

**Noise Sensitivity and Diminished Health:
The Role of Stress-Related Factors**

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A thesis submitted to
the Auckland University of Technology
in fulfillment of the requirements for the degree of
Doctor of Philosophy (PhD)

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August 2012

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Abbreviations

ACTH – Adrenocorticotrophic Hormone

AUT – Auckland University of Technology

AUTEC – Auckland University of Technology Ethics Committee

BCa CI – Bias Corrected and Accelerated Confidence Interval

BFI – Big Five Inventory

CAR – Cortisol Awakening Response

CARauc – Cortisol Awakening Response (area under the curve)

CARi – Cortisol Awakening Response (with respect to increase)

CRF – Corticotropin-Releasing Factor

DASS – Depression and Anxiety Stress Scale

dB – Decibel

EM - Expectation Maximisation

EPI – Eysenck Personality Inventory

GAS – General Adaptation Syndrome

GHQ – General Health Questionnaire

GR – Glucocorticoid Receptor

HPA – Hypothalamic-Pituitary-Adrenal

HRQOL – Health-Related Quality of Life

HSPS – Highly Sensitive Person Scale

HYENA – Hypertension and Exposure near Airports (Study)

MCAR – Missing Completely at Random

MMPI – Minnesota Multiphasic Personality Inventory

MR – Mineralocorticoid Receptor

MVA – Missing Value Analysis

NoiSeQ – Noise Sensitivity Questionnaire

NZ – New Zealand

PSQI – Pittsburgh Sleep Quality Index

PSS – Perceived Stress Scale

PTSD – Post-Traumatic Stress Disorder

PVN – Paraventricular Nucleus

QOL – Quality of Life

REM – Rapid Eye Movement

SAM – Sympathetic-Adrenal-Medullary

SCN – Suprachiasmatic Nucleus

SF-36 – Short-Form Health Survey

SHC – Subjective Health Complaints

SHCI – Subjective Health Complaints Inventory

SPL – Sound Pressure Level

SPS – Sensory Processing Sensitivity

SPSS – Statistical Package for the Social Sciences

VIF – Variance Inflation Factor

WHO – World Health Organization

WHOQOL – World Health Organization Quality of Life

WNS – Weinstein Noise Sensitivity Scale

3-NS – 3-Item Noise Sensitivity Scale

Attestation of Authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person, nor material which to a substantial extent has been submitted for the award of any other degree or diploma of a university or other institution of higher learning.

Erin Hill _____

Date _____

Acknowledgements

I would like to take this opportunity to thank those who have helped me in this academic pursuit. First, sincerest thanks to my supervisors – Dr. Rex Billington, for his unfailing support throughout my time at AUT, and Dr. Chris Krägeloh, for his patience and keen insight that helped in crafting this thesis. I am grateful to have worked with both of you. I have greatly appreciated your candidness and I truly admire your drive to conduct thorough and relevant scientific research.

I would like to acknowledge the financial support of the Commonwealth Scholarship and Fellowship Plan, administered by the Vice-Chancellors' Committee of New Zealand. Without this doctoral scholarship, I would not have been able to pursue this degree in New Zealand and expand my research experience and collaboration to this side of the world. Not only am I thankful to have gained academic experience in NZ, I am also grateful to have been able to live here and explore such a beautiful country over the past few years.

I would also like to thank those who helped me with the early stages of this research project. Thanks to Dr. Daniel Shepherd (AUT) for his resourcefulness in helping with initiating the research programme and data collection. I am grateful for the support from the AUT Faculty of Health and Environmental Sciences Contestable Research Fund, which provided funding for Study 2. Thanks also to Dr. Kim Dirks (University of Auckland) and Dr. David McBride (University of Otago) for their financial support for the community-based study (Study 1). I would also like to acknowledge Dr. David Welch (University of Auckland) for help with questionnaire design for Study 1. Thanks to the North Shore Times for showing an interest in my doctoral research programme and publishing an article on it. Thanks also to my participants; without the generous donation of their time, such

psychological research would not be possible. Both studies were approved by the Auckland University of Technology Ethics Committee (Study 1 reference: 10/271, Study 2 reference: 10/270, both approved 18.02.2011).

Finally, thank you to family and friends, both here in New Zealand and back home in Canada, for the ongoing support. Sincerest thanks to Erinn Squires (Carleton University, Canada), who has not only been a great friend to me throughout this degree, but also assisted me in using the Preacher and Hayes's statistical models and SPSS macros used in Study 1. I am indebted to my partner, Adam, who has helped me every step of the way. From spending hours discussing the intricate details of my thesis to getting up at the break of dawn on several occasions to help me hand-deliver thousands of questionnaires, Adam's support has been endless. Thanks to my sister, Steph, and my brother-in-law, Sam, who have continuously shown me love and support throughout my studies. Finally, I would like to extend deepest thanks to my Mom and Dad. I am ever grateful to have such supportive parents, whose guidance and encouragement helped propel me through this degree.

Abstract

The concept of noise sensitivity emerged from public health and psychoacoustic research to help explain individual differences in reactions to noise. Noise sensitivity is frequently included as a personality variable in public health studies investigating the influence of environmental noise on health and well-being. Interestingly, noise sensitivity appears to be associated with diminished health, independent of environmental noise exposure. However, the mechanisms underlying the relationship have not been adequately explained. Noise sensitivity has been described as a variable that may increase an individual's vulnerability to noise and other stressors. Therefore, the association between noise sensitivity and diminished health might be explained by excessive psychological stress and stress-related physiological changes. As such, this thesis addressed an important gap in the literature by examining the role of stress-related factors in the association between noise sensitivity and diminished health.

Two studies were conducted to test the hypothesis that stress and stress-related physiological changes are involved in the association between noise sensitivity and diminished health. Study 1 was a large ($n = 1102$; 367 males, 713¹ females aged 18-94 years) survey-based study designed to test a model of noise sensitivity and self-reported health involving measures of subjective health complaints, self-reported hypertension, and mental health complaints (anxiety and depression). Perceived stress and sleep problems were tested as mediators in the model, while gender and environmental noise exposure were tested as moderators. Perceived stress and sleep problems mediated the association between noise sensitivity and subjective health complaints, while the moderators did not

¹ Gender information for 22 participants was not provided.

influence the overall model. Neuroticism, included in the models as a covariate, accounted for the association between noise sensitivity and mental health complaints. No association was found between noise sensitivity and self-reported hypertension.

Study 2 was conducted to assess the role of stress-related physiological changes that may be involved in the associations among noise sensitivity, perceived stress and health complaints in a community sample ($n = 107$; 51 males, 56 females aged 18-78 years). Specifically, using a subsection of the sample ($n = 92$), Study 2 tested the association between noise sensitivity and functioning of the hypothalamic-pituitary-adrenal (HPA) axis, a major stress system of the body, through assessment of the cortisol awakening response (CAR). The CAR captures general HPA axis functioning, which can be dysregulated under chronic stress, and may have detrimental effects on health and well-being. There was no significant relationship between noise sensitivity and CAR levels. However, gender emerged as the strongest predictor of overall cortisol output across the awakening period, with females exhibiting greater output than males. Females also reported greater perceived stress, noise sensitivity, and subjective health complaints than males.

Overall, this thesis significantly contributes to the understanding of the relationship between noise sensitivity and diminished health. Perceived stress and sleep problems mediated the association between noise sensitivity and subjective health complaints, while neuroticism accounted for the relationship between noise sensitivity and mental health complaints (anxiety and depression). HPA axis functioning was not related to noise sensitivity or health complaints. Results are discussed with reference to the importance of considering perceived stress and sleep problems in noise sensitivity research, and future directions for investigations on the association between noise sensitivity and diminished health.

Chapter 1: Introduction

1.1. Background

Exposure to environmental noise has been identified as a risk factor for the development of health problems, such as a cardiovascular disease (Brink, 2011; Selander, 2010; van Kempen & Babisch, 2012), and the exacerbation of mental health issues such as excessive anxiety and irritability (Hardoy et al., 2005; Matheson, Stansfeld, & Haines, 2003; Stošić & Blagojević, 2011). However, the effects of noise, such as noise annoyance and sleep disturbance, vary considerably across the population (Fields, 1993; Weinstein, 1980). In light of these individual differences in reaction to noise, acoustic and public health researchers frequently include measures of ‘personal noise sensitivity’ in their investigations, usually in the form of a self-report questionnaire (e.g., Al-Mutairi, Al-Attar, & Al-Rukaibi, 2011; Fyhri & Aasvang, 2010; Paunovic, Jakovljević, & Belojević, 2009). Noise sensitivity is regarded as a stable trait that increases an individual’s reactivity to noise and susceptibility to annoyance (Job, 1999; Stansfeld, 1992). While noise sensitivity is seldom the focus of public health research, there is mounting evidence to suggest that noise sensitivity may be an independent predictor of diminished health (Fyhri & Klæboe, 2009; Kishikawa et al., 2009; Nivison & Endresen, 1993; Stansfeld, 1992).

The association between noise sensitivity and diminished health is unlikely to be direct (Fyhri & Klæboe, 2009). Rather, various intermediary factors are presumably involved. There is evidence to suggest that noise sensitivity may be associated with a vulnerability to a variety of stressors in addition to noise (Stansfeld, 1992; Weinstein, 1978). Chronic stress may be more frequently experienced by noise sensitive individuals, and in turn, may be a link in the association between noise sensitivity and health problems. Indeed, health psychology research has demonstrated that chronic stress affects long-term

physiological changes and health problems (Cohen, Janicki-Deverts, & Miller, 2007; Cohen, Tyrrell, & Smith, 1991; McEwen, 1998b; McEwen & Seeman, 1999). Therefore, the relationship between noise sensitivity and diminished health² could be accounted for by elevated stress, and related physiological changes.

Broadly, this thesis was designed to address the following research question: Do stress-related factors account for the association between noise sensitivity and diminished health? Two studies were employed to address this question. Each study was designed to investigate a particular aspect of stress in the relationship between noise sensitivity and diminished health. The first study involved an investigation of the relationship among noise sensitivity, perceived stress, and self-reported health in a large community sample. The use of a large community-based study also allowed for the investigation of the role of environmental noise and additional health and psychological factors (e.g., neuroticism, sleep problems involved in the association between noise sensitivity and diminished health.

The second study was designed to measure the relationship between noise sensitivity and activity of the hypothalamic-pituitary-adrenal (HPA) axis, a major component of the neuroendocrine system involved in the initiation and regulation of the stress response. The HPA axis can be altered as a result of chronic stress (Schmidt-

² Health is widely regarded as a broad term encompassing an individual's physical, mental, and social well-being (Jadad & O'Grady, 2008). For the purpose of this thesis, health was operationalised as the absence of physical and mental health problems (i.e., absence of complaints, disease). This definition of health was applied, in part, to remain consistent with previous noise sensitivity research investigating its relation to self-reported health problems, as assessed with subjective health complaints (e.g, Fyhri & Klæboe, 2009; Nivison & Endresen, 1993).

Reinwald et al., 1999), and may be involved in the development of mental and physical health problems (Clow, Hucklebridge, & Thorn, 2010; Dedovic et al., 2010). The use of an objective measure of neuroendocrine functioning (the CAR) was chosen in order to investigate a potential physiological mechanism involved in the association between noise sensitivity and diminished health.

1.2. Thesis Rationale

Noise sensitivity is characterised as an attitudinal trait that can impact an individual's reaction to noise (Ellermeier, Eigenstetter, & Zimmer, 2001; Job, 1999; Zimmer & Ellermeier, 1999) and their surrounding environment (Stansfeld, 1992; Weinstein, 1978). While some psychiatric and psychophysiological research has acknowledged that noise sensitivity increases an individual's likelihood of experiencing psychological or physiological stress (Heinonen-Guzejev et al., 2004; Ljungberg & Neely, 2007; Stansfeld, 1992), few studies have addressed the association empirically, or tested it in relation to health outcomes.

If, as hypothesised, psychological and physiological stress parameters help explain the association between noise sensitivity and diminished health, this research has implications in health psychology and public health research. Noise sensitivity may be a particularly important trait in health psychology research, a field of study that attempts to discern the influence of psychological factors on mental and physical health. Therefore, better understanding the stress-related psychological and physiological factors involved in the relationship between noise sensitivity and diminished health is an important endeavour.

Noise sensitivity is often dismissed in environmental noise policy because little is known about the trait itself. For example, noise policy regarding the distance at which motorways and airports should be built from residential communities is often based on

average annoyance ratings (Staples, 1997), thus discounting the possible ill-effects on noise sensitive community members. Further, because noise sensitivity is closely related to negative affectivity (i.e., the propensity to experience negative emotions; Smith et al., 2002; Weinstein, 1980), noise annoyance and other complaints are often dismissed as artefacts of a negative psychological state. Therefore, identifying more clearly the mechanisms at play in the association between noise sensitivity and diminished health will help inform acousticians, policy makers, and health professionals wishing to protect the well-being of people most sensitive to noise.

1.3. Originality of the Thesis

This thesis adds to the limited body of literature on the relationship between noise sensitivity and diminished health through an investigation of stress-related factors that may be involved in the association.

- Despite noise sensitivity having been described as a trait that can increase an individual's stress vulnerability (Heinonen-Guzejev, 2009; Stansfeld, 1992), no research has tested perceived stress as a mediator of the relationship between noise sensitivity and health outcomes (e.g., subjective health complaints, hypertension, mental health problems).
- In accordance with the 'stress vulnerability' framework of the thesis, a relatively novel physiological measure of HPA axis activity (the CAR) was used in order to investigate a health-relevant physiological marker in relation to noise sensitivity.

- According to the available research, no previous research has studied noise sensitivity in relation to the CAR in a naturalistic setting³, or within the context of the relationship between noise sensitivity and diminished health.

1.4. Thesis Organisation

In order to provide an adequate background for this research programme, the following three chapters of the thesis are literature reviews. Chapter 2 provides information on stress and the health effects of chronic stress. Chapter 3 is a review of the literature on reactions to environmental noise and related health effects. While the focus of the thesis is not on environmental noise exposure, this research is presented to provide a thorough background on environmental noise – the field from which the concept of noise sensitivity emerged. In the final section of the literature review, Chapter 4 discusses research on noise sensitivity. It is noteworthy that some of the research presented in Chapter 3 and Chapter 4 overlap due to the presentation of closely-related noise topics (e.g., noise annoyance, sleep disturbance). The findings are presented separately as related to environmental noise (Chapter 3) or noise sensitivity (Chapter 4) in order to keep the two topics distinct in the literature review.

Chapter 5 presents Study 1, “Noise sensitivity and self-reported health: An investigation of potential mediators and moderators” in which the intermediary influence of perceived stress in the association between noise sensitivity and self-reported health is

³ In contrast to laboratory experiments. The only previous study measuring both the CAR and noise sensitivity was a small ($n = 12$) male-only laboratory study on the effects of night-time noise exposure (see Persson Waye, Clow, Edwards, Hucklebridge, & Rylander, 2003). Limitations of this study are discussed in Chapter 6.

examined. An additional mediator (sleep problems) and moderators (gender, noise exposure) were included in the model to further investigate other factors in the relationship between noise sensitivity and diminished health. Chapter 6 presents Study 2, “Noise sensitivity and the cortisol awakening response”, which examined the association between these two variables, and in relation to perceived stress, health complaints and self-reported sleep disturbance. Finally, the general discussion is presented in Chapter 7 of the thesis.

Chapter 2: Literature Review – Stress Physiology and Health

2.1. Stress

Stress has become a ubiquitous term used in both everyday language and scientific research to describe negative emotional states and the accompanying physical changes (McEwen & Stellar, 1993). From an evolutionary perspective, the physical response to stress is adaptive – it provides an individual with biological resources and the ability to deal with a potentially life-threatening situation (Korte, Koolhaas, Wingfield, & McEwen, 2005). While the stress response was adaptive for survival in ancestral times, the human body did not evolve to cope with chronic stress. Chronic stress negatively impacts overall health; it has been linked to the development of chronic diseases including cardiovascular disease and autoimmune disorders (McEwen & Seeman, 1999). The risk and pervasiveness of chronic stress in modern society highlights the need to understand the physiological and psychological factors involved in the relationship between stress and health (Baum, 1990).

2.1.1. Historical origins of the ‘stress’ concept. The term ‘stress’ was first used in the context of health by Hans Selye (1936), an endocrinologist, who inadvertently stumbled upon a unique pattern of biological changes in his lab rats. Specifically, Selye, possibly due to inexperience in working with laboratory animals, mishandled the rats throughout the experiments, unintentionally causing the animals to experience considerable discomfort and distress (Gurung, 2010). Because the rats in both the experimental and control conditions exhibited striking physical abnormalities (e.g., enlarged adrenal glands, deformed lymph nodes), Selye began to suspect that the reason for the physical changes was the stressful environment of the rats (i.e., continuously being mishandled), rather than the substance with which they were being injected.

In good scientific form, Selye and his research team continued experimenting; they exposed more rats to a variety of stressors, including extreme heat and cold, noise and water (Gurung, 2010). Results across the various stressor conditions were the same; the rats exhibited significant changes to the endocrine and immune systems (Selye, 1936). Borrowing the term from the engineering and mechanical sciences to describe forces acting on matter, Selye (1946) proposed that the rats had experienced ‘stress’ – a non-specific response of the body to demands upon it. Based on these astute observations, Selye described the General Adaptation Syndrome (GAS) – a theory that laid the groundwork for later research on physiological and health changes associated with stress in humans (Fink, 2009).

2.1.2. General adaptation syndrome and the ‘fight or flight’ response. Selye’s GAS was developed from the foundations of Cannon (1929), who had previously described the physiological patterns associated with homeostasis and the ‘fight or flight’ response. Homeostasis is a term used to describe the delicate balance of the body’s biochemical and physiological processes (Cannon, 1929). In order to maintain this balance, Cannon observed, for example, that when an animal would develop low blood sugar, hunger would prompt ingestion of food, and when an animal’s core body temperature would lower, shivering would occur in order to create heat from the muscle contractions (Cannon, 1929). These adaptive processes allowed the body to maintain homeostasis. Stress, from the perspective of GAS, was simply defined as a threat to homeostasis – physiological changes that move the animal’s body away from this optimal balance (Selye, 1946).

Selye’s (1946) GAS describes the progression of acute stress to chronic stress. In the first phase (the alarm phase), an animal is faced with a stressor, which, in turn, causes physiological changes. The acute stress response can be adaptive; rapid bodily changes

such as the increase of stress hormones, heart rate, blood pressure, and the increased availability of blood glucose help provide the animal with energy and the capability to ‘flee’ or ‘fight’ the stressor (e.g., predator). When stress is acute, the animal also experiences positive cognitive side effects such as vigilance and attention (Charmandari, Tsigos, & Chrousos, 2005).

The animal/organism can only maintain this level of physiological arousal for a short period of time – a period that Selye (1946) called the resistance phase. However, these adaptive bodily changes are not sustainable. Eventually, the animal will enter the stage of exhaustion, during which it develops physical side effects from the sustained physiological arousal (e.g., enlargement of the adrenal cortex, shrinking thymus). Therefore, an animal enduring exposure to chronic stressors will eventually be at risk for the development of these pathological physical changes, and possibly early mortality (Daruna, 2004).

2.1.3. The physiology of the stress response. The stress response involves activation of the endocrine system and the autonomic nervous system. The physiological changes resulting from the stress response are adaptive, designed to restore equilibrium through the mobilisation of bodily resources, and maximise survival (Kyrou & Tsigos, 2009).

2.1.3.1. Activation of the sympathetic-adrenal-medullary system. The autonomic nervous system consists of two complementary branches: the sympathetic nervous system, associated with the typical fight or flight stress response, and the parasympathetic nervous system, associated with restoration and relaxation (Kalat, 2007). During a stress response, the sympathetic-adrenal-medullary (SAM) system, a subsidiary of the sympathetic nervous system initiates the secretion of catecholamines – adrenaline (epinephrine) and

noradrenaline (norepinephrine; see Figure 1). Adrenaline is released from the adrenal medulla, an endocrine gland located just above the kidney. Noradrenaline is also released from this site, but most noradrenaline secreted during the stress response originate from post-ganglionic neurons. These neurons directly innervate blood vessels and other organ sites (Brophy, Scarlett-Ferguson, & Webbers, 2010). Both catecholamines act on various organs in the body during the stress response. Adrenaline and noradrenaline stimulate the cardiovascular system (increasing blood flow and heart rate). Additionally, adrenaline also stimulates dilation of the bronchials and helps to mobilise energy stores through the stimulation of glycogenolysis – the breakdown of glycogen into glucose (Sherwood, 2012).

2.1.3.2. Activation of the hypothalamic-pituitary-adrenal axis. During the stress response, the amygdala also activates the HPA axis, an important component of the neuroendocrine system. Following the recognition of a stressor, the hypothalamus initiates a cascade of events that leads to the eventual secretion of stress hormones (glucocorticoids). The hypothalamus secretes the corticotropin-releasing factor (CRF), which then triggers the release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary gland (Pruessner et al., 2010). ACTH then travels through the bloodstream to bind with the adrenal cortex, where it stimulates the release of glucocorticoids into the blood.

Glucocorticoid is the broad term used for corticosteroid hormones, the most common being cortisol in humans and cortisone in rodents (Kemeny, 2003). Cortisol activates a number of adaptive physiological changes during the stress response such as the increase of blood glucose and the mobilisation of energy stores through the upregulation of glucose, lipid, and amino acid metabolism (Sherwood, 2012). Overall, cortisol is the main indicator of HPA axis functioning. For this reason, cortisol is a popular biomarker in stress

research (e.g., Hellhammer, Wüst, & Kudielka, 2009; Wüst, Federenko, Hellhammer, & Kirschbaum, 2000a; Wüst et al., 2000b).

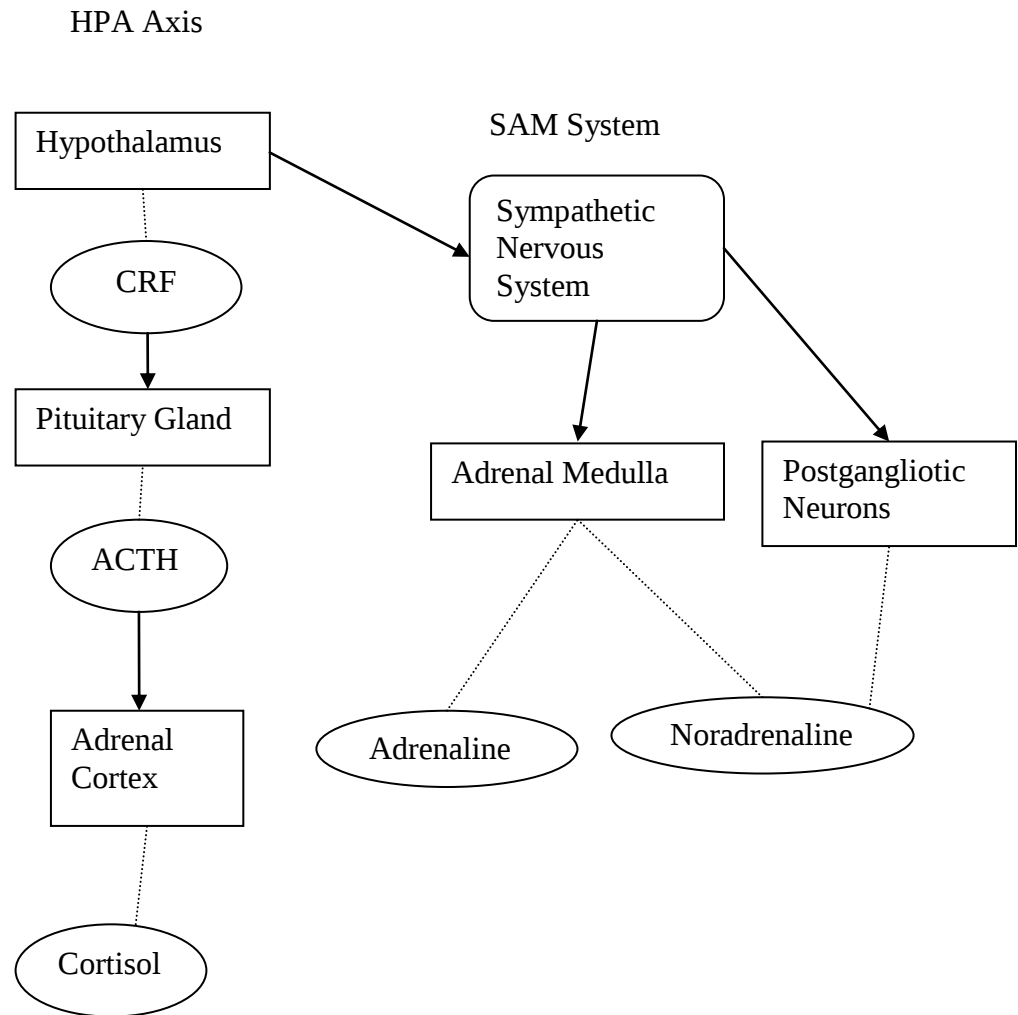


Figure 1. Stress response cascades of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic-adrenal-medullary (SAM) system.

Note. Dotted lines indicate release from a gland/neuron. Arrows indicate signalling between physiological components. The release of noradrenaline from the post-ganglionic neurons is included for completeness; it is not technically a component of the SAM system.

Abbreviations: HPA = hypothalamic-pituitary-adrenal; SAM = sympathetic-adrenal-medullary; CRF = corticotropin-releasing factor; ACTH = adrenocorticotrophic hormone.

2.1.3.3. Regulation of the stress response. Proper regulation of the stress response is necessary for maintaining homeostatic balance in the body, and in turn, maintaining good health (McEwen, 2007). The stress response is beneficial to the organism in the short-term (i.e., for survival), but the organism must be able to terminate the stress response when a threat is no longer present. Glucocorticoids (e.g., cortisol in humans) and the hippocampus, a component of the limbic system and an important memory centre in the brain, are involved in stress response regulation (Lovallo, 2005). The regulatory function of the hippocampus depends on the binding of cortisol to mineralocorticoid receptors (MR) and glucocorticoid (GR) receptors. MRs are largely involved with initiating the stress response, while GRs influence its appropriate termination (de Kloet, Joels, & Holsboer, 2005). When cortisol binds to the hippocampal GRs, this communication pathway is blocked and CRF is not secreted from the hypothalamus. When there is no binding of cortisol to the hippocampal GRs, the hippocampus continues to communicate with the hypothalamus via relay neurons to promote the secretion of CRF and thus the continuation of a stress response (Fink, 2009).

2.1.3.4. Dysregulation of the stress response. The initiation and termination processes of the stress response are not consistently straightforward, however. Chronic stress has been identified as a clear culprit in the dysregulation of the stress response (McEwen, 2001; Miller, Chen, & Zhou, 2007; Sapolsky, Krey, & McEwen, 1986). More specifically, sustained elevations of glucocorticoids can cause hippocampal GRs to lose their function, and potentially lead to hippocampal atrophy (Lovallo, 2005; O'Connor, O'Halloran, & Shanahan, 2000; Rylander, 2004; Sapolsky, Krey, & McEwen, 1985). Without the binding of cortisol to the hippocampal GRs, the hippocampus continues to promote the secretion of CRF via the hypothalamus. Desensitisation of the hippocampal

GRs causes dysregulation of the negative feedback loop of the stress response – leading to prolonged cortisol release (Sapolsky et al., 1986).

2.1.3.4.1. Glucocorticoid cascade hypothesis. Hippocampal dysregulation of the stress response negative feedback system was the foundation for the ‘glucocorticoid cascade hypothesis of aging’ (Sapolsky et al., 1986), a theory still reported in neuroscientific research (e.g., de Kloet et al., 2005; Garrido, de Blas, Del Arco, Segovia, & Mora, 2012). Sapolsky (1986) proposed that the hypersecretion of glucocorticoids (resulting from the dysregulation of the stress response) leads to pathogenesis in the brain and accelerates the aging process (e.g., dendritic atrophy, hippocampal neuron loss; Sapolsky, Uno, Rebert, & Finch, 1990). Further, once desensitisation of the hippocampal GRs begins, an individual will become increasingly vulnerable to both sustained glucocorticoid secretion and further hippocampal receptor damage (Sapolsky et al., 1990). This “cascade” of glucocorticoids results in damage to important brain centres (Sapolsky et al., 1986).

The glucocorticoid cascade hypothesis provided a neurobiological framework to study diseases of the brain and the aging process. Glucocorticoid-induced damage to the hippocampus, resulting in memory and learning deficits, has been demonstrated in studies on rats (e.g., Lupien et al., 1998; McEwen, 2007). That is, while the plasticity of the hippocampus can be potentially adaptive in navigating and coping with acute stressors, its malleability increases its susceptibility to stress-related damage (McEwen, 2007). Further demonstrating this relationship, extreme psychological stress in humans, as in the case of post-traumatic stress disorder (PTSD), has been linked to hippocampal damage (identified with neuroimaging) in both cross-sectional (Shin, Rauch, & Pitman, 2006) and longitudinal research (Carrion, Weems, & Reiss, 2007). The stress-induced brain alterations depicted in

the glucocorticoid cascade hypothesis of aging fall under the umbrella of allostatic load, a broad theoretical framework currently used to describe stress-related disease and physiological damage in various systems of the body.

2.2. Allostatic Load

Short-term physiological adaptation is referred to as ‘allostasis’, which refers to the maintenance of stability through change (Sterling & Eyer, 1988). That is, while homeostasis refers to the balance of physiological processes, allostasis refers to the physiological changes that occur to maintain a homeostatic state (Sterling & Eyer, 1988). Therefore, allostasis is an adaptive mechanism, akin to the ‘alarm’ state of General Adaptation Syndrome in that it helps the body cope with threats to homeostasis such as hunger and physical activity (McEwen & Seeman, 1999).

While stress hormones and physiological changes associated with allostasis can be helpful in coping with short-term stressors (i.e., providing adequate energy to escape from a dangerous situation), in the long-term, such changes can damage the body (McEwen, 1998b; Thayer & Sternberg, 2006). These long-term physical changes to the body have been termed ‘allostatic load’ (McEwen, 1998b; Seeman, McEwen, Rowe, & Singer, 2001). For example, continuous elevated blood pressure as result of chronic stress at an individual’s workplace can eventually lead to cardiovascular disease (Nordstrom, Dwyer, Merz, Shicore, & Dwyer, 2001). That is, allostatic load refers to the physical changes to the body that result from chronic stress. Allostatic load not only refers to damaged stress response systems (e.g., neuroendocrine, cardiovascular), but it also encompasses damage to other systems of the body such as the digestive system (e.g., stomach ulcers, irritable bowel syndrome), and diminished immune system functioning (e.g., Chrousos, 2009; Kemeny, 2003; McEwen & Seeman, 1999; Sapolsky, 2004; Segerstrom & Miller, 2004).

2.2.1. Pathways in the development of allostatic load. McEwen and Seeman

(1999) described four possible pathways in which allostatic load can develop (see Figure 2). The first (“a: repeated hits”; see Figure 2) involves frequent stress – more specifically, exposure to chronic stressors, such as noise, crowding or even social psychological stressors such as being a long-term caregiver for an individual with chronic illness (Evans & Stecker, 2004; Son et al., 2007). Therefore, because of continual exposure to stressors, an organism experiences the physiological stress response too frequently for the body to adequately recover.

The second pathway (“b: lack of adaptation”; see Figure 2) refers to lack of physical adaptation to the stressor. That is, over time, many individuals adapt to a stressor and do not show a stress response to the same degree as the first time they were exposed. However, in others, the stress response persists and no adaptation occurs. This process by which allostatic load can develop therefore encompasses the notion that individual differences may impact one’s susceptibility to the health effects of stressors.

The third possible mechanism McEwen and Seeman (1999) described involves the exhaustion of the allostatic system (“c: prolonged response”; see Figure 2). More specifically, the body loses its effectiveness in appropriately turning off the stress response, a mechanism possibly linked to damage to the hippocampal region of the brain, an area of the brain often associated with memory functions (McEwen, 2007). This type of allostatic load might occur under severe psychological stress or depression, whereby an individual’s body does not return to a state of recovery (McEwen & Seeman, 1999).

Finally, the fourth possible pathway in which allostatic load can develop (“d: inadequate response”; see Figure 2) involves lack of an appropriate physiological response (e.g., cortisol not being produced during exposure to a stressor). Indeed, in some cases of

severe stress, such as a PTSD, diminished cortisol output has been observed (Kolassa et al., 2007; Meewisse, Reitsma, de Vries, Gersons, & Olff, 2007; Wessa, Rohleder, Kirschbaum, & Flor, 2006). When this type of allostatic load occurs, individuals can develop compensatory physiological responses (e.g., excess pro-inflammatory cytokines) as a result of chronic exposure to allostatic mechanisms (e.g., chronic exposure to cortisol). For example, in some cases, chronic exposure to cortisol may result in overactivity of other systems (e.g., immune system). Supporting this theoretical pathway, Miller et al. (2002) found that pro-inflammatory cytokines were more likely to be produced by individuals under chronic psychological stress. The fact that excessive inflammation, characteristic of a variety of health problems including autoimmune and cardiovascular diseases, often worsens under psychological stress (Miller et al., 2002), also provides some evidence for this type of allostatic load.

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Figure 2. The four pathways in the development of allostatic load.

Illustration taken from McEwen and Gianaros (2011).

2.2.2. The measurement of allostatic load. Allostasis involves the activation of various physiological systems including the nervous, endocrine, and cardiovascular systems. As a result, measuring allostatic load can involve collecting information on physiological components as well as health outcomes in order to fully understand factors that may be influencing illness development. While metabolic (e.g., cholesterol levels) and neuroendocrine biomarkers (e.g., cortisol) are among the most commonly used measures of allostatic load, Hawkley et al. (2011) suggested that in order to fully assess allostatic load, a composite measure should be used.

Composite measures of allostatic load include a variety of biomarkers, representing physiological changes in various bodily systems. Allostatic load composite measures include physiological parameters that assess cardiovascular and respiratory function (e.g., blood pressure, heart rate, peak expiratory flow), anthropometric outcomes (e.g., body mass index), neuroendocrine components (e.g., cortisol), metabolic biomarkers (e.g., insulin, cholesterol) and immune system components (e.g., cytokines; Juster, McEwen, & Lupien, 2010; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997).

2.2.3. The cortisol awakening response. The assessment of physiological parameters can provide considerable insight into the impact of stress on the body. In particular, because cortisol is the primary output of the HPA axis (Pruessner et al., 2010), it is widely used to assess stress-related physiological changes. Indeed, cortisol is released in response to an acute stressor (Kemeny, 2003; Kirschbaum & Hellhammer, 1994), but it also follows a diurnal pattern – the production sharply increases at approximately 20-45 minutes following awakening and decreases throughout the day (Clow, Thorn, Evans, & Hucklebridge, 2004; Pruessner et al., 1997; Wilhelm, Born, Kudielka, Schlotz, & Wüst, 2007).

This peak of cortisol release in the morning period is referred to as the CAR. There is increasing evidence to suggest that dysregulation of this response may be related to psychological and health issues (e.g., Bruehl, Wolf, & Convit, 2009; Greaves-Lord et al., 2007; Kudielka & Kirschbaum, 2003), and thus may be an indicator of allostatic load (Kirschbaum & Hellhammer, 1999; Wüst et al., 2000b). Figure 3 provides information on the basic pattern cortisol exhibits over the awakening period. Individual differences are widely reported on the CAR; the figure provided to display the general pattern with the peak of cortisol release occurring approximately 30 minutes following awakening.

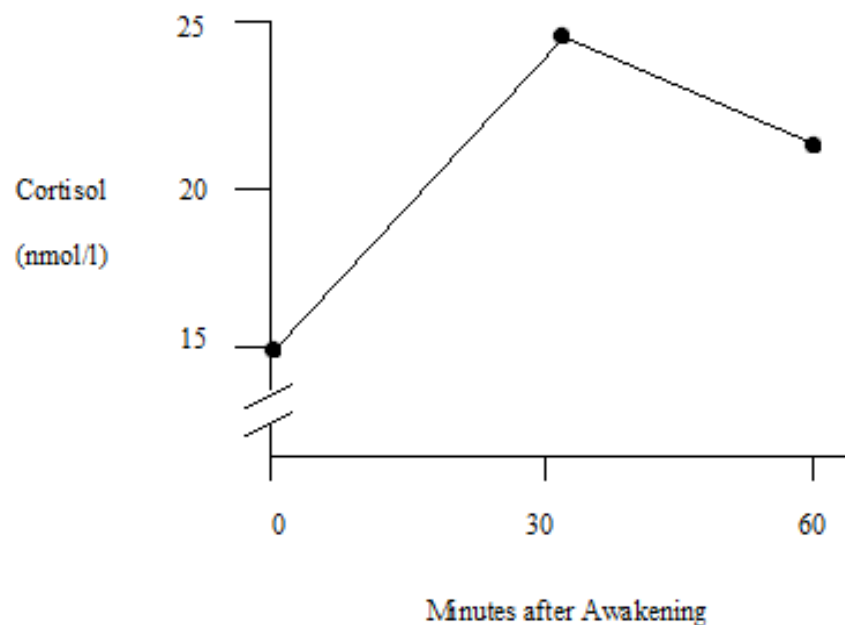


Figure 3. Average cortisol awakening response pattern.

Note. Figure adapted from Wüst et al. (2000b). Cortisol levels decline throughout the remainder of the day. This figure depicts only the expected pattern over the awakening period. Values vary across individuals.

The CAR first became widespread in the literature following a publication by Pruessner, Kirschbaum, and Hellhammer (1997) that reported increases in the concentration of salivary cortisol of 50-100% within the first 30 minutes of awakening. Since its first

introduction in the psychological literature, research on the CAR has increased tremendously; more than 280 articles were published on the topic between 1995 and 2009 (Clow et al., 2010). Research in the area continues to flourish with recent publications on various psychological and health issues, such as acculturation (Mangold, Mintz, Javors, & Marino, 2012), perseverative cognition (Zoccola, Dickerson, & Yim, 2011), and coronary artery disease (Bhattacharyya, Molloy, & Steptoe, 2008), among others (see Kudielka & Wüst, 2010).

2.2.3.1. Measuring the CAR. Cortisol levels can be measured using a variety of methods including through the collection of blood and urine. However, researchers in the field frequently opt to collect samples through saliva, a method that is regarded as non-invasive, consistent, and one that shows good intra-individual stability across time (Pruessner et al., 1997; Quirin, Pruessner, & Kuhl, 2008; Wüst et al., 2000b). The use of the CAR contrasts with the measurement of cortisol in a laboratory setting, usually in response to a psychosocial stressor (e.g., the Trier Social Stress task; Kirschbaum, Pirke, & Hellhammer, 1993) or a physical stressor (e.g., noise). The CAR is a more naturalistic measure; that is, it reflects processes of the neuroendocrine system in a natural setting since most samples are taken in a domestic environment (Clow et al., 2010).

While the CAR is largely viewed as a measure of overall functioning of the HPA axis (Clow et al., 2004), there is some research that shows that the CAR may be influenced by both state and trait factors (Hellhammer et al., 2007; Maina, Bovenzi, Palmas, Rossi, & Filon, 2011; Stalder, Hucklebridge, Evans, & Clow, 2009; Thorn, Hucklebridge, Evans, & Clow, 2009; Walker, O'Connor, Schaefer, Talbot, & Hendrickx, 2011; Zoccola et al., 2011). Specifically, the CAR may be influenced by demands of the upcoming day, as

evidenced by different patterns occurring between weekdays and weekends (Kunz-Ebrecht, Kirschbaum, Marmot, & Steptoe, 2004).

The CAR is, arguably, more ecologically valid than laboratory measurements of cortisol (e.g., following exposure to an acute stressor), but it cannot easily be tested in experimental conditions. That is, the CAR reflects overall functioning of the HPA axis, and therefore is largely a distinct entity from the stressor-induced changes in cortisol levels (e.g., in response to a physical or acute psychological stressor). However, due to the location of sample collection (i.e., domestic setting), the measurement of CAR is also susceptible to non-adherence to study protocol among participants (Broderick, Arnold, Kudielka, & Kirschbaum, 2004; Clow et al., 2010).

Although the CAR has been linked to a variety of psychological and behavioural variables (Chida & Steptoe, 2009b; Fries, Dettenborn, & Kirschbaum, 2009; Kudielka & Wüst, 2010), the underlying biological mechanisms involved in the CAR are not yet fully understood (Clow et al., 2010). The CAR may reflect cognitive processes of awakening (i.e., memory function and adaptation to awake status) and alterations in immune system functioning during the sleep-to-wake transition (for review, see Clow et al., 2010).

