 4- Higher professionals (new) – employees; 4- Higher professionals (traditional) - self-employed; 5- Higher professionals (new) - self-employed; 6- Lower professionals and higher technical (traditional) – employees; 7- Lower professionals and higher technical (new) – employees; 8- Lower professionals and higher technical (traditional) - self-employed; 9- Lower professionals and higher technical (new) - self-employed; 10- Lower managerial; 11-Higher supervisory; 12- Intermediate clerical and administrative; 13- Intermediate sales and service; 14- Intermediate technical and auxiliary; 15- Intermediate engineering; 16- Employers in small organisations (non-professional); 17- Employers in small organisations (agriculture); 18- Own account workers (non- professional); 19- Own account workers (agriculture); 20- Lower supervisory; 21- Lower technical craft; 22- Lower technical process operative; 23- Semi-routine sales; 24- Semi-routine service; 25- Semi-routine technical; 26- Semi-routine operative; 27- Semi-routine agriculture; 28- Semi-routine clerical; 29- Semi-routine childcare; 30- Routine sales and service; 31- Routine production; 32- Routine technical; 33- Routine operative; 34- Routine agricultural; 35- Never worked; 36- Long-term unemployed; 37- Full-time students; 38- Occupations not stated or inadequately described; 39- Not classifiable for other reasons.

(b) Thinking now about all kinds of drink, how often have you had an alcoholic drink of any kind in the last 12 months?

Response categories: 1- Almost every day; 2- Five or six days a week; 3- Three or four days a week; 4- Once or twice a week; 5- Once or twice a month; 6- Once every couple of months; 7- Once or twice a year; 8- Not at all in the last 12 months. 1 – 7 coded as 1, 8 coded as 0.

(c) Do you smoke cigarettes at all nowadays?

Response categories: Yes / No

(d) In the past three weeks, have you had any respiratory infections such as influenza, pneumonia, bronchitis or a severe cold?

Response categories: Yes / No

Variables from the National Health and Nutrition Examination Survey

(a) What is the highest grade or level of school you have completed or the highest degree you have received?

Response categories: 1- Less Than 9th Grade; 2 9-11th Grade (Includes 12th grade with no diploma); 3- High School Grad/GED or Equivalent; 4- Some College or AA degree; 5- College Graduate or above.

(b) In the past 12 months, how often did you drink any type of alcoholic beverage?

Response categories: Yes / No

(c) Do you now smoke cigarettes?

Response categories: Yes / No

(d) i. Did you have a head cold or chest cold that started during those 30 days?

Response categories: Yes / No.

ii. Did you have a stomach or intestinal illness with vomiting or diarrhea that started during those 30 days?

Response categories: Yes / No.

iii. Did you have flu, pneumonia, or ear infections that started during those 30 days?

Response categories: Yes / No.

Chapter 6:

Self-rated Health

(a) Regarding your current state of health, do you feel it is excellent, good, average, not so good, or poor?

Earthquake-related questions

(a) i. Did the earthquake cause any damage to the respondents house?

Response categories: Yes / No.

ii. Did the respondent have to temporarily move away from home because of the earthquake?

Response categories: Yes / No.

(b) Were you scared by the earthquake?

Response categories: 1- Not scared; 2- A little scared; 3- Somewhat scared; 4- Very scared; 5- Extremely scared.

Health Behaviour

(a) Did you smoke in the past six months?

Response categories: Yes / No.

(b) Is the respondent using any long-term medications?

Response categories: Yes / No.

Demographics

(a) Education- recoded to: 0- Illiterate or literate with no education; 1- Primary education only (1st – 6th year); 2- Junior high (7th – 12th year); 3- College education (1st – 4th year and above).

(b) Ethnicity- coded as follows: 1- Fukienes; 2- Hakka; 3- Mainland.

Chapter 7

Health Behaviour

(a) Did you smoke in the past six months?

Response categories: Yes / No.

(b) In the past six months did you drink alcohol…

Response categories: 0- No; 1- Sometimes; 2- Frequently; 3- Everyday.

(c) How many times do you exercise per week?

Response categories: 0- None; 1- less than once a week; 2- l to 2 tims per week; 3- 3 to 5 times per week; 4- six or more times per week.

Note: "Exercise" refers to regular or periodical physical exercise, it does not include household activities or labor.

(d) Is the respondent using any long-term medications?

Response categories: Yes / No.

Demographics

(a) Education- recoded to: 0- Illiterate or literate with no education; 1- Primary education only (1st – 6th year); 2- Junior high (7th – 12th year); 3- College education (1st – 4th year and above).

(b) Ethnicity- coded as follows: 1- Fukienes; 2- Hakka; 3- Mainland.