Additionally, there is some evidence to suggest that the CAR is influenced, at least in part, by genetic factors (Wüst et al., 2000a). Supporting this notion, Vreeburg et al. (2010) found that compared to controls, individuals with a parental history of depression and anxiety had elevated CAR levels similar to individuals with clinical levels of depression or anxiety. Therefore, Vreeburg et al. (2010) proposed that the CAR may be a physiological parameter marking an individual's vulnerability to stress-related and psychological disorders.

2.2.3.2. Neurobiological mechanisms of the CAR. The CAR reflects the increased activity of the hypothalamic suprachiasmatic nucleus (SCN) during the sleep-to-wake transition (Clow, Hucklebridge, Stalder, Evans, & Thorn, 2010; Wilhelm et al., 2007). Upon awakening, the SCN, a light-sensitive regulatory centre of the brain, communicates with the paraventricular nucleus (PVN) of the hypothalamus to activate the HPA axis and subsequent secretion of CRF and ACTH.

In addition to playing an important role in controlling the release of cortisol in acute stress, the hippocampus may influence the CAR as well. Hippocampal damage has been linked to an absent CAR (Buchanan, Tranel, & Kirschbaum, 2009), as well as a blunted CAR among individuals with diabetes mellitus (Bruehl et al., 2009) and depressive symptomatology (Dedovic et al., 2010). Although the specific role of the hippocampus in the CAR is not yet clear, these results suggest strong involvement (Fries et al., 2009).

2.2.3.3. The CAR and psychological factors. An elevated CAR is typically associated with either situational stress or a vulnerability to stressors and negative emotions (for review, see Fries et al., 2009). Indeed, an increased CAR has been positively associated with situational factors including chronic stress and worry (Wüst et al., 2000a; Zoccola et al., 2011), as well as work overload and job strain (Schulz, Kirschbaum, Pruessner, & Hellhammer, 1998; Steptoe, Cropley, Griffith, & Kirschbaum, 2000). Further, a recent study found elevated morning cortisol levels among individuals suffering from Alzheimer's disease as well as their caregivers (Wahbeh, Kishiyama, Zajdel, & Oken, 2008). However, there are some mixed findings in the literature with some research indicating a negative association between CAR and psychological states such as anticipatory anxiety (Walker et al., 2011).

In addition to situational factors, psychological traits have also been linked to the CAR. A positive association between morning cortisol levels and trait anxiety was reported in a study of university students and young adolescents (Lai & Wan, 2009). Similarly, negative psychological traits including neuroticism (Portella et al., 2005), trait negative affect (Polk, Cohen, Doyle, Skoner, & Kirschbaum, 2005), and harm avoidance (Rademaker, Kleber, Geuze, & Vermetten, 2009) have also been linked to an elevated cortisol awakening pattern. Similarly, Greaves et al. (2007) reported a positive correlation between CAR levels and persistent anxiety problems among young adolescents (aged 10-12 years).

While an elevated CAR appears to be linked to psychological stress and negative affectivity, a different pattern tends to emerge for severe psychological disorders such as PTSD and clinical depression (Huber, Issa, Schik, & Wolf, 2006; Vythilingam et al., 2010). A flattened CAR has been linked to PTSD (Lauc, Zvonar, Vuksic-Mihaljevic, & Flögel, 2004; Neylan et al., 2005; Wessa et al., 2006; Yehuda, 2006) and severe depression (Huber et al., 2006), a pattern that suggests that severe chronic stress and psychological trauma may eventually result in hypoactivity of the HPA axis. Further, the positive association between depressive symptomatology and CAR found in a study by Pruessner et al. (2003) was stronger when clinically depressed participants were excluded from the data analyses. That is, the clinically depressed participants' cortisol levels were lower than subclinical participants, thus weakening the correlation reported for the full sample.

2.2.3.4. The CAR and health conditions. Hypoactivity of the HPA axis reflects a neuroendocrine system that is not appropriately adapting to day-to-day environmental and psychological demands. A blunted CAR (i.e., limited rise in cortisol levels following awakening) may therefore be a health risk, possibly for the development of both psychiatric

and physical health problems (e.g., Bruehl et al., 2009; Hellhammer, Schlotz, Stone, Pirke, & Hellhammer, 2004).

In addition to various psychological conditions and traits, the CAR has been associated with chronic and acute health conditions (Clow et al., 2010; Clow et al., 2004; Kudielka & Kirschbaum, 2003). An elevated CAR has been observed among patients with coronary artery disease (Bhattacharyya et al., 2008; Whitehead, Perkins-Porras, Strike, Magid, & Steptoe, 2007), as well as individuals suffering from a chronic health condition (Kudielka & Kirschbaum, 2003). Additionally, an elevated response has been found among women who suffer from musculoskeletal pain (Riva, Mork, Westgaard, & Lundberg, 2012). Indeed, there is evidence that the CAR may be an important physiological agent involved in both physical health and psychological well-being (Clow et al., 2010; Lovallo, 2011). However, the field is currently in its infancy, and therefore, further research on the CAR in relation to various health and psychological conditions will help elucidate its role in affecting well-being.

2.2.4. Chronic stress and health. Allostatic load has become an inclusive term, one that encompasses a broad range of physiological parameters that, taken together, measure the bodily “wear and tear” that results from chronic stress (Juster et al., 2010). In assessing allostatic load, biological parameters (e.g., stress hormone levels, changes in brain functioning) are often investigated in conjunction with the occurrence of illness or health problems (e.g., cardiovascular disease, decline in cognitive or physical functioning; Evans, 2003; Johnston-Brooks, Lewis, Evans, & Whalen, 1998; Mair, Cutchin, & Kristen Peek, 2011; Seeman et al., 2001). While allostatic load is a relatively new term, evidence for the detrimental health effects of chronic stress has been mounting over the past few decades.

2.2.4.1. Chronic stress and the immune system. The study of the effects of stress on the functioning of the immune system is often referred to as ‘psychoneuroimmunology’ (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). The central nervous system, nervous system, endocrine system, and immune system interact to impact bodily responses to viruses and other foreign invaders. Sympathetic nerve fibres have direct connections with immune system organs including the thymus, bone marrow, and spleen (Segerstrom & Miller, 2004). Further, stress hormones directly communicate with immune cells, impacting their functioning (Gouin, Hantsoo, & Kiecolt-Glaser, 2008).

All lymphocytes have adrenergic receptors that have the ability to bind with adrenaline and noradrenaline (Segerstrom & Miller, 2004). These catecholamines work in conjunction with glucocorticoids in impacting immune function. Specifically, catecholamines have been linked to suppression of T-lymphocytes, a white blood cell regularly on the frontline against foreign invaders (Elenkov, 2008). The distribution and function of B cells, natural killer cells, neutrophils and macrophages – white blood cells that identify and destroy foreign substances or tumour cells – are also affected considerably by catecholamines (Elenkov, 2008). Chronic elevations of catecholamines may be particularly disruptive to immune system function and regulation (Padgett & Glaser, 2003).

In addition to adrenaline and noradrenaline, glucocorticoids (e.g., cortisol) also have the capacity to downregulate immune functioning. Glucocorticoids regulate inflammation by acting on cytokines, the proteins involved in intercellular communication (Kalat, 2007). Specifically, glucocorticoids inhibit the production of inflammatory cytokines, and promote the production of anti-inflammatory cytokines (Buford & Willoughby, 2008).

Psychosocial stressors activate both the HPA axis and SAM system as part of the stress response. The chronic activation of these systems has been linked to health outcomes

– including depression, cardiovascular disease and the progression of HIV/AIDS (Cohen et al., 2007). Stress from university exams and interpersonal stress from marital issues have also been linked to decreased wound healing (Kiecolt-Glaser et al., 2005; Marucha, Kiecolt-Glaser, & Favagehi, 1998). Further, caregiving for a chronically-ill spouse, a life circumstance characterised by considerable psychosocial stress, has been linked to decreased effectiveness of vaccinations among caregivers (Glaser, Kiecolt-Glaser, Malarkey, & Sheridan, 1998). These examples provide support for the immunosuppression model of stress – the notion that stress down-regulates immune system functions leading to susceptibility to illness.

Chronic stress is not only linked to immunosuppression, but also to autoimmune diseases characterised by chronic inflammation including arthritis, irritable bowel syndrome, and Crohn's disease (Barnes & Adcock, 2009). As such, Miller, Cohen, and Ritchey (2002) developed a complementary model to explain the link between stress and inflammatory diseases. The glucocorticoid resistance model states chronic stress diminishes the immune system's sensitivity to the anti-inflammatory properties of glucocorticoids. Chronically elevated levels of glucocorticoids (e.g., cortisol) lead to the downregulation of GRs on lymphocytes, which, in turn, inhibit anti-inflammatory effects.

Supporting this model, Miller et al. (2002) found that the ability of cortisol to inhibit the production of pro-inflammatory cytokines was reduced among parents of children with cancer (high stress condition) compared to controls. Additionally, longitudinal research has also demonstrated a relationship between chronic stress (caregiving for a spouse with dementia) and elevated levels of pro-inflammatory cytokines (Kiecolt-Glaser et al., 2003). The relationship between psychological stress and immune

function is clearly complex, involving both inhibition and activation of various immune system components.

2.2.4.2. Chronic stress and the brain. While various regions of the brain become activated as a result of stressors, there are three main areas of the brain that are closely involved with the stress response: the amygdala, hippocampus, and the prefrontal cortex. The amygdala is the emotion-centre of the brain, governing emotions such as fear and anxiety (McEwen & Gianaros, 2011). While the amygdala is involved in initiating the stress response, the hippocampus plays a major role in the termination of the stress response (McEwen & Gianaros, 2011). The prefrontal cortex plays a regulatory role in the more complex psychological factors involved in stress, namely decision making and coping with the stressor.

The amygdala, a brain region that does not reach full development until the late 20s (Lupien, McEwen, Gunnar, & Heim, 2009), can be affected considerably by chronic stress. Chronic stress has been found to result in enhanced excitability of amygdala neurons (Rosenkranz, Venheim, & Padival, 2010) and overall amygdala reactivity (McEwen, 2007). Stress-induced increases in neuronal connections in the amygdala result in structural changes and increased volume (Lupien et al., 2009). These changes to the amygdala have been implicated in alterations in cognitive performance as well as vulnerability to pathological anxiety (Roozendaal, McEwen, & Chattarji, 2009).

The hippocampus is arguably the brain region most affected by chronic stress. As previously discussed in reference to the ‘glucocorticoid cascade hypothesis’ (see p. 13), the structure of the hippocampus is particularly malleable to environmental demands and chronic stress (Sapolsky et al., 1986). Chronic stress has been found to both temporarily

and permanently alter hippocampal circuitry, thus affecting both memory function and HPA regulation (McEwen & Gianaros, 2011).

The prefrontal cortex, the brain region involved in higher cognitive function (e.g., working memory, executive functioning), is particularly important in appraising and coping with stressors (McEwen & Gianaros, 2011). There is evidence to suggest that chronic stress can induce considerable changes in the structure and complexity of the prefrontal cortex (Lupien et al., 2009). Specifically, chronic elevations of cortisol have been linked to reduction in the volume and synaptic connection complexity of the prefrontal cortex (McEwen & Gianaros, 2011). These structural changes can lead to impairments in cognitive flexibility and decision making (McEwen & Gianaros, 2011) – alterations that could impede effective coping with stressors. In sum, the plasticity of the brain regions involved in stress – the amygdala, hippocampus, and prefrontal cortex – may be adaptive in the short-term, but also increase their susceptibility to stress-induced damage (McEwen, 2007).

2.3. Cognitive Theories of Stress

As reviewed in this chapter, chronic stress is not only implicated in acute illness, but also in long-term pathology and disease development (McEwen, 2007, 2008). To complement this discussion, it is important to note that the relationship between stress and health is likely not unidirectional, but also involves an individual's reaction to the stressor. As such, cognitive theories of stress are discussed, in part, to bring to light the role of the individual's perceptions in dealing with stressors.

2.3.1. Appraisal theory. Appraisal theory was developed by Lazarus and Folkman (1984) to help explain individual differences in responding to stressors. Stress, according to Lazarus and Folkman (1984), is produced from a complex interplay between the demands

of the environment and the coping resources of the individual. Lazarus and Folkman (1984) define psychological stress as a “particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being” (p. 19).

Central to the theory is the concept of appraisal – an individual’s assessment of the surrounding environment and his or her capability to deal with the environment. According to appraisal theory, the meaning of a stressor is determined by both primary and secondary appraisals (Lazarus & Folkman, 1984). The primary appraisal is the initial evaluation of the stressor or event (e.g., sighting a bear while hiking), and the secondary appraisal involves the evaluation of options for coping with the stressor (e.g., the ability to run away, radio for help, make lots of noise). Whether or not the environmental demand (stressor) leads to psychological or physical symptoms of stress depends on the individual’s appraisal, coping strategies, and reappraisal of the situation.

Lazarus and Folkman (1984) further identified three subtypes of stress-eliciting situations. Stressful situations can be appraised as a harm/loss, threat, or challenge. During harm/loss, the damage, be it physical (e.g., pain) or psychological (e.g., embarrassment), has already occurred. For threats and challenges, the perception of these events is future-oriented. During a threat, the harm is anticipated, and as such, the individual works towards preemptive strategies to reduce the ill-effects of the stressor (e.g., during divorce, in anticipation of the anxiety and sadness associated with adapting to new single life, an individual may seek out friends for continual support during the transition period). A challenge occurs when an individual believes he or she can confidently and effectively cope with the stressor.

Secondary appraisals refer to coping strategies, of which Lazarus and Folkman (1984) initially identified two types – problem-focused coping and emotion-focused coping. Problem-focused coping deals with the interaction between the individual and environment and possible alterations in the relationship to ease the stress (Lazarus & Folkman, 1984). For example, if you come across a dangerous animal while hiking (e.g., bear), problem-focused coping may involve strategies to create considerable distance between oneself and the animal.

Alternatively, emotion-focused coping was originally described as the regulation of emotions resulting from the stressor. This may include reducing distress and reframing the problem. However, emotion-focused coping may also include problematic emotion-based cognitions, such as self-blame and magnification of the problem (Collins, Sorrocco, Hala, Miller, & Lovullo, 2003).

Additionally, Folkman et al. (1986) later identified avoidance coping as another method by which individuals cope with a stressor. This may involve avoiding the stressor through ‘escape-avoidance’ strategies (e.g., wishing the problem would disappear, avoiding people). Similar to emotion-focused coping, this method is largely regarded as maladaptive because such strategies do not involve the employment of problem-solving behaviours to effectively deal with the stressor (e.g., Rippeto & Rogers, 1987).

2.3.2. Cognitive activation theory of stress. The Cognitive Activation Theory of Stress (CATS) proposes that the perception of stress is based on the discrepancy between what an individual expects in a given situation and the actual experience (Ursin & Eriksen, 2004). There are four aspects to a stressful situation: (1) the stress stimulus (stressor), (2) the stress experience, (3) the stress response, and (4) the person’s experience. The perception of a stress stimulus depends on a cognitive process similar to Lazarus and

Folkman's (1984) primary appraisal; it involves the individual's perception of the stressor based on his or her previous experience with the stress stimulus. The stress experience involves the individual labelling the stimulus as negative or threatening. The stress response involves physiological arousal and increased wakefulness, akin to Selye's (1946) alarm phase of the General Adaptation Syndrome. Lastly, the person's experience of the stressor involves transmitting information back to the brain and readjusting outcome expectancies based on the experience.

Based on the experience of a stressor, an individual develops a positive, negative or no outcome expectancy, each relating to cognitive responses – coping, hopelessness, and helplessness respectively. Ursin and Eriksen (2004) note that positive outcome expectancies result in decreased arousal. That is, effectively coping with a stress stimulus will decrease its physiological and psychological toll on the body. Based on the model, health is threatened by high arousal levels (Eriksen & Ursin, 2004; Ursin & Eriksen, 2010), following a similar framework as the allostatic load model (McEwen, 1998b). More specifically, uncertain and uncontrollable stress stimuli (stressors) produce high arousal (Ursin & Eriksen, 2010), and thus are the greatest health risk.

The CATS framework has been used to explain the psychological processes underlying subjective health complaints (Ursin & Eriksen, 2010). Subjective health complaints may be the result of increased physiological sensitisation, and an abnormal activation of the cognitive networks related to pain and illness (Ursin & Eriksen, 2010). Through both the cognitive and psychological components, the CATS framework helps explain the individual differences in subjective health complaints and other stress-related health issues.

Chapter 3: Literature Review – The Effects of Environmental Noise Exposure

3.1. Environmental Stressors

An environmental stressor is anything in the physical environment that is unwanted by an individual (Evans, 1984). Public health and environmental psychology research continues to highlight the important role of the environment in health and well-being (Adler & Newman, 2002; Cohen, Evans, Stokols, & Krantz, 1986; Evans & Cohen, 1987; Hill, Shepherd, Welch, Dirks, & McBride, 2012; Stokols, 1992; Taylor & Repetti, 1997). Major environmental stressors, particularly those affecting residents in urban environments, are noise, crowding, and air pollution. The effects of environmental stressors are often compounded due to the relationship among the stressors themselves. For example, individuals who are exposed to noise in their residential community are likely to be exposed to air pollution as well, especially if road traffic is the primary cause of both of these environmental stressors. However, Cohen (1985) argues that psychological factors such as attitudes towards the environmental stressor best predict the reaction and subsequent effects (e.g., annoyance, distraction from task performance).

Campbell (1983) coined the term ‘ambient stressors’ to describe environmental stressors that typically exist in the background, but which are also chronic and perceptible. Ambient stressors are also regarded as ‘non-urgent’ because they usually do not cause immediate harm to residents (Campbell, 1983). However, due to sustained emotional and physiological arousal, chronic exposure and continuous attempts to cope with an ambient stressor can put an individual at risk for health problems (Baum, Singer, & Baum, 1984). Indeed, there is clear evidence for the impact of environmental stressors on allostatic load, health, and well-being (e.g., Evans, 2003; Hill et al., 2012; Mair et al., 2011).

Noise has been identified by the World Health Organization (WHO) as one of the fastest growing environmental stressors (Berglund, Lindvall, & Schwela, 1999).

Urbanisation and industrialisation have led to considerable increases in noise exposure over the past century (Goines & Hagler, 2007). Although noise exposure has long been identified as a public nuisance (King & Davis, 2003), relatively little attention has been given to the issue of noise exposure in health promotion policy development (Moudon, 2009; Staples, 1996). Because of its negative impact on health and well-being, and its continual growth in modern society (Goines & Hagler, 2007), noise remains an important stressor to examine in public health research.

3.2. Sound versus Noise

Sound is defined simply as the change in air pressure levels as detected by the ear, whereas noise is defined as unwanted sound (Catlin, 1965). Cohen and Weinstein (1981) suggested that any sound that is potentially damaging to one's physiology can be termed 'noise'. In essence, it is the assumption that if a sound can be potentially harmful, it is unwanted. Therefore, there is no physical difference between sound and noise, but rather, this difference is determined by the perception of the individual or whether the sound could be physiologically damaging. As such, hearing loss is termed 'noise-induced hearing loss' regardless of whether the soundscape that contributed to the damage to the auditory system was wanted or not. The main acoustic properties of sound are the frequency (pitch) and the sound pressure level (SPL; loudness).

3.2.1. Frequency. The frequency of a sound refers to the number of vibrations per second, measured in Hertz (Hz). The range of hearing for humans spans from 20-20000 Hz, though we are most sensitive to frequencies between 1000 to 5000 Hz (Jacko & Sears, 2003). Thus, a high-pitched tone (e.g., squeak) has a frequency of approximately 4000 Hz,

and a low-pitched tone (e.g., washing machine) would have a frequency close to 200 Hz (Passchier-Vermeer & Passchier, 2000). Low frequency noise (< 100 Hz) is regarded as particularly troublesome to health and well-being. Specifically, at low frequencies, noise is more likely to annoy and interrupt cognitive tasks such as reading (Broner, 1978; Persson Waye, Bengtsson, Kjellberg, & Benton, 2001), and cause headaches among other health complaints (Berglund, Hassmen, & Soames Job, 1996; Leventhall, 2009). Further, low frequency noise is increasing in prevalence in both home and office environments, and therefore is of particular concern from a public health perspective (Berglund et al., 1999; Bolin, Bluhm, Eriksson, & Nilsson, 2011; Broner, 1978; Pedersen & Persson Waye, 2008; Persson & Rylander, 1988; Persson Waye, Agge, Clow, & Hucklebridge, 2004; Persson Waye et al., 2001).

3.2.2. Sound pressure level. SPL specifically refers to the deviation of the sound pressure from atmospheric pressure (Passchier-Vermeer & Passchier, 2000). The SPL is expressed in decibels (dB SPL), a logarithmic scale that quantifies the intensity of the sound. Because the human ear is less sensitive to low frequency sounds than high frequency sounds, sound pressure levels in scientific and epidemiological studies are often expressed using an A-weighting dB scale (dB(A)). The A-weighting system places less emphasis on lower frequency sounds than mid to high frequency sounds, simply because the human ear is not as sensitive to them (Berglund et al., 1999). There is also a C-weighting correction for high intensity sounds (e.g., explosions), but it is far less common in scientific studies (King & Davis, 2003).

3.2.3. Evolution of auditory reflexes. The auditory system has two primary functions: (1) for communication, and (2) as a warning system to identify sources of danger in the surrounding environment (Spreng, 2004). The hearing system has two reflexes: the

orienting reflex and the startle reflex (Westman & Walters, 1981). The orienting response simply refers to the innate reflex that involves the eyes orienting toward a sound source. The startle reflex refers to the sudden contraction of limb muscles and the middle ear muscles in response to a loud or unexpected sound, a response usually occurring in less than one second from exposure to the sound (Westman & Walters, 1981).

Additionally, a third innate auditory reaction is the defence response, which is regarded as an extension of the orienting and startle reflexes (Westman & Walters, 1981). Specifically, this reaction is, in essence, a stress response involving an increase in blood pressure and heart rate, and the release of stress hormones (Westman & Walters, 1981). For example, an animal may be alerted to a sudden noise in the bush (orienting and startle reflexes activated), which then prompts fleeing from the noise source (defence response). The defence response to sound occurs typically between 70 and 120 dB, but can also vary according to the significance of the sound source (i.e., whether the source signifies a threat; Westman & Walters, 1981). Westman and Walters (1981) also discussed that chronic noise exposure could potentially elicit a defensive response analogous to the General Adaptation Syndrome (alarm, resistance, exhaustion), which has the capacity to put an animal at risk for development of disease (Selye, 1946).

3.3. Health Effects of Noise Exposure

Early research in the field of noise and health largely focused on the effects of excessive noise on the auditory system (e.g., Farr, 1967; Lawrence, Gonzalez, & Hawkins, 1967). Noise-induced hearing loss is, indeed, a major health problem and remains a prevalent irreversible occupational hazard (Berglund et al., 1999). However, noise-induced hearing damage is only one of the many health effects associated with noise. Over the past 40 years, a myriad of public health and psychoacoustic researchers have investigated the

various non-auditory health effects associated with noise. It has become widely accepted that noise can have a negative impact on health and well-being, communication, sleep, and overall quality of life (Babisch, 2003; Clark & Stansfeld, 2007; Clausen, Christensen, Lund, & Kristiansen, 2009; Dratva et al., 2010; Ising & Kruppa, 2004; Jonsson & Hansson, 1977; King & Davis, 2003; Lundberg, 1999; Morell, Taylor, & Lyle, 1997). Further, there has also been an increase in research and evidence for the negative physiological changes linked to both acute and chronic noise exposure (Evans, Lercher, Meis, Ising, & Kofler, 2001; Rylander, 2004, 2006).

3.3.1. Sleep disturbance. One of the most pronounced and important health effects of noise is sleep disturbance (Fyhri & Aasvang, 2010; Lercher et al., 2010; Miedema & Vos, 2007; Ouis, 2002; Stansfeld, Haines, & Brown, 2000; Wilkinson, 1984). Sleep disturbance is a broad term describing any problem associated with sleeping including: difficulty getting to sleep, waking throughout the night, and decreased quantity or quality of sleep (Langdon & Buller, 1977; Zaharna & Guilleminault, 2010). Sleep disturbance is linked to poor daily functioning due to tiredness and cognitive deficits, including memory problems and a decreased ability to focus (Killgore, 2010). Poor sleep is also acknowledged as a significant risk factor for the development of some health problems (e.g., obesity, hypertension, diabetes; Lockley, 2010; Marshall & Stranges, 2010; World Health Organization, 2009), which, in turn, could also be linked to physiological changes to the immune system (Wright, Erblich, Valdimarsdottir, & Bovbjerg, 2007).

Although still the subject of debate, it is clear that sleep helps us function in our daily lives through the conservation of energy, consolidation of memory, and restoration of cellular processes (Mignot, 2008; Siegel, 2005). Sleep involves a change in consciousness, reflected in changes in the electrophysiology of the brain (Lockley, 2010). Each night, an

individual experiences two distinct types of sleep – non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep (sometimes referred to as paradoxical sleep; Luppi et al., 2012). When people fall asleep, they transition through the stages of NREM sleep (N1, N2, N3), which is marked by parasympathetic dominance, and thus, a lower body temperature and a slower breathing rate. In contrast, REM sleep involves more active physiological processes including faster breathing, faster heart rate, and increased blood flow to the brain (Lockley, 2010). Each night, we experience approximately 4-5 sleep cycles, alternating between NREM sleep and REM sleep every 45 minutes. A healthy sleep cycle is characterised by twenty-five percent REM sleep – the stage where dreaming most often occurs (Lockley, 2010).

While the main purpose of REM sleep remains unclear (Stickgold, 2011), both REM and NREM sleep are believed to play an important role in memory consolidation and restoration (Bonnet, 2005; Siegel, 2001). NREM sleep is divided into 3 stages – N1, N2 and N3, with the first two stages referring to lighter sleep, and the third stage the deepest stage of sleep (Lockley, 2010). The N3 stage involves slow wave sleep, the deepest sleep pattern, believed to be particularly important for homeostasis and restoration of the body (Lockley, 2010). Sleep disturbance throughout the night can lead to considerable deprivation of slow wave sleep, which is believed to be a risk factor in the development of chronic illnesses such as diabetes (Dijk, 2008) and hypertension (Fung et al., 2011).

Human sleep is not restorative if it is frequently interrupted (Bonnet, 2005). There have been an array of studies – both epidemiological and lab-based – exploring the relationship between noise and sleep, and the link to daily functioning and physiological changes (e.g., Aasvang, Moum, & Engdahl, 2008; Aasvang, Overland, Ursin, & Moum, 2011; Arber, Bote, & Meadows, 2009; Basner, Samel, & Isermann, 2006; Griefahn, Marks,

& Robens, 2006; Hume, 2010; Kawada, 2011; Lercher et al., 2010; Marks & Griefahn, 2007). Environmental noise exposure can impact sleep in a variety of ways: increased sleep latency time, reduced sleep quantity and quality, changes in sleep stages, and increased night-time body movement (Passchier-Vermeer & Passchier, 2000).

Griefahn, Marks and Robens (2006) used an experimental design to explore the impact of aircraft, rail and road traffic noise on sleep quality and after-effects including reaction time to tasks and overall performance on tasks. Participants were exposed to each type of noise in a sleep laboratory, and were monitored and tested during the mornings. Aircraft, rail and road traffic noise were linked to similar amounts of decreased subjective sleep quality and increased reaction time. However, slow wave sleep appeared to be most negatively affected by rail noise, suggesting that rail noise may be particularly detrimental to objective sleep quality.

A tightly controlled lab study on rats found a relationship between noise exposure and decreased amounts of slow wave sleep, which, in turn, was linked to an increase in locomotor reactivity to novelty (Rabat, Bouyer, Aran, Le Moal, & Mayo, 2005). Following exposure to environmental noise for nine consecutive days, the rats experienced a permanent change in sleep patterns (i.e., disturbance of slow wave and paradoxical sleep). An additional interesting result of the study was that, based on their responses to environmental noise, the rats could be classified as ‘vulnerable’ or ‘resistant’ to environmental noise. The ‘vulnerability’ differences observed among the rats suggest that biological factors might also cause the individual differences in noise reactions observed in humans (i.e., noise sensitivity), in addition to the wide variety of psychosocial influences on noise reactions (Guski, 1999).

While laboratory studies of human participants offer the benefits of a controlled experiment, generalisations of the results are often limited because of small sample size (e.g., less than 40 participants) and lack of ecological validity. Supplementing laboratory research, many epidemiological and community-based studies have explored the relationship between noise and sleep disturbance (e.g., Langdon & Buller, 1977; Muzet, 2007; Nivison & Endresen, 1993). Similar to laboratory studies, noise exposure in community-based studies has been linked to both primary effects such as decreased sleep quality, prolonged sleep latency and increased awakenings throughout the night, as well as after-effects including negative moods and decreased cognitive performance ability (Berglund & Lindvall, 1995; Eberhardt, 1988; Eberhardt, Strale, & Berlin, 1987; Griefahn, 2002; Job, 1996; Muzet, 2007). As with laboratory studies, the effects of noise on sleep disturbance are dependent on a variety of factors including the noise source, the nature of the noise, and individual differences among the participants (Pearsons, Barber, Tabachnick, & Fidell, 1995).

Noise exposure can also disturb sleep through the induction of physiological changes (Griefahn, Brode, Marks, & Basner, 2008; Pirrera, De Valck, & Cluydts, 2010). Intermittent or ambient noise can cause a stress response, leading to increased blood pressure and the release of stress hormones. During sleep, the threshold for the activation of the stress response is much lower than when a person is awake (Zaharna & Guilleminault, 2010). Therefore, while a person may not report sleep disturbance or annoyance in reference to his or her noise-exposed environment, the noise could be inducing physiological changes during the night, which, in turn, has potential negative health implications (Muzet, 2007).

Although the long-term health effects of noise exposure on sleep are still not clear, further investigations, including longitudinal studies, will help to further elucidate the mechanisms involved in the relationship. Vallet, Gagneux, Blanchet, Favre and Labiale (1983) found evidence for long-term sleep disturbance as a result of traffic noise. Their experimental research was conducted over 4 years, and the results suggested that long-term exposure to environmental noise may lead to deficits in N3 (deep) sleep among young adults, while REM sleep may be affected in the elderly. Similarly, Eberhardt (1988) reported that participants who had experienced years of night-time noise exposure did not exhibit habituation, as assessed by the frequency of arousal reactions throughout the night and lack of improvements in sleeping patterns (measured by an electroencephalogram) with reduction of sound levels. Therefore, even after years of night-time noise exposure, physiological habituation was not evident.

There are various pathways by which noise-related sleep disturbance could lead to health detriments, though the relationship among the variables has not yet been confirmed. As mentioned previously, obtaining an adequate amount of good quality sleep is important for maintaining good health (Pirrera et al., 2010; Zaharna & Guilleminault, 2010). Alterations in sleep schedules, as well as diminished quantity or quality of sleep due to noise exposure could lead to health-risk related physiological changes, such as increased blood pressure (Haralabidis et al., 2008; McEwen, 2006). Sleep disturbance also leads to performance deficits and decreased mood, both of which could be ongoing stressors for an individual, which, in turn, could lead to stress-related physiological changes and subsequent health problems (McEwen, 2006). Given the wide range of negative health and cognitive effects of sleep disturbance (Bonnet, 2005; Lockley, 2010), the influence of noise on sleep quality remains an important research avenue.

3.3.2. Noise annoyance. Noise annoyance is regarded as one of the most pervasive negative effects of environmental noise exposure (Bluhm, Nordling, & Berglind, 2004; Cohen & Weinstein, 1981; Miedema & Vos, 1999; Ouis, 2001; Vallet, Maurin, Page, Favre, & Pachiaudi, 1978; Weinstein, 1982). Passchier-Vermeer and Passchier (2000) define noise annoyance as “the feeling of resentment, displeasure, discomfort, dissatisfaction, or offense when noise interferes with an individual’s thoughts, feelings or actions” (p. 126). Some research suggests a dose-response relationship between noise exposure and annoyance (Babisch et al., 2009; Bluhm et al., 2004; Rylander, 2006; Schultz, 1978; Tarnopolsky & Morton-Williams, 1980), though this relationship has not been replicated in other studies (de Jong, 1990; Hall, 1984). Guski (1999) proposed that approximately one third of the variance in noise annoyance can be attributed to environmental noise, while another third of the variance is likely related to personal or social variables⁴.

3.3.2.1. Psychological factors in noise annoyance. A variety of psychosocial and non-acoustical factors have been linked to noise annoyance (Kroesen, Molin, & Van Wee, 2010). Noise sensitivity, a personality trait influencing a person’s reaction to noise, has been positively associated with noise annoyance (Guski, 1999; Jakovljević, Paunovic, & Belojević, 2009; Kjellberg, Landstrom, Tesarz, Soderberg, & Akerlund, 1996). Even after controlling for noise exposure, noise sensitivity has been identified as an independent predictor of noise annoyance (van Kamp et al., 2004). Therefore, noise sensitivity is clearly

⁴ Guski (1999) did not specify what variables might explain the remaining third of variance in noise annoyance.

an important psychological factor related to noise annoyance. The relationship between noise sensitivity and noise annoyance is further discussed in Chapter 4 (p. 79).

Fear of the noise source has been acknowledged as another important factor in the relationship between environmental noise and annoyance (Fields, 1993; Miedema & Vos, 1999; Stallen, 1999). This relationship may be most relevant to perceptions of aircraft noise; studies on aircraft noise exposure have found that fear of an aircraft crashing in their neighbourhood is associated with participants' ratings of noise annoyance (Graeven, 1974; Guski, 1999).

Similarly, noise annoyance is also elevated among those individuals who are most concerned about the psychological and health effects of noise exposure (Morell et al., 1997) as well as the potential damage to environmental quality (Michaud, Keith, & McMurchy, 2008; Staples, Cornelius, & Gibbs, 1999). It is possible that the relationship between noise annoyance and these psychological factors is, at least partially, explained by overall concern for the well-being of the community, or possibly related to underlying negative emotions or anxiety leading to both fear and annoyance reactions. Regardless of the precise influence of each psychological factor, it is clear that the impact of noise annoyance can be influenced by a variety of sources in addition to the actual acoustic properties of the noise.

3.3.2.2. Social factors in noise annoyance. In her social-psychological model of noise annoyance, Maris (2008) argued that social factors, in addition to psychological factors and physical features of the noise source, have considerable impact on annoyance. Similarly, Guski (1999) discussed that a variety of social influences can impact noise annoyance including perceived misfeasance and trust in the authorities, and general evaluations of the noise source.

Through the use of laboratory experiments, Maris et al. (2007) demonstrated that perceived misfeasance and unfairness significantly influence annoyance reactions to noise. Participants were randomly assigned to either the ‘unfair’ or ‘neutral’ condition in the experiment. In the ‘unfair’ condition, participants were exposed to aircraft noise (50 to 70 dB(A)) despite being told they could listen to the sound of their choice (e.g., bird song, radio sound, or aircraft sound). Those in the neutral condition were told they would be listening to the aircraft noise. As hypothesised, participants in the ‘unfair’ procedure reported greater annoyance than those in the ‘neutral’ condition.

Similar results have been observed in field studies, including those exploring perceptions of wind turbine farms and airport development (Staples et al., 1999). The development of wind turbine farms has become a politically-relevant and timely issue because of the concern with establishing renewable energy resources in favour of less sustainable (e.g., coal) and more potentially dangerous options (e.g., nuclear power). Despite the general public’s relatively favourable perception of wind turbines, the low frequency noise emitted from them causes nearby residents considerable annoyance and disturbance (Pedersen & Persson Waye, Bolin et al., 2011; 2004; Persson Waye & Öhrström, 2002; Shepherd, McBride, Welch, Dirks, & Hill, 2011). Further, perceptions of loss of control and negative interactions with wind turbine authorities (e.g., energy companies) have also been linked to increased noise annoyance (Pedersen, Hallberg, & Waye, 2007).

Related issues have also been raised with respect to airport development and management of aircraft noise levels (Guski, 1999; Hatfield et al., 2001; Kroesen, Molin, & van Wee, 2011; Staples et al., 1999). The ‘importance of the source’ has been cited as a moderator of the relationship between aircraft noise exposure and annoyance (Flindell &

Stallen, 1999; Guski, 1999), a variable that may have particular relevance to wind turbine noise research as well.

Specifically, individuals who perceive the noise source as important are less likely to perceive annoyance. Further, some research suggests that people weigh the negative environmental effects and possible economic benefits of the noise source in evaluating the importance of the noise source (Kroesen et al., 2011; Staples et al., 1999). Therefore, an individual may be less likely to report noise annoyance due to a nearby airport if he or she believes that it helps to provide economic stability for the community.

Many of the social and political factors that can influence annoyance are largely related to perceptions of control over one's environment, another important determinant of noise reactions (e.g., Glass & Singer, 1972b; Glass, Singer, Leonard, Krantz, & Cohen, 1973). While noise annoyance typically increases with noise exposure (Rylander, Bjorkman, Ahrlin, Arntzen, & Solberg, 1986), social and psychological factors interact to influence the relationship, and therefore have important implications for noise policy development and implementation.

3.3.2.3. Demographic factors and noise annoyance. In addition to social and psychological factors, some studies have explored demographic factors associated with noise annoyance (e.g., Fields, 1993; Fyhri & Klæboe, 2006; Michaud, Keith, & McMurchy, 2005; Miedema & Vos, 1999). Fields (1993) conducted analyses on an aggregate of 36 studies, and although he found limited evidence for the role of demographic factors, more recent studies have reported contrary findings. In community-based studies, women have reported being more annoyed than men by environmental noise (Dratva et al., 2010; Michaud et al., 2005), a finding that some have suggested could be attributed to the greater amount of time women spend in the home compared to men (e.g., Fields, 1993; Nivison &

Endresen, 1993; Willich, Wegscheider, Stallmann, & Keil, 2006). However, the influence of social or biological factors that may be involved in this relationship remains unclear.

Michaud et al. (2005) found an inverse U-shaped relationship between income and annoyance, with people in the middle-income bracket (\$20,000-\$49,999 Canadian dollars) being more likely to report annoyance than individuals in lower or higher income brackets. Although this relationship may be complex and difficult to explain, it is possible that individuals of middle or high socioeconomic status may have higher expectations for quiet (Michaud et al., 2005), but only individuals in the higher income brackets are able to “free” themselves of noise exposure (e.g., purchasing or renting a home away from noise source; Fyhri & Klæboe, 2006). Both environmental exposure to noise (e.g., location of residence) and psychological factors (e.g., expectations) may be important moderators in the relationship between income and noise annoyance.

An inverted U-shaped pattern has also been reported for the relationship between noise annoyance and age (Miedema & Vos, 1999; Van Gerven, Vos, Van Boxtel, Janssen, & Miedema, 2009). Miedema and Vos (1999) reported that both the relatively young and the relatively old are less annoyed by environmental noise than middle-aged individuals. Similarly, using aggregate data from international studies, Van Gerven et al. (2009) reported that noise annoyance was greatest among individuals in the middle-age bracket, peaking around 45 years. Although Miedema and Vos (1999) suggest that individuals in the most elderly group may be less annoyed than younger individuals because of decreased acuity of senses (i.e., hearing loss), it is also possible that those highly annoyed individuals may be less likely to survive to late adulthood (Chida & Steptoe, 2008; Ndrepepa & Twardella, 2011).

3.3.2.4. Noise annoyance and health. Although noise annoyance is widely regarded as a negative health outcome in and of itself (Michaud et al., 2008; Paunovic et al., 2009), it has also been identified as a risk factor for the development of cardiovascular disease (Babisch, 2003; Babisch, Beule, Schust, Kersten, & Ising, 2005; Belojević & Saric-Tanaskovic, 2002; Sobotova, Jurkovicova, Stefanikova, Sevcikova, & Aghova, 2010). A recent meta-analysis conducted on eight studies revealed that noise annoyance was associated with an increased risk of hypertension, though there is no conclusive relationship between noise annoyance and ischemic heart disease (Ndrepepa & Twardella, 2011). Therefore, individuals who frequently or chronically experience noise annoyance may be straining their cardiovascular system, which, in turn, puts them at risk for the development of hypertension.

In addition to being a potential risk factor for the development of cardiovascular problems, noise annoyance has also been linked to other health problems including loss of sleep and physical ailments (Fooladi, 2012; Fyhri & Aasvang, 2010; Wallenius, 2004). Noise annoyance can lead to sleep disturbance, but the relationship can indeed be bidirectional; loss of sleep can lead to noise annoyance as well (Job, 1996). Additionally, compared to less annoyed individuals, highly annoyed participants are more likely to report greater gastric and mental health complaints (Nivison & Endresen, 1993; Ohrström, 2004), as well as respiratory health issues and migraines (Niemann et al., 2006).

Noise annoyance has also been linked to an increased risk for depression and reduced health-related quality of life (Dratva et al., 2010; Shepherd, Welch, Dirks, & Mathews, 2010). It may also worsen pre-existing mental health issues (Cohen, Glass, & Phillips, 1979). However, diminished mental health has been found to increase susceptibility to annoyance (Stansfeld & Matheson, 2003). The extent of health damage

associated with noise annoyance is not yet fully understood, though it is possible that annoyance may be a risk factor for allostatic load – the wear and tear on the body as a result of chronic stress (McEwen & Stellar, 1993). Noise annoyance, an emotional response linked to physiological arousal (Ndrepepa & Twardella, 2011), may lead to a wide variety of health problems due to prolonged or recurrent allostatic processes.

Noise annoyance is a common and often underestimated health effect of noise exposure (Niemann et al., 2006). Not only is it linked to both mental and physical health problems, but it can also interfere significantly with daily functioning (Rylander, 2004). Noise annoyance can negatively impact cognitive processes such as concentration, oral communication, and relaxation (Chrström, 2004; Ouis, 2002), which may be particularly important in achieving lowered stress levels and subsequently maintenance of good health. Further, noise annoyance has increased over the years (Babisch et al., 2009), and will likely continue to increase with urbanisation, accentuating its importance in psychological and public health research on environmental noise exposure.

3.3.3. Physiological effects of noise exposure. When the noise source is sudden or loud (e.g. > 70dB(A)), a stress response can occur automatically in an organism (Westman & Walters, 1981). Additionally, a negative emotional reaction to the noise source, including fear or annoyance, can also lead to allostasis (Rylander, 2004). Exploring the effect of both acute and chronic noise exposure on physiological changes provides insight as to the potential mechanisms involved in the relationship between noise exposure and ill-health (Ising, Babisch, & Kruppa, 1999; Lundberg, 1999; Prasher, 2009; Spreng, 2000a). Specifically, both acute and long-term changes to the sympathetic nervous and neuroendocrine system activity may provide information about allostatic processes that occur as a result of noise exposure (Ising & Braun, 2000).

Both laboratory and community-based studies have demonstrated a relationship between noise exposure and activation of the sympathetic nervous system. Exposure to occupational noise (e.g., textile industry noise, electrical drill noise) has been linked to an increase in acute and chronic elevations in adrenaline and noradrenaline (Babisch, Fromme, Breyer, & Ising, 2001; Cavatorta et al., 1987; Goyal, Gupta, & Walia, 2010; Ising et al., 1999; Ising, Babisch, Kruppa, Lindthammer, & Wiens, 1997). Elevations in heart rate as a result of noise exposure have been found among experimental studies on the impact of environmental noise exposure as well (Griefahn et al., 2008; Raggam et al., 2007).

Further, activation of the sympathetic nervous system has also been observed in noise-exposed children. In a naturalistic study, Cohen, Evans, Krantz, and Stokols (1980) found that children exposed to aircraft noise had significantly higher systolic and diastolic blood pressure compared to children in the control group. This finding has since been replicated (Belojević, Jakovljević, Stojanov, Paunovic, & Ilic, 2008; Evans, Bullinger, & Hygge, 1998; Evans et al., 2001; van Kempen et al., 2006). However, van Kempen et al. (2006) only found a significant relationship between elevated blood pressure and noise exposure at home, and not with noise exposure at school. Although sociodemographic and biological factors (e.g., age, gender, body mass index) remain strong predictors of blood pressure levels, exposure to noise appears to be an additional environmental factor to consider (Babisch, 2000; Paunovic, Stansfeld, Clark, & Belojević, 2011).

A particularly important finding in the field of noise and cardiovascular health is that noise-induced activation of the sympathetic nervous system can occur during the night (i.e., the individual does not necessarily need to be actively appraising the noise source for it to influence the stress response; Griefahn et al., 2008). Studies conducted as part of the HYENA (Hypertension and Exposure to Noise near Airports) project found that an increase

in both systolic and diastolic blood pressure were observed during exposure to night-time aircraft (> 35 dB) and road traffic (> 45 dB) noise (Haralabidis et al., 2008; Jarup et al., 2008). Therefore, even during sleep, noise can impact sympathetic activation, which can have long-term implications for overall cardiovascular health (Rosenlund, Berglund, Pershagen, Jarup, & Bluhm, 2001; van Kempen et al., 2002).

Noise-induced allostatic processes involve the activation of both the sympathetic nervous system and the neuroendocrine system, specifically, the HPA axis. As discussed earlier, the main output of the HPA axis is cortisol, a stress hormone that helps mobilise energy stores (Sherwood, 2012). Ising and Braun (2000) suggest that hypercortisolism may be the culprit in elevating cardiovascular disease risk, as well as risk for other health problems among noise-exposed individuals (Prasher, 2009). Elevations in cortisol have been reported in studies on occupational noise (Gitanjali & Ananth, 2003; Melamed & Bruhis, 1996), as well as in community-based studies on environmental noise exposure (Evans et al., 2001; Ising & Ising, 2002; Spreng, 2004).

Similarly, van Raaij et al. (1997) reported alteration of neuroendocrine functioning (i.e., increased ACTH) following chronic exposure to noise (i.e., 540 minutes per day across 8 days) among rats. Further, a relationship between aircraft noise exposure and morning cortisol levels was found among women in a large epidemiological study (HYENA; Selander et al., 2009a). Therefore, although there is some evidence to suggest a relationship between noise exposure and elevated cortisol (Ising & Ising, 2002; Selander et al., 2009a), its link to cardiovascular risk has not yet been confirmed.

While the negative health implications of chronic exposure to elevated blood pressure and increased catecholamines are well-established (van Kempen et al., 2002; Vasan et al., 2001), the impact of noise-induced cortisol release affecting well-being is not

as clear. In addition to being linked with a variety of mental and physical health issues (Spreng, 2000b), cortisol may have an important influence on sleep patterns.

Glucocorticoids inhibit slow wave sleep (Prasher, 2009), the type of sleep thought to be most restorative (Tasali, Leproult, Ehrmann, & Van Cauter, 2008). However, laboratory studies have found that following night-time exposure to noise, poorer reported sleep quality and increased tiredness were linked to lowered cortisol levels (Persson Waye et al., 2003).

It is possible that the health consequences associated with chronic activation of the HPA axis may involve a complex interplay among environmental (e.g., noise) and psychological (e.g., noise sensitivity, noise annoyance) factors (Maschke, Rupp, & Hecht, 2000). Large epidemiological studies, like the HYENA project, will help in further clarifying the interplay among physiological, environmental and psychological factors in noise-related health problems.

3.3.4. Noise and mental health. Early research exploring the relationship between noise exposure and mental health largely focused on the association between aircraft noise exposure and psychiatric symptoms (e.g., Jenkins, Tarnopolsky, Hand, & Barker, 1979; Tarnopolsky, Barker, Wiggins, & McLean, 1978; Watkins, Tarnopolsky, & Jenkins, 1981). These studies found associations between high levels of aircraft noise and headaches, restless nights, irritability, and edginess. Further, a number of studies have found an association between living near an airport and admissions to psychiatric hospitals (Abey-Wickrama, A'Brook, Gattoni, & Herridge, 1969; Meecham & Smith, 1977), though many of these studies were criticised because of a potentially spurious relationship between the two variables. The association between environmental noise exposure and psychiatric illness was further studied as a part of the Caerphilly Study, a large epidemiological study

conducted in England. However, no relationship between level of road traffic noise and psychiatric disorder was found (Stansfeld, Sharp, Gallacher, & Babisch, 1996; Stansfeld, Gallacher, Babisch, & Shipley, 1993).

Although early research on the association between noise exposure and mental health problems was criticised for failing to take potential confounders, such as socioeconomic status, into account (Cohen & Weinstein, 1981), more recent studies have provided evidence for an association between noise exposure and anxiety and use of psychotropic medication (Floud et al., 2011; Hardoy et al., 2005; Stansfeld, Haines, Burr, Berry, & Lercher, 2000). Exposure to aircraft noise has also been linked to prevalence of Generalised Anxiety Disorder (Hardoy et al., 2005), increased use of anxiolytic medication (Floud et al., 2011), depressive and nervous symptoms (Öhrström, 1991; Stošić & Blagojević, 2011), and diminished overall mental well-being (Black, Black, Issarayangyun, & Samuels, 2007).

Hardoy et al. (2005) proposed that increased prevalence of anxiety among residents exposed to aircraft noise could be due to chronic activation of the stress response. Chronic elevations of the corticotropin-releasing factor (CRF), secreted as part of the neuroendocrine response to stress, have been implicated in the development of anxiety and psychiatric disorders (Abrorelius, Owens, Plotsky, & Nemeroff, 1999; Heim & Binder, 2012). Alternatively, annoyance and other negative emotional reactions to environmental noise source may exacerbate symptoms of a pre-existing mental illness (Cohen & Weinstein, 1981).

3.3.5. Noise and perceived stress. An important research question that will help in addressing the relationship between noise exposure and health problems is whether noise exposure is linked to individuals' perceived stress levels. That is, because noise can be both

a physical and psychosocial stressor (Babisch, 2003), it may increase an individual's level of psychological stress. Specifically, perceived stress is regarded as an important intermediary factor between stressor exposure and health problems, as depicted in McEwen's (1998b) allostatic load model (see Figure 4).

Although some researchers question whether subjective ratings of stress accurately reflect physiological stress parameters (e.g., Brant, Wetherell, Lightman, Crown, & Vedhara, 2010; Noto, Sato, Kudo, Kurata, & Hirota, 2005), congruence between subjective ratings and objective measures of stress (e.g., salivary cortisol) has been reported in social stress (e.g., Oldehinkel et al., 2011) and noise research (Hebert & Lupien, 2009). While the relationship between noise exposure and perceived stress has been explored to an extent among nurses and patients in hospital settings (e.g., Short, Ahern, Holdgate, Morris, & Sidhu, 2010; Topf, 1985, 1989; Topf & Thompson, 2001), there have only been a limited number of community-based studies (i.e., large scale or epidemiological designs) conducted to assess the association (Haines, Stansfeld, Brentnall, et al., 2001; Meister & Donatelle, 2000).

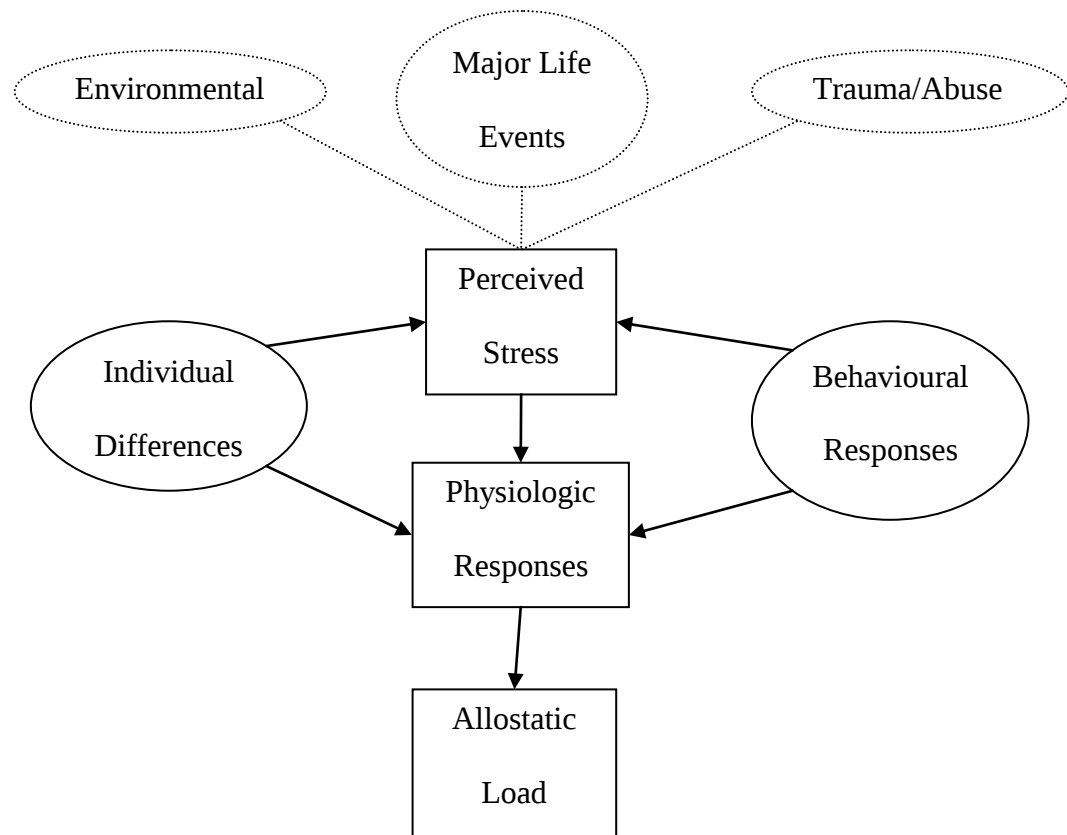


Figure 4. Allostatic load framework adapted from McEwen (1998a).

Meister and Donatelle (2000) found a positive relationship between self-rated stress, as measured by a stress scale from the United States of America Center for Disease Control and exposure to commercial aircraft noise. A similar relationship was found between aircraft noise exposure and perceived stress among noise-exposed children (Haines, Stansfeld, Brentnall, et al., 2001), suggesting that the stress levels of both children and adults may be influenced by noise exposure. Further, the results supported a relationship between aircraft noise and perceived stress, but not stressful events. Therefore, the children reporting high perceived stress levels did so for reasons other than stressful life events that had occurred recently, thereby suggesting the elevated stress levels could be a result of the chronic exposure to aircraft noise.

The anxiety and tension that can develop as a result of sustained arousal among noise-exposed individuals (Abrorelius et al., 1999) could also lead to increased perceived stress levels and possibly diminished coping abilities. As previously suggested by Babisch (2003), perceived stress may be an important psychological outcome of noise exposure, or possibly a mediator that could explain the relationship between noise exposure and ill-health (Meister & Donatelle, 2000).

3.3.6. Noise exposure and children's well-being. The association between mental health and environmental noise exposure has also been explored among children, a sector of the population that may be particularly vulnerable to the ill-effects of noise exposure (Babisch, Schulz, Seiwert, & Conrad, 2010; Haines, Stansfeld, Job, Berglund, & Head, 2001a; Matheson et al., 2003; van Kempen et al., 2010). A study of Austrian school children aged 8-11 years found a dose-response relationship between noise exposure and self-reported psychological distress (Lercher, Evans, Meis, & Kofler, 2002). However, previous research among English school children reported inconsistent findings (Haines, Stansfeld, Brentnall, et al., 2001; Haines, Stansfeld, et al., 2001a; Haines, Stansfeld, Job, Berglund, & Head, 2001b).

Haines et al. (2001a) reported a weak positive association between aircraft noise exposure and hyperactivity, a finding that has been replicated in a recent study (Stansfeld et al., 2009). However, the authors cautioned against drawing causal conclusions about this association. Stansfeld et al. (2009) proposed that because of the unpredictability and distraction of intermittent bouts of aircraft noise, it is more likely that noise exposure exacerbates symptoms of hyperactivity rather than causing them.

While children's mental health does not appear to be greatly affected by noise exposure, noise-exposed children do exhibit a range of cognitive deficits, including

impaired speech perception, memory problems, and impaired reading ability (Ana, Shendell, Brown, & Sridhar, 2009; Clark et al., 2006; Clark & Stansfeld, 2007; Cohen, Glass, & Singer, 1973; Evans & Maxwell, 1997; Haines, Stansfeld, Brentnall, et al., 2001; Hygge, Evans, & Bullinger, 2002; Stansfeld, Head, Clark, van Kamp, & Barrio, 2005). Longitudinal studies show that children do not appear to habituate to the noise source, as evidenced by chronic ill-effects of noise including reading difficulty and annoyance (Cohen, Evans, Krantz, Stokols, & Kelly, 1981; Haines, Stansfeld, et al., 2001b).

A recent study reported significant improvement in the long-term memory of children following the closure of a nearby airport (Padungtod et al., 2011), which suggests that children could recover if removed from the noise source or if the noise levels were better controlled. However, other studies have not found a clear association between chronic noise exposure and cognitive impairments (Haines, Stansfeld, et al., 2001a; Matsui, Stansfeld, Haines, & Head, 2004). Overall, a cautious conclusion is that children do appear to be a vulnerable sector of the population if exposed to excessive and chronic noise exposure.

3.3.7. Noise and quality of life. Increasingly, public health researchers are paying attention to quality of life as an important indicator of overall well-being in noise research. Quality of life (QOL) is defined as:

An individual's perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns. It is a broad ranging concept affected in a complex way by the persons' physical health, psychological state, level of independence, social relationships and their relationship to salient features of their environment.

(WHOQOL Group, 1995, p. 1403)

However, there are various definitions of the term ‘quality of life’, and in turn, a variety of assessment tools being used. For example, the World Health Organization Quality of Life instruments (WHOQOL Group, 1998) and the Short Form Health Survey (SF-36; Ware & Sherbourne, 1992) both purport they assess “health-related quality of life” (HRQOL). Specifically, the WHOQOL instrument (WHOQOL Group, 1998) assesses feelings across various domains of everyday life (psychological, physical, environmental, social), while the SF-36 assesses physical and mental health status. As such, research on the impact of environmental noise exposure on HRQOL, albeit limited, involves a variety of measurement tools. For succinctness, the literature review of noise exposure and QOL for this thesis involves an overview of research on QOL, as defined and measured by the researchers. The comparative validity of these definitions will not be discussed.

Broadly, noise exposure has been linked to diminished HRQOL (Dratva et al., 2010; Issarayangyun, Black, Black, & Samuels, 2005; Shepherd et al., 2011). Specifically, noise annoyance to both wind turbine and aircraft noise have been negatively associated with HRQOL (Dratva et al., 2010; Schreckenber, Meis, Kahl, Peschel, & Eikmann, 2010b; Shepherd et al., 2011; Shepherd et al., 2010). Further, both longitudinal (Bullinger, Hygge, Evans, & von Mackensen, 1999; Evans et al., 1998) and cross-sectional (Lercher et al., 2002) research have shown diminished QOL among children exposed to aircraft noise.

Seidman and Standring (2010) proposed that physical QOL could be depleted through the physiological changes associated with noise exposure. Noise exposure can lead to chronically elevated blood pressure or increased cortisol, physiological changes that may lead to chronic health problems (Ising et al., 1999; Spreng, 2000b). Suffering from chronic conditions that are brought on or exacerbated by environmental noise exposure, could, in turn, lead to diminished QOL.

Dratva et al. (2010) reported an interaction between chronic illness and noise annoyance on HRQOL in her study of residents exposed to aircraft noise. Among those participants with a diagnosed chronic illness (e.g., hypertension, heart disease, stroke, diabetes, migraine, asthma, chronic bronchitis/lung emphysema, kidney disease, arthritis and depression), the negative relationship between HRQOL and noise annoyance was stronger than among those participants without chronic illness. It is possible that chronic illness leads to an increased vulnerability to the negative health effects of noise annoyance. Alternatively, compared to relatively healthy individuals, those with chronic illness may lack the coping skills or the ability to deal with unwanted noise (e.g., physical capacity to close a window). It is well-documented that excessive or chronic noise can negatively impact exposed individuals, and there is mounting evidence to suggest QOL is yet another health outcome that is likely affected by noise exposure.

3.3.8. Noise and physical disorders. Noise has been implicated in the aggravation and development of stress-related disorders, including migraines, hypertension, coronary artery disease, peptic ulcers and irritable bowel syndrome (Babisch, 2002; Rosenlund et al., 2001). It is well-established that noise is an environmental stressor that can influence the body's physiological systems both directly (e.g., loud/chronic noise inducing a stress response) and indirectly – through emotional reactions to the noise source (e.g., noise annoyance). While cardiovascular disease is arguably the most pervasive and well-researched disease in noise research, exposure to noise has also been linked to general ratings of poor health (Brink, 2011; Franssen, van Wiechen, Nagelkerke, & Lebrecht, 2004).

Further, noise has been identified as a stressor that may be “immunotoxic” (Prasher, 2009) – an environmental risk that may be negatively impacting the immune system. Early psychoneuroimmunology research determined a relationship between psychological stress

and a decreased ability to fight off infection (Cohen et al., 1991). Later studies on chronic psychological stress (e.g., being a caregiver for an ill loved one) confirmed a negative association between stress and number of active immune cells (Irwin, Daniels, Risch, Bloom, & Weiner, 1988). Considering the evidence for the relationship between psychological stress and immune system functioning, it is not surprising that noise has been identified as another potentially immune system-degrading source of stress.

Prasher (2009) described the biological effects on the immune system that could occur as a result of chronic noise exposure. Because psychological stress can induce the production of regulatory (suppressor) T cells, which suppress immune system functioning (Manuck, Cohen, Rabin, Muldoon, & Bachen, 2001), Prasher (2009) proposed that the same process could occur as a result of chronic noise exposure. While noise has been linked to a variety of health complaints such as fatigue, headache, and digestive issues (Yoshida et al., 1997), no research to date has explored the role of environmental noise in immune function. However, with the increase in research on the relationship between noise and cortisol (Rylander, 2004; Selander et al., 2009a), a well-known immune suppressor (Segerstrom & Miller, 2004; Taylor, 2010), this relationship may receive further attention in the near future.

3.3.9. Noise and cardiovascular disease. Cardiovascular disease is a broad term used to describe any ailment of the heart or blood vessels, including hypertension, stroke, and myocardial infarction – also known as a heart attack (Gurung, 2010). It is the number two cause of death in New Zealand, second only to cancer (Ministry of Health, 2010), and the number one cause of death worldwide (World Health Organization, 2012). The relationship between noise annoyance and cardiovascular problems was reviewed in this chapter (see p. 46). Additionally, there appears to be a strong evidence for an association

between environmental noise exposure and risk for cardiovascular disease (Babisch, 2006; Barregard, 2011; Bendokiene, Grazuleviciene, & Dedele, 2011; van Kempen & Babisch, 2012).

The relationship between environmental noise exposure and hypertension has been demonstrated in a large number of studies, both cross-sectional (Aydin & Kaltenbach, 2007; Belojevic, Jakovljevic, Stojanov, Slepcević, & Paunovic, 2008; Belojevic & Saric-Tanaskovic, 2002; de Kluizenaar, Gansevoort, Miedema, & de Jong, 2007; Haralabidis et al., 2008; Jarup et al., 2008; Sorensen et al., 2011; Stansfeld & Crombie, 2011) and longitudinal (Babisch, Ising, Gallacher, Sweetnam, & Elwood, 1999; Barregard, Bonde, & Ohrström, 2009; Beelen et al., 2009; Eriksson et al., 2007; Selander et al., 2009b). However, in a recent population-based study, Dratva et al. (2012) found an adverse effect of railway noise on blood pressure only among vulnerable members of the population (i.e., those already suffering from diabetes/cardiovascular disease).

Recent research in this field has benefited from the availability of personal noise dosimeters – portable devices that can measure individual noise exposure throughout the day and night. Two recent studies using personal dosimeters have found positive correlations between noise exposure and blood pressure (Chang, Lai, Hsieh, Lai, & Liu, 2009; Weinmann, Ehrenstein, von Kries, Nowak, & Radon, 2011). Interestingly, mean daytime noise exposure was 74 dB(A) and 80 dB(A) for adults and children respectively in the study by Weinman et al. (2011), exceeding the minimal threshold for a noise-induced stress response (> 70 dB; Westman & Walters, 1981).

The mean noise exposure was lower (56.6 dB(A)) in the study of young adults conducted by Chang et al. (2009), and interestingly, their findings suggested an interaction between noise exposure and gender. Their results suggested that females may be more

susceptible to the cardiovascular effects of noise exposure than males, a finding supported by some community-based studies (Bluhm, Berglind, Nordling, & Rosenlund, 2007; Selander et al., 2009b). However, the impact of gender is not clear; other studies have reported associations between environmental noise exposure and hypertension only among men (Babisch et al., 2005; Barregard et al., 2009; Eriksson, Bluhm, Hilding, Ostenson, & Pershagen, 2010).

Further supporting the association between noise exposure and cardiovascular risk, some studies have reported a link with myocardial infarction (Babisch et al., 2005; Gan, Davies, Koehoorn, & Brauer, 2012; Selander et al., 2009b; Willich et al., 2006). However, the meta-analysis conducted by van Kempen et al. (2002) indicated that the link between noise exposure and hypertension is stronger than the association between noise exposure and myocardial infarction. With the increase in evidence for the association between noise exposure and hypertension, some researchers suggested that the relationship is indeed causal (Barregard, 2011; Neus & Boikat, 2000). That is, the increase in catecholamines, stress hormones and blood pressure that accompanies a noise-induced stress response, when sustained, can lead directly to hypertension.

While the relationship between sustained physiological arousal and cardiovascular risk is not novel (Curtis & O'Keefe, 2002), Fyhri and Klæboe (2009) proposed that the relationship between noise exposure and cardiovascular risk is spurious. They suggested that personal factors, such as noise sensitivity, adequately explain the association, despite previous research demonstrating a clear link between noise exposure and cardiovascular risk, even after considering subjective reactions (e.g., Black et al., 2007; Willich et al., 2006). Indeed, noise sensitivity has been linked to both increased reactivity to noise (Persson Waye et al., 2002) and cardiovascular risk (Fyhri & Klæboe, 2009; Heinonen-

Guzejev, 2009). However, because research on this topic remains relatively sparse, further noise research that assesses the relationship among noise exposure, noise sensitivity and cardiovascular risk will help to further elucidate this topic (Lercher, Botteldooren, Widmann, Uhrner, & Kammeringer, 2011). The relationship between noise sensitivity and health will be further discussed in Chapter 4 (p. 93).

3.3.10. Noise and perceived control. Psychological and social factors are important moderators of the ill-effects of chronic noise exposure. Among the myriad of potential moderators including noise sensitivity and noise annoyance (Fields, 1993; Hatfield et al., 2001; Ndrepepa & Twardella, 2011), perceived control is yet another modifier of the effects of noise exposure (Hatfield et al., 2002; Stallen, 1999). Perceptions of control can considerably reduce the psychological, cognitive and physical effects of noise exposure (Hatfield et al., 2002).

However, the powerful effects of perceived control are not novel in stress research. Various laboratory experiments have demonstrated that perceived control can markedly impact physiological, psychological, and the after-effects of noise exposure. In their landmark study in this field, Glass and Singer (1972a) discovered the extent of after-effects of noise exposure (i.e., reduced performance) and the impact of perceived control over these outcomes. Results of their experiment showed that participants who perceived the noise to be uncontrollable were more likely to suffer the after-effects of noise exposure such as poorer task performance. Although Glass and Singer (1972a; 1973) did not report an effect of perceived control on physiological parameters of the stress response (e.g., galvanic skin response), later research reported an association between perceptions of lack of control and increased response of the SAM system (heart rate, skin conductance;

Bugental & Cortez, 1988) as well as elevated activity of the HPA axis (Dickerson & Kemeny, 2002).

Perceived control is widely acknowledged as a moderator of the effects of stress (e.g., Glass & Singer, 1972a). However, it has received relatively little attention in the field of noise research (Hatfield et al., 2002). Chronic noise exposure can lead to psychological responses akin to ‘learned helplessness’ – characterised by depressive symptomatology and lack of motivation (Evans & Stecker, 2004; Hatfield et al., 2002). Originally defined by Seligman (1975), learned helplessness was coined to describe the defeat and dejection that animals experienced in situations where they consistently had no control over their environment.

In their experiments, Seligman, Maier and Geer (1968) observed that dogs repeatedly exposed to seemingly inescapable electric shock regimes eventually failed to attempt to escape the shocks. Mainly applied to clinical depression treatment and research among humans, this paradigm has also become an important framework for studying the role of perceived control in the face of a variety of environmental stimuli (e.g., Evans & Jacobs, 1981; Hatfield et al., 2002).

Perceptions of control may be closely linked to coping skills, especially when considered in the context of noise exposure. Environmental noise exposure is not often controllable, so believing one has control over a noise-exposed situation may involve employing considerable coping skills (Smith, 2003). For instance, if the noise source is under the control of a neighbour (e.g., lawnmower, electric tools, music at a party), an individual who is confident in politely discussing the issue of noise with the neighbour may be more likely to perceive greater control over the noise than a person who would not want to engage in such a discussion. Perceived control may also relate to the perceived

effectiveness of engaging in behaviours such as closing a window, putting in ear plugs, or relocating one's activities to a quieter room of the house. Further, because perceived control is linked, at least in part, to resilience to depression and anxiety (Becker & Chorpita, 2008), it is plausible that negative emotions may influence perceptions of control.

Hatfield et al. (2002) explored the role of perceived control over aircraft noise among residents of Sydney, Australia. Although results of this epidemiological study did not show differences in perceived control between noise-exposed and non-noise-exposed individuals, perceived control was negatively correlated with sleep disturbance, reading disturbances, and general health symptoms. Interestingly, however, perceived control was not associated with symptoms of anxiety or depression. To summarise, although limited research has been conducted on perceived control in epidemiological studies (Bobak, Pikhart, Hertzmann, Rose, & Marmot, 1998; Evans & Carrere, 1991), it remains an important factor to consider in investigations on the effects of noise exposure.

Chapter 4: Literature Review – Noise Sensitivity

4.1. The Concept of Noise Sensitivity

Job (1999) described noise sensitivity as a personal trait encompassing internal factors (e.g., physiological, psychology, attitudinal) that increase an individual's susceptibility to the effects of noise. The concept of noise sensitivity was first used in a survey of noise around London's Heathrow Airport conducted by McKennell (1963). Results from the survey revealed that individuals who self-identified as noise sensitive⁵ were more likely to be annoyed by noise than participants who were not sensitive to noise. In psychoacoustics and public health research, individual differences in noise reactions (e.g., noise annoyance) emerged (e.g., Griffiths & Delauzan, 1977; Griffiths & Langdon, 1968; Griffiths & Raw, 1986; Raw & Griffiths, 1988), with researchers eventually embracing the concept of 'noise sensitivity' to help explain these differences (e.g., Aniansson, Pettersson, & Peterson, 1983; Geen, McCown, & Broyles, 1985; Moreira & Bryan, 1972; Stansfeld, Clark, Jenkins, & Tarnopolsky, 1985a; Stansfeld, Clark, Turpin, Jenkins, & Tarnopolsky, 1985b).

Noise sensitivity is, indeed, a trait in which people can range from low to high on the continuum. However, in order to understand prevalence of the trait, many researchers define noise sensitivity as those who consider themselves to be considerably affected or oversensitive to noise (e.g., Matsumura & Rylander, 1991; Olsen Widen & Erlandsson, 2004). Using that definition, there has been general agreement that in the total population,

⁵ Noise sensitivity was measured using the question "Would you say you were more sensitive or less sensitive than other people are to noise" with response options: (1) more sensitive, (2) less sensitive, (3) same, (4) don't know. Those who were classified as noise sensitive chose response option 1.

approximately 20-25% of individuals are noise sensitive – considerably affected by excessive or unwanted noise (Matsumura & Rylander, 1991; Olsen Widen & Erlandsson, 2004).

In addition to having a greater emotional response to noise, noise sensitive people are also more likely to attend to noises, discriminate between noises and find noises more threatening than less noise sensitive individuals (Stansfeld, 1992). It is perhaps not surprising then that in his detailed investigation of noise sensitivity among psychiatric patients, Stansfeld (1992) suggested that noise sensitivity is an indicator of vulnerability to noise, and also to stressors in general. Stansfeld (1992) found that among his sample of psychiatric patients, noise sensitivity⁶ levels declined as the patients recovered from depression, but still remained relatively high following recovery. Based on the results of this investigation, Stansfeld (1992) proposed that noise sensitivity involved a negative affectivity component as well as a vulnerability to effects of noise.

The concept of ‘stress vulnerability’ in relation to noise reactions, however, is not a novel concept in this field of research. Because of their observation that at any given noise level there are likely some individuals that are greatly disturbed by noise, and some who do not notice it, Tarnopolksy et al. (1980) proposed the ‘vulnerability hypothesis’. Tarnopolsky et al. (1980) observed that not all individuals in their study were equally affected by noise, and that an individual ‘vulnerability’ appeared to have a moderating effect on noise reactions. The ‘vulnerability hypothesis’, then, may be closely related to the

⁶ measured with the WNS

concept of noise sensitivity, which, in turn, helps to explain individual differences in response to stressors such as noise (e.g., Stansfeld, 1992; Weinstein, 1978).

In Job's (1999) review of noise sensitivity research, he noted that definitions of noise sensitivity, at least in research in the 1980s and 1990s, differed across studies (e.g., degree of susceptibility to annoyance, attitudinal characteristics), which, in turn, presents a considerable challenge for psychoacoustic and public health researchers who wish to identify noise sensitive individuals in their research. As such, Job (1999) proposed that noise sensitivity is a complex and multifaceted construct, one that likely encompasses physiological reactivity to noise, psychological reactivity and coping mechanisms.

4.2. Psychoacoustic Correlates of Noise Sensitivity

An important research question in this field of research is whether noise sensitive individuals actually perceive the acoustic world differently from non-noise sensitive individuals. Acousticians have sought to address this question by studying both subjective experiences of acoustic stimuli and sensory perceptions. Although only a handful of studies have addressed this topic, there appears to be little evidence supporting a relationship between noise sensitivity and auditory acuity (Ellermeier et al., 2001; Heinonen-Guzejev et al., 2011).

Overall, noise sensitive individuals do not appear to have superior auditory ability compared to non-noise sensitive individuals, as assessed on a variety of psychoacoustic variables including absolute hearing thresholds, intensity discrimination, and auditory reaction time (Ellermeier et al., 2001; Moreira & Bryan, 1972; Stansfeld et al., 1985b). However, noise sensitivity does appear to have some relationship with the uncomfortable loudness level and discomfort thresholds (Öhrström, Björkman, & Rylander, 1988; Thomas & Jones, 1982).

Noise sensitive individuals seem to be more easily distracted and disturbed by acoustic stimuli at lower thresholds than non-noise sensitive individuals. In an audiology study, Abel et al. (1990) found that individuals with noise sensitivity⁷ were more likely to be affected by low frequency sounds masking the detection of high frequency sounds. In terms of auditory discomfort, Öhrström et al. (1988) found small positive correlations between noise sensitivity⁸, annoyance to traffic noise, and discomfort thresholds for sound. Additionally, a 5-year case study of a 34-year-old man with high sensitivity to noise⁹, precipitated by stress-related psychosomatic disorder (melancholia along with dizziness and vomiting), explored the relationship between discomfort threshold and mental state (Murata & Sakamoto, 1995). Interestingly, the discomfort threshold was considerably lower (40-50 dB) when the participant's psychosomatic symptoms were elevated, compared to when the symptoms had improved (70-95 dB). It is possible that mental health and noise sensitivity interact to influence auditory discomfort (Stansfeld, 1992), however, results of this case study have not been replicated in a larger study.

⁷ 'Noise sensitive' participants were referred to the study from a practising otolaryngologist. It is likely then that noise sensitivity was determined from participant-reported noise complaints. However, Abel et al. (1990) did not specify the criteria or process by which the otolaryngologist labelled the participant as 'noise sensitive'.

⁸ Assessed using the WNS and an open scale with two end points: (1) not sensitive at all, (2) extremely sensitive.

⁹ Assessed through clinical interviews.

Recently, Heinonen-Guzejev et al. (2011) found an association between self-reported noise sensitivity¹⁰ and self-reported hearing disability (among participants aged 31-88 years), despite lack of an association between auditory acuity and noise sensitivity. Interestingly, noise sensitivity was also associated with the use of hearing protectors in the workplace, and therefore it is unclear as to the mechanisms by which noise sensitivity could contribute to hearing disability. Overall, the evidence in this field has led some researchers to conclude that noise sensitivity is more of an attitudinal trait than an acoustic one (Ellermeier et al., 2001; Stansfeld, 1992). Therefore, it is possible that noise sensitive individuals are more likely to express discomfort or annoyance than non-noise sensitive individuals (Stansfeld et al., 1993).

Within the context of noise sensitivity and auditory perception, it is noteworthy to distinguish noise sensitivity from hyperacusis, an auditory condition in which an individual experiences considerable discomfort to everyday sounds (Roberts et al., 2010). Hyperacusis is regarded as the result of an over-sensitive auditory system, which may be related to hyperactivity of cortical neurons (Sun, Deng, Jayaram, & Gibson, 2012). The condition can occur as a result of auditory disorders, such as tinnitus (Gu, Halpin, Nam, Levine, & Melcher, 2010), and neurological conditions including traumatic brain injury (Ruff, Iverson, Barth, Bush, & Broshek, 2009), William's syndrome (Barozzi et al., 2012), and autism (Stiegler & Davis, 2010). Due to the severity of hyperacusis, it can significantly

¹⁰ Noise sensitivity was assessed with the question: "People experience noise in different ways. Do you experience noise generally as (1) very disturbing, (2) quite disturbing, (3) not especially disturbing, (4) not at all disturbing or (5) can't say?". Participants indicating option (1) or (2) were classified as noise sensitive, and participants indicating options (3) or (4) were classified as non-noise sensitive.

decrease quality of life and lead to social isolation (Jastreboff & Jastreboff, 2010; Shabana, Selim, El Refaie, El Dessouky, & Soliman, 2011).

Although both hyperacusis and noise sensitivity relate to the ways an individual perceives his or her acoustic environment, noise sensitivity is regarded as a separate entity from hyperacusis (Heinonen-Guzejev et al., 2011). While hyperacusis is a condition of the auditory system, noise sensitivity is regarded as psychological trait that can influence an individual's vulnerability to noise and other stressors (Stansfeld, 1992; Weinstein, 1978).

4.3. Noise Sensitivity and Other Sensory Sensitivity

Noise sensitivity has been associated with annoyance to a variety of environmental stressors (Langdon, 1976; Miedema & Vos, 1999; Öhrström, Björkman, et al., 1988; Stansfeld et al., 1993; Weinstein, 1978), and as such, the relationship between noise sensitivity and other sensory sensitivities has been explored. Providing some evidence for a neurophysiological basis for noise sensitivity, Öhrström et al. (1988) found weak yet significant associations between sensitivity to noise and discomfort thresholds for cold, light, and heat, each assessed experimentally. Moreover, Winneke and Neuf (1992) proposed that noise sensitivity may be more accurately reflected by the term 'trait environmental annoyance', which describes a personality predisposition to become easily annoyed by various environmental stressors such as tobacco smoke and odour.

Other investigations have also found positive correlations between chemical sensitivity and noise sensitivity¹¹ (Andersson, Johansson, Millqvist, Nordin, & Bende, 2008; Nordin, Millqvist, Löwhagen, & Bende, 2003). Further, experimentally-tested

¹¹ Assessed with the WNS in both studies.

sensitivity to capsaicin (the hot substance in chilli peppers) has been positively correlated with noise sensitivity, as assessed by the Weinstein Noise Sensitivity Scale (WNS; Andersson et al., 2008). Therefore, although the research is still relatively sparse, evidence points to a possible association between noise sensitivity and other sensory sensitivities.

4.4. Noise Sensitivity and Sensory Processing Sensitivity

Sensory processing sensitivity (SPS), a relatively new concept that could relate to noise sensitivity, has emerged recently in the psychological literature (Aron & Aron, 1997; Benham, 2006; Smolewska, McCabe, & Woody, 2006). Although it has not yet been explored in reference to noise sensitivity, based on the previously discussed relationship between noise sensitivity and general sensitivity (e.g., Andersson et al., 2008; Nordin et al., 2003; Öhrström, Björkman, et al., 1988; Topf, 1994; van Kamp & Davies, 2008), it is possible that the two constructs are closely related.

Aron and Aron's (1997) definition of SPS broadly describes the trait as the proneness to overstimulation from environmental stimuli and feelings of deep emotion and empathy. That is, Aron and Aron (1997) propose that SPS not only relates to physical sensitivity, but also emotional sensitivity. The scale designed to assess SPS, the Highly Sensitive Person Scale (HSPS), includes items such as "Do you get rattled when you have a lot to do in a short amount of time?", and "Are you made uncomfortable by loud noises?" – the latter item seemingly tapping into the construct of noise sensitivity.

While original psychometric testing revealed that the HSPS measured a unidimensional construct (Aron & Aron, 1997), a later evaluation of the scale found different results. Smolewska et al. (2006) proposed that the scale actually measures three dimensions, which they labeled aesthetic sensitivity, low sensory threshold, and ease of

excitation. Therefore, sensitivity to environmental stimuli may not be as uniform as Aron and Aron (1997) initially proposed.

Interestingly, Smolewska et al. (2006) found that ease of excitation and low threshold sensitivity (the dimension that includes the noise sensitivity-related item) both correlate with neuroticism. Similarly, other studies have linked both ease of excitation and low threshold sensitivity to anxiety and depression (Aron, Aron, & Davies, 2005; Liss, Mailloux, & Erchull, 2008; Liss, Timmel, Baxley, & Killingsworth, 2005) as well as SPS to stress and physical symptoms (Benham, 2006). Further provoking a possible relationship with SPS, noise sensitivity has also been linked to some of the previously described health outcomes: anxiety and depression (Kishikawa et al., 2009; Nivison & Endresen, 1993), and physical health complaints (Fyhri & Klæboe, 2009).

The concept of SPS has been described in relation to biological sensitivity, a theory that attempts to explain individual differences in stress reactivity and arousability (Boyce & Ellis, 2004). With a strong foothold in evolutionary theory, the ‘biological sensitivity in context’ theory proposes that a high degree of sensitivity and reactivity has advantages for survival (Ellis, Jackson, & Boyce, 2006). Even though noise sensitivity does not involve superior auditory acuity, it has been linked to stress reactivity (e.g., Griefahn & Di Nisi, 1992; Persson Waye & Ohrström, 2002), which may have been particularly adaptive in monitoring the surrounding environment for danger during ancestral times (Rylander, 2004; Spreng, 2004).

Although it has not been tested empirically, based on the similar patterns of relationship with health outcomes and stress reactivity, noise sensitivity may be closely related to SPS. The SPS concept is still relatively new, however, and arguably may not be the uniform construct Aron and Aron (1997) first proposed. Regardless, the biological

sensitivity theory, which provided a theoretical grounding for SPS, appears to have relevance to noise sensitivity as well, particularly with regards to the elevated stress reactivity (Stansfeld, 1992).

4.5. Assessments of Noise Sensitivity

In the earliest study involving trait noise sensitivity, McKennell (1963) classified participants as ‘noise sensitive’ or ‘non-noise sensitive’ based on their self-perceived sensitivity to noise (see p. 64 footnote for the item used). However, researchers now acknowledge that the trait falls along a spectrum – with individuals who would report a high degree of noise sensitivity at one end, and individuals who would report a low degree of noise sensitivity at the other end. Although the concept of noise sensitivity had not yet been defined as a stable trait, the Broadbent-Gregory Annoyance Battery (Bowsher, Johnson, & Robinson, 1966) and the General Noise Annoyance Questionnaire (Anderson, 1971) were arguably the first continuous measures of noise sensitivity. The General Noise Annoyance Questionnaire and Broadbent-Gregory Annoyance Battery both seemed to tap into the construct of noise sensitivity (Heinonen-Guzejev, 2009), but neither managed to gain much traction in noise research.

Weinstein (1978) designed the first scale to measure noise sensitivity exclusively. The scale is composed of 21 items, and to each item the participant responds on a 6-point Likert scale ranging from (1) “agree strongly” to (6) “disagree strongly”. Weinstein (1978) reported that the scale had good internal reliability (.84-.87), test-retest reliability (.75), and later research confirmed that the scale had good external validity (Ekehammar & Dornic, 1990). The WNS has since been used in numerous lab (Ljungberg & Neely, 2007; Persson Waye et al., 2003) and field studies (e.g., Dornic & Ekehammar, 1990; Fyhri & Aasvang, 2010; Matsumura & Rylander, 1991; Staples et al., 1999; van Kamp et al., 2004).

Additionally, shortened versions of the scale have been developed to help reduce questionnaire length in community-based studies. The 10-item WNS scale was developed shortly following the conception of the 21-item WNS scale (Weinstein, 1980), and Kishikawa et al. (2006) composed a 6-item to improve upon the 10-item scale. Recently, Benfield et al. (2012) developed and validated a 5-item version of the WNS, which the authors noted may be particularly useful for research in field settings (e.g., community-based studies in neighbourhoods and parks).

Zimmer and Ellermeier (1998) also developed a continuous measure of noise sensitivity. The 52-item German scale was designed to measure sensitivity to noise across seven areas: everyday life, recreation, health, sleep, communication, work, and noise in general. Participants respond to each item on a 4-point Likert scale ranging from “strongly agree” to “strongly disagree”. Although Zimmer and Ellermeier (1999) determined that their scale more accurately assessed noise sensitivity than the WNS, it did not replace the WNS as the noise sensitivity measure of choice.

The multidimensional design of Zimmer and Ellermeier’s (1998) scale did, however, influence the development of the Noise Sensitivity Questionnaire (NoiSeQ; Schütte, Marks, Wenning, & Griefahn, 2007). The NoiSeQ was designed to measure global noise sensitivity, as well as noise sensitivity in five domains: leisure, work, habitation, communication, and sleep. A confirmatory factor analysis determined that the items measured separate noise sensitivity domains (Schütte, Sandrock, & Griefahn, 2007). However, the leisure domain may require further psychometric examination. Excellent internal reliability (.90) was determined for the scale in a cross-national sample (Sandrock, Schütte, & Griefahn, 2007). The NoiSeQ has been used recently in field studies on the

impact of aircraft noise on well-being (Schreckenberg et al., 2010b; Shepherd et al., 2010) and task performance research (e.g., Sandrock, Schütte, & Griefahn, 2010)

More recently, in an effort to alleviate burden on participants, much shorter assessments of noise sensitivity have been used. Some large community-based studies have opted for using a 1-item measure of noise sensitivity (e.g., Fyhri & Aasvang, 2010; Fyhri & Klæboe, 2009; Shepherd et al., 2011). The format of the 1-item measures of noise sensitivity have included 3-point Likert scales (Fyhri & Klæboe, 2009; Shepherd et al., 2011), 5-point Likert scales (Heinonen-Guzejev et al., 2004) and 6-point Likert scales (Fyhri & Aasvang, 2010).

Amann, Lercher, Weichbold, and Eisenmann (2007) discussed some of the issues with 1-item assessments of noise sensitivity. Although the 1-item measures have adequate validity, they tend to underestimate noise sensitivity, and may not capture the increase of noise sensitivity that might occur with age (Fyhri & Aasvang, 2010; Matsumura & Rylander, 1991; Nivison & Endresen, 1993). As such, Amann et al. (2007) proposed that a 3-item measure of noise sensitivity may be an appropriate choice for epidemiological studies; it is not too lengthy as to discourage participation among volunteers, and reliably captures noise sensitivity. Nivison and Endresen (1993) had previously used a 3-item measure of noise sensitivity in their community-based study, and found the index had adequate internal reliability (.70).

Using the WNS, Belojević and Jakovljević (2001) found that the trait of noise sensitivity in their sample followed a Gaussian (normal) distribution, a pattern reported in other studies as well (Ekehammar & Dornic, 1990; Shepherd et al., 2010). However, a threshold for noise sensitivity has been set in some research programmes (e.g., Heinonen-Guzejev et al., 2011; Heinonen-Guzejev et al., 2004). In such cases, noise sensitive

individuals are often classified as such if they report noise as “very disturbing” or “quite disturbing”, while non-noise sensitive individuals indicated they found noise “not especially disturbing” or “not at all disturbing”.

4.6. Psychophysiological Correlates of Noise Sensitivity

Related to the concept of ‘vulnerability’ to stress, Eysenck’s (1967) arousal theory states that individuals elevated on certain personality traits (i.e., introversion, neuroticism) exhibit higher basal arousal (i.e., lower thresholds for limbic system activity, greater reactivity of sympathetic nervous system; Ryckman, 2007), and as such, are easily affected by psychological or environmental stressors such as noise. Indeed, some research supports a possible relationship between Eysenck’s arousal theory and noise sensitivity (Belojević, Jakovljević, & Slepcević, 2003; Ising, Dienel, Gunther, & Markert, 1980; Persson Waye et al., 2002).

Noise sensitive individuals, as with individuals elevated on neuroticism and introversion traits, tend to exhibit a higher basal level of arousal (Ising et al., 1980; Kelly, 1986), which, in turn, may help explain the relationship between noise sensitivity and both emotional regulation and hyperactivation of stress response physiological parameters. In other words, individuals with high levels of noise sensitivity react more easily to stressors such as noise (e.g., Di Nisi, Muzet, & Weber, 1987; Griefahn & Di Nisi, 1992). Much of the early research in this field has assessed the association between cardiovascular system measurements (e.g., blood pressure) and noise sensitivity, while recent research has begun to consider catecholamines and stress hormones as well.

Some psychophysiological research, in both laboratory and naturalistic settings, support the notion that noise sensitivity is linked to greater activation of the SAM system. In a lab experiment involving the completion of mental tasks, high noise sensitive¹² individuals had a faster heart rate compared to low noise sensitive individuals (Di Nisi et al., 1987). Further, another lab experiment, using exposure to noises (e.g., traffic noise, gunfire), found larger changes in heart rate and peripheral blood flow at 80 dB(A) among the noise sensitive¹³ participants (Griefahn & Di Nisi, 1992). Similarly, in an experiment using male volunteers, Ising et al. (1980) found that self-reported noise sensitivity (measured on a 5-point Likert scale) was positively correlated with increased heart rate and blood pressure during day-long exposure to traffic noise.

More recent psychophysiological lab experiments have begun to investigate the relationship between noise sensitivity and cortisol. Persson Waye et al. (2002) designed a lab experiment in which participants were exposed to noise while completing a performance task. Cortisol is a particularly difficult physiological correlate to measure in a lab setting, mainly because levels of the stress hormone decline throughout the day as a part of the normal circadian pattern. Persson Waye et al. (2002) measured each participant's cortisol levels at six time points on two separate lab visits in order to accurately assess the

¹² Noise sensitivity was measured with a 12-point Likert scale ranging from 1 (not sensitive to noise) and 12 (very sensitive to noise). Participants with a score below seven were classified as low sensitive and participants with a score of eight or above were classified as high sensitive.

¹³ Noise sensitivity was measured with 3 items, each with response options ranging from (0) “disagree strongly” to (9) “agree strongly”. The total noise score was calculated by taking the mean of the three responses. Participants were classified as “resistant” (0-3), “indifferent” (4-6) and “sensitive” (7-9).

impact of noise on stress hormones. The results showed that noise sensitive¹⁴ individuals not only reported greater subjective stress (measured with a 12-item adjective rating scale) and had poorer task performance during the lab experiment, but their cortisol levels were attenuated in the low frequency noise condition. In the low frequency condition, the cortisol levels of the noise sensitive participants did not follow the typical circadian pattern of decline, but rather, were higher than normal, thereby indicating a sustained stress response. Low noise sensitive participants exhibited the typical decline of cortisol during exposure to the low frequency noise.

More recently, Ljungberg and Neely (2007) found some evidence that noise sensitivity was associated with both perceived stress and objective stress, as measured by lab cortisol levels. In the experiment, 24 male participants were exposed to noise stimuli and a series of cognitive tasks (e.g., memory, reasoning). Noise sensitivity was measured using the Swedish version of the WNS, and participants were categorised as noise sensitive based on a median split of the data. Compared to low noise sensitive participants, noise sensitive participants exhibited higher levels of cortisol during the experiment and reported greater stress, as measured by the Borg CR-10 rating scale (Borg, 1998).

However, a follow-up experiment, reported within the same manuscript (Ljungberg & Neely, 2007), could not replicate the cortisol results. Noise sensitive participants were, however, more likely to report greater subjective stress than low-noise sensitive participants in both experiments. Overall, results of their study partially support the ‘stress vulnerability’ hypothesis, which posits that noise sensitive individuals may simply be more

¹⁴ Dichotomous categorisation based on a principal component analysis involving participant self-reports of noise sensitivity (single items and the WNS). See Persson Waye et al. (2002) for details.

vulnerable to stress (Heinonen-Guzejev et al., 2009; Stansfeld, 1992). However, the relationship between perceptions of stress and physiological stress parameters (e.g., cortisol) is not entirely clear.

Although some research supports the notion that noise sensitivity may be associated with an elevated basal arousal level, research in this area has not been conclusive. Stansfeld et al. (1985a) assessed various physiological measures and psychiatric outcomes in a sample of women from the 1977 West London Survey. Although noise sensitive¹⁵ women were more likely to report psychiatric symptoms, unexpectedly, a follow-up study using the same data (Stansfeld et al., 1985b) revealed that high noise sensitive¹⁶ women had slower heart rates compared to women with lower sensitivity. Further, no other physiological differences were found. Similarly, in a lab-based study, Öhrström et al. (1988) found no differences in heart rate between noise sensitive and non-noise sensitive participants¹⁷.

With noted exceptions, much of the psychophysiological research on noise sensitivity supports the notion that noise sensitive individuals are more likely to experience hyperarousal and excessive activation of the stress response in the face of stressors (e.g., noise, mental task performance) compared to low-noise sensitive individuals. Possibly due

¹⁵ Noise sensitivity was assessed with various measures: McKennell's 'list of annoying noises', a single item measure, the WNS, and the General Noise Questionnaire. There was a high degree of agreement in results for the scales and psychiatric outcomes. Results are generalised for the discussion above.

¹⁶ Participants were classified as "high", "intermediate" and "low" noise sensitive based on data from Stansfeld et al. (1985a).

¹⁷ Assessed using the WNS and an open scale with two end points: (1) not sensitive at all, (2) extremely sensitive.

to innate or biological differences, noise sensitive individuals might have a uniquely structured nervous system – one which allows the stress response to be initiated either too quickly or too frequently (Bell, Hardin, Baldwin, & Schwartz, 1995). However, the nature of this relationship is not well-understood and requires further attention.

4.7. Noise Sensitivity and Noise Annoyance

In both community and laboratory-based research noise sensitivity correlates positively with noise annoyance (e.g., Al-Mutairi et al., 2011; Bodin, Bjork, Öhrström, Ardo, & Albin, 2012; Guski, 1999; Jakovljević et al., 2009; Job, 1988; Lam, Chan, Chan, Au, & Hui, 2009; Matsumura & Rylander, 1991; Meijer, Knipschild, & Salle, 1985; Nijland, Hartemink, van Kamp, & van Wee, 2007; Paunovic et al., 2009; Pierrette et al., 2012; Ryu & Jeon, 2011; Weinstein, 1978). Noise sensitivity and annoyance typically exhibit weak to moderate positive correlations of .15 to .45 (Guski, 1999), suggesting that the concepts, while related, are indeed unique. Specifically, noise sensitivity is largely regarded as a trait that increases an individual's susceptibility to the effects of noise (Job, 1999), while noise annoyance is a negative emotional reaction to noise. Noise sensitivity is believed to be an important moderator of the relationship between noise exposure and noise annoyance, with noise sensitivity accounting for approximately 10% of variance in noise annoyance ratings (Guski, 1999). Importantly, while noise annoyance is influenced by noise exposure (Birk, Ivina, von Klot, Babisch, & Heinrich, 2011; Passchier-Vermeer & Passchier, 2000), noise sensitivity is regarded as largely independent (Heinonen-Guzejev et al., 2000; Job, 1988; van Kamp et al., 2004).

Providing insight on the associations among noise exposure, noise sensitivity and noise annoyance, Guski (1999) noted that at the extreme ends of noise exposure (i.e., very little noise, excessive noise), individual reaction to noise is not markedly variable.

However, in the middle spectrum (i.e., some noise exposure), there is considerable variation in noise annoyance, likely influenced by personal factors such as noise sensitivity. In an examination of the association among the three variables (exposure, annoyance, sensitivity) around three international airports, van Kamp et al. (2004) found that noise sensitivity¹⁸ was strongly predictive of noise annoyance across their regression models (explaining 21-38% of variance in noise annoyance). Their research supported the notion that noise sensitivity is, indeed, independent of noise exposure, and that noise sensitivity may lead to aversive reactions even with low exposure to noise.

4.8. Noise Sensitivity and Age

The association between age and noise sensitivity is not clear. Some studies have reported that noise sensitivity increases with age (Fyhri & Aasvang, 2010; Ising et al., 1980; Matsumura & Rylander, 1991; Nivison & Endresen, 1993), while another study reported a peak around middle age (Stansfeld et al., 1985a), similar to noise annoyance and age (see p. 45). However, some research has indicated no relationship between age and noise sensitivity (e.g., Belojević & Jakovljević, 2001; Moreira & Bryan, 1972), and one study reported an inverse association between age and noise sensitivity among participants aged 31-70 years (Heinonen-Guzejev et al., 2004).

An association between noise sensitivity and age could be supported, theoretically, by cognitive research. With age, the ability to discriminate between task-relevant and task irrelevant stimuli declines (Grady, Springer, Hongwanishkul, McIntosh, & Winocur, 2006). Therefore, older adults, with a diminished capacity to filter unwanted environmental

¹⁸ The Sydney study used a 'sensitivity to loud noises' scale, the Amsterdam study used a single-item 11 point scale, and the London study used the 10-item WNS.

stimuli, may experience greater interruption of daily activities (e.g., reading the newspaper, watching television) and annoyance due to excessive processing of auditory stimuli (e.g., aircraft noise, lawnmower noise). Therefore, noise should negatively affect older adults more than younger adults because of their difficulty in filtering unwanted auditory stimuli. As previously discussed, however, while some noise sensitivity research supports this cognitive theory (Fyhri & Aasvang, 2010; Matsumura & Rylander, 1991; Nivison & Endresen, 1993), other research does not (Heinonen-Guzejev et al., 2004; Moreira & Bryan, 1972; Stansfeld et al., 1985a).

However, noise sensitivity has also been found to peak around middle-age (i.e., approximately 40-55 years old) in some studies (Booi & van den Berg, 2012; Stansfeld et al., 1985a). From a developmental perspective, middle-age is a period of considerable growth and challenges. Middle-aged people have been found to report more stress and daily hassles than older adults (Almeida & Horn, 2004; Darbonne, Uchino, & Ong, 2012). Therefore, because of elevated stress levels, individuals may be less likely, or possibly less willing, to cope with unwanted environmental noise. Additionally, sensitivity to low frequency noise (e.g., low humming sounds) has been found to increase around middle-age (Leventhall, 2004).

Another possible reason for the reported peak of noise sensitivity around middle-age may be due to premature mortality among noise sensitive individuals. Noise sensitivity has been linked to coronary heart and cardiovascular mortality in women (Heinonen-Guzejev et al., 2007). Therefore, it is possible that studies have found greater noise sensitivity among middle-aged individuals because, compared to those who are less noise sensitive, fewer noise sensitive individuals survive to older adulthood. As such, the average

rating of noise sensitivity would be higher among middle-aged individuals simply because of overrepresentation of noise sensitive individuals in that group.

Changes in lifestyle factors in middle adulthood, such as career and interpersonal stress, as well as age-related cognitive changes in auditory processing could also influence the prevalence of self-reported noise sensitivity in different age groups. However, while noise sensitivity may increase slightly in middle-age and older adulthood, it should be noted that noise sensitivity is generally regarded as a relatively stable trait (Ellermeier et al., 2001; Miedema & Vos, 2003; Tennant, 2001).

4.9. Noise Sensitivity and Gender

Some research has reported gender differences in noise sensitivity, with females reporting greater sensitivity (e.g., Novak, La Lopa, & Novak, 2010; van Kamp et al., 2004), but other studies have found no gender differences (Belojević et al., 2003; Enmarker & Boman, 2004; Kjellberg et al., 1996; Widen, Bohlin, & Johansson, 2011). Heinonen-Guzejev (2009) proposed that females may be more prone to environmental noise, related to an “evolutionary born alertness” (p. 56). In other words, the auditory system of females may be more attuned to responding to environmental noise in order to appropriately react to potential threats or distressed offspring. However, this notion is largely theoretical.

Other noise research has reported greater reaction to noise among females (McFadden, 1998; Velle, 1987). Rhudy and Meagher (2001), in an experiment on noise and pain thresholds, found that women were more likely to experience fear and exhibit physiological arousal (greater skin conductance, higher heart rate) to noise stress than men. In a related vein, women also reported being more disturbed by noise than men. Ellermeier et al. (2001) reported that females were more likely to report feelings of unpleasantness when exposed to the sounds of the auditory experiment. Similarly, Rogers et al. (2003)

found that females had lower “most comfortable listening levels” to speech and lower acceptance of background noise compared to males. However, in a recent study on factors influencing an individual’s “need for quietness”, Booij and van den Berg (2012) reported no influence of gender.

Gender differences in noise attitudes and sensitivity have also been reported among adolescents and young adults. Olsen Widen et al. (2004) found that adolescent girls (aged 13-19 years) reported experiencing noise sensitivity¹⁹ to a greater extent than adolescent boys. Widen et al. (2006) also reported differences in attitudes toward noise among young men and women (aged 17 to 21 years) as measured using the Youth Attitude to Noise Scale (YANS). Although there were no gender differences in self-reported ability to concentrate in a noisy environment, women were more likely to report negative attitudes to noise associated with youth culture (e.g., concerts, nightclubs) and daily noise (e.g., from fans, refrigerators, traffic noise).

Widen et al. (2006) proposed that this difference in tolerance for a variety of noise sources could have important implications for behaviour-based protection against noise-induced hearing loss. Indeed, men are more likely than women to experience hearing loss, a relationship that persists even among individuals in low noise occupations (Pearson et al., 1995). Therefore, although noise sensitivity is associated with mental and physical health problems (e.g., Fyhri & Klæboe, 2009; Kelly, 1986; Kishikawa et al., 2009; Stansfeld, 1992), it may be a protective factor against auditory system damage in that noise sensitive individuals are more likely to avoid risky auditory environments.

¹⁹ Measured with the item: "Do you consider yourself to be oversensitive to noise?" (yes/no).

4.10. Pathophysiology of Noise Sensitivity

As previously discussed, noise sensitivity is a complex trait that influences an individual's response to noise and vulnerability to a variety of stressors (Job, 1999; Stansfeld, 1992). Possibly due to its complexity, the pathophysiology of noise sensitivity is not well understood (Yablon, 2007). It is possible that noise sensitivity is influenced by genetic factors. In a study on the Finnish Twin Cohort, Heinonen-Guzejev et al. (2005) explored a possible genetic component of noise sensitivity, as assessed with a single self-report item (see p. 75). Using genetic models, the research team estimated that noise sensitivity has a heritability of 36%, allowing them to conclude a likely genetic component of the trait. Further, when hearing-impaired participants were excluded from the analyses, the estimate of heritability rose to 40%. Supporting the notion of a genetic link with noise sensitivity, monozygotic twins (identical) reported more similar noise sensitivity than dizygotic twins (fraternal) in the study.

Although a possible genetic influence on susceptibility to hearing damage has been suggested (Davis, Kozel, & Erway, 2003), the genetic study on the Finnish Twin Cohort (Heinonen-Guzejev et al., 2005) focused specifically on a potential genetic influence on noise sensitivity. While no further genetic studies have been conducted to confirm these findings, van Kempen et al. (2009) found that children's annoyance to aircraft and road traffic noise followed a similar exposure-response pattern (i.e., degree of annoyance) as their parents. While this finding may be an artefact of similar noise exposure among children and their parents, it could also be a reflection of genetic influence of noise sensitivity.

However, other research has reported differences in noise sensitivity between younger and older adolescents, indicating that the trait may not become a noticeable or

stable trait until late adolescence. Olsen Widen et al. (2004) found that older adolescents in their study (16-19 years of age) were more likely to report noise sensitivity²⁰ than younger adolescents (13-15 years of age). Interestingly, adolescence is a period in which individuals begin to experience an increase in anxiety and negative emotions (Lupien et al., 2009), which, in turn, are correlated with trait noise sensitivity (e.g., Kishikawa et al., 2009; Nivison & Endresen, 1993). This probes the question as to whether the factors that contribute to mental health problems also influence noise sensitivity.

Notably, much of the research on noise exposure and noise sensitivity is focused on adult populations (e.g., Belojević & Jakovljević, 2001; Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2007; Kishikawa et al., 2009; Schreckenberg, Griefahn, & Meis, 2010; van Kamp et al., 2004), and as such, the progression of noise sensitivity through childhood and adolescence is unclear. Further research, specifically on the development of noise sensitivity and familial trends, may help clarify these results. However, for the purpose of this thesis, which deals with stress-related factors in adult samples, noise sensitivity is treated as a stable trait, consistent with related public health research (e.g., Belojević & Jakovljević, 2001; Fyhri & Klæboe, 2009; Heinonen-Guzejev, 2009; Marks & Griefahn, 2007; Schreckenberg et al., 2010; Shepherd et al., 2010; Stansfeld et al., 1993).

4.11. Noise Sensitivity and Task Performance

It is well-established that noise negatively impacts concentration and attention, and in turn, the ability of an individual to carry out a specific task (Cohen & Weinstein, 1981; Smith, 1989). Additionally, noise sensitivity can be a hindrance in performing tasks,

²⁰ Measured with the item: "Do you consider yourself to be oversensitive to noise?" (yes/no).

especially if they are undertaken in the presence of noise (Belojević et al., 2003; Belojević, Ohrström, & Rylander, 1992). Belojević et al. (1992) found that highly noise sensitive²¹ participants performed similarly to less noise sensitive participants on a variety of performance tasks (e.g., memory, spatial reasoning, mental arithmetic) when they were conducted in quiet (30 dB(A)) settings. However, when exposed to noise (≥ 55 dB(A)) during the tasks, highly noise sensitive participants performed more poorly on the short-term memory tasks and mental arithmetic.

Other studies have found a similar relationship between noise sensitivity and impaired task performance (e.g., Belojević et al., 2003; Sandrock et al., 2007; Sandrock, Schütte, & Griefahn, 2009; Sandrock et al., 2010). Interestingly, Sandrock et al. (2010) found that noise sensitivity²² negatively influenced task performance, regardless of noise exposure. The authors proposed that the tendency for noise sensitive participants to experience a greater level of mental strain (e.g., tension, lack of concentration) during tasks may have contributed to the decrement in performance.

Similarly, Smith and Stansfeld (1986) found a positive association between noise sensitivity²³ and frequency of everyday errors such as forgetting why you went from one part of the house to the other, daydreaming rather than concentrating, and having trouble

²¹ Participants were classified as “tolerant” “moderate” or “highly sensitive” based on a modified WNS.

²² Participants were classified as high and low noise sensitive based on the 50th percentile of NoiSeQ norm data (Schütte, Marks, et al., 2007).

²³ Based on scores of the McKennell (1963) “list of annoying noises” in which participants indicated which of seven common noises annoyed them (e.g., dripping tap, barking dog). Participants with scores of 5 or greater were classified as high noise sensitive, and 4 or less as low sensitive.

making up your mind. On the other hand, in their exploration of individual differences in susceptibility to the “irrelevant speech effect”²⁴, Ellermeier and Zimmer (1997) reported only a weak association between noise sensitivity²⁵ and impaired serial recall. Similarly, other studies have found no association between noise sensitivity and impaired task performance (Ljungberg & Neely, 2007; Sandrock et al., 2009)²⁶. However, in the experiment by Sandrock et al. (2009), noise sensitive participants exhibited greater annoyance and mental strain in completing the tasks.

Noise sensitivity may increase an individual’s vulnerability to impaired cognitive performance due to an easily activated sympathetic nervous system, which detracts the individual from focusing on the task at hand (for review, see Belojević et al., 2003). As demonstrated in psychophysiological experiments, individuals with elevated noise sensitivity²⁷ seem to be more easily aroused by stressors (previously discussed, see p. 75; Hebert & Lupien, 2009; Ising et al., 1980; Persson Waye et al., 2002; Stansfeld et al., 1985a). Noise sensitivity may be a significant barrier to concentration, attention, and achieving work-related tasks in a timely fashion, especially in noise-exposed environments – a finding that has direct implications in occupational settings. Further, Belojević et al.

²⁴ Difference in recall errors during speech compared to silence.

²⁵ Participants in the highest quartile (measured by the WNS and a 52-item questionnaire by Zimmer & Ellermeier; see p. 87) were contrasted with the lowest quartile.

²⁶ Noise sensitivity measured with the WNS (Ljungberg & Neely, 2007) and based on the 50th percentile of norm data of the NoiSeQ (Schütte, Marks, et al., 2007) in the experiment by Sandrock et al. (2009).

²⁷ Measured with the 10-item WNS (Weinstein, 1980). Noise sensitivity classification used in Persson Waye et al. (2002) was previously summarised (see p. 91).

(2003) proposed that the attention and performance difficulties associated with noise sensitivity may heighten anxiety, and possibly result in diminished mental health. The relationship between noise sensitivity and diminished mental health is discussed later on in this chapter (p. 94).

4.12. Noise Sensitivity and Personality

While noise sensitivity has been described as a personality trait in its own right (Anderson, 1971; Heinonen-Guzejev, 2009; Stansfeld, 1992), studies have also been conducted on the association between personality and noise sensitivity. Moreira and Bryan (1972) were among the first researchers to investigate the relationship. They assessed personality with three measures: the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1951), the Eysenck Personality Inventory (EPI; Eysenck & Eysenck, 1968), and the Rorschach Projection Test (Rorschach, 1942). Noise sensitivity, or rather, “susceptibility to noise annoyance”²⁸ as termed by the authors, was not associated with the MMPI or the EPI. However, they noted that results from the Rorschach Projection Test suggested that noise sensitive individuals were more likely to exhibit empathy, creativity and intelligence than individuals who were not noise sensitive. However, the results of the study were not replicated, likely due to the lack of psychometric reliability and validity of the Rorschach Test (Wood & Lilienfeld, 1999).

Later research in the field of personality and noise sensitivity, while limited, has largely been conducted using the framework of Eysenck’s theory of personality (e.g., Belojević & Jakovljević, 2001; Belojević, Jakovljević, & Aleksić, 1997; Campbell, 1992;

²⁸ Measured with an annoyance ranking scale to six noises. Participants rated noises as “quiet”, “noticeable”, “intrusive”, “annoying”, “very annoying” and “unbearable”.

Dornic & Ekehammar, 1990; Heinonen-Guzejev et al., 2004; Öhrström, Björkman, et al., 1988; Weinstein, 1978). However, recently Benfield et al. (2012) used the Big Five Inventory (BFI) – measuring openness to experience, conscientiousness, extraversion, agreeableness, and neuroticism – to assess the relationship between personality and noise sensitivity. Some studies have reported an association between introversion and noise sensitivity²⁹ (Benfield et al., 2012; Campbell, 1992; Dornic & Ekehammar, 1990); however, others have reported no association (e.g., Belojević et al., 1997; Griffiths & Delauzan, 1977). Additionally, Weinstein (1978) found that among his sample of students living in a college dorm, those who were noise-sensitive³⁰ were also more likely to be introverted, as assessed with the EPI. Noise sensitive students were also less comfortable in social situations and had a strong desire for privacy.

Neuroticism may be another personality factor associated with noise sensitivity. Öhrström et al. (1988) found that noise sensitivity was associated with both noise annoyance and neuroticism, as measured by the EPI. Similarly, Iwata (1984) investigated health and personality differences between high and low noise sensitive³¹ undergraduate students and found that participants who met the diagnosis for neurosis, as determined by the Cornell Medical Index, were more likely to be high noise sensitive. Further, noise-

²⁹ Measured using the WNS in Campbell (1992) and Dornic and Ekehammer (1990), measured with a 5-item version of the WNS in Benfield et al. (2012).

³⁰ Noise sensitivity was assessed using the 21-item WNS, administered prior to participants beginning the school year at Rutgers University. Noise sensitive participants ($n = 31$) were randomly selected from the top 30% of scorers, and non-noise sensitive participants were randomly selected from the bottom 30% of scorers.

³¹ Classified as high and low noise sensitive based on a scale developed by Iwata (1981).

sensitive individuals were more likely to have a maladjusted personality, as measured by the Yatabe-Guilford Personality Inventory. Specifically, noise sensitive individuals were more likely to report depressive symptoms, feelings of inferiority, nervousness, and lack of cooperativeness – characteristics often associated with neuroticism (McCrae & Costa, 1987).

More recently, in a study comparing noise sensitivity (measured with the WNS) between participants living in a noisy neighbourhood to those living in a relatively quiet neighbourhood, neuroticism emerged as the best predictor of noise sensitivity among males and females in the noisy neighbourhood, and also among females in the quiet neighbourhood (Belojević & Jakovljević, 2001). Other studies have reported correlations between neuroticism and noise sensitivity³² (Dornic & Ekehammar, 1990; Heinonen-Guzejev et al., 2004), although in these studies the relationship between the two variables disappeared after other factors (e.g., extraversion, stress, hostility) were taken into account.

³² Measured with the WNS in Dornic and Ekehammar (1990); using the single item from the Finnish Twin Cohort Study in Heinonen-Guzejev et al. (2004).

4.13. Noise Sensitivity and Negative Affectivity

Neuroticism is viewed as a personality trait that accurately captures negative affectivity³³ (McCrae, 1990; Miller, Vachon, & Lynam, 2009; Smith et al., 2002) – the tendency to experience negative emotions including anxiety, depression and anger (Watson et al., 1988). Perhaps not surprisingly then, in addition to being linked with neuroticism, noise sensitivity has also been associated with negative affective states including anger and hostility (Nivison & Endresen, 1993; Ramirez, Alvarado, & Santisteban, 2004). Based on their findings, Ramirez et al. (2004) proposed that noise stress may induce negative emotion reactions, such as anger, among noise sensitive individuals in particular.

In addition to negative affective states, noise sensitivity is also linked to general trait-based negative affectivity and related dispositions. Among the first studies on noise sensitivity, Weinstein (1978) found that noise sensitive university students (see p. 89 for sample and measurement details) in his study were more likely to report a desire for privacy, discomfort in social interactions, and annoyance to a variety of nuisances. Drawing upon these findings among university students, and further research among the general population, Weinstein (1980) proposed that noise sensitivity should be viewed as a reflection of an individual's critical appraisal of his or her environment. Weinstein (1980) proposed the concept of a 'critical-uncritical' dimension. The position on the critical-uncritical dimension indicates how an individual will perceive his or her environment with

³³ In this section negative affectivity is treated as a broad term regarding the propensity to experience negative emotions in a variety of situations. The previous section discussing neuroticism reflected research conducted on the personality trait, as defined by the researchers. Nonetheless, negative affectivity and neuroticism are closely related constructs (see Watson, Clark, & Carey, 1988).

regards to a variety of different characteristics – noise, air pollution, privacy, and safety (Weinstein, 1980). Therefore, high noise sensitivity reflects an individual's position on the 'critical' end of the spectrum.

Supporting this theoretical position, Meijer et al. (1985) found that noise sensitive participants had less appreciation for their living environment than non-noise sensitive individuals. Meijer et al. (1985) collected information on noise annoyance, noise sensitivity³⁴, sleep disturbance, and satisfaction with their home or neighbourhood from 3445 individuals living in Amsterdam, the Netherlands. Compared to less sensitive participants, those who reported being noise sensitive were more likely to report noise annoyance, sleep disturbance, and dissatisfaction with their neighbourhood and home. Similarly, in a study of 200 residents of Maarssenbroek, the Netherlands, Nijland et al. (2007) found that noise sensitive³⁵ participants reported feeling less satisfied with their home and living environment than non-noise sensitive participants.

Although Weinstein's (1980) 'critical-uncritical' dimension did not receive a lot of traction in environmental psychology or much support in noise research (e.g., Miedema & Vos, 2003; Schreckenberg et al., 2010), it could be argued that this concept relates more generally to negative affectivity. Stansfeld (1992) proposed that negative affectivity may, in

³⁴ Measured with the question: "Some people are very sensitive to sounds, others are not. In general, are you sensitive or insensitive to sounds, or are you in between?" (sensitive/in-between/non-sensitive response options).

³⁵ Assessed with a 10-item questionnaire. Noise sensitive people were classified as such if they scored 72 or higher out of a possible 100 points on the questionnaire.

fact, be a component of noise sensitivity. Additionally, Smith et al. (2002) contended that negative affectivity accounts for the association between noise sensitivity and poor health.

Using a community sample ($n = 543$) in the United Kingdom, Smith et al. (2002) measured negative affectivity with the Neuroticism Scale of the EPI, and found that the association between noise sensitivity³⁶ and mental health, measured by the General Health Questionnaire (GHQ; Goldberg, 1972), and self-reported illness disappeared when neuroticism was included as a covariate. Indeed, negative affectivity is a well-established correlate of negative physical symptoms and self-reported health (Watson & Pennebaker, 1989), and therefore remains an important covariate in noise sensitivity and health research.

4.14. Noise Sensitivity and Health

The inclusion of noise sensitivity as a personality variable in various noise-based epidemiological studies has led researchers to recognise noise sensitivity as an important health determinant in its own right. Noise sensitivity, although not often the focus of public health studies, has been linked to mental health outcomes, sleep problems, reports of subjective health complaints and health status, as well as cardiovascular disease (Booi & van den Berg, 2012; Fyhri & Aasvang, 2010; Fyhri & Klæboe, 2009; Kishikawa et al., 2009; Nivison & Endresen, 1993; Schreckenberg et al., 2010). Further, noise sensitivity has been reported as a stronger predictor of self-reported health compared to noise annoyance (Fyhri & Klæboe, 2009), thereby accentuating the importance of considering this trait in health research.

³⁶ Measured with a 7-item scale with items asking the participant's annoyance to various noise sources (e.g., knife grating on a plate, banging door, dog barking).

4.14.1. Noise sensitivity and mental health. Given the association between noise sensitivity and negative affectivity, it is not surprising that noise sensitivity has also been linked to diminished mental health (Kelly, 1986; Krog, Engdahl, & Tambs, 2010; Nivison & Endresen, 1993; Stansfeld, 1992; Tennant, 2001). Among the first studies to focus on noise sensitivity, Stansfeld (1992) explored the relationship among noise sensitivity³⁷ and stress among psychiatric patients. Compared to the controls, the depressed patients reported greater noise sensitivity. Further, as the patients' depression symptoms decreased, so did their noise sensitivity. However, noise sensitivity levels did remain relatively high. Interestingly, this trend, of mental health improvement leading to decreased noise sensitivity, was also replicated in a case study of an individual with a psychosomatic disorder (Murata & Sakamoto, 1995).

Epidemiological studies have also demonstrated an association between noise sensitivity and mental ill-health. Using the population-based Caerphilly Collaborative Survey, Stansfeld et al. (1993) found an association between noise sensitivity, assessed with the 10-item WNS (Weinstein, 1980), and psychiatric morbidity, assessed with the GHQ. A similar association had been previously reported by Tarnopolosky et al. (1978). Both studies found no interaction between noise sensitivity and noise exposure, and as such, Stansfeld et al. (1993) proposed that diminished mental health, linked with noise sensitivity, likely does not relate to noise reactions. Rather, Stansfeld et al. (1993) proposed that noise sensitivity, like trait anxiety, appears to be a stable trait relating more generally to a vulnerability to stressors and mental illness development.

³⁷ Measured with the WNS.

Other community-based studies have found a link between noise sensitivity and diminished mental health. In an unpublished doctoral thesis, Kelly (1986) investigated the associations between aircraft noise exposure, noise sensitivity³⁸, and physical and mental health among 216 older adults (aged 65 years and older). As hypothesised, noise sensitivity was associated with anxiety, depression, and diminished overall psychological health. Similarly, studies assessing the association among road traffic noise, noise sensitivity, and health outcomes have reported associations between elevated anxiety and noise sensitivity (Fyhri & Aasvang, 2010; Kishikawa et al., 2009; Nivison & Endresen, 1993; Stansfeld et al., 1993).

Recent research on the association between aircraft noise (Shepherd et al., 2010) and wind turbine noise exposure (Shepherd et al., 2011) have linked noise sensitivity³⁹ to lowered HRQOL, an important indicator of overall well-being (WHOQOL Group, 1998). More specifically, noise sensitivity was associated with diminished psychological HRQOL in both studies, further supporting the relationship between noise sensitivity and lowered mental health. Additionally, in a study conducted on the impact of noise on children in the classroom, Boman and Enmarker (2004) reported an association between noise sensitivity⁴⁰

³⁸ Measured with a 13-item modified version of the WNS.

³⁹ Measured with the NoiSeQ in Shepherd et al. (2010); measured with 1-item assessment in which participants classified themselves as “not noise sensitive”, “moderately noise sensitive” or “very noise sensitive” in Shepherd et al. (2011).

⁴⁰ Assessed with a 5-point Likert scale ranging from (1) much less sensitive to (5) much more sensitive to noise than others.

and ‘stress symptoms’, a variable that measured both mental health-related issues (e.g., irritation, tension), as well as physical complaints (e.g., headache, tiredness, energy loss).

Although noise sensitivity and lowered mental health have been linked in various studies, the nature of the relationship is unclear. Specifically, there is little evidence to suggest that noise sensitivity precedes mental ill-health. It could be that noise sensitivity and mental health are both related to a biological predisposition to enhanced stress reactivity, which, in turn, impacts both sensory processes and emotional reactions (Boyce & Ellis, 2004). Given the limited research dedicated to understanding this relationship, it is clear that further investigation on this topic is needed.

4.14.2. Noise sensitivity and sleep disturbance. Both laboratory and field studies have revealed that noise sensitivity has a considerable impact on sleep (Aasvang et al., 2008; Belojević et al., 1997; Lercher & Kofler, 1996; Nivison & Endresen, 1993; Öhrström, 1995; Öhrström, Björkman, & Rylander, 1990; Shepherd et al., 2010; Wilkinson, 1984). Marks and Griefahn (2007), in their laboratory-based study, found an association between noise sensitivity⁴¹ and decreased sleep quality. Specifically, noise sensitive individuals were more likely to exhibit increased body movements through the night, and difficulty falling asleep after being awoken. Surprisingly, there was no relationship between noise sensitivity and any of the polysomnogram parameters, such as sleep latency, wakefulness after sleep onset and sleep efficiency. However, in comparison to normative data, Marks and Griefahn (2007) noted that their 24 participants were not

⁴¹ Measured with the NoiSeQ.

particularly sensitive to noise – possibly an artefact of participation bias in this type of research.

Community-based studies have also found an association between noise sensitivity and diminished sleep quality. Noise sensitive individuals are more likely to report sleep disturbance when exposed to road traffic noise (Belojević et al., 1997; Nivison & Endresen, 1993; Öhrström, 1995; Öhrström et al., 1990; Öhrström, Rylander, & Björkman, 1988), railway noise (Aasvang et al., 2008), and aircraft noise (Shepherd et al., 2010). Additionally, other studies have found an association between noise sensitivity⁴² and the use of sleep medication and other psychotropic drugs (Heinonen-Guzejev et al., 2004).

Noise sensitivity has been linked to some negative after-effects from nocturnal noise exposure as well. After-effects of noise exposure including increased morning tiredness and decreased mood have been found to be more pronounced in noise sensitive⁴³ participants, compared to non-noise sensitive participants (Öhrström & Björkman, 1988). However, the same study found that non-noise sensitive participants were more likely to have decreased performance (reaction-time tests) following night-time noise exposure, contrasting later research on noise sensitivity and performance during noise exposure (e.g., Belojević et al., 2003; Belojević et al., 1992).

Sleep disturbance is a well-established risk factor for a range of health problems (Meerlo, Sgoifo, & Suchecki, 2008), and therefore is a particularly important variable to

⁴² Measured with the single item assessment of noise sensitivity used in the Finnish Twin Cohort Study (see p. 68).

⁴³ Participants were classified as “noise sensitive” if they reported being “quite” or “very sensitive to noise”, and “non-noise sensitive” if they considered themselves to be “not very sensitive to noise”.

consider in exploring associations among noise, noise sensitivity and health problems.

Although Fyrhi and Aasvang (2010) found an association between noise sensitivity⁴⁴ and traffic-related sleep disturbance, no association between noise or noise sensitivity and cardiovascular disease, the main health outcome of the study, was found. Similarly, Smith et al. (2002) found associations among noise exposure, noise sensitivity and health outcomes, but the relationship among the variables was largely accounted for by negative affectivity (measured by the EPI Neuroticism Scale).

Other research has investigated the relationship among noise sensitivity, sleep disturbance and mental health problems (e.g., Fyhri & Klæboe, 2009; Nivison & Endresen, 1993; Öhrström et al., 1990; Shepherd et al., 2010; Smith et al., 2002), but none of the studies explored the influence of sleep as a potential mediator of the relationship. Noise sensitive individuals may be particularly susceptible to sleep disturbance because of general increased reactivity (arousability) to stressors (e.g., Griefahn & Di Nisi, 1992; Ising et al., 1980), however, this has not been demonstrated adequately in the literature (e.g., Marks & Griefahn, 2007).

4.14.3. Noise sensitivity and cardiovascular disease. As previously discussed, the evidence for the association between noise exposure and cardiovascular disease has been mounting in the past decade (Babisch, 2011; Selander et al., 2009b). Elevated physiological arousal resulting from chronic noise exposure has been identified as the likely culprit in the development of cardiovascular disease risk among noise exposed individuals. Noise sensitivity has also been linked to an elevated stress reactivity to noise (Ising et al., 1980;

⁴⁴ Assessed with the single item “I am sensitive to noise” to which participants responded on a 6-point Likert scale ranging from “disagree strongly” to “agree strongly”.

Persson Wayne et al., 2002), and therefore, could theoretically compound the cardiovascular risk – or even be an independent risk factor.

Significant positive correlations between objectively measured blood pressure and noise sensitivity have been found in community-based studies of young to middle-aged adults (Neus, Ruddel, & Schulte, 1983), as well as older adults (Kelly, 1986). Noise sensitivity has also been linked to self-reported hypertension (Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2004), though a recent study did not find an association (Fyhri & Aasvang, 2010). Notably, Heinonen-Guzejev et al. (2007), in a recent investigation on the Finnish Twin Cohort, reported an association between noise sensitivity (see footnote on p. 68 for measurement of variable) and cardiovascular mortality among women.

Babisch (2010) recently assessed the association between noise sensitivity (assessed with the WNS) and cardiovascular health outcomes in three large epidemiological studies – the Caerphilly and Speedwell Studies, the Berlin Traffic Noise Study, and the HYENA study. Although his analyses on some of the data suggested an interaction between noise exposure and noise sensitivity in predicting cardiovascular risk, other findings suggested a protective effect of noise sensitivity. Overall, based on his aggregate findings, Babisch (2010) concluded that although noise sensitivity may increase cardiovascular risk, chronic noise exposure is a more potent risk factor. Taken together with other research on the topic (Heinonen-Guzejev et al., 2004, 2007; Kelly, 1986), noise sensitivity may be an independent risk factor for cardiovascular disease, and is therefore an important variable to consider in noise-health studies.

4.14.4. Noise sensitivity and diminished physical health. Noise sensitivity, in addition to being linked to diminished mental health and sleep disturbance, has also been linked to reports of reduced physical health. Although the association between noise

sensitivity and health is largely understudied, research points to a relationship between the two variables (Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2004, 2007; Lercher & Kofler, 1996; Nivison & Endresen, 1993). Noise sensitivity has been linked to diminished physical HRQOL among residents exposed to aircraft (Shepherd et al., 2010) and wind turbine noise (Shepherd et al., 2011). Similarly, among residents living in the vicinity of the Frankfurt airport, noise sensitivity⁴⁵ has been negatively correlated with physical health status, as measured with the SF-36 (Schreckenberg et al., 2010; Schreckenberg et al., 2010b).

Noise sensitivity has also been positively correlated with subjective health complaints. Nivison and Endresen (1993), studying a sample of 94 Norwegian residents, found an association between noise sensitivity⁴⁶ and various health complaints, as assessed with the Subjective Health Complaints Inventory (SHCI; Ursin, Endresen, & Ursin, 1988). In their study, gender significantly moderated the association. Specifically, among women, noise sensitivity was positively correlated with overall health complaints, muscle complaints, intestinal complaints, cold and flu, nervous symptoms, and heart-related complaints. Among men, noise sensitivity was only marginally associated with long-term (i.e., 3 years) allergy complaints.

A gender effect on the association between noise sensitivity and self-reported health was also found in a recent study. Babisch (2010) reported that the presence of self-reported

⁴⁵ Measured with a single 5-point item: “not”, “a little”, “moderately”, “rather”, “a lot” in Schreckenberg, Meis et al. (2010), and assessed with the NoiSeQ in Schreckenberg, Griefahn et al. (2010).

⁴⁶ Assessed with a 3 item scale.

doctor-diagnosed disease was positively associated with noise sensitivity⁴⁷ among women, but not men. Therefore, there appears to be some evidence that noise sensitive women may be particularly vulnerable to ill-health. However, some research has suggested that women report more health complaints (e.g., Gobina et al., 2011; Indregard, Ihlebaek, & Eriksen, 2012; Koopmans & Lamers, 2007) and greater noise sensitivity than men (e.g., Ellermeier et al., 2001; van Kamp et al., 2004), which suggests that it is possible that this finding may be an artefact of gender bias in health care seeking and symptom reporting.

Other recent investigations have also reported on the relationship among road traffic noise exposure, noise sensitivity and health complaints. In a large study of 1842 residents of Oslo, Norway, Fyhri and Klæboe (2009) found that noise sensitivity⁴⁸ was associated with reports of nervousness, weariness, sore throat, headache, high blood pressure, and chest pain. Intriguingly, their results suggested no direct relationship between road traffic noise exposure and cardiovascular health, but rather, Fyrhi and Klæboe (2009) proposed that noise sensitivity may account for the association.

Similarly, in an investigation of the health and environmental disturbances associated with aircraft noise, Schreckenberget al. (2010) found an association between noise sensitivity and a variety of health complaints. Specifically, noise sensitivity was significantly linked to reports of exhaustion, stomach complaints, limb complaints, and cardiac complaints. It should be noted, however, that self-reported health complaints garner some criticism such as the possibility of incongruence between actual health and reported

⁴⁷ Measured with the WNS.

⁴⁸ Measured with the 3-point single item “Would you say you are highly, somewhat or not sensitive to noise?”

health (e.g., Johnston, Propper, & Shields, 2009). Additionally, some people have stated that self-reports of physical complaints are merely an artefact of negative affectivity (Watson & Pennebaker, 1989). However, in addition to being a cost-effective way to gather health status information in epidemiological studies, Eriksen and Ursin (2002) suggest that health complaints, because of their impact on daily functioning and productivity, are an important health outcome in their own right.

Chapter 5: Study 1 – Noise Sensitivity and Self-Reported Health: An Investigation of Potential Mediators and Moderators

5.1. Introduction

Stansfeld (1992) described noise sensitivity as a trait that increases one's vulnerability to noise and other stressors. There is evidence to suggest that noise sensitive individuals have a lower threshold for physiological stress reactivity (e.g., Griefahn & Di Nisi, 1992; Ljungberg & Neely, 2007; Persson Waye et al., 2002), in addition to having a greater emotional response to stressors (Ljungberg & Neely, 2007; Nivison & Endresen, 1993; Stansfeld, 1992; Weinstein, 1978). Chronic activation of the stress response has been implicated in the development of both physical (Barnes & Adcock, 2009; Cohen et al., 2007) and mental illness (Hardoy et al., 2005), and therefore one possible explanation for the association between noise sensitivity and diminished health is the bodily wear and tear associated with chronic stress (McEwen & Stellar, 1993).

As discussed in Chapter 4 (p. 93), noise sensitivity may be an important health risk factor for the development of mental and physical health problems. However, the mechanisms of this relationship remain understudied. Using the allostatic load framework (see p. 14; Figure 4, p. 53), this study tested a model of noise sensitivity and diminished health that was developed through identification of potential mediators and moderators in the literature (see Figure 5, p. 105). In developing the model for the present study, perceived stress and sleep problems were identified as factors that can impact health and well-being (Juster et al., 2010; Lahey, 2009; Segerstrom & Miller, 2004; Zaharna & Guilleminault, 2010), and thus, were tested as potential mediators in the overall model. The psychological and health effects of noise sensitivity may differ between males and females; therefore, gender was tested as a moderator. Finally, while some studies suggest that noise

sensitivity is a health risk factor independent of actual environmental noise exposure (Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2004, 2007; Schreckenberg, Griefahn, & Meis, 2010a), an interaction between noise exposure and noise sensitivity has been reported (Kishikawa et al., 2009). Therefore, noise exposure was tested in the model as a moderator.

It was hypothesised that the relationship between noise sensitivity and diminished health, assessed through self-reported health complaints and hypertension in this study, would be explained by perceived stress and sleep problems – both previously identified as risk factors for health problems (Juster et al., 2010; Lahey, 2009; Segerstrom & Miller, 2004; Zaharna & Guilleminault, 2010). Specifically, it was hypothesised that as noise sensitivity increases, so does the experience of perceived stress, which, in turn, contributes to health problems. It was also hypothesised that noise sensitivity would be associated with sleep problems, which would increase the likelihood of developing health problems. These mediation relationships may differ depending on noise exposure, in that the effect of noise sensitivity on sleep problems may be worse among individuals exposed to environmental noise. The mediation relationship may also differ across gender, as females have previously reported greater health problems (Heinonen-Guzejev et al., 2007) and noise sensitivity compared to males (Nivision & Endresen, 1993; van Kamp et al., 2004). Therefore the relationship between noise sensitivity and mediators may be stronger among females compared to males.

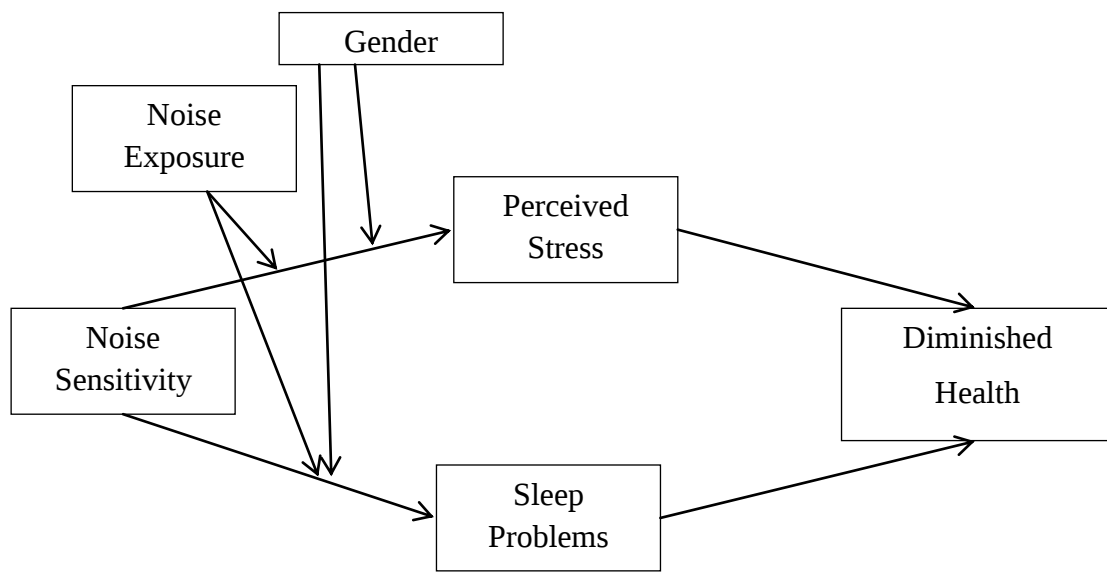


Figure 5. Noise sensitivity and diminished health model.

Note. The generic term ‘diminished health’ is used to display the overall model. Four health outcomes were assessed: subjective health complaints, hypertension, anxiety complaints and depression complaints.

5.2. Method

5.2.1. Participants. Participants were residents of Auckland and surrounding suburbs. Questionnaires were hand-delivered between April and August 2011 to letterboxes of noise-exposed households (situated near high traffic volume roads) and households in control areas (not situated near high road traffic volume roads; information on traffic data is presented in the procedure, see p. 110). Due to sampling being based on the participant's proximity to arterial roads, it should be noted that the sample was not representative of the Auckland population. The study was approved by the AUT Ethics Committee (reference: 10/271; see Appendix A, p. 250). Participants were informed on the Participant Information Sheet and Questionnaire that by completing the questionnaire anonymously, they were providing consent for participation in the study (see Participation Information Sheet in Appendix B, p. 252). On the information sheet provided, residents of the households were invited to participate in the study if they were aged 18 years or older.

5.2.2. Measures

5.2.2.1. Noise sensitivity. A 3-item noise sensitivity scale was used in Study 1. Previous noise studies have used 3-item measures to assess noise sensitivity (e.g., Amann et al., 2007; Nivison & Endresen, 1993), and the brevity of such scales helps diminish burden on participants (Amann et al., 2007). After being in contact with Dr. Peter Lercher, a co-author of the Amann et al. (2007) paper, a translated version of the German scale (3-NS) was sent for use in Study 1.

The 3-NS involves three statements to which participants respond on Likert scales ranging from (1) completely agree to (5) completely disagree. This Likert format was slightly modified from the 4-point scale used in the original study (Amann et al., 2007) in order to maintain consistency with other noise perception items in the overall questionnaire

(discussed on p. 109). A total score is calculated by summing the Likert scale ratings for the three items. Amann et al. (2007) reported good internal consistency and validity for the scale. In the present study, internal consistency of the 3-NS was adequate (Cronbach's $\alpha = 0.68$). Refer to Appendix C (p. 254) for the questionnaire.

5.2.2.2. Subjective health complaints. The Subjective Health Complaints Inventory (SHCI) is a 29-item scale that measures the intensity and frequency of common health complaints in the last 30 days (Eriksen, Ihlebaek, & Ursin, 1999; Ursin et al., 1988). The questionnaire collects information regarding musculoskeletal pain (headaches, neck pain, lower back pain, arm pain, shoulder pain, migraine and leg pain), pseudoneurological complaints (extra heart beats, hot flushes, sleep problems, tiredness, dizziness, anxiety and sadness/depression), gastrointestinal problems (heartburn, stomach discomfort, diarrhoea, constipation), allergy complaints (asthma, breathing difficulties, eczema, allergies and chest pain), and flu complaints (colds or flu, cough). The scale has been used previously in investigations on noise exposure, noise sensitivity, and health (Fyhri & Aasvang, 2010; Nivison & Endresen, 1993).

For each health complaint item, participants rated the extent to which they were suffering from the condition (the severity rating) on a 4-point Likert scale ranging from 0 (not at all) to 3 (seriously), and they also indicated the number of days they were suffering from the health problem in the past month. There are a variety ways to score the SHCI (see Eriksen et al., 1999). A composite score for the scale is computed by adding the total severity ratings for all the items. In the present study, the sleep problems severity rating was used as a separate mediator variable in the analyses. Therefore, the total health complaints score was calculated by summing the severity ratings of the remaining 28 items, a method similarly employed in previous noise sensitivity and health research (e.g., Fyhri

& Aasvang, 2010)⁴⁹. The anxiety and depression severity ratings were also treated as outcome variables in the analyses. The questionnaire has also been used in large samples and has been reported to have adequate validity and reliability (Eriksen et al., 1999; Ihlebaek, Eriksen, & Ursin, 2002). In this study, the overall scale had good internal consistency (Cronbach's $\alpha = 0.83$). Refer to Appendix D (p. 255) for the complete questionnaire.

5.2.2.3. Perceived stress. The Perceived Stress Scale (PSS) is a 10-item self-report measure of psychological stress that was designed for community samples with at least intermediate school level education (Cohen & Williamson, 1988). It measures the extent to which participants consider their lives to have been unpredictable, unmanageable and generally stressful within the past month. In completing the PSS, participants replied to each question on a 5-point Likert scale ranging from 0 (never) to 4 (very often). An example question is: "In the last month, how often have you found that you could not cope with all the things you had to do?". Scores are calculated by summing the Likert scale scores after appropriately coding each item. The scale is psychometrically sound, exhibiting adequate reliability and validity (Cohen & Williamson, 1988). In the present study, the PSS had excellent internal consistency (Cronbach's $\alpha = 0.87$). Refer to Appendix E (p. 256) for the complete questionnaire.

5.2.2.4. Neuroticism. The Neuroticism Scale of the Big Five Inventory (John, Donahue, & Kentle, 1991) was used to collect information on this personality trait. For each of the eight items of the scale, participants were asked to respond on a Likert scale

⁴⁹ Fyhri and Aasvang (2010) used 27 of the 29 items of the Subjective Health Complaints Inventory in their study.

ranging from (1) strongly disagree to (5) strongly agree. The total neuroticism score is calculated by taking the mean Likert scale rating across the items. As previously discussed (see p. 93), Smith et al. (2002) suggested that the associations between noise sensitivity and health problems are accounted for by negative affectivity. Therefore, assessing negative affectivity alongside noise sensitivity was particularly important in the present study. The use of a neuroticism scale to assess negative affectivity was applied in the noise sensitivity research conducted by Smith et al. (2002), and has been used in other health and psychological studies (e.g., Bouchard & Poirier, 2011; Pasch, Bradbury, & Davila, 1997; Shackman et al., 2011). Good internal consistency (.75-.90), test-retest reliability and validity have been previously reported for the BFI (Pervin & John, 2001). The Neuroticism Scale had acceptable internal consistency in the present study (Cronbach's $\alpha = 0.76$). Refer to Appendix F (p. 257) for scale items.

5.2.2.5. Noise perception and residence questions. Both objective and self-reports of noise exposure are important considerations, especially in studies pertaining to noise sensitivity (Heinonen-Guzejev et al., 2000). Participants were asked to rate the noisiness of their home and work environments, the controllability of the noisiness in their home and work environments, and their annoyance to the noise in their home and work environments (total of 6 items). Participants responded to each question on a 5-point Likert scale. Additionally, participants were asked to indicate the length of time they have lived in their current residence (years), the length of time that they have been working at their current workplace (years), and approximately how many hours per day they spend at home and work. Refer to Appendix G (p. 258) for the questions.

5.2.2.6. Demographics and lifestyle questions. The questionnaire included items regarding gender, age, ethnicity, level of education, and current employment status.

Participants were also asked to indicate their current smoking status (non/ex/occasional/daily), and whether they currently suffer from hypertension (yes/no), a variable that has been linked to both noise exposure and noise sensitivity (e.g., Babisch, 2006; Heinonen-Guzejev et al., 2004; van Kempen & Babisch, 2012). Refer to Appendix H (p. 259) for the questions from the demographics and lifestyle section (7 items).

5.2.3. Procedure. Traffic volumes of Auckland's arterial and side roads were determined through maps and data collected during 2009 by Auckland City Council. The traffic volumes were used as a proxy for road traffic noise exposure in the present study, a method used in previous noise-related community-based research (e.g., Willich et al., 2006). Prior to distribution to households, questionnaires were coded according to traffic volume exposure (see Table 1). All houses included in the noise-exposed conditions (defined as > 10,000 vehicles/24 hour) had at least one side of the house within 20 metres of the road (determined with the use of google maps), as residents of households within 20 metres of an arterial road have previously reported ill-effects of noise exposure (e.g., Kageyama et al., 1997). Questionnaires (7500) were hand-delivered to letterboxes of households in Auckland (see Table 1) along with a postage paid envelope; 1106 were returned (response rate: 14.75%). The questionnaire took approximately 10 minutes to complete.

Table 1. *Questionnaires Delivered and Returned across Road Traffic Volume Groups*

Road Traffic Volume (24 hour period)	Questionnaires Delivered	Questionnaire Returned
< 10,000	2000	466 (44.8% ^a)
10,000 – 20,000	1700	158 (14.3%)
20,000 – 30,000	1600	250 (22.6%)
> 30,000	2200	189 (17.1%)
Missing*	-	13 (1.2%)
Total	7500	1106 (100%)

^aPercentage of total questionnaires returned.

**Note.* Water damage on the coversheet of 13 returned questionnaires prevented knowledge of original traffic volume group.

5.3. Results Overview

For the purpose of providing detailed information about both the characteristics of the sample and in addressing the goals of the study, the results are presented in two sections. The first section of the results provides general information on the sample. Descriptive statistics of the sample are provided. Additionally, information on health variables, psychological variables, noise perceptions, gender differences, and bivariate correlations are presented in order to describe the study sample. The second section of the results involves the testing of the health outcome models (the initial model is presented in Figure 5, p. 105). The description of the statistical methods and the results of the analyses are described in Section 2 (see p. 124).

5.3.1. Summary of data screening and preliminary analyses. Prior to conducting the analyses, data were screened for data entry errors, missing values, and regression assumptions. Preliminary analyses were also conducted to assess whether delivery season (i.e., winter or autumn) influenced participant responses. Details of the analyses are presented in

Analyses were conducted in SPSS (Statistical Package for the Social Sciences) version 19.0. For each full-length questionnaire (i.e., 3-NS, PSS, Neuroticism Scale of BFI, SHCI), any case that had missing values for more than 30% of the scale was deleted for that scale. For the remaining cases, missing value analysis (MVA) was conducted using the Little's MCAR test to determine whether values were missing completely at random (see Appendix L, p. 267, for details). Expectation Maximisation (EM) was used to estimate values that were missing at random (see Newman, 2003; Schafer & Graham, 2002). After removing four multivariate outliers, determined through the use of Mahalanobis distance

(Tabachnick & Fidell, 2007), the final sample size for the analyses was 1102. Listwise deletion was applied to the analyses so that only cases with complete data on all variables were used.

5.4. Results Section 1. Descriptive Statistics and Characteristics of the Sample

5.4.1. Descriptive statistics. The final sample used in the main analyses included 1102 participants who ranged in age from 18 to 94 years ($M = 51.39$, $SD = 16.42$). Table 2 provides descriptive statistics for the demographic information. The majority of the sample was female (64.7%) and New Zealand European (64.9%). A small subsection reported belonging to more than one ethnic group⁵⁰ ($n = 51$, 4.6%). In particular, 29 participants identified as both New Zealand European and Māori (2.6%). These participants are presented as belonging to the Māori ethnic group in Table 2. Seven participants (.6%) identified as both New Zealand European and Pacific Islander (i.e., Samoan, Cook Island Māori, Tongan, or Niuean). Similarly, these participants are presented as belonging to the Pacific Islander ethnic group in Table 2. The remaining participants (15) who identified as belonging to two ethnic groups are classified as belonging to the minority ethnic group in Table 2 (i.e., not New Zealand European/European). If the participant identified as a member of two minority groups, the participants are classified as the first ethnicity group listed in the questionnaire (see Appendix H, p. 259). Compared to NZ census data, people of European decent were overrepresented in the sample (56.5% of Auckland population

⁵⁰ It should be noted that ethnicity information was collected by self-report and therefore reflects the self-designated identity of the participant. This section of the thesis was written to give a broad description of the sample.

vs.74.1%⁵¹ of the sample), while Asian (18.9% of Auckland population vs. 13.9%⁵² of the sample), Pacific (14.4% of Auckland population vs. 3.1% of the sample), and Māori (11.1 % of Auckland population vs. 4.8% of the sample) ethnic groups were underrepresented (Statistics New Zealand, 2006b).

Over half of the sample had completed some university post-secondary education (51.0%). Compared to NZ census data, which indicated that 40% of the New Zealand population has a post-secondary school qualification (Statistics New Zealand, 2006c), the sample was well-educated (71.2% had completed university or polytechnic education). Most participants reported having either full-time (45.3%) or part-time employment (18.8%), and approximately one-fifth of participants were retired (19.4%). Descriptive statistics on the number of years the participants have been living in their home, the average hours they spent at home and work each day, and the length of time they had been at their current place of employment are reported in Table 3.

⁵¹ Including individuals identifying as NZ European, European, North American, or Australian ethnic groups.

⁵² Including individuals identifying as Chinese, Indian, or Other Asian ethnic groups.

Table 2. *Socio-demographics of the Study 1 Sample*

Variable	Category	<i>n</i>	(%)	(Valid %) ^a
Gender	Valid			
	Male	367	(33.3)	(34.0)
	Female	713	(64.7)	(66.0)
	Total	1080	(98.0)	(100.0)
	Missing	22	(2.0)	
	Total	1102	(100.0)	
Ethnicity	Valid			
	NZ European	715	(64.9)	(65.7)
	Māori	53	(4.8)	(4.9)
	Pacific Islander	34	(3.1)	(3.1)
	Chinese	46	(4.2)	(4.2)
	Indian	58	(5.3)	(5.3)
	European	79	(7.2)	(7.3)
	North American	9	(.8)	(.8)
	Middle Eastern	11	(1.0)	(1.0)
	African	10	(.9)	(.9)
	Australian	13	(1.2)	(1.2)
	Central/South American	3	(.3)	(.3)
	Other Asian	48	(4.4)	(4.4)
	Other	10	(.9)	(.9)
	Total	1089	(98.8)	(100.0)
	Missing	13	(1.2)	
	Total	1102	(100.0)	
Education (Completed)	Valid			
	Secondary school	289	(26.2)	(26.9)
	Polytechnic	223	(20.2)	(20.8)
	University	562	(51.0)	(52.3)
	Total	1074	(97.4)	(100.0)
	Missing	28	(2.6)	
	Total	1102	(100.0)	
Employment Status	Valid			
	Full-time work	499	(45.3)	(45.6)
	Part-time work	207	(18.8)	(18.9)
	Retired	214	(19.4)	(19.6)
	Student	59	(5.4)	(5.4)
	Unemployed	31	(2.8)	(2.8)
	On leave or sick-leave	15	(1.4)	(1.4)
	Own household work	63	(5.7)	(5.8)
	Other	6	(.5)	(.5)
	Total	1094	(99.3)	(100.0)
	Missing	8	(.7)	
	Total	1102	(100.0)	

^a valid percentages are presented to display frequencies for available data (excluding missing data)

Table 3. *Descriptive Statistics for Residential and Employment Information*

Variable	<i>n</i>	Mean (<i>SD</i>)	Minimum	Maximum
Years at current residence	1102	12.8 (12.1)	0	73
Hours spent at home per day	1070	15.8 (3.8)	5	24
Hours spent at work per day	807	8.5 (3.7)	0	24*
Years at current workplace	839	7.5 (7.7)	0	50

**Note.* Participants who worked full-time at home reported spending the majority of the day at their work (also their home environment).

5.4.2. Health and psychological variables. Descriptive statistics are presented for the health and psychological variables in Table 4 – including noise sensitivity, perceived stress, neuroticism, and subjective health complaints. In terms of smoking behaviours, the large majority of the sample (75.4%) reported being non-smokers. The rest of the sample reported being ex-smokers (14.8%), occasional smokers (3.4%) or daily smokers (4.5%). A small percentage of the sample (1.9%) did not provide information on their smoking status. While most of the sample (81.2%) did not report having hypertension, 190 participants (17.2%) reported having the condition (1.6% of the sample did not indicate whether they had hypertension or not).

Table 4. *Descriptive Statistics for Health and Psychological Variables*

Variable	<i>n</i>	Mean (<i>SD</i>)	Range	Possible Range
Noise Sensitivity	1088	8.95 (2.66)	3-15	3-15
Perceived Stress	1087	15.38 (6.33)	0-36	0-40
Neuroticism	1074	2.55 (.77)	1-5	1-5
Subjective Health Complaints	1088	11.17 (8.82)	0-75	0-84
Sleep Problems	1088	.84 (.98)	0-3	0-3
Anxiety	1086	.58 (.84)	0-3	0-3
Depression	1088	.58 (.81)	0-3	0-3

Table 5 displays the frequency of self-reports of any (> 0) or serious (3) health complaints. Participants seldom reported severe (3) health complaints. However, 10.8% of the sample did report seriously suffering from the health complaint ‘tiredness’ in the past 30 days. Further, 62.3% of participants reported suffering from tiredness in the past 30 days making it the most prevalent health complaint. Over one-third of the sample reported suffering, at least a little, from the following health conditions: headache (50.8 %), neck pain (38.9%), pain in the lower back (41.7%), gas (33.8%), sleep problems (42.9%), anxiety (35.3%), and sadness/depression (36.9%).

Table 5. *Frequency of Any (> 0) or Serious (3) Subjective Health Complaints*

	Health Complaint	<i>n</i>	Any (%)	Severe (%)
1	Cold/flu	1088	28.4	4.0
2	Cough/bronchitis	1088	20.4	1.8
3	Asthma	1089	7.2	1.1
4	Headache	1088	50.8	3.3
5	Neck pain	1086	38.9	3.3
6	Pain – upper back	1088	27.2	2.8
7	Pain – lower back	1088	41.7	6.5
8	Pain – arms	1088	17.7	2.1
9	Pain – shoulders	1086	28.4	3.7
10	Migraine	1089	7.3	2.1
11	Extra heart beats	1088	12.6	1.0
12	Chest pain	1089	8.5	.4
13	Breathing difficulties	1089	11.3	1.4
14	Pain in feet during exercise	1087	20.3	2.4
15	Heart-burn	1088	19.3	1.3
16	Stomach discomfort	1089	29.7	2.1
17	Gastritis	1088	7.5	1.1
18	Stomach pains	1088	14.5	2.0
19	Gas	1089	33.8	2.1
10	Diarrhoea	1088	17.5	.9
21	Constipation	1087	16.8	1.6
22	Eczema	1089	8.6	1.4
23	Allergic skin problems	1088	12.9	2.2
24	“Flushes”/heat sensations	1087	15.6	2.1
25	Sleep Problems	1088	42.9	7.1
26	Tiredness	1087	62.3	10.8
27	Dizziness	1088	15.6	1.3
28	Anxiety	1086	35.3	3.5
29	Sad/Depressed	1088	36.9	3.1

Note. Actual percentages are presented. Approximately 1% of data was missing for each item.

5.4.3. Noise and noise-related variables.

Frequency statistics for the noise perception variables are presented in Table 6. Only 27.2% of participants reported that their home environment was “quite noisy” or “very noisy”, and only 13.0% of participants reported being “quite” or “very annoyed” by noise in the home environment. Most participants (81.1%) reported having at least some control over the noise in their home environment. The majority of participants (53.2%) reported their workplace environment

was “not noisy” or “not especially noisy”, and similarly, many participants (66.6%) were “not annoyed” or “not especially annoyed” by noise at work.

Table 6. *Noise Perceptions in the Home and Work Environment*

Noisiness	<i>Not noisy</i>	<i>Not especially noisy</i>	<i>Somewhat noisy</i>	<i>Quite noisy</i>	<i>Very noisy</i>
At home	107 (9.8%)	361 (33.2%)	324 (29.8%)	217 (19.9%)	79 (7.3%)
At work ^a	119 (12.7%)	380 (40.5%)	219 (23.3%)	165 (17.6%)	56 (5.9%)
Annoyed by Noise	<i>Not at all</i>	<i>Not especially</i>	<i>Somewhat</i>	<i>Quite</i>	<i>Very</i>
At home	152 (14.0%)	519 (47.7%)	275 (25.3%)	110 (10.1%)	32 (2.9%)
At work ^a	171 (18.6%)	442 (48.0%)	228 (24.8%)	65 (7.1%)	14 (1.5%)
Perceived Control over Noise	<i>No control</i>	<i>Very little</i>	<i>Some</i>	<i>Quite a bit</i>	<i>A lot</i>
At home	22 (2.0%)	184 (16.9%)	456 (41.8%)	307 (28.2%)	121 (11.1%)
At work ^a	134 (14.5%)	280 (30.3%)	281 (30.5%)	175 (19.0%)	53 (5.7%)

^aA subsection of the sample was used for the workplace statistics (those who were currently working full-time or part-time). Valid percentages are presented.

A Spearman’s *rho* correlation was conducted to assess the association between self-reported noisiness of the home environment and the noise level category (as determined by the Auckland City Council road traffic information). A cross-tabulation of the variables is presented in Table 7. A significant relationship was observed between traffic volume and perceived noise exposure in the home environment, $\rho = .253$, $p < .001$. Perceptions of noisiness (quite noisy, very noisy) were more frequently reported among participants with homes in high traffic areas (> 20,000 vehicles/24 hours), while those participants exposed to less traffic were more likely to report a home environment that is “not noisy” or “not especially noisy”, thus suggesting that the use of arterial road traffic volume was a reasonable proxy measure of noise exposure in the home environment. Additionally, noise

sensitivity was not significantly correlated with noise exposure, $\rho = -.021$, $p = .484$, similar to previous research (Babisch, 2010; van Kamp et al., 2004).

Table 7. *Cross-tabulation of Perceptions of Home Noisiness and Road Traffic Volume near Home*

	Road Traffic Volume (24 hour)			
	30,000+	20,000 - 30,000	10,000- 20,000	Less than 10,000
Not at all noisy	9 (4.8%)	21 (8.5%)	12 (7.7%)	65 (13.2%)
Not especially noisy	51 (27.0%)	50 (20.3%)	57 (36.5%)	203 (41.1%)
Somewhat noisy	53 (28.0%)	84 (34.1%)	44 (28.2%)	141 (28.5%)
Quite noisy	45 (23.8%)	68 (27.6%)	36 (23.1%)	67 (13.6%)
Very noisy	31 (16.4%)	23 (9.3%)	7 (4.5%)	18 (3.6%)
Total	189 (100%)	246 (100%)	156 (100%)	494 (100%)

5.4.4. Gender differences. Due to the unequal number of males and females in the study (367 males; 713 females), a non-parametric test (Mann-Whitney U) was used to assess gender differences across variables. A Bonferroni correction was applied because of the number of tests conducted (12); the new critical alpha level was determined to be .004. Overall, females in the study were more noise sensitive than males, and they reported more subjective health complaints and more anxiety complaints (Table 8).

Table 8. *Gender Differences across Noise and Health Variables*

Variable	Mann-Whitney <i>U</i>	<i>p</i>	Mean (SD)	
			Males	Females
Noise Sensitivity	143,525.0	<.001	8.55 (2.68)	9.16 (2.63)
Perceived Stress	133,166.5	.350	15.11 (6.45)	15.5 (6.31)
Neuroticism	140,055.5	.017	2.46 (.77)	2.60 (.77)
Subjective Health Complaints	149,730.0	<.001	10.32 (7.86)	12.76 (8.93)
Sleep Problems	134,297.5	.165	.78 (.93)	.89 (1.00)
Anxiety Complaints	140,100.0	.003	.48 (.90)	.63 (.86)
Depression Complaints	137,443.0	.027	.54 (.85)	.61 (.80)
Perceived Noise (Home)	134,750.0	.203	2.75 (1.06)	2.85 (1.10)
Noise Annoyance (Home)	130,583.0	.615	2.39 (.93)	2.41 (.96)
Perceived Control (Home)	124,892.5	.362	3.33 (.91)	3.28 (.96)
Perceived Noise (Work)	100,103.5	.451	2.61 (1.17)	2.64 (1.05)
Noise Annoyance (Work)	96,383.0	.449	2.22 (.92)	2.26 (.87)
Perceived Control (Work)	91,450.0	.388	2.76 (1.15)	2.68 (1.08)

5.4.5. Correlations. Bivariate correlations among the noise, health and psychological variables are presented in Table 9. Age was also included in the correlation matrix. All health, psychological, and noise-related variables were significantly correlated. Noise sensitivity was positively correlated with perceived stress ($r = .235, p < .001$), health complaints ($\rho = .202, p < .001$), anxiety complaints ($\rho = .156, p < .001$), and depression complaints ($\rho = .153, p < .001$); however, the correlations were weak. Noise sensitivity had the strongest correlation with annoyance to noise in the home environment ($\rho = .404, p < .001$). Perceived stress was moderately correlated with neuroticism ($r = .657, p < .001$) as well as health complaints ($\rho = .504, p < .001$).

Table 9. *Bivariate Correlations among Stress, Neuroticism, Health, and Noise Variables*

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. 3-NS													
2. Stress	.235**												
3. Neurot	.270**	.657**											
4. SHC	.202**	.504**	.441**										
5. Sleep Problems	.148**	.283**	.289**	.389**									
6. Anxiety	.156**	.501**	.480**	.539**	.380**								
7. Depression	.153**	.553**	.509**	.499**	.328**	.541**							
8. Work Noise	.146**	.203**	.149**	.218**	.118**	.161**	.164**						
9. Work Annoy	.350**	.298**	.201**	.266**	.175**	.192**	.243**	.465**					
10. Work Control	-.078*	-.200**	-.203*	-.129**	-.084*	-.085*	-.120**	-.260**	-.188**				
11. Home Noise	.126**	.241**	.174**	.177**	.070*	.152**	.175**	.194**	.196**	-.126**			
12. Home Annoy	.404**	.289**	.237**	.251**	.181**	.203**	.256**	.187**	.391**	-.136**	.474**		
13. Home Control	-.091*	-.254**	-.205**	-.143**	-.072*	-.167**	-.180**	-.149**	-.159**	.303**	-.444**	-.335**	
14. Age	.001	-.199**	-.112**	-.110**	.088**	-.031	-.129**	-.136**	-.021	.143**	-.232**	-.133**	.203**

Note. 3-NS = noise sensitivity (Amann et al., 2007), Stress = Perceived Stress Scale (Cohen et al., 1988), Neurot = Neuroticism Scale of BFI (John et al., 1991), SHC = Subjective Health Complaints Inventory Composite Severity Score (Eriksen et al., 1999), Sleep Problems = Sleep Problems item from SHCI (Eriksen et al., 1999), Anxiety = Anxiety Complaints item from SHCI; Depression = Depression Complaints item from SHCI; Work Noise = perceived noise exposure at work, Work Annoy = annoyance to noise at work, Work Control = perceived control over noise at work, Home Noise = perceived noise exposure at home, Home Annoy = annoyance to noise at home, Home Control = perceived control over noise at home. Correlations listed under SHC, Sleep Problems, Anxiety, Depression, Work Noise, Work Annoy, Work Control, Home Noise, and Home Annoy have been calculated using Spearman's ρ for non-parametric data. All others are Pearson's r correlations.

** $p < .001$ * $p < .05$

5.5. Results Section 2. Testing Models of Noise Sensitivity and Diminished Health

5.5.1. Statistical approach. In order to test the model of the relationship between noise sensitivity and diminished health (see Figure 5, p. 105), indirect effects were tested using bootstrapping estimates for 5000 resamples (see Preacher & Hayes, 2008; Preacher, Rucker, & Hayes, 2007). The bootstrapping method involves resampling the data in order to obtain an estimate of the indirect effect (ab). Preacher and Hayes (2008) recommend using 5000 resamples for main analyses. The bootstrapping statistical method has considerable advantages compared to previous mediation approaches such as Baron and Kenny (1986) and the Sobel test (Hayes, 2009). The method is a nonparametric procedure, and therefore does not assume a normal sampling distribution of the indirect effect (unlike the Sobel test; Hayes, 2009). Additionally, multiple mediators can be tested in the model, thus reducing the risk of type 1 errors. Mediation models were tested using the INDIRECT SPSS macro (Preacher et al., 2007) and the moderated mediation model was tested using the PROCESS SPSS macro for 5000 samples provided by Preacher and Hayes (2008)⁵³.

The INDIRECT macro calculates coefficients for the unique pathways among the independent variable (IV), the dependent variable (DV) and the mediator variable(s) (MV) in the model (see Figure 7 as an example, p. 130). Coefficients are calculated for the total effect (c path; the association between IV and DV), the direct effect (c' path; the association between IV

⁵³ Instructions for the SPSS procedures are provided in the cited articles by Preacher and Hayes (e.g., Preacher & Hayes, 2008; Preacher et al., 2007). Further information and the macro download is available from Dr. Andrew Hayes's website: <http://www.afhayes.com/spss-sas-and-mplus-macros-and-code.html>

and DV after controlling for MVs), and the indirect effect (*ab* path; the influence of the MV in the association between IV and DV). The output of the macro provides unstandardised regression coefficients and associated *p* values, as well as bias corrected and accelerated confidence intervals (BCa CI) for the indirect effects. A significant indirect effect occurs when the confidence interval does not cross zero. Additionally, the macros calculate pairwise contrasts of the indirect effects, and therefore the magnitude of the indirect effect through each of the mediators can be compared (Preacher & Hayes, 2008).

The PROCESS macro allows for conditional process modelling to be conducted on a dataset – commonly referred to as mediated moderation or moderated mediation regression analyses (Hayes, 2012). It allows for assessing moderator effects in mediation models, such as that in Figure 6 (p. 127). In this study, the moderating influence of gender and noise exposure (assessing using road traffic volume as a proxy) can be examined in the model. The PROCESS macro produces unstandardised regression coefficients and associated *p* values for predictor variables as well as interactions between the moderator and select model variables. Conditional indirect effects and corresponding bias corrected confidence intervals are also generated in the output.

5.5.1.1. Controlling for Confounding Variables. Neuroticism was a particularly important covariate to include in the models as negative affectivity is closely related to noise sensitivity (Smith et al., 2002; Stansfeld, 1992). Additionally, for any models testing the influence of personality and environmental variables on health, it is theoretically advantageous to control for the influence of age and socioeconomic status (Pickett & Pearl, 2001). In the present study, education was used as a proxy for socioeconomic status, as the collection of such information is often preferred over asking participants to indicate income or value of their assets

(see Grzywacz, Almeida, Neupert, & Ettner, 2004). Age was also entered as a covariate in each of the models.

5.5.2. Noise sensitivity and health complaints moderated mediation model. The overall model (Figure 6) was significant, $F(6, 1008) = 91.76, p < .001, R^2 = .353$. The moderated regression analyses (i.e., the influence of gender/noise exposure) revealed that the *noise sensitivity x noise exposure* and *noise sensitivity x gender* interactions were not significant in predicting the model mediators (see Table 10; interaction p values $> .05$). Because of the non-significant interaction between noise sensitivity and the moderators, the conditional indirect effects were not examined (Preacher et al., 2007). The indirect effect of noise sensitivity on health complaints through perceived stress and sleep problems did not depend on noise exposure or gender.

The overall model was also conducted with perceived noise exposure as a moderator in place of objective noise exposure (as estimated by road traffic volume). This analysis was completed to determine whether noise perceptions moderated the relationship between noise sensitivity and each of the mediators. The interaction between noise sensitivity and the moderators did not change significantly in this model (i.e., the results did not change from that presented in Table 10). Results of this analysis are summarised in Appendix M (p. 288).

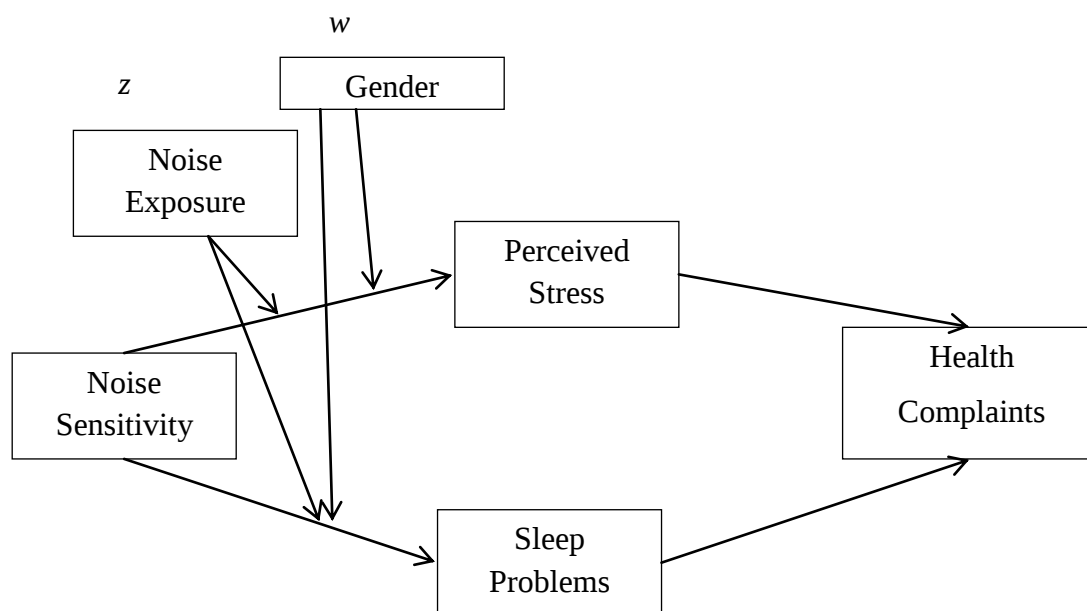


Figure 6. Noise sensitivity and health complaints moderated mediation model.

Note: Noise Exposure (*z*) was treated as an ordinal variable in the model, and Gender (*w*) was a dichotomous variable.

Covariates: neuroticism, age, education

n = 1015

Table 10. *Moderated Regression Results for the Effect of Gender and Noise Exposure on the Relationship between Noise Sensitivity and Perceived Stress and Sleep Problems*

	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>
Moderated Multiple Regression of Noise Sensitivity on Perceived Stress				
Noise Sensitivity	.244	.222	1.101	.271
Noise Exposure	-.350	.435	-.805	.421
Noise Exposure x Noise Sens.	.052	.047	1.102	.271
Gender	.337	1.059	.318	.751
Gender x Noise Sens.	-.108	.116	-.933	.351
Neuroticism	5.183	.199	25.993	< .001
Age	-.050	.010	-5.225	< .001
Education	-.167	.177	-.945	.345
<i>F</i> (8, 1006) = 110.16, <i>p</i> < .001, <i>R</i> ² = .467				
Moderated Multiple Regression of Noise Sensitivity on Sleep Problems				
Noise Sensitivity	-.0003	.044	-.007	.142
Noise Exposure	.031	.087	.359	.720
Noise Exposure x Noise Sens.	-.0008	.009	-.084	.933
Gender	-.098	.212	-.462	.645
Gender x Noise Sens.	.021	.023	.905	.366
Neuroticism	.382	.040	9.575	< .001
Age	.008	.002	3.890	< .001
Education	-.024	.035	-.672	.502
<i>F</i> (8, 1006) = 17.66, <i>p</i> < .001, <i>R</i> ² = .123				

Note. Noise Sens. = Noise sensitivity; *n* = 1015

5.5.3. Noise sensitivity and health complaints mediation model. Due to the lack of significant moderator effects in the overall moderated mediation model (Figure 6, p. 127; Table 10, p. 128), a mediation model of noise sensitivity and health complaints was tested to investigate the influence of the mediators in the model (see Figure 7, p. 130). Results indicated a significant model, $F(6, 1014) = 93.32, p < .001$, with predictors and mediators explaining 35.2% of the variance in health complaints, $adj. R^2 = .352$. The total effect (c path) of noise sensitivity on health complaints was significant, $c = .326, p < .001$. The direct effect (c' path; controlling for the influence of mediators in the model) of noise sensitivity on health complaints was not significant, $c' = .171, p = .053$ (after mediators were tested in the model), indicating significant influence of the indirect effects of perceived stress and sleep problems in the model. In terms of the influence of the covariates on health complaints, neuroticism was positively predictive of health complaints, $B = 1.316, p < .001$. Neither age, $B = -.008, p = .580$, nor education, $B = -.177, p = .513$, was significant in the model.

The total indirect effect was significant, $ab_{total} = .155$, 95% BCa CI: [.074, .249]. The indirect effects of noise sensitivity on health complaints via perceived stress, $a_1b_1 = .088$, 95% BCa CI: [.024, .137] and sleep problems, $a_2b_2 = .077$, 95% BCa CI: [.023, .143], were significant. Pairwise contrasts indicated there was no difference in strength between the indirect effect through perceived stress (a_1b_1) compared to that via sleep problems (a_2b_2), $B = -.0004$, 95% BCa CI: [-.075, .074].

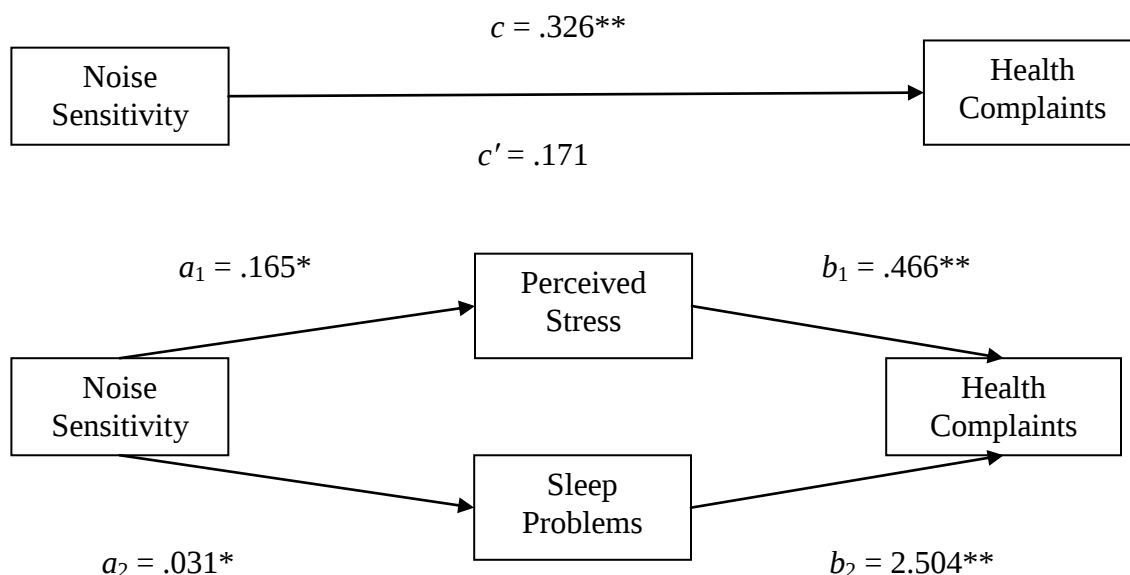


Figure 7. Noise sensitivity and health complaints mediation model.

Note. Unstandardised coefficients (B) are presented for the pathways between independent variables, mediators and dependent variable⁵⁴.

a pathway = relationship between noise sensitivity and mediator (perceived stress/sleep problems)

b pathway = relationship between mediator (perceived stress/sleep problems) and health complaints

c pathway = relationship between noise sensitivity and health complaints (total effect)

c' pathway = relationship between noise sensitivity and health complaints after controlling for model variables (direct effect)

Covariates: neuroticism, age, education

$n = 1021$ ** $p < .001$ * $p < .05$

5.5.4. Noise sensitivity and hypertension mediation model. Cardiovascular disease has been identified as a health outcome linked to both noise exposure (e.g., Rosenlund et al., 2001; van Kempen & Babisch, 2012) and noise sensitivity (Heinonen-Guzejev et al., 2004, 2007). The previously identified mediation model was applied to a model with hypertension (present/not

⁵⁴ However, Preacher and Hayes (2008) note that the unstandardised regression coefficients should not be the focus of interpretation of regression results. Instead, interpretation should be based on indirect effects (ab coefficients).

present) as the health outcome of interest (Figure 8). Due to the use of a binary outcome, a logistic regression was applied to the overall model. Moderators were not tested in the model; interactions involving gender and noise exposure were previously tested on the mediators (see Figure 6, p. 127). The only change to the model was the outcome variable (hypertension); because of the position of the moderators (set to interact with noise sensitivity along path *a* rather than path *b*), their influence would not be different from the previous health complaints model. Therefore, gender and noise exposure were not included in the remaining health models.

Results of the regression analysis revealed that the mediation model of noise sensitivity and hypertension (with covariates included) was significant, $\chi^2(6) = 138.82, p < .001, R^2$ (Nagelkerke) = .215. The significance of the overall model resulted from the predictive value of age in the development of hypertension, $B = .067, p < .001$. As age increased, the risk of hypertension also increased. No other variables were significant predictors in the model. Education was not significantly predictive of occurrence of hypertension, $B = -.108, p = .317$, nor was neuroticism, $B = .091, p = .566$. The total effect of noise sensitivity on hypertension (*c* path) was not significant, $c = -.0001, p = .999$, nor was the direct effect of noise sensitivity on hypertension (*c'* path), $c' = -.008, p = .821$, indicating that there was no relationship between noise sensitivity and hypertension even after controlling for the mediator variables. The total indirect effect was not significant, $ab_{\text{total}} = .006$, 95% BCa CI: [-.002, .018], indicating no significant difference between the *c* and *c'* pathways (Hayes & Preacher, 2010). The individual indirect effects of the mediators were also not significant: perceived stress, $a_1b_1 = .006$, 95% BCa CI: [.000, .017] and sleep problems, $a_2b_2 = .0004$, 95% BCa CI: [-.007, .009].

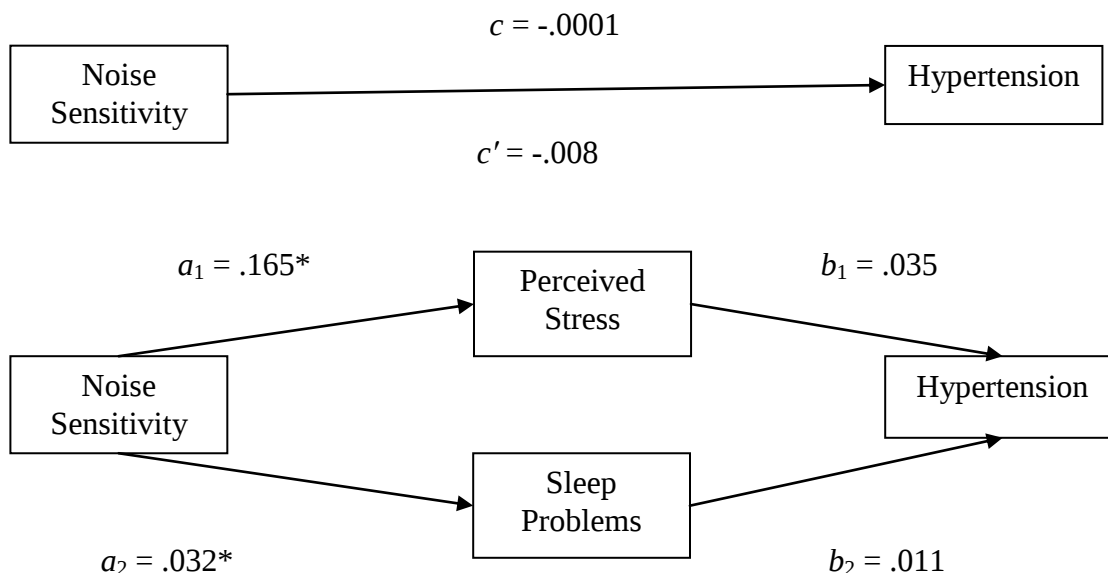


Figure 8. Noise sensitivity and hypertension mediation model.

Note. Unstandardised coefficients (B) are presented for the pathways between independent variables, mediators and dependent variable.

a pathway = relationship between noise sensitivity and mediator (perceived stress/sleep problems)

b pathway = relationship between mediator (perceived stress/sleep problems) and hypertension

c pathway = relationship between noise sensitivity and hypertension (total effect)

c' pathway = relationship between noise sensitivity and hypertension after controlling for model variables (direct effect)

Covariates: neuroticism, age, education

$n = 1029$ $*p < .05$

5.5.5. Noise sensitivity and mental health complaints. Mental health problems have been strongly associated with noise sensitivity (e.g., Kishikawa et al., 2009; Stansfeld, 1992). Therefore, it was important to not only consider general subjective health complaints and hypertension, but mental health problems as well. In order to test this, the anxiety and depression severity ratings from the SHCI were used as outcome variables in the regression models.

5.5.5.1. Noise sensitivity and anxiety complaints mediation model. The noise sensitivity and anxiety complaints model is presented in Figure 9. The results of the mediation model of noise sensitivity and anxiety complaints indicated a significant model, $F(6, 1013) = 110.30, p <$

.001, with the model variables explaining 39.0% of the variance in anxiety complaints, $adj. R^2 = .390$. However, notably neither the total effect ($c = .012, p = .173$) nor the direct effect ($c' = -.001, p = .902$) was significant, thereby suggesting that the significance of the model cannot be attributed to the mediators. Rather, the covariates accounted for the variance in anxiety complaints. Specifically, neuroticism was significant in predicting anxiety complaints, $B = .259, p < .001$, with elevated neuroticism being predictive of anxiety complaints. Age was also positively predictive of anxiety complaints, $B = .004, p = .009$, with increases in age being associated with increased anxiety. Education was not significant in the overall model, $B = .042, p = .093$.

The overall indirect effect was significant, $ab_{total} = .013$, 95% BCa CI: [.006, .021], a result suggesting a significant difference between the total (c path) and direct effects (c'). This difference was accounted for by the indirect effect via both perceived stress, $a_1b_1 = .007$, 95% BCa CI: [.003, .013], and the indirect effect via sleep problems, $a_2b_2 = .006$, 95% BCa CI: [.001, .011]. Pairwise contrasts revealed no significant difference in strength between the indirect effect via perceived stress compared to sleep problems, $B = .001$, 95% BCa CI: [-.005, .008].

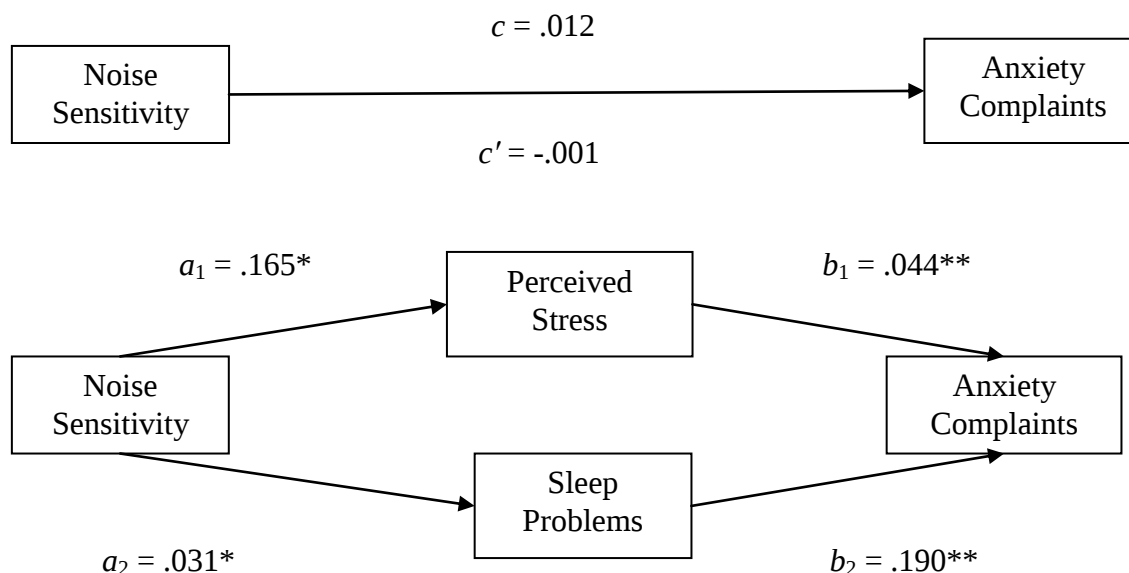


Figure 9. Noise sensitivity and anxiety complaints mediation model.

Note. Unstandardised coefficients (*B*) are presented for the pathways between independent variables, mediators and dependent variable.

a pathway = relationship between noise sensitivity and mediator (perceived stress/sleep problems)

b pathway = relationship between mediator (perceived stress/sleep problems) and anxiety complaints

c pathway = relationship between noise sensitivity and anxiety complaints (total effect)

c' pathway = relationship between noise sensitivity and anxiety complaints after controlling for model variables (direct effect)

Covariates: neuroticism, age, education

n = 1020 ** *p* < .001 **p* < .05

5.5.5.2. Noise sensitivity and depression complaints mediation model. The mediation model of noise sensitivity and depression complaints was significant, $F(6, 1014) = 112.53$, $p < .001$, with model variables explaining 39.6% of variance in depression complaints, $adj. R^2 = .396$. Neither the total effect of noise sensitivity on depression complaints ($c = .003$, $p = .683$) nor the direct effect ($c' = -.008$, $p = .284$) was significant. Neuroticism, included in the model as a covariate, was significant in predicting depression complaints, $B = .270$, $p < .001$. The

remaining covariates, age ($B = -.001, p = .464$) and education ($B = .007, p = .781$), were not significant in predicting depression complaints.

The overall indirect effect of noise sensitivity on depression complaints through the mediators was significant, $ab_{\text{total}} = .012$, 95% BCa CI: [.006, .019] indicating a significant difference between the c and c' paths. The indirect effects via perceived stress, $a_1b_1 = .007$, 95% BCa CI: [.003, .013], and sleep problems were significant, $a_2b_2 = .004$, 95% BCa CI: [.001, .009]. Pairwise contrasts of the indirect effects revealed that the indirect effect via sleep problems was not stronger than that via perceived stress, $B = .003$, 95% BCa CI [-.003, .009].

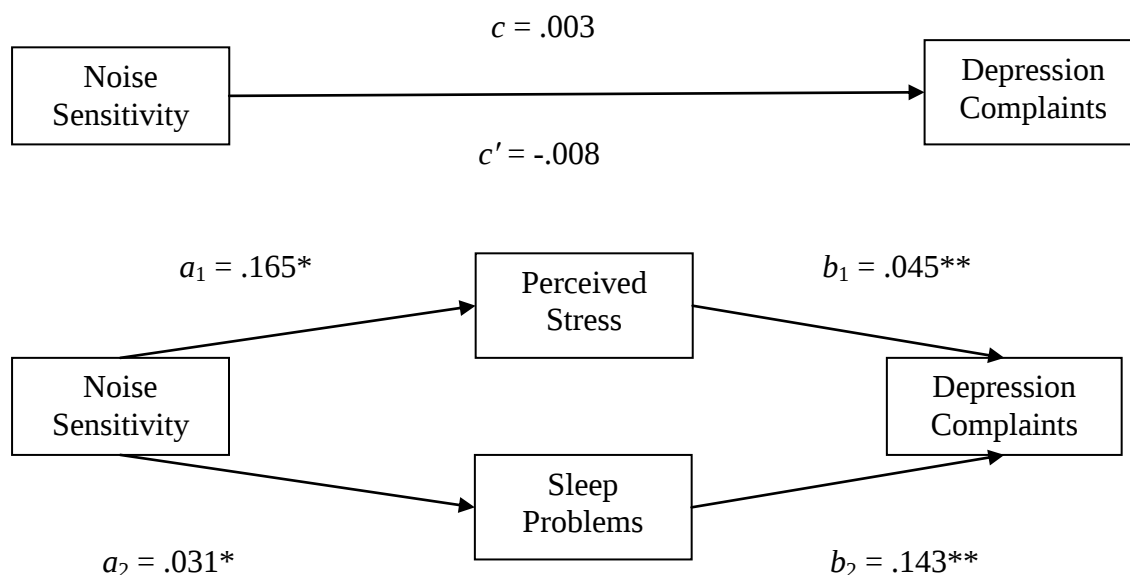


Figure 10. Noise sensitivity and depression complaints mediation model.

Note. Unstandardised coefficients (B) are presented for the pathways between independent variables, mediators and dependent variable.

a pathway = relationship between noise sensitivity and mediator (perceived stress/sleep problems)

b pathway = relationship between mediator (perceived stress/sleep problems) and depression complaints

c pathway = relationship between noise sensitivity and depression complaints (total effect)

c' pathway = relationship between noise sensitivity and depression complaints after controlling for model variables (direct effect)

Covariates: neuroticism, age, education

$n = 1021$ ** $p < .001$ * $p < .05$

5.6. Discussion

The present study tested a model of mediators and moderators potentially involved in the association between noise sensitivity and self-reported health. As expected, noise sensitivity was associated with health complaints (i.e., greater noise sensitivity was associated with more health complaints). Results of the study indicated that the proposed moderators – gender and noise exposure – did not impact the regression model significantly. Rather, perceived stress and sleep problems were significant mediators in the association between noise sensitivity and subjective health complaints. The results for the mental health complaints models indicated that neuroticism, included in the models as a covariate, largely explained the association between noise sensitivity and anxiety and depression complaints.

It is notable that noise sensitivity did not interact with noise exposure – assessed with objective measures (road traffic volumes) or subjective ratings (perceptions of noise in one's home) – in predicting perceived stress and sleep problems (see Figure 6, p. 127 and Appendix M., p. 288). Therefore, individuals elevated on noise sensitivity, who were also exposed to – or perceived – high levels of noise in the home environment, were not more likely to report perceived stress or sleep problems than those less exposed to environmental noise. The lack of interaction between these two variables may be due, in part, to the complex nature of noise itself. That is, noise could be considered in absolute (e.g., high vs. low SPL) or relative terms. For example, an individual who lives in a quiet neighbourhood may experience considerable disturbance when a neighbour starts up his lawnmower, while a person who lives near a busy road may not be affected by a lawnmower in the same way because of the high level of background noise already outside his home. In other words, the difference between background noise exposure and changes in one's environmental noise exposure (e.g., from a lawnmower,

dogs barking, child playing) over the course of a day may be an important variable in determining noise reactions.

Public health and psychoacoustic research largely focuses on research on absolute noise exposure levels (dB), rather than relative noise exposure, in order to make claims about the ill-effects of environmental noise (e.g., Ana et al., 2009; Birk et al., 2011; Flindell & Stallen, 1999; Haines, Stansfeld, et al., 2001a; Kishikawa et al., 2009; Öhrström et al., 1990; Öhrström, Skanberg, Svensson, & Gidlof-Gunnarsson, 2006; Passchier-Vermeer & Passchier, 2000; Selander et al., 2009a). This approach arguably oversimplifies the concept of ‘noise exposure’ by reducing it to its SPL. The various qualities of noise (e.g., intermittent, high vs. low frequency, the SPL change relative to background noise) and the psychological make-up of the individual perceiving the noise are all factors that could impact disturbance and other ill-effects of noise exposure (Flindell & Stallen, 1999).

The relative noisiness of the environment may be particularly relevant to noise sensitive individuals, who are affected by a variety of noises. Indeed, Ryu and Jeon (2011) found that noise sensitivity, assessed with a 20-item scale in their study, was strongly predictive of annoyance to indoor noises (e.g., children jumping/running, people talking, flushing toilets). Further, they reported that noise sensitivity was more strongly associated with indoor noise annoyance than with outdoor noise annoyance (i.e., road traffic noise), thus emphasising the importance of context-specific noise perceptions in such investigations. It is possible that noise sensitive individuals who live in quiet neighbourhoods may be disturbed by relative changes in noise exposure (e.g., lawnmower, dogs barking) in their home environment. In this thesis, noise exposure information was collected in absolute terms (i.e., with road traffic volumes, perceptions of overall noisiness of the home environment) in line with current environmental noise exposure

research (e.g., Bodin et al., 2009; de Kluizenaar, Janssen, van Lenthe, Miedema, & Mackenbach, 2009; Floud et al., 2011; Sorensen et al., 2011; Stansfeld & Crombie, 2011; Stošić & Blagojević, 2011). Although beyond the scope of this thesis, the concepts of intermittent noise exposure and relative noisiness are clearly important to consider in noise sensitivity and noise research, and therefore should be considered in future studies.

Noise sensitivity was also significantly positively correlated with ratings of noise annoyance, and noisiness of the work and home environment, and negatively correlated with perceptions of control. These results corroborate previous research indicating that noise sensitivity may be largely an attitudinal trait (Alimohammadi, Nassiri, Azkhosh, & Hoseini, 2010; Ellermeier et al., 2001; Stansfeld, 1992), which, in turn, influences an individual's perception of their environment. Because there was no interaction between noise sensitivity and noise exposure in the moderated mediation models tested (see Figure 6, p. 127, and Appendix M, p. 288), the influence of the mediators – perceived stress and sleep problems – on the relationship between noise sensitivity and health outcomes was the focus of the results of Study 1.

The significant mediation of the relationship between noise sensitivity and subjective health complaints is particularly noteworthy (see Figure 7, p. 130). This finding is congruent with the allostatic load model, which posits that chronic stress can increase an individual's vulnerability to health problems (McEwen, 1998b). Therefore, according to the results of this study, noise sensitivity is linked to an increased vulnerability to perceived stress, which, in turn, places an individual at risk for health problems. Further, these results were found even after controlling for the influence of neuroticism in the model, thereby countering the previous

contention by Smith et al. (2002) that the relationship between noise sensitivity and self-reported health is accounted for by negative affectivity.

While the relationships among noise sensitivity, physiological stress (i.e., activation of the stress response), and psychological stress (i.e., perceived stress levels) have been tested within laboratory settings (e.g., Ljungberg & Neely, 2007; Persson Waye et al., 2003), the present study explored the role of perceived stress within a large community-based study, and in relation to the association between noise sensitivity and diminished health. Laboratory studies have demonstrated a relationship between noise sensitivity and stress reactivity, as evidenced by hyperactivation of the SAM system and HPA axis in response to mental or environmental stressors (Di Nisi et al., 1987; Ising et al., 1980; Ljungberg & Neely, 2007). To supplement this line of research, the positive association between perceived stress and noise sensitivity provides some evidence that the noise sensitivity-related physiological stress ‘vulnerability’, as exhibited in laboratory setting (e.g., Persson Waye et al., 2003), may translate into psychological stress as well.

The relationship between reports of psychological stress (e.g., perceived stress) and physiological stress (i.e., activation of the stress response) may be bidirectional. While it is a common assumption within health psychology research that psychological stress leads to activation of the physiological stress response (Miller, Chen, & Cole, 2009), there is some evidence that chronic exposure to stress hormones contributes to psychological stress. Specifically, chronic exposure to stress hormones may exacerbate anxiety and worry (e.g., Abrorelius et al., 1999; Binder & Nemeroff, 2010). Therefore it is possible that noise sensitive individuals, because of their easily-aroused stress response system (Di Nisi et al., 1987; Griefahn & Di Nisi, 1992; Ljungberg & Neely, 2007; Persson Waye et al., 2002), might experience

increased stress due to neurobiological factors. However, this research question extends beyond the purpose of the present study. The role of the HPA axis in noise sensitivity and diminished health will be pursued in Study 2 of the thesis.

Although it is well known that adequate sleep may help buffer risk for health problems (Lockley, 2010), results of the current study apply this finding to the association between noise sensitivity and diminished health (Heinonen-Guzejev, 2009; Kishikawa et al., 2009; Schreckenberg et al., 2010a; Schreckenberg et al., 2010b). In other words, noise sensitivity is associated with sleep problems, which, in turn, can lead to poor health. Indeed, sleep disturbance has been associated with noise sensitivity in both field and laboratory studies (e.g., Aasvang et al., 2008; Marks & Griefahn, 2007). Noise sensitive individuals may experience greater sleep disturbance because of greater stress reactivity (Griefahn & Di Nisi, 1992; Persson Waye et al., 2002), which, in turn, may contribute to increased awakenings throughout the night. These awakenings could be due to night-time noise both inside or outside the home, or possibly other psychological factors such as anxiety and stress.

Kishikawa et al. (2009), authors of a recent study on the association among noise exposure, noise sensitivity, and minor psychiatric disorder (as assessed by the GHQ; Goldberg, 1972), proposed that the likely mechanism explaining the link between noise sensitivity and mental health problems was sleep disturbance. In their study, noise sensitive individuals who were exposed to considerable road traffic noise (> 55 dB) were more likely to report psychiatric complaints (e.g., anxiety and insomnia, somatic symptoms). Due to the relationship between noise sensitivity and mental health problems, particularly among noise-exposed participants, it is reasonable that Kishikawa et al. (2009) suggested sleep disturbance as a likely mediator. However, noise exposure was not a significant moderator in the present study, and therefore we

cannot conclude that the relationship between noise sensitivity and health complaints is attributable to sleep disturbance due to road traffic noise.

The relationship between noise sensitivity and mental health complaints in the present study was largely accounted for by neuroticism (see Figure 9, p. 134; Figure 10, p. 136). This replicates the previous research by Smith et al. (2002), who reported that the relationship between noise sensitivity and self-reported health could be accounted by negative affectivity. Smith et al. (2002) used self-reported physical illness and the GHQ, which measures anxiety, severe depression, social dysfunction, and somatic complaints, as health outcomes. Specifically, the use of a largely mental health-orientated scale (GHQ) may account for the similar results for the mental health complaints models in the present study (see Figure 9, p. 134; Figure 10, p. 136). Indeed, the findings of this study suggest that the relationship between noise sensitivity and mental health complaints is accounted for neuroticism. However, to contrast the research findings of Smith et al. (2002), in this study, neuroticism did not have the same influence over the relationship between noise sensitivity and subjective health complaints (i.e., sleep problems and perceived stress mediated the relationship even after controlling for neuroticism; see Figure 7, p. 130).

Indeed, neuroticism is a well-established risk factor for the development of mental health problems, including anxiety and depression (Cuijpers et al., 2010; Malouff, Thorsteinsson, & Schütte, 2005). Therefore it is not surprising that it emerged as a significant predictor in the anxiety and depression complaints models. Stansfeld (1992) had previously discussed that there is a negative affectivity component to noise sensitivity (p. 65). The results of this study suggest that this component of noise sensitivity accounts for the association between noise sensitivity and mental health problems. However, results of the subjective health complaints model

emphasise that noise sensitivity is not merely a symptom of negative affectivity. Rather, even after controlling for neuroticism, noise sensitivity and subjective health complaints were significantly correlated – a relationship that was mediated by perceived stress and sleep problems.

The theoretical framework of this study was grounded in the allostatic load model. The allostatic load model posits that chronic stress has the capacity to damage the body, leading to physical changes such as diminished immune functioning and vulnerability to cardiovascular disease (Miller et al., 2009). Although beyond the scope of Study 1, it is possible that the relationship between noise sensitivity and health complaints exists due to the bodily wear and tear resulting from chronic stress. Indeed, chronic stress has been linked to the development of cold symptoms (e.g., Cohen et al., 1991; Miller et al., 2009) and gastrointestinal issues (e.g., Mayer, 2000), health problems measured by the SHCI. Although the measurement of health complaints is a common method to assess health problems (e.g., Lovell, Moss, & Wetherell, 2011; van den Berg, Maas, Verheij, & Groenewegen, 2010; Witvliet, Kunst, Stronks, & Arah, 2012), it should be noted that the measure is a subjective health rating, and therefore limits the conclusions that can be drawn about the real physical state of participants.

Further, contrary to previous research (Heinonen-Guzejev et al., 2004), noise sensitivity was not associated with hypertension, and perceived stress was not a significant mediator in the hypertension model. Previous research has identified noise sensitivity as a potential risk factor for the development of cardiovascular disease (Babisch, 2010; Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2004) and cardiovascular disease-related mortality (Heinonen-Guzejev et al., 2007). Although the present study did not support this association, various factors could be at play.

First, self-reported information was used, rather than measured blood pressure or medical reports, and therefore it is possible that the prevalence of hypertension in the sample was underreported. Second, although the sample contained many middle-aged and elderly participants, who are typically at higher risk for cardiovascular disease than younger individuals, the participants of the sample might have been healthier than other members of the general population. For example, only 8% of the sample reported being current daily or occasional smokers, compared to 22% of the general New Zealand population (aged 15-64 years; Ministry of Health, 2010). Indeed, participants of community-based survey studies are often healthier and more educated than the general population (Galea & Tracy, 2007). Further research on the association between noise sensitivity and cardiovascular disease, possibly conducted with objective health measures (e.g., measured blood pressure, medical reports), may be advantageous in further understanding the impact of cardiovascular-specific allostatic mechanisms in the relationship between noise sensitivity and diminished health.

5.6.1. Limitations. While the present study examined factors involved in the relationship between noise sensitivity and diminished health relationship using a large ($n > 1000$) community-based study, there are some limitations with the use of this research design. First, in order to decrease burden on participants in the study, and in turn encourage participation in the study (Galea & Tracy, 2007), the questionnaire was designed to be as short as possible, using as few questions as possible to address the goals of the study.

While the use of a relatively short questionnaire is an advantageous approach in conducting large survey-based studies (Galea & Tracy, 2007), the depth of understanding associations among variables may be compromised. For example, information on noise sensitivity was gathered using a 3-item scale (Amann et al., 2007). While using a small number

of items limited burden on participants, a longer scale (e.g., 35-item Noise Sensitivity Questionnaire; Schütte et al., 2007) would have allowed the assessment of various aspects of noise sensitivity (e.g., in relation to the home environment and during sleep). However, previous research has relied on a single item to measure noise sensitivity (Fyhri & Aasvang, 2010; Fyhri & Klæboe, 2009; Shepherd et al., 2011), therefore, in comparison, the 3-item scale (3-NS) captured the construct of noise sensitivity adequately in this study. Further, the variable was normally distributed, similar to findings of previous noise sensitivity research (e.g., Belojević & Jakovljević, 2001; Ekehammar & Dornic, 1990; Shepherd et al., 2010), and the scale had adequate internal consistency (Cronbach's $\alpha = .68$).

The limitation of lack of breadth in measuring study variables also applies to the assessment of sleep and mental health problems. Sleep problems, anxiety, and depression complaints were each assessed using one question from the SHCI. Again, while this helped to reduce burden on participants, it limited the extent to which these health issues could be explored. It would be useful for future studies to replicate the current study with the use of longer validated questionnaires, such as the short form of the Depression Anxiety and Stress Scale (DASS-21; Henry & Crawford, 2005). Similarly, the use of a longer sleep scale (e.g., 4-item Jenkins Sleep Scale; Jenkins, Stanton, Niemcryk, & Rose, 1988) in large community-based studies may be better for understanding specific sleep issues (i.e., insomnia, sleep latency) in relation to noise sensitivity and health outcomes.

However, as mentioned above, while longer and more reliable scales may be beneficial in attempting to capture various facets of constructs, the advantage of using such scales would need to be considered against the possible loss of participation and interest of respondents due to the increased burden of answering lengthy questionnaires (Galesic & Bosnjak, 2009). Although the

questionnaire was designed to be as brief as possible, it did involve answering over 70 items, which, in turn, may have discouraged participation among some individuals. Further, if lengthier scales (e.g., NoiSeQ, DASS-21, 4-item Jenkins Sleep Scale) were used in this study, the questionnaire would have been over 100 questions, thus considerably increasing the time required for participants to complete it.

The primary goal of the study was to further elucidate the association among noise sensitivity, perceived stress and diminished health. However, many factors that can influence health and well-being, such as physical activity, diet, coping mechanisms, and type of employment (Evans, Barer, & Marmor, 1994; Ogden, 2007) were not tested in the present study. While additional health influences would be interesting to assess within the context of noise sensitivity, stress, and health, the present study tested but a subsection of variables that could influence health, consistent with the allostatic load model of stress and health (McEwen, 1998b). In particular, future research on coping mechanisms, employed by noise sensitive individuals, in relation to both daily and environmental stressors may be helpful to further understand possible mitigating factors against stress. The topic of noise sensitivity and coping is further discussed in the general discussion (see p. 192).

As with all cross-sectional studies, the assumption of causality within the study models should be considered with caution. While the models were structured to suggest that noise sensitivity precedes perceived stress, sleep problems, and health complaints, it is not possible to confidently confirm the direction of the associations. Further research on the topic, longitudinal designs in particular, would be beneficial in discerning the nature of relationship between noise sensitivity and diminished health.

In this study, a health psychological model was applied to the relationship between noise sensitivity and diminished health. Specifically, noise sensitivity was treated as a stable personal trait, and its relationship to perceived stress and health complaints was examined. This approach to understanding noise sensitivity is unique from public health research, which frequently investigates the influence of noise exposure and noise reactions (e.g., noise annoyance) on health and well-being (e.g., Babisch et al., 2009; Birk et al., 2011; Klæboe, Amundsen, Fyhri, & Solberg, 2004; Pierrette et al., 2012; Willich et al., 2006). The comparability to previous noise research is therefore limited because of the focus on noise sensitivity as a causal agent influencing stress and health problems. Nonetheless, the investigation of the trait noise sensitivity, because of its association with health problems, remains an important research topic in further understanding psychological factors involved in health and well-being.

Additionally, community-based postal surveys are susceptible to self-selection bias. In other words, although the questionnaires were delivered to a variety of neighbourhoods in Auckland, only a subsection of participants who received the questionnaires completed the study. Compared to data collected by Statistics New Zealand, the sample was well-educated, women were overrepresented and members of the Asian, Pacific Islander and Māori ethnic groups were somewhat underrepresented. This limits the extent to which the results can be generalised to the population of Auckland or New Zealand. However, this research was conducted to assess the relationship between noise sensitivity and diminished health, and potential moderators and mediators of this association, rather than ethnic or socioeconomic differences in the variables.

Finally, the low response rate (14.8%) of this study must be acknowledged. Galea and Tracy (2007) previously discussed the issue of declining participation rates that has become particularly noticeable in recent years. They suggest that members of the general population may

be less likely to volunteer for studies due to the steep increase in survey-based research conducted by universities and government bodies. In other words, the declining participation rates for studies in general may be due to members of the population feeling continuously bothered to participate in research. Or it may be that potential participants consider that there is nothing to be gained personally from them participating.

With a population of 1.3 million, Auckland is the largest city in New Zealand (Statistics New Zealand, 2006a), and home to three universities as well as various polytechnic institutions. Therefore, the low response rate of this study may be a result of oversampling of the Auckland population. Some factors can improve response rates such as offering financial incentives or extensive follow-up with participants. Unfortunately, these strategies were not employed in Study 1. Instead, the survey was kept as brief as possible (i.e., no consent form) and anonymous, in hopes of encouraging interest and willingness to participate. Further, prospective participants were chosen based on the distance between their home and arterial roads, rather than through contacting the participant directly (e.g., after gathering contact information from NZ Electoral Rolls). Therefore, follow-up was not feasible in this study due to lack of available contact information (e.g., phone number).

Despite having a low response rate, this study did involve over 1,000 participants. This large sample size allowed for the detection of small correlations among study variables. Further, the distribution of noise sensitivity was similar to those in previous studies (e.g., Belojević & Jakovljević, 2001; Ekehammar & Dornic, 1990; Shepherd et al., 2010), thereby indicating good range of data in the sample used. Nonetheless, the limitations of this study should be kept in mind in interpreting the results.

5.6.2. Conclusion. While previous researchers have reported association between noise sensitivity and diminished health (Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2004, 2007; Nivison & Endresen, 1993), there has been limited attention as to the mechanisms underlying the association. The present study fills an important gap in the literature through the assessment of mediators and moderators of the association between noise sensitivity and diminished health using a large community-based study.

Perceived stress and sleep problems significantly mediated the association between noise sensitivity and subjective health complaints. Specifically, it is possible that noise sensitive individuals are more likely to experience psychological stress, which, in turn, places them at risk for developing health problems. Further research to elucidate physiological agents that may be involved would help provide more understanding. That is the purpose of Study 2 of the thesis. The results of Study 1 also indicated that the relationship between noise sensitivity and mental health complaints was accounted for by neuroticism, and that there was no evidence for an association between noise sensitivity and hypertension in the present study. Overall, Study 1 provided considerable insight into the psychological factors involved in the relationship between noise sensitivity and diminished health.

Preface to Study 2

Study 1 of the thesis tested the role of perceived stress, among other variables, in the association between noise sensitivity and diminished health. As hypothesised, perceived stress was a significant mediator in the association between noise sensitivity and subjective health complaints. This research sought to substantiate previous studies reporting an association between noise sensitivity and psychological stress (Ljungberg & Neely, 2007; Persson Waye et al., 2002), and further apply it to the relationship between noise sensitivity and health problems. Although noise sensitivity does appear to impact an individual's acute response to both physiological and psychological stressors (Ljungberg & Neely, 2007; Persson Waye et al., 2002; Stansfeld, 1992), there is limited research on the biological underpinnings of the 'stress vulnerability' component of the trait noise sensitivity (Fyhri & Klæboe, 2009; Stansfeld, 1992). Study 2, therefore, sought to investigate this further.

The CAR has emerged in the past decade as a relevant and promising biomarker of HPA axis activity (Clow et al., 2010). The CAR has been positively associated with chronic stress and work overload (Schlotz, Hellhammer, Schulz, & Stone, 2004; Schulz et al., 1998; Wüst et al., 2000a), among various health conditions such as respiratory illnesses and psychiatric conditions (Chida & Steptoe, 2009b; Kudielka & Kirschbaum, 2003). The association between noise sensitivity and both perceived stress and health complaints may be linked to chronic overactivity of the HPA axis. That is, the 'stress vulnerability' aspect of noise sensitivity may be exhibited in the CAR, and thus, provide some insight into the physiological factors that may be involved in the association between noise sensitivity and diminished health. As such, Study 2 of the thesis sought to build upon the results of Study 1 through an investigation of the relationship between noise sensitivity and the CAR.

Chapter 6: Study 2 – Noise Sensitivity and the Cortisol Awakening Response

6.1. Introduction

Cortisol, a stress hormone released by the HPA axis of the neuroendocrine system, has been identified as a valid and useful physiological marker of allostatic load (Juster et al., 2010). Specifically, elevated cortisol has been identified as an immunosuppressant (Taylor, 2010), and a significant predictor of physical health decline (McEwen, 1998b). In terms of discerning the impact of chronic stress on physiological systems, the assessment of cortisol levels upon awakening (CAR) has been acknowledged as a useful index of HPA axis activity (Clow, Hucklebridge, & Thorn, 2010; Clow et al., 2004; Kudielka & Kirschbaum, 2003; Kudielka & Wüst, 2010; Wüst et al., 2000a; Wüst et al., 2000b). Specifically, CAR levels have been positively associated with chronic stress and worrying (Schlotz et al., 2004; Wüst et al., 2000a), and appear to be closely related to an individual's health and psychological well-being⁵⁵ (Chida & Steptoe, 2009b; Clow et al., 2004; Karlson, Eek, Hansen, Garde, & Ørbaek, 2011; Kudielka & Kirschbaum, 2003).

Chronic stress can result from a variety of situational or stable factors such as family troubles, economic hardship, or coping with terminal illness (Schluz & Northridge, 2004). Stable factors, such as an individual's personality also have the capacity to impact chronic stress, and in turn, the functioning of the neuroendocrine system (Cohen, Kamarck, & Mermelstein, 1983; Whitehead et al., 2007). Noise sensitivity has been identified as a personality trait that may influence the reactivity of the neuroendocrine and autonomic nervous system (Belojević et al.,

⁵⁵ The physiological processes involved in CAR have been reviewed in Chapter 2 (p. 22).

2003; Hebert & Lupien, 2009; Persson Waye et al., 2002; Stansfeld, 1992; Stansfeld et al., 1985a; Stansfeld & Shine, 1993). In other words, people with elevated noise sensitivity are more likely to experience greater arousal of the stress systems than those lower in noise sensitivity. Research on this topic has been reviewed in Chapter 4 (p. 75). Therefore, noise sensitivity not only has the capacity to influence state-based reactions to environmental stressors (e.g., heart rate increase, annoyance due to environmental noise), but being noise sensitive might also be linked to a long-term alteration of the neuroendocrine system.

To date, only one study has included noise sensitivity as a variable in an investigation on the CAR. Persson Waye et al. (2003) assessed the impact of exposure to night-time low frequency noise on the CAR and subjective sleep quality with 12 males who slept in a noise laboratory during the experiment. The objective of their study was to examine the influence of exposure to low frequency noise on the CAR and subjective sleep quality. Additionally, the researchers measured noise sensitivity with the Weinstein Noise Sensitivity Scale (WNS), and also with single (5-point Likert scale) items regarding general noise sensitivity and sensitivity to low frequency noise. Their pilot study showed evidence that low frequency noise may attenuate levels of cortisol over the awakening period, possibly related to tiredness or excess cortisol secretion throughout the night. There was no evidence for a relationship between noise sensitivity and CAR, or interactions between noise sensitivity (tested on all measures of noise sensitivity) and noise on CAR. However, the small sample size and experimental design of the study limited the scope and findings of the study. Therefore, to more clearly understand the association between noise sensitivity and the CAR, a larger, more naturalistic study should be employed.

It is possible that the neuroendocrine system of noise sensitive individuals differs from that of non-noise sensitive individuals (Ljungberg & Neely, 2007; Persson Waye et al., 2002). Specifically, noise sensitivity, a trait that may increase a person's likelihood of experiencing acute and chronic stress (Ljungberg & Neely, 2007; Stansfeld, 1992), may result in dysregulation of the HPA axis. Therefore, in the present study, it was hypothesised that noise sensitivity would be positively associated with an elevated CAR – evidence for HPA axis dysregulation. In turn, the assessment of the CAR in relation to noise sensitivity may further elucidate factors that may be involved in the association between noise sensitivity and diminished health.

6.2. Method

6.2.1. Participants. Participants ($n = 107$), aged 18 years and older, were recruited using advertisement of the study through media (e.g., local newspapers) and electronic and paper-based posters at community centres (i.e., local theatres, churches) and academic institutions. Most ($n = 90$) participants were recruited through the use of an advert distributed to community centres and academic institutions (see Appendix N, p. 290). An article written on the research programme in a local newspaper (North Shore Times; see Appendix O, p. 291) was also used to help recruit participants for the research project. This strategy was employed to hopefully attract a pool of participants with a broad range of noise sensitivity, including those most sensitive to noise (approximately 20% of the population; Matsumura & Rylander, 1991; Olsen Widen & Erlandsson, 2004). Current smokers and individuals on steroid medication or hormone-replacement therapy were not invited to participate in the study as these factors have been found to influence the CAR (see Chida & Steptoe, 2009b). Participants received a petrol or supermarket voucher (value of \$20) for completing the study.

6.2.2. Procedure. Participants were provided with a total of three salivettes (Sarstedt Ltd, Aktiengesellschaft & Co, Germany, D-51588), which are small plastic test tubes that include a cotton swab. Detailed written instructions regarding appropriate salivary cortisol collection were provided to the participants (i.e., chewing on the cotton swab until fully saturated and then placing it in the salivette; see Appendix Q, p. 294). Cortisol samples were collected during a weekday morning (Monday-Friday). Participants were instructed to not eat, drink or brush their teeth until testing had been completed, but otherwise, they were free to go about their morning tasks as normal (as per recommended protocol for assessing CAR, see Wüst et al., 2000b).

Although research indicates that menstrual status (i.e., whether a female is in the luteal or follicular phase) does not significantly impact the CAR (Bouma, Riese, Ormel, Verhulst, & Oldehinkel, 2009; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Kudielka & Kirschbaum, 2003; Quirin et al., 2008), a recent study found that cortisol levels may peak around ovulation (Wolfram, Bellingrath, & Kudielka, 2011). As such, participants were instructed to collect their samples within 10 days of their next expected period (i.e., post-ovulation, which usually occurs around day 14 of a typical 28-day cycle; Selgrade, 2010). Instructions to all participants stated that measurements were to be collected upon awakening, and 30 and 60 minutes following awakening (see Appendix Q, p. 294), a similar protocol to previous studies assessing the CAR (e.g., De Vente, Olf, Van Amsterdam, Kamphuis, & Emmelkamp, 2003; Nater et al., 2008; Pruessner et al., 2003; Williams, Magid, & Steptoe, 2005).

Participants were instructed to write the time and date of collection on the label of each salivette, a protocol recommended in the collection of CAR levels (see Chida & Steptoe, 2009b). Participants stored the three samples in a freezer (e.g., their home freezer) until returned to the research team. Samples were kept on ice or with an ice-pack during delivery, thus kept frozen as

much as possible. This is the recommended protocol (see Wüst et al., 2000b), although salivary cortisol samples can be stored for up to four weeks at room temperature with no impact on their levels (Kirschbaum & Hellhammer, 1999). Participants completed a series of questionnaires (described in next section) on the same day they collected their saliva samples. Participants returned the questionnaires and cortisol samples to the research team within two weeks of completing the sampling.

In order to guard against non-adherence to sampling protocol, which is a potential issue with the collection of bodily fluids in a domestic setting (Thorn et al., 2006), the following strategies were employed: (1) participants were given flexibility in the collection of the saliva sample (i.e., a regular work day during the week was the criterion; the date was not pre-determined by the research team), and (2) participants were asked to record the date and time of awakening on their survey form and on the salivettes. These are recommended strategies in protecting against protocol non-adherence (Chida & Steptoe, 2009b). Having the participant record the date and time of collection acted to prompt the participant to collect samples at the appropriate time. It also allowed for monitoring of non-adherence to protocol (i.e., to ensure participants collected samples 30 minutes apart). The study protocol was approved by the Auckland University of Technology Ethics Committee (AUTEC reference: 10/270; see Appendix P, p. 292). Participants were provided with detailed information about the study procedure in the Participant Information Sheet (see Appendix Q, p. 294) and completed the Consent Form prior to participating in the study (see Appendix R, p. 297). The series of questionnaires took approximately 30-45 minutes to complete.

6.2.3. Measures

6.2.3.1. Demographics and sleep-related questions. Participants were asked to indicate their gender, age, ethnicity, education and work status (e.g., full-time, retired, student, etc.). Additionally, participants provided information regarding the date that they collected their saliva samples, the time of awakening that day, the number of hours they had slept that night, sleep quality that night, and whether or not the participants had used an alarm to wake up (see Appendix I, p. 260, for the list of questions).

6.2.3.2. Noise sensitivity. The NoiSeQ (Schütte et al., 2007) was used to collect information about noise sensitivity. The scale was developed to measure global noise sensitivity as well as sensitivity to noise across five everyday life situations: ‘leisure’, ‘work’, ‘habitation’, ‘communication’, and ‘sleep’ (Schütte, Marks, et al., 2007). Participants responded to each item on a 5-point Likert scale ranging from (1) strongly disagree to (5) strongly agree. The total noise sensitivity score is derived by calculating the mean average response for the 35 items. Additionally, subscale scores can be calculated (7 items per subscale).

Psychometric properties of the scale were tested based on Generalisability (G) theory (Schütte et al., 2007). In developing the scale, Schütte et al. (2007) reported that the work, sleep, communication, and habitation subscales have adequate reliability and validity, but the leisure subscale had questionable reliability. Schütte et al. (2007) suggested that the low reliability of the leisure subscale likely has to do with the wide range of leisure activities in which people participate. In this study, work (Cronbach’s $\alpha = .71$), communication (Cronbach’s $\alpha = .84$), habitation (Cronbach’s $\alpha = .79$), leisure (Cronbach’s $\alpha = .73$), and sleep (Cronbach’s $\alpha = .86$) subscales had adequate internal consistency.

The NoiSeQ has been used in recent studies on environmental noise exposure and noise sensitivity (e.g., Sandrock et al., 2009; Schreckenberg et al., 2010a; Schreckenberg et al., 2010b; Shepherd et al., 2010). A cross-national analysis indicated that the overall measure has strong reliability, ranging from 0.90 to 0.91 (Sandrock et al., 2007). In this study, the global NoiSeQ scale had excellent internal consistency (Cronbach's $\alpha = .92$). Please see Appendix J (p. 261) for complete questionnaire.

6.2.3.3. Subjective health complaints. The SHCI was described previously (see Study 1 Method section, p. 107; measure included in Appendix D, p. 255). In the present study, the total health complaints (severity) score was used. The internal consistency of the scale was good in Study 2 (Cronbach's $\alpha = .83$).

6.2.3.4. Perceived stress. The PSS was described previously (see Study 1 Method section, p. 108; measure included in Appendix E, p. 256). It was included to assess the overall psychological stress in the participant's life during the previous 30 days. In this study, internal consistency for the scale was very good (Cronbach's $\alpha = .88$).

6.2.3.5. Neuroticism. The Neuroticism Scale of the BFI was developed by John, Donahue, and Kentle (1991) to measure the personality dimension. The scale was described previously (see Study 1 Method section, p. 108; measure included in Appendix F, p. 257). It was used in Study 1 to capture negative affectivity, a strong correlate of noise sensitivity (Iwata, 1984; Öhrström, Björkman, et al., 1988; Smith et al., 2002). The neuroticism scale was used in Study 2 for the same purpose. In this study, the internal consistency was good (Cronbach's $\alpha = .84$).

6.2.3.6. Sleep quality. The Pittsburgh Sleep Quality Index (PSQI) is a widely-used 19-item measure of subjective sleep quality over a one-month period (Buysse, Reynolds, Monk,

Berman, & Kupfer, 1989). Seven component scores are generated from the items, with higher scores indicating greater severity of sleep problems (all component scores range from 0-3; see Buysse et al., 1989). Subjective sleep quality, sleep latency (i.e., the amount of time it takes to fall asleep), sleep duration, sleep efficiency (i.e., the number of hours typically awake during the day), sleep disturbance, use of sleeping medication, and daytime dysfunction (i.e., tiredness during daily activities) are the component scores that can be generated. The total score of the PSQI is calculated through adding together the seven component scores (possible range: 0-21). Adequate internal consistency, test-retest reliability and validity have been reported for the PSQI (Buysse et al., 1989). In the present study, the PSQI had acceptable internal consistency (Cronbach's $\alpha = .65$). Please see Appendix K (p. 263) for questionnaire.

6.2.3.7. Salivary cortisol. In order to determine cortisol concentration (nmol/l), salivary cortisol assays were performed commercially (LabPlus, Auckland City Hospital) using cortisol electrochemiluminescence immunoassay (ECLIA) kits (Roche Diagnostics, Switzerland). Participant salivettes were delivered to LabPlus by the research team. Cortisol concentrations (nmol/l) at the time of awakening, at 30 minutes and at 60 minutes after awakening were determined for each participant.

Using the cortisol values determined by LabPlus, Auckland City Hospital (at awakening, at 30 minutes, and at 60 minutes after awakening), summary CAR values were calculated (see Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). These calculations are based on the overall release of cortisol across the morning sampling period (see Figure 11). The two values calculated were: the CAR area under the curve with respect to the ground (CAR_{auc}; the total volume of cortisol released over the awakening period), and the area under the curve with

respect to increase (CARI; the total volume of cortisol released relative to awakening cortisol level; Pruessner et al., 2003).

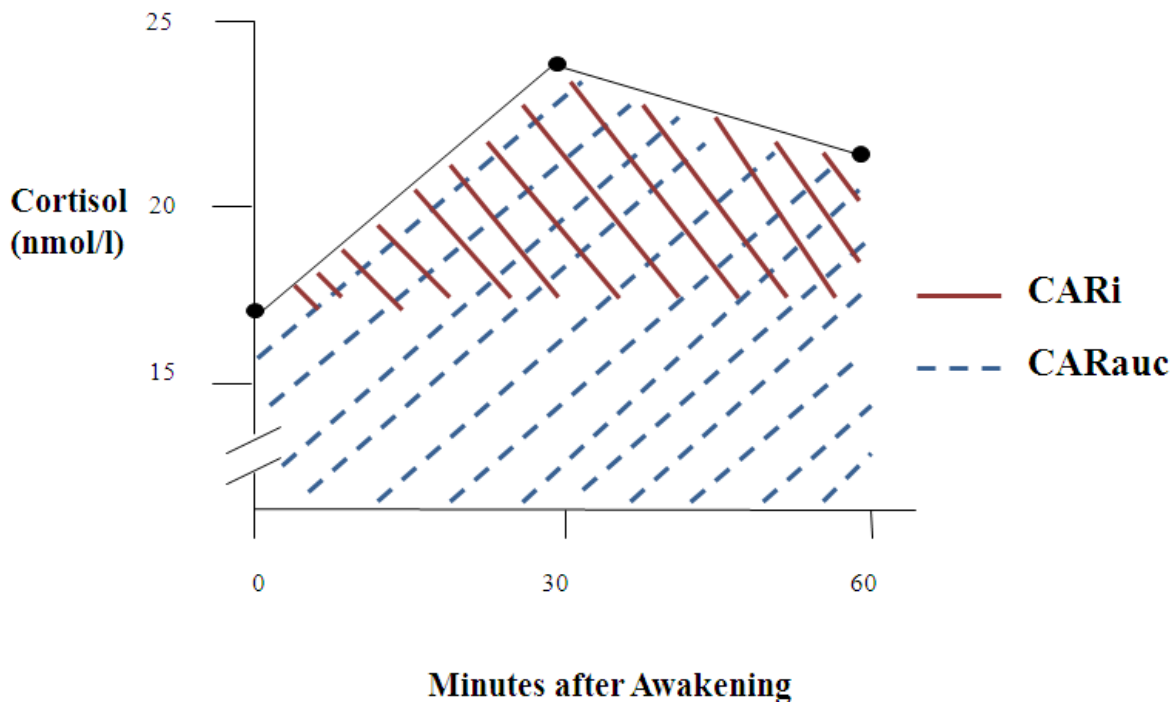


Figure 11. Visual depiction of CAR with respect to increase (CARI) and CAR area under the curve (CARauc).

The CARauc reflects the overall secretion of cortisol from awakening to 60 minutes after awakening, and is calculated by the formula: $((0 \text{ minute cortisol} + 30 \text{ minute cortisol})/2) \times .5) + ((30 \text{ minute cortisol} + 60 \text{ minute cortisol})/2) \times .5$. The CARI reflects the area under the curve with reference to the increase in cortisol following awakening, calculated using the formula: $\text{CARauc} - 0 \text{ minute cortisol}$ (for details regarding the calculations, see Pruessner et al., 2003). Whereas the CARauc provides information about the overall cortisol increase over the awakening period, CARI may provide more accurate information about the reactivity of the HPA axis to awakening (Chida & Steptoe, 2009b).

6.3. Results

6.3.1. Summary of data screening. Data ($n = 107$) were screened for missing values, normality, outliers and multicollinearity. Details of the data screening procedures are presented in Appendix S (p. 298). Due to non-adherence to protocol or issues with cortisol analysis (e.g., not collecting salivary cortisol at 30 minute intervals, not providing enough saliva), the data for 15 participants were not included in the calculation of CAR values. The subjective health complaints variable was positively skewed, and following a square root transformation, skewness was improved. Listwise deletion was applied, and therefore the subsection of the sample used for regression analyses involving cortisol data ($n = 92$) was smaller than the full sample ($n = 107$). Analyses were conducted in SPSS version 19.0.

6.3.2. The sample. Descriptive statistics of the sample are presented in Table 11. The final sample included 107 participants ranging in age from 18 to 78 years ($M = 33.6$; $SD = 15.4$). The sample was composed of 51 men (47.7%) and 56 women (52.3%). New Zealand European was the most frequently reported ethnicity (43.9%), and most participants were either students (44.9%) or working full-time (32.7%). A small subsection reported belonging to more than one ethnic group⁵⁶ ($n = 6$; 5.6%). Two participants (1.9% of sample) identified as both New Zealand European and Māori, and are presented as belonging to the Māori ethnic group in Table 11. The remaining participants (4) who identified as belonging to two ethnic groups are classified as belonging to the minority ethnic group in Table 11 (i.e., not New Zealand European/European).

⁵⁶ It should be noted that ethnicity information was collected by self-report and therefore reflects the self-designated identity of the participant. This section of the thesis was written to give a broad description of the sample.

If the participant identified as a member of two minority ethnic groups, the participant is classified as the first ethnicity group listed in the questionnaire.

Table 11. *Socio-demographics of the Study 2 Sample*

Variable	Category	<i>n</i>	(%)
Gender	Male	51	47.7
	Female	56	52.3
Ethnicity	NZ European	47	43.9
	Māori	8	7.5
	Pacific Islander	3	2.8
	Chinese	7	6.5
	Indian	7	6.5
	European	14	13.1
	North American	2	1.9
	Middle Eastern	2	1.9
	African	2	1.9
	Central/South American	2	1.9
	Asian	10	9.3
	Other	3	2.8
Education (Completed)	Secondary school	45	42.0
	Polytechnic	20	18.7
	University	42	39.3
Employment Status	Full-time work	35	32.7
	Part-time work	14	13.1
	Retired	8	7.5
	Student	48	44.8
	Unemployed	2	1.9

Note. There were no missing data for sociodemographic variables.

6.3.3. The cortisol awakening response. Cortisol levels across the awakening period are presented in Table 12. Bivariate correlations were tested between the CAR values and sleep information (time of awakening, number of hours slept). Awakening times ranged from 4:30am to 10:05am ($M = 7:08\text{am}$, $SD = 1:15$), and the number of hours slept ranged from 3.4 to 11 ($M = 7.40$, $SD = 1.21$). There was no correlation between CARauc and awakening time ($r = .014$, $p =$

.897) or the number of hours slept ($\rho = -.002, p = .987$), nor was there a correlation between CARi and awakening time ($r = .032, p = .764$) and the number of hours slept ($\rho = -.037, p = .734$).

Table 12. *Cortisol Levels across the Awakening Period*

	Mean (SD)	Minimum	Maximum
0 min cortisol (nmol/l)	13.72 (4.45)	5.00	26.80
30 min cortisol (nmol/l)	17.80 (6.11)	5.80	30.20
60 min cortisol (nmol/l)	13.63 (6.31)	5.00	34.60
CARauc	15.74 (4.81)	5.88	30.40
CARi	2.02 (4.55)	-9.97	12.75

Fifty-one participants used an alarm to wake up (55.4% of total sample; 38 participants did not use an alarm, 18 did not complete the question). Independent t-tests were used to test whether the CAR was associated with using an alarm to wake up. No differences were found on the CARauc ($t(87) = 1.78, p = .078$) or on the CARi ($t(87) = -.81, p = .419$). Therefore, there was no significant difference in the CAR between those who used an alarm ($M = 16.41, SD = 4.46$ for CARauc; $M = 1.68, SD = 7.73$ for CARi) and those who did not ($M = 14.62, SD = 14.62$ for CARauc; $M = 2.47, SD = 4.38$ for CARi).

6.3.4. Descriptive statistics and bivariate correlations. Descriptive statistics are reported in Table 13 for the variables of interest in the present study: noise sensitivity, perceived stress, subjective health complaints, neuroticism, and sleep quality (PSQI total score). Correlations between the CAR values and the variables of this study are presented in Table 14. A moderate positive correlation was found between CARauc and CARi ($r = .549, p < .001$).

Unexpectedly, the negative correlation between CARauc and perceived stress approached significance ($r = -.195, p = .065$) indicating that as perceived stress levels increased, the overall cortisol released during the awakening period decreased. The correlation between CARauc and sleep-related noise sensitivity ($r = .186, p = .077$) and leisure-related noise sensitivity ($r = .185, p = .077$) both approached significance, indicating that as these types of noise sensitivity increased so did the CARauc. However these trend associations were both very weak ($< .2$). Global noise sensitivity had a weak positive correlation with perceived stress ($r = .195, p = .047$) and neuroticism ($r = .228, p = .019$), as well as a moderate positive correlation with subjective health complaints ($\rho = .441, p < .001$), PSQI ($r = .349, p < .001$), and age ($\rho = .455, p < .001$).

Table 13. *Descriptive Statistics for Health and Psychological Variables*

Variable	n	Mean (SD)	Range	Possible Range
Noise Sensitivity (Global)	107	3.10 (.58)	1.57-4.69	1-5
Noise Sensitivity (Sleep)	107	3.12 (.64)	1.71-4.57	1-5
Noise Sensitivity (Communication)	107	2.93 (.92)	1.00-4.71	1-5
Noise Sensitivity (Work)	107	3.13 (.82)	1.14-5.00	1-5
Noise Sensitivity (Leisure)	107	3.20 (.60)	1.71-4.71	1-5
Noise Sensitivity (Habitation)	107	3.12 (.64)	1.71-4.57	1-5
Perceived Stress	105	15.92 (6.57)	2.00-33.00	0-40
Subjective Health Complaints	107	11.79 (8.06)	.00-39.00	0-87
Subjective Health Complaints ^T	107	3.23 (1.61)	.00-6.24	0-9.33
Neuroticism	107	2.79 (.80)	1.00-4.75	1-5
Sleep Quality (PSQI total score)	102	5.33 (2.75)	1.00-13.00	0-21

Note. Subjective Health Complaints^T = Total Severity Score of Subjective Health Complaints Inventory (square root transformation applied to address positive skew; Eriksen et al., 1999).

Table 14. *Bivariate Correlations among CAR values, Health and Psychological Variables, and Age*

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. CARauc												
2. CARi	.549**											
3. Noise Sens	.105	.039										
4. NS (Sleep)	.186†	.083	.805**									
5. NS (Comm)	-.050	-.101	.810**	.511**								
6. NS (Work)	.104	.057	.742**	.475**	.534**							
7. NS (Leisure)	.185†	.154	.847**	.587**	.614**	.555*						
8. NS (Habit)	.004	-.009	.778**	.509**	.532**	.444**	.698**					
9. PSS	-.195†	-.019	.195*	.146	.164†	.205*	.172†	.089				
10. SHC	.088	.150	.441**	.404**	.260*	.188†	.343**	.330*	.509**			
11. Neurot	-.073	.095	.228**	.236**	.180†	.184†	.155	.133	.654**	.513**		
12. PSQI	.015	.052	.349**	.345**	.283**	.165	.222*	.232*	.365**	.542**	.313*	
13. Age	-.051	-.018	.455**	.402**	.535**	.371**	.516**	.535**	.020	.198*	-.024	.108*

Note. CARauc = cortisol awakening response with respect to the area under the curve, CARi = cortisol awakening response with respect to increase following awakening, Noise Sens = global noise sensitivity score (Noise Sensitivity Questionnaire (NoiSeQ; Schütte et al., 2007), NS (Sleep) = sleep subscale of NoiSeQ; NS (Comm) = communication subscale of NoiSeQ; NS (Work) = work subscale of NoiSeQ; NS (Leisure) = leisure subscale of NoiSeQ; NS (Habit) = habitation subscale of NoiSeQ; SHC = Subjective Health Complaints total severity score (Eriksen et al., 1999), Neurot = Neuroticism scale (BFI; John et al., 1991); PSQI = Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989). Note that higher PSQI scores indicate poorer sleep quality. Spearman's ρ correlations were calculated for the Subjective Health Complaints Inventory, the PSQI, and age due to non-normality of data. ** $p < .001$ * $p < .05$ † $p < .10$

Spearman's ρ correlations were conducted on noise sensitivity and the PSQI subscales (Table 15). Notably, sleep disturbance had a weak positive correlation with noise sensitivity ($\rho = .304, p < .001$), and a weak positive correlation with sleep quality ($\rho = .400, p < .001$).

Table 15. *Bivariate Correlations between Global Noise Sensitivity and PSQI Subscales*

Variable	1	2	3	4	5	6	7
1. NS (global)							
2. Duration	.175†						
3. Disturbance	.304**	-.031					
4. Latency	.117	.278*	.146				
5. Dysfunction	.137	.238*	.192*	.110			
6. Efficiency	.252*	-.188†	.018	-.063	.013		
7. Quality	.400**	.102	.518**	.039	.192*	.002	
8. Medication	.167†	.272*	.240*	.452**	.267*	-.175†	.104

Note. All PSQI subscales are scored such that higher scores indicate greater sleep problems. NS global = global score of Noise Sensitivity Questionnaire (NoiSeQ; Schütte et al., 2007), Duration = Sleep duration subscale of Pittsburgh Sleep Quality Inventory (PSQI; Buysse et al., 1989), Disturbance = Sleep disturbance subscale of PSQI, Latency = Sleep latency subscale of PSQI, Dysfunction = Daytime dysfunction subscale of PSQI, Efficiency = Sleep Efficiency of PSQI, Quality = Sleep quality subscale of PSQI, Medication = Use of sleep medication subscale of PSQI. Note Spearman's ρ correlations are applied due to non-normality of PSQI subscales.

** $p < .001$

* $p < .05$

† $p < .10$

6.3.5. Regression analyses – CAR and noise sensitivity. In order to assess the influence of noise sensitivity on the CAR, hierarchical linear regression analyses were employed. CARauc and CARi were entered in two separate regression models as the outcome variables. Age, gender (female coded 2, male coded 1), and neuroticism were

entered into the model as covariates (block 1), followed by noise sensitivity (block 2). The hierarchical approach was employed to assess the influence of noise sensitivity on CAR beyond the variance explained by the covariates. A total of four predictors were used in each model, in accordance with the regression sample size guidelines of Green (1991; $N > 50 + 8k$).

6.3.5.1. CARauc and noise sensitivity. Table 16 presents the results of the regression of noise sensitivity (and covariates) on the CARauc. The first block (covariates only) of the regression was significant, $F(4, 88) = 6.10, p = .001$, with the covariates explaining 14.4% of the variance in the CARauc ($adj. R^2 = .144$). Only gender emerged as a significant predictor in the first block ($\beta = .417, p < .001$), with females having higher CARauc than males. Neuroticism approached significance in the first block of the model ($\beta = -.172, p = .085$).

The second block of the model, including covariates and noise sensitivity, was also significant, $F(4, 87) = 4.87, p = .001$, with predictors explaining 14.5% of the variance in CARauc ($adj. R^2 = .145$). However, the model was not significantly improved with the inclusion of noise sensitivity, $R^2_{change} = .011, p = .287$. Similar to the previous block, gender was the strongest predictor of CARauc ($\beta = .386, p = .001$). Neuroticism was near significant in the second block of the model ($\beta = -.204, p = .051$), with higher neuroticism scores predictive of lower CARauc values.

Table 16. *Hierarchical Linear Regression of Noise Sensitivity and Covariates on CARauc*

Predictor	β	p	R	R^2	adjusted R^2	F	df	p
<i>Block 1</i>			.415	.172	.144	6.10	4, 88	.001
Gender	.417	<.001						
Age	-.142	.158						
Neuroticism	-.172	.085						
<i>Block 2</i>			.428	.183	.145	4.87	4, 87	.001
Gender	.386	.001						
Age	-.216	.078						
Neuroticism	-.204	.051						
Noise Sensitivity	.140	.287						

6.3.5.2. CARi and noise sensitivity. Table 17 displays the results for the regression of noise sensitivity and covariates on CARi. Neither block 1 of the model nor the overall model (block 2) was significant.

Table 17. *Hierarchical Linear Regression of Noise Sensitivity and Covariates on CARi*

Predictor	β	p	R	R^2	adjusted R^2	F	df	p
<i>Block 1</i>			.221	.049	.016	1.50	3, 88	.220
Gender	.218	.048						
Age	-.067	.530						
Neuroticism	.031	.772						
<i>Block 2</i>			.221	.049	.005	1.12	4, 87	.353
Gender	.223	.052						
Age	-.055	.675						
Neuroticism	.036	.746						
Noise Sensitivity	-.023	.870						

6.3.6. Post-hoc analyses on gender. Gender emerged as a strong predictor of CARauc in the present study. Specifically, females had a significantly higher CARauc than males. In order to further understand relevant gender differences in the primary variables of the study, independent t-tests were conducted. Results are displayed in Table 18. Females reported greater perceived stress, more health complaints, and greater noise sensitivity (global and across the five subscales), and poorer sleep quality than the males in the sample.

Table 18. *Independent t-tests of Differences between Males and Females on Study**Variables*

	<i>n</i>	Males Mean (SD)	Females Mean (SD)	<i>t</i>	df	<i>p</i>
Noise Sensitivity	51M, 56F	2.87 (.51)	3.29 (.57)	-4.02	105	<.001
NS (Sleep)	51M, 56F	2.54 (.83)	3.27 (.87)	-.4.42	105	<.001
NS (Comm)	51M, 56F	2.94 (.82)	3.27 (.79)	-2.15	105	.034
NS (Work)	51M, 56F	2.88 (.62)	3.31 (.63)	-3.52	105	.001
NS (Leisure)	51M, 56F	3.03 (.56)	3.35 (.59)	-2.96	105	.004
NS (Habit)	51M, 56F	2.97 (.62)	3.25 (.63)	-2.36	105	.020
Perceived Stress	51M, 54F	14.40 (5.98)	17.36 (6.82)	-2.36	103	.020
Neuroticism	51M, 56F	2.62 (.75)	2.92 (.82)	-1.95	104	.054
Health Complaints ^T	51M, 56F	2.95 (.99)	3.49 (1.25)	-2.47	105	.015
PSQI total score	49M, 53F	4.65 (2.56)	5.96 (2.79)	-2.46	100	.016

Note. Noise Sensitivity = global score of Noise Sensitivity Questionnaire (NoiSeQ; Schütte et al., 2007), NS (Sleep) = sleep subscale of NoiSeQ, NS (Comm) = communication subscale of NoiSeQ; NS (Work) = work subscale of NoiSeQ; NS (Leisure) = leisure subscale of NoiSeQ; NS (Habit) = habitation subscale of NoiSeQ, Perceived Stress = score of Perceived Stress Scale (Cohen & Williamson, 1988), Sleep Quality = total score of Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989; higher scores indicate poorer sleep quality), Neuroticism = Neuroticism Scale of Big Five Inventory (John et al., 1991), Health Complaints^T = Total Severity Score of Subjective Health Complaints Inventory (SHCI; Eriksen et al., 1999; square root transformation applied to address positive skew).

6.4. Discussion

The purpose of Study 2 was to assess the association between noise sensitivity and HPA axis activity through measurement of the CAR. Contrary to the hypothesis that noise sensitivity would be associated with an elevated CAR, the results did not indicate an association between global noise sensitivity and the CAR values (CAR_{auc}, CAR_i). While none of the health or psychological variables was associated with the CAR_i, there were some trend associations ($p < .10$) found for the CAR_{auc} (perceived stress, sleep-related and leisure-related noise sensitivity; see Table 14, p. 165). Notably, gender emerged as the strongest predictor of CAR_{auc}, with females having significantly higher CAR_{auc} than males. Further, females reported greater perceived stress, greater noise sensitivity, more health complaints, and poorer sleep quality than males (see Table 18, p. 170), indicating that there may be a complex interplay between gender and psychosocial and health factors in predicting the CAR.

Previous psychophysiological research has demonstrated that noise sensitive individuals appear to react more easily to environmental stressors (e.g., noise), and take longer to habituate (Persson Waye et al., 2002; Stansfeld, 1992; Stansfeld & Shine, 1993). Furthermore, noise sensitivity has been associated with elevated cortisol levels during mental task performance (Ljungberg & Neely, 2007; Persson Waye et al., 2002). However, reactivity of the HPA axis (e.g., to an environmental stressor) is regarded as distinct from HPA axis regulation (measured through the CAR). That is, HPA axis dysregulation (i.e., elevated or diminished CAR_{auc} or CAR_i) may occur as a result of chronic stress and related personality traits, among other psychosocial factors (Chida & Steptoe, 2009b). Although there is evidence for a relationship between noise sensitivity and neuroendocrine system reactivity (e.g., Ljungberg & Neely, 2007; Persson Waye et al., 2002), the results of

Study 2 indicate that global noise sensitivity is not associated with HPA axis dysregulation as shown by the non-significant correlation (see Table 14, p. 165) and non-significant regression results for noise sensitivity and CAR values (see Table 16, p. 168; Table 17, p. 169). Similarly, Carlsson et al. (2006) did not find that self-reported environmental annoyance (to electricity and common smells), subjective stress or health complaints were associated with the CAR.

Interestingly, however, there were near-significant positive correlations between CARauc and both sleep-related and leisure-related noise sensitivity (see Table 14, p. 165). That is, increases in sleep-related and leisure-related noise sensitivity were associated with increased CARauc. These trend associations (correlations $p < .10$) are very weak and difficult to explain, especially given the lack of association between global noise sensitivity and CAR despite a very high positive correlation ($> .8$) between the global and the noise sensitivity subscales.

The association between sleep-related noise sensitivity and CARauc could be related to the notion that the CAR occurs because of the transition sleep to awakening (Clow, Hucklebridge, Stalder, et al., 2010; Wilhelm et al., 2007). This transition may be particularly difficult among individuals with high sleep-related noise sensitivity, who may be more likely to experience sleep disturbance. Indeed, the NoiSeQ sleep subscale items largely measure issues such as noise-related difficulty falling asleep or waking during the night when exposed to unwanted noise (e.g., thunder). However, despite a weak-moderate significant correlation between sleep-related noise sensitivity and poor sleep quality (i.e., PSQI total score; see Table 14, p. 165), there was no association was found between sleep quality and the CAR. Therefore, it is unclear as to the reason for the trend association between sleep-related noise sensitivity and CARauc. It is possible though that these small

associations observed between CARauc and the noise sensitivity subscales could be an artefact of the high cortisol levels exhibited by females, who also reported greater noise sensitivity than men. Gender differences found in the present study will be reviewed later in the Study 2 discussion.

The results of the study also suggest a possibly complex interplay among gender, neuroticism, perceived stress and the CAR. Specifically, contrary to some previous research (e.g., Pruessner et al., 2003; Schulz et al., 1998; Wüst et al., 2000a), perceived stress had a near-significant negative association with the CARauc (Table 14, p. 165). That is, elevated stress levels were, unexpectedly, associated with lower overall cortisol levels across the awakening period. Similarly, in the regression analyses, neuroticism approached significance ($p = .051$) in predicting CARauc, indicating that elevated scores on neuroticism were associated with decreased cortisol levels. This association contrasts previous findings that have reported an association between neuroticism and an elevated CAR (e.g., Portella et al., 2005). However, neuroticism has been linked to a blunted cortisol response to a psychological stress test (Oswald et al., 2006), thereby making it difficult to determine the impact of negative affectivity on HPA axis activity and reactivity. Specifically, Oswald et al. (2006) found that, among women, lowered cortisol levels following a psychological stress test (i.e., 5 minute speech and 5 minute mental arithmetic) were associated with higher neuroticism.

Gender emerged as the strongest predictor of CARauc, with females exhibiting significantly higher cortisol levels across the awakening period. However, females also reported greater perceived stress, greater noise sensitivity, more health complaints, and poorer sleep quality than males. Therefore, some of the psychosocial factors associated with gender (i.e., noise sensitivity, perceived stress being elevated among females) were

antagonistically related to CAR_{auc}. In other words, even though there was a positive correlation between noise sensitivity and perceived stress, the variables had opposing correlations with the CAR_{auc}; sleep-related noise sensitivity was associated with higher cortisol levels, while perceived stress was associated with lower levels. However, the psychosocial variables included in the present study did not account for the association between gender and cortisol levels across the awakening period. Instead, gender remained an independent and strong predictor of CAR_{auc} in the regression analyses. Therefore, the gender differences in noise sensitivity, perceived stress and health complaints did not explain the divergence of cortisol awakening patterns across gender.

Similar to the results of the present study, some previous research has indicated an association between gender and the CAR (e.g., Almeida, Piazza, & Stawski, 2009; Griefahn & Robens, 2010; Kunz-Ebrecht et al., 2004; Pruessner et al., 1997; Wright & Steptoe, 2005). Kunz-Ebrecht et al. (2004) reported that on work days (Monday-Thursday), women displayed a greater CAR than men; however, no differences were reported for weekends. More recently, Almeida et al. (2009) found that there are large gender differences in CAR in young to middle adulthood, with the difference closing with age as men begin to exhibit cortisol levels more similar to women.

Interestingly, Almeida et al. (2009) proposed that the association between aging and CAR, most notably exhibited by men, may reflect allostatic processes. In other words, with age, the HPA axis becomes dysregulated, possibly reflecting years of engagement of the stress response and related bodily wear and tear. Women, because of their reported propensity to experience greater levels of psychological stress than men (Matud, 2004; McDonough & Walters, 2001), may be more likely to exhibit allostatic load much earlier in life than men. This theoretical explanation may also explain the greater number of health

complaints reported by women compared to men. However, results from the present study do not support a correlation between age and CAR (see Table 14, p. 165). Therefore, although Almeida et al. (2009) proposed that HPA axis dysregulation might reflect the bodily wear and tear of allostatic load that increases with aging, there is little empirical evidence in this study to support the contention.

Although women are more likely than men to report stress and health complaints (Malmusi, Artazcoz, Benach, & Borrell, 2011; Shih & Eberhart, 2010), these psychological and health factors did not explain the association between CAR and gender. Women were more likely to experience higher levels of perceived stress than men. However, because perceived stress was negatively associated with CAR (albeit very weakly), elevated perceived stress did not adequately explain the propensity for women to exhibit elevated cortisol levels. Although this contradicts some previous research (e.g., Pruessner et al., 2003; Wüst et al., 2000a), similar to results of Study 2, Lovell et al. (2011) reported a trend association between perceived stress, also assessed using the 10-item PSS, and flatter cortisol output across the awakening period. In their study, Lovell et al. (2011) separated the sample ($n = 32$) into two “stress” groups (16 per group) – high stress (PSS scores ranged from 16-26) and lower stress (PSS scores ranged from 3-14). In plotting cortisol levels across the awakening period, Lovell et al. (2011) demonstrated that the peak of cortisol (at 30 minutes post awakening) was lower among the “high stress” participants. However, this finding only approached significance ($p = .09$). Therefore, the precise role of perceived stress in HPA axis regulation remains unclear.

In addition to psychological factors possibly influencing gender differences in CAR, biological factors must also be considered. Compared to men, women produce higher levels of estrogen, a steroid hormone that appears to influence cortisol levels (Wolfram et al.,

2011). A recent study found that gender differences in basal cortisol levels begin to appear with the advent of puberty (Stroud, Papandonatos, Williamson, & Dahl, 2011), which suggests that sex hormones and other biological changes may influence the functioning of the HPA axis. However, in their review of research on the CAR, Fries et al. (2009) contended that the impact of hormones on the CAR is likely negligible. While the biological mechanisms underlying this observation remain unclear, Stroud et al. (2011) proposed that change in HPA axis regulation that begins to occur at puberty may be a factor contributing to the increased risk of anxiety and depression that occurs as females reach adolescence (Piccinelli & Wilkinson, 2000). However, the present study included adults only ($M_{age} = 33.6$; $SD = 15.4$), so this notion could not be examined. Therefore the influence of hormonal factors on CAR, and its relation to mental well-being, requires further study.

The relationship between gender and the CAR may also reflect neurobiological factors, involving the GRs and MRs that serve in the regulation of the HPA axis (de Kloet, Vreugdenhil, Oitzil, & Joels, 1998; Kudielka & Kirschbaum, 2005; Kumsta et al., 2007; Muhtz, Zyriax, Bondy, Windler, & Otte, 2011; van Leeuwen et al., 2010). Cortisol acts on the brain and body via these receptors, and thus the observed gender differences in the CAR may be linked to gender-specific differential functioning or presence of these receptors (Muhtz et al., 2011). While the MRs are believed to influence basal activity of the HPA axis, GRs are involved in the feedback mechanisms following a stress response (Spencer, Kim, Kalman, & Cole, 1998). van Leeuwen et al. (2010) reported that gender-specific gene expression of MRs may help to explain gender differences in cortisol secretion. Additionally, the hyperactive stress response of females could also be linked to sex differences in the limbic system (Kudielka & Kirschbaum, 2005), which, in turn, may help

explain gender differences in anxiety and perceived stress. Further research on this topic would help to clarify sex differences in both HPA axis activity and related neurobiological structures.

In addition to the cortisol levels across the awakening period, gender differences were also found for noise sensitivity, health complaints, and sleep quality (see Table 18, p. 170). Previous research has reported gender differences in noise sensitivity, with females reporting greater sensitivity (e.g., Novak et al., 2010; Rhudy & Meagher, 2001; van Kamp et al., 2004; Widen et al., 2006). However, other research has found no gender differences (Belojević et al., 2003; Enmarker & Boman, 2004; Kjellberg et al., 1996). There is some evidence for a superior auditory system, in terms of tone and loudness discrimination, among females (Sax, 2010; Velle, 1987), which, in turn, may be influenced by sex-related differences in hormones (Rammsayer & Troche, 2011). However, because noise sensitivity is largely regarded as attitudinal (i.e., involving thoughts and emotions) rather than a result of auditory acuity (Ellermeier et al., 2001; Zimmer & Ellermeier, 1999), this biological difference does not adequately explain the observed gender differences. Further, Heinonen-Guzejev et al. (2005) did not report a gender-specific genetic component for noise sensitivity. Therefore, the mechanisms underlying the link between gender, noise sensitivity and health are not yet clear.

Women are more likely to experience distress than men (Mirowsky & Ross, 1995), ruminate about problems (Nolen-Hoeksema, Larson, & Grayson, 1999), and employ emotion-focused coping strategies, which are often less effective than problem-focused coping strategies (Tamres, Janicki, & Helgeson, 2002). In other words, broadly, women appear to be more vulnerable to stress, a key factor in noise sensitivity (Stansfeld, 1992). Therefore, it is possible that women, because of their increased likelihood to experience

stress and distress, are also more likely to report noise sensitivity. Widen et al. (2006) noted that gender differences in noise sensitivity are difficult to explain possibly due to the limited amount of research on gender differences in noise attitudes and noise sensitivity.

In addition to reporting greater noise sensitivity, women also reported more health complaints and poorer sleep quality. These findings are not novel; it is well-established that women report poorer physical and mental health, as well as greater sleep disturbance, than men (e.g., Arber et al., 2009). While there has been some debate as to whether this observation is merely an artefact of women simply being more likely to report distress than men (Mirowsky & Ross, 1995), a recent study suggested that this conclusion may not hold true. Malmusi et al. (2011) reported that women were more likely to experience poorer health than men (38.8% of women reported poor health vs. 27% of men, $p < .001$), and that this difference that could be accounted by the fact that women suffered from chronic illnesses such as high cholesterol, chronic pain, and cardiovascular disease, to a greater extent than men (gender and health condition prevalence chi-square analysis values $p < .001$).

Various social and psychological factors have been cited to explain the discrepancies in health reported between men and women. Women are more susceptible to chronic strain, depressive symptoms, and have lower feelings of control than men (Nolen-Hoeksema et al., 1999), which, in turn, are risk factors for the development of allostatic load (Steptoe & Willemsen, 2004). Yet, there remains a gender gap in mortality rates, with men dying younger than females (Owens, 2002). This reality is in contrast to the findings on gender differences in psychological and physical health. However, it is possible that women live longer with chronic conditions than men. Results of the present study corroborate previous research in that women were more likely to report more health

complaints, noise sensitivity and stress than men (Nivison & Endresen, 1993). However, the precise role of HPA axis dysregulation in the association between gender and health requires further attention.

Noise sensitivity was also positively associated with poorer sleep quality and greater sleep disturbance. Indeed, previous laboratory-based and community surveys have revealed similar findings. Individuals elevated on noise sensitivity have been found to exhibit increased body movements throughout the night (Marks & Griefahn, 2007), and report greater sleep disturbance to environmental noise (e.g., road traffic, railway, aircraft; Aasvang et al., 2008; Nivison & Endresen, 1993; Öhrström et al., 1990; Shepherd et al., 2010).

Adding to this field of research, the present study used the multidimensional PSQI, which allowed for the assessment of a range of possible sleep disturbances. Interestingly, global noise sensitivity was only correlated with sleep disturbance, poor sleep efficiency, and poor sleep quality (see Table 15, p. 166). Correlations were not found for the remaining four PSQI subscales: sleep duration, sleep latency, dysfunction the following day, or use of medication. Therefore, although individuals with elevated noise sensitivity do not report less sleep than those with lower levels of noise sensitivity, noise sensitivity was associated with some sleep problems.

6.4.1. Limitations. Several limitations of Study 2 must be acknowledged. First, the measurement of the CAR is an exploratory physiological biomarker in that the precise role for the dramatic increase in cortisol following awakening is still unknown (Clow, Hucklebridge, & Thorn, 2010). However, over the past 15 years, it has become clear that HPA axis dysregulation may be a potential risk factor for health and psychological problems (for reviews, see Chida & Steptoe, 2009b; Fries et al., 2009). Indeed, various

researchers in the field have tested cortisol levels in relation to a variety of state and trait psychological factors (e.g., Thorn et al., 2009; Walker et al., 2011). Therefore, the study of the association between noise sensitivity and the CAR is nonetheless an important research endeavour.

However, discouragingly, there is limited acknowledgement as to what exactly marks an elevated CAR. Wüst et al. (2000b) in his study of 509 participants, presented normal values for cortisol across the awakening period. Yet, these normal values are not typically cited as reference values in latter studies. Rather, many studies cite patterns of elevations among subgroups (e.g., participants high on neuroticism, work stress, depressive symptomatology) in comparison to control/remaining participants with little reference to what would be expected for HPA dysregulation (e.g., Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009; Huber et al., 2006; Ong, Fuller-Rowell, Bonanno, & Almeida, 2011; Schlotz et al., 2004; Thorn, Hucklebridge, Esgate, Evans, & Clow, 2004; Wessa et al., 2006).

In comparison to the normal values presented by Wüst et al. (2000b), the mean cortisol levels in the present study are low (e.g., 30 minute cortisol = 22.95nmol/l vs. 17.80 nmol/l). However, the cortisol values found in the present study are similar to other studies on the CAR (e.g., Dedovic et al., 2009; Oskis, Loveday, Hucklebridge, Thorn, & Clow, 2009; Shea et al., 2007; Thorn et al., 2004). Therefore, there appears to be little consensus about what constitutes normal cortisol patterns across the awakening period, relevant to comparative studies such as this one. The trend associations found for psychosocial factors (i.e., sleep-related and leisure-related noise sensitivity, perceived stress) and the CAR should be considered with this limitation in mind. Future research, possibly focused on identifying pathology in neurobiological structures involved in the HPA axis (e.g.,

hippocampus), may help to clearly define cortisol volumes that are reflective of HPA axis dysregulation.

Although it was hypothesised that noise sensitivity would be predictive of an elevated CAR (i.e., a positive correlation between noise sensitivity and CAR_{auc}/CAR_i), it is noteworthy that such a relationship might be difficult to detect in samples with highly stressed participants. While elevated cortisol levels are regarded as evidence for HPA axis dysregulation, in cases of extreme stress (e.g., PTSD), cortisol levels can be substantially lower than what would be expected for a normal CAR – also evidence of HPA axis dysregulation (see p. 23; Lauc et al., 2004; Neylan et al., 2005; Wessa et al., 2006; Yehuda et al., 2006). Therefore, it is possible that in any given sample, highly stressed participants (i.e., those who score very highly on the PSS) could contribute to an observed curvilinear relationship between psychological stress and the CAR. Thus, non-significant findings from linear regression analyses could result.

However, in this study, the PSS scores were not significantly different from those in Study 1 (Mann-Whitney $U = 59,623$, $p = .448$; $M = 15.38$, $SD = 6.33$ in Study 1; $M = 15.92$, $SD = 6.57$ in Study 2). Further, there was no evidence of a curvilinear relationship between the CAR values and PSS scores through a visual assessment of a scatterplot of the associations (see Figure T.1. and Figure T.2., p.327 and 328). Therefore, it does not appear that the relationship between perceived stress and the CAR in Study 2 was curvilinear. Nonetheless, the complex nature of the CAR should be considered in studies in order to appropriately assess its relation to the psychosocial variables of interest.

Another limitation that may have influenced this study is participant non-adherence to saliva collection protocol (Thorn, Hucklebridge, Evans, & Clow, 2006). Although strategies were employed to help protect against issues of non-adherence (e.g., flexibility in

date of collection, recording of date and time of collection in order to encourage timely collection of data), as with most research that involves the collection of samples outside of the laboratory, strict adherence cannot be guaranteed. Recently, Griefhan and Robens (2011) noted that delays of up to 10 minutes will not significantly impact CAR results, but delays greater than this may lead to a flattened observed CAR.

Some laboratories have invested in electronic monitoring devices to track when participants remove the lid of the salivette (Kudielka, 2003). This information can be compared to the time the participants recorded taking the sample on the salivette label. However, this method does not guarantee that participants are taking the samples immediately after they wake up. Rather, it only allows for comparison between self-reported collection time and the time of collection as indicated on the electronic monitor (Clow et al., 2004).

The collection of data in the social and biological sciences can be a challenge. In reality, similar to the collection of information through self-report questionnaires, to an extent, researchers are at the mercy of their participants to be truthful in their responses, and similarly, correctly adhere to research protocol. Therefore, in designing such psychobiological studies, it is important to emphasise the importance of adherence to protocol to the participants. Additionally, it would be advantageous for researchers to monitor potential non-adherence (e.g., with electronic monitoring devices, comparing times recorded on salivette to awakening time reported in the questionnaire) in order to identify participants who might not have followed the study protocol appropriately. In this study, participants who had not adhered to study protocol (e.g., did not collect saliva samples at 30 minute intervals) were excluded from the analyses due to potential inaccuracy of data.

While the CAR is regarded as a measurement with good intra-individual stability (Klok et al., 2011; Pruessner et al., 1997; Quirin et al., 2008; Wüst et al., 2000b), there is some debate as to whether more than one set of saliva samples should be collected for CAR studies (e.g., Eek, Karlson, Garde, Hansen, & Orbaek, 2012; Hellhammer et al., 2007; Mikolajczak et al., 2010). The use of the average cortisol response across the two (or more) days is thought to increase reliability of the measure (Hellhammer et al., 2007). In the present study, only one day of collection was chosen, similar to previous research on the topic (de Kloet et al., 2007; Pruessner et al., 2003; Steptoe, Brydon, & Kunz-Ebrecht, 2005; Weekes et al., 2008).

In their recent meta-analysis of CAR and psychosocial factors, Chida and Steptoe (2009b) reported that 45.6% of CAR studies in their sample involved collection on one day, while the remaining involved sample collection across two days or more. The expenses associated with cortisol analyses (e.g., approximately \$18.00 NZD per cortisol sample) can be a limiting factor in this type of research. Indeed, other than the single case-study conducted on the CAR (Stalder et al., 2009), studies have involved sample sizes as low as 12 participants (e.g., Thorn et al., 2004), and several below 50 participants (e.g., Ebrecht et al., 2004; Harris, Ursin, Murison, & Eriksen, 2007; Kuehner, Holzhauer, & Huffziger, 2007; Munafò et al., 2006; Pruessner et al., 2003; Quirin et al., 2008; Therrien et al., 2008; Thorn et al., 2006; Weekes et al., 2008; Williams et al., 2005). These numbers are possibly related to the financial limitations of this method. Because the present study focused on a psychological trait (noise sensitivity), a large sample size (> 100) was sought, in part, to ensure a sufficient range of noise sensitivity scores, and to obtain a large enough sample for the linear regression analyses. Thus, only one day of saliva collection was used in this study. Researchers interested in further investigating HPA axis activity and noise

sensitivity may wish to collect information across a number of days in order to thoroughly assess the potential interaction between noise sensitivity and state factors (e.g., morning activities, state stress).

Limitations in the recruitment method used in Study 2 should also be noted. As mentioned in the method section (p. 153), one of the ways participants were recruited was through a newspaper article about “people who are sensitive to noise” (see Appendix O, p. 291). While this method encouraged the participation of individuals with elevated noise sensitivity, and therefore was a proactive strategy in attempting to diminish potential issues with data range restriction, this approach may have created some bias in the data. Specifically, recruitment through the newspaper article might have contributed to the high correlation between noise sensitivity and age, as many of the participants recruited using this method were middle-aged or elderly women. However, similar to results of Study 1, noise sensitivity was normally distributed (see Figure S.5., p. 306) which has also been reported in other noise sensitivity studies (e.g., Belojević & Jakovljević, 2001; Ekehammar & Dornic, 1990; Shepherd et al., 2010). In sum, the results should be interpreted with these limitations in mind.

6.4.2. Recommendations for future research. Many of the limitations of this study stem from the challenges of measuring cortisol through saliva samples. This method may be susceptible to non-adherence and the CAR may be impacted by both state and trait factors (Griefahn & Robens, 2011; Hellhammer et al., 2007). Recently, the measurement of cortisol through hair samples has emerged as a promising biomarker in stress research (Gow, Thomson, Rieder, Van Uum, & Koren, 2010; Karlen, Ludvigsson, Frostell, Theodorsson, & Faresjo, 2011; Sauvé, Koren, Walsh, Tokmakejian, & Van Uum, 2007; Van Uum et al., 2008). In contrast to saliva cortisol measurements, the assessment of

cortisol in hair is unaffected by participant adherence to protocol. Hair grows at the rate of approximately 1cm per month, and therefore long-term cortisol release can be determined (Sauvé et al., 2007). Especially given that noise sensitivity is regarded as a stable personality trait, which may contribute to a vulnerability to stressors in general (Heinonen-Guzejev, 2009; Stansfeld, 1992), cortisol production in hair may allow for better assessment of long-term cortisol output. This would provide valuable information about chronic heightened HPA axis activity in relation to stress vulnerability-related personality traits such as noise sensitivity.

It may also be advantageous for future research to consider both HPA axis activity (as measured by the CAR) in conjunction with stress reactivity of the HPA axis. Although previous research indicates that noise sensitivity is associated with stress reactivity of the HPA axis and SAM system (Griefahn & Di Nisi, 1992; Ljungberg & Neely, 2007), the relation between the acute stress response and general functioning of the HPA axis in relation to noise sensitivity has not been adequately tested. It is possible that noise sensitivity impacts acute stress responses, but does not impact general HPA axis functioning (e.g., as measured through the CAR). Further research studying both stress system activity and reactivity would help to establish neuroendocrine reactivity and functioning in relation to noise sensitivity. Further recommendations for the overall programme of research are discussed in the general discussion (see p. 199).

6.4.3. Conclusion. Overall, the results of Study 2 did not provide evidence for an association between noise sensitivity and HPA axis dysregulation. Global noise sensitivity was not associated with the CAR summary values (CARauc/CARi). However, there were trend associations between sleep-related and leisure-related noise sensitivity and the CARauc. In particular, the trend association between sleep-related noise sensitivity and the

CARauc probes the role of noise-disturbed sleep and the sleep-to-wake transition in HPA axis regulation. This association, however, was difficult to explain because of the lack of association between sleep quality and the CAR. Notably, gender emerged as the strongest predictor of CARauc. Compared to males, females had significantly higher cortisol levels across the awakening period. Females also reported greater noise sensitivity, greater perceived stress, more health complaints and poorer sleep quality. Despite no evidence for an association between HPA axis activity and global noise sensitivity, the results of this study revealed considerable gender differences in neuroendocrine functioning, as well as psychological and health factors including noise sensitivity.

Chapter 7: General Discussion

The present thesis was designed to further elucidate the mechanisms underlying the association between noise sensitivity and diminished health. Results from Study 1 provide considerable evidence for the influence of psychological factors and sleep problems in the association between noise sensitivity and health complaints. Study 2 revealed that the association between noise sensitivity and health complaints may not be explained by dysregulation of the HPA axis. However, results from Study 2 provide insight into the relationship among gender, noise sensitivity, and the CAR. The general discussion of the thesis has been structured to provide an overview of the thesis findings, and place them within the context of related public health and health psychology research. Limitations of the thesis and recommendations for future research in the field are further discussed.

7.1. Stress Factors in the Relationship between Noise Sensitivity and Diminished Health

There has been increasing evidence for the association between noise sensitivity and diminished health (Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2004, 2007; Kishikawa et al., 2009). Using an allostatic load framework (see p. 14), this thesis sought to determine the role of stress factors – both psychological and physiological – in this association. Results of the thesis indicate that the relationship between noise sensitivity and self-reported health problems (measured with subjective health complaints) is mediated by perceived stress and sleep problems. However, this mediation did not apply to the mental health complaints models. Rather, neuroticism accounted for the relationship between noise sensitivity and anxiety and depression complaints. Noise sensitivity has previously been linked to mental health conditions (Kishikawa et al., 2009; Stansfeld, 1992), and the results of this thesis implicate neuroticism as a major factor in this relationship.

However, results of the thesis do not provide evidence for the role of HPA axis regulation, as measured by the CAR, in the association between noise sensitivity and self-reported health. Overall, this thesis provides considerable insight into psychological factors (e.g., perceived stress, neuroticism) that influence the relationship between noise sensitivity and diminished health. Further, sleep problems also play an important role in mediating the association, which, in turn, could initiate an additional avenue for further understanding the impact of noise sensitivity on health.

7.2. Concerns of Spuriousness in Stress-Health Research

Before further discussing the findings of the thesis, it is pertinent to recognise the concern of spuriousness in stress-health research. Specifically, there is the concern that the correlations between self-reported psychological stress and diminished health are influenced by the propensity or willingness of participants to report distress (e.g., Lazarus, DeLongis, Folkman, & Gruen, 1985; Watson & Pennebaker, 1989). In other words, the correlation between self-reported physical disorders and psychological stress might actually reflect psychological distress rather than an actual relationship between the two variables.

Watson and Pennebaker (1989) discussed the problematic contamination of negative affectivity in the association between stress and health complaints. Specifically, in their review of research on stress, distress and health complaints, they proposed that negative affectivity largely explains the association between self-reported stress and health complaints. Because noise sensitivity is largely a complex attitudinal trait, which may contain a negative affectivity component (Brooker, 2010; Ellermeier et al., 2001; Job, 1999; Stansfeld, 1992), this issue is relevant to the results of this thesis. As previously discussed in the literature review (p. 93), the issue of spuriousness has been broached in previous studies on the relationship between noise sensitivity and diminished health (Babisch, 2010;

Smith et al., 2002). In their survey on noise, sleep and health, Smith et al. (2002) reported that the association between noise sensitivity and self-reported health disappeared when negative affectivity (measured by a neuroticism scale) was taken into account.

Results of this thesis confirm that negative affectivity, indeed, plays a role in the association between noise sensitivity and self-reported mental health (anxiety and depression complaints). Similar to previous research, in this thesis negative affectivity was measured with a neuroticism scale (e.g., Bouchard & Poirier, 2011; Pasch et al., 1997; Shackman et al., 2011). However, in contrast to what Smith et al. (2002) proposed, the results of Study 1 revealed that the association between noise sensitivity and subjective health complaints is not fully accounted for by negative affectivity. Rather, sleep problems and perceived stress mediated the relationship, even after controlling for the influence of neuroticism in the model (see Figure 7, p. 130).

Watson and Pennebaker (1989) discussed that measures of health complaints may be particularly vulnerable to contamination by negative affectivity in comparison to more objective health outcomes (e.g., lifestyle choices such as smoking or drinking). This thesis involved a measure of health complaints, in part, to allow comparisons to the limited research on the association between noise sensitivity and diminished health, (e.g., Fyhri & Aasvang, 2010; Nivison & Endresen, 1993), which also used subjective health complaints as a health outcome. Therefore, to help combat the possible influence of negative affectivity in the association between noise sensitivity and health complaints, neuroticism was included as a covariate in the statistical models in both studies. Indeed, this is the recommended protocol in assessing traits and outcomes that may be influenced by negative affectivity (e.g., Cohen & Williamson, 1988; MacKinnon & Luecken, 2008; Watson & Pennebaker, 1989).

Although negative affectivity was assessed in both studies of the thesis, the measurement of psychological traits in relation to stress and health is nonetheless limited by self-report questionnaire approaches. Future research on the association between noise sensitivity and diminished health may benefit from using other health criteria (e.g., laboratory-tested medical conditions), in addition to health complaints, in order to gain a better understanding of the relationship between noise sensitivity and diminished health (Fyhri & Aasvang, 2010; Fyhri & Klæboe, 2009; Kishikawa et al., 2009; Nivison & Endresen, 1993).

However, it also must be emphasised that measures of health complaints, although subjective, do provide a relevant and reliable indication of the participant's health status (e.g., Irish, Kobayashi, & Delahanty, 2010; Maas, van Dillen, Verheij, & Groenewegen, 2009; van den Berg et al., 2010). Health complaints scales have been used to monitor recovery from acute illness such as a cardiac event (e.g., Denollet, 1994; Pelle, Pedersen, Szabo, & Denollet, 2009) and as a health outcome in public health studies (e.g., Berg-Beckhoff et al., 2009; Levin, Inchley, Currie, & Currie, 2012; Maas et al., 2009; van den Berg et al., 2010). Additionally, subjective health complaints are strong predictors of visits to a medical physician and taking sick leave from work (Eriksen & Ursin, 2004), thus highlighting its relevance to public health research. Despite the limitations of subjective health complaints measures, such as potential contamination by negative affectivity (Watson & Pennebaker, 1989), it is clear that such self-report measures are suitable options for assessing ill-health, particularly in large survey-based studies. Further, the influence of negative affectivity was taken into account in this thesis, thus allowing the explicit association between noise sensitivity and diminished health to be illuminated.

Further, it is notable that much of the literature discussing the role of negative affectivity in the relationship between stress and health date over 20 years ago (e.g., Cohen & Williamson, 1988; Lazarus et al., 1985; Watson & Pennebaker, 1989). Research since then has convincingly demonstrated that the relationship between stress and health is not insignificant. For example, the groundbreaking study conducted by Cohen et al. (1991) on the impact of psychological stress on susceptibility to developing cold symptoms provided clear empirical evidence for the deleterious effects of psychological stress on immune functioning. Recent meta-analyses have shown that having a stress-prone personality is, indeed, a risk factor in the development of cancer and cancer-related mortality (Chida, Hamer, Wardle, & Steptoe, 2008) as well as coronary heart disease (Chida & Steptoe, 2009a). Such research thus provides considerable insight into the actual impact of negative affectivity and related personality traits on health and disease.

7.3. Gender, Coping, and Noise Sensitivity

Across both studies of this thesis, gender differences in noise sensitivity and health complaints were found. As discussed in the literature review (p. 82), gender differences in noise sensitivity have been previously reported (e.g., Novak et al., 2010; van Kamp et al., 2004). However, there are other studies that have reported no gender differences (Belojević et al., 2003; Enmarker & Boman, 2004; Widen et al., 2011). Additionally, some research on noise sensitivity and diminished health has highlighted that the association may be particularly strong among women (Babisch, 2010; Nivison & Endresen, 1993). Thus, further understanding the gender differences in noise sensitivity is relevant in health research.

Study 1 attempted to assess the moderating role of gender in the association between noise sensitivity and the model mediators (perceived stress, sleep problems).

Gender did not emerge as a significant moderator in the model. However, assessment of direct gender differences in both studies revealed that females reported greater noise sensitivity and health complaints than males. Additionally, Study 2 revealed that females also report poorer sleep quality and exhibited higher CAR levels than males. Therefore, gender differences in health-related variables and noise sensitivity would seem to merit further investigation.

The gender differences in noise sensitivity and subjective health complaints could also be linked to coping styles. Matud (2004) revealed that women reported greater emotional and avoidance focused coping strategies than men. Relevant to this thesis, women reported greater somatic complaints and psychological distress than men. Given that noise sensitivity is a complex variable (Job, 1999; van Kamp et al., 2004), it is possible that coping mechanisms play a role. In other words, it is possible that gender differences emerged in noise sensitivity and health complaints due to contrasting coping styles between men and women. It could be that emotion-based coping increases an individual's susceptibility to the effects of noise as well as other stressors. In other words, an individual may self-perceive as noise sensitive because they are more likely to become upset by noise exposure, or lack in problem-focused coping strategies in dealing with noise (for a more detailed discussion, see p. 62).

Noise sensitivity was also significantly associated, albeit weakly, with lack of perceived control over sources of noise at home and at work in Study 1 (see Table 9, p. 123), further suggesting that coping could be impacting an individual's self-reported noise sensitivity. Avoidant coping has been associated with noise sensitivity (Pulles, Siesiot, & Stewart, 1990), and there has been some discussion of the relationship between noise sensitivity and coping strategies particularly among nurses working in hospital (Topf, 1985,

1989). Specifically, Topf (1989) found that noise sensitivity was inversely related to psychological hardiness among her sample of nurses. Noise sensitivity was also linked to lack of coping strategies in dealing with noise-induced stress in the hospital setting. This avenue of research may not only be applicable to noise stress in hospital workers, but it may be helpful in better understanding the relationship between noise sensitivity and diminished health, as well as the relationship among noise sensitivity, stress, and coping.

7.4. Implications of Noise Sensitivity Research in Improving Health

As previously discussed, there has been a limited body of research addressing the association between noise sensitivity and negative health outcomes (e.g., Fyhri & Klæboe, 2009; Heinonen-Guzejev et al., 2004, 2007). One possible reason for this is that the policy implications of such research findings are not as easy to implement as those on the health effects of noise exposure. In public health research on noise exposure (e.g., aircraft, railway, motorway), there are policy changes, such as noise by-laws and guidelines for building noise-generating infrastructure that can be implemented (e.g., location of flight pathways, distance between residential communities and wind turbines). In contrast, noise sensitivity is a trait variable, which, by its nature, is less easy to address or change. Arguably then, noise sensitivity research complicates rather than aids in the designing of simple policy-based solutions for the increasing problem of noise pollution (Berglund et al., 1999; Flindell & Stallen, 1999; World Health Organization, 2009).

Nonetheless, given the increasing volume of research emphasising the influence of noise on health and well-being (e.g., Babisch et al., 2009; Gan et al., 2012; Selander et al., 2009b; World Health Organization, 2009), investigating the role of noise sensitivity as a moderator of the noise-health relationship, and as an independent predictor of health outcomes, will be an important area for further research. Results of this thesis indicate that

the association between noise sensitivity and subjective health complaints is mediated by perceived stress and sleep problems. Further, this relationship was not modified by objective or perceived noise exposure. These results suggest that individuals elevated on noise sensitivity may experience more health problems as a result of excessive perceived stress and sleep problems.

Across both studies of the thesis, noise sensitivity was associated with self-reported sleep problems. One limitation of the two studies is that the nature of the sleep problems was not sufficiently clarified. Specifically, it is not clear whether the sleep problems reported were associated with noise or other problems such as anxiety, which is also strongly associated with noise sensitivity (Nivison & Endresen, 1993; Stansfeld, 1992). In previous research, noise sensitive individuals have reported noise disturbance to both quiet and loud noises (Job, 1999). If noise sensitive individuals suffer from sleep problems more than those less sensitive due to common neighbourhood and household noises (e.g., dogs barking, noisy flatmates, snorers), some interventions could possibly help. For example, some noise exposure within houses can be improved with structural changes (e.g., replacing single or double-pane windows with triple-pane, insulation in the walls), which, in turn, may improve the living conditions of those vulnerable to a variety of noise disturbances. However, because people generally move into previously-inhabited houses, any changes to the house may require additional funds and hence rely on the financial capacity of the individual residents. Further, some of the most disturbing noises that occur during the night may come from within the home (e.g., snorers), which may require various individual-based strategies to reduce noise-related disturbance (e.g., wearing ear plugs, sleeping in a spare bedroom if available).

In a related vein, there is evidence that having access to a quiet space within one's home can ameliorate the ill-effects of noise exposure (Klæboe, 2007; Öhrström et al., 2006). Access to quiet is believed to impact psychological stress recovery (Pedersen & Persson Waye, 2008), which may be particularly beneficial for those most sensitive to noise. Similarly, in recent years, there has been a surge of evidence for the benefit of restorative soundscapes and natural physical environments (e.g., access to green space) in health and psychological stress recovery (e.g., Gidlöf-Gunnarsson & Öhrström, 2007; Miles, Coutts, & Mohamadi, 2012; Ulrich et al., 1991; van den Berg et al., 2010). This thesis identified perceived stress as one of the mechanisms linking noise sensitivity to reported health problems. Building upon these results, the interaction among restorative environments, noise sensitivity, stress, and health may be a particularly fruitful avenue for research directed to protecting the health of those most sensitive to noise.

It must be emphasised that although the inclusion of noise sensitivity in environmental noise research complicates understanding, this thesis does not undermine the importance of noise reduction strategies for the benefit of population health. Rather, in order to protect the health and well-being of those most sensitive to noise in the population, more creative strategies need to be employed at both the individual and community level. Specifically, drawing upon the results of this thesis, reducing sleep disturbance as well as improving recovery from psychological stress in relation to both acoustic and physical environments may be particularly important.

7.5. Clarifying the Concept of Noise Sensitivity

This thesis was designed to examine the role of stress in the relationship between noise sensitivity and diminished health. As discussed in Chapter 4 (p. 64), noise sensitivity is typically regarded as a stable personality trait that affects an individual's vulnerability to noise and other stressors (Heinonen-Guzejev, 2009; Stansfeld, 1992). This assumption underlies the central research question of the thesis. However, the complexity of the concept must be realised when analysing the results of this thesis.

Several researchers have noted that the concept of noise sensitivity itself is not well understood (Job, 1999; Miedema, 2007; Miedema & Vos, 2003; van Kamp et al., 2004). For example, in his discussion of the trait of noise sensitivity, Job (1999) proposed that noise sensitivity influences sensitivity to loud noises as well as sensitivity to distractions. However, noise sensitivity does not appear to be related to auditory processes, but rather, is largely a psychological trait (Ellermeier et al., 2001).

Beyond the few studies investigating the psychoacoustic and neurophysiological correlates of noise sensitivity (e.g., Ellermeier et al., 2001; Öhrström, Björkman, et al., 1988), there has been limited discussion of the concept of noise sensitivity itself in the literature (Smith, 2003). Instead, researchers within the field of public health acknowledge that noise sensitivity is an individual difference variable that can impact a person's response to noise-related disturbance (Belojević & Jakovljević, 2001; Heinonen-Guzejev, 2009). As such, public health-orientated studies often opt to measure noise sensitivity using self-report questionnaires, and assess or control for its influence in the analyses (e.g., Babisch et al., 1999; Birk et al., 2011; Bodin et al., 2012; Brooker, 2010; Jakovljević et al., 2009; Klæboe et al., 2004; Willich et al., 2006).

A clearer understanding of noise sensitivity in terms of its stability and relationship with emotions and coping strategies may be helpful in developing interventions to improve the health and stress levels of those most sensitive to noise. Noise sensitivity largely encompasses the individual differences in noise reactions, and therefore may involve individual differences in coping, and emotional and sensory processes. Further understanding these factors will not only aid in clarifying the nature of the trait itself, but will also help in understanding the association between noise sensitivity and diminished health.

7.6. Strengths and Limitations

Limitations of each of the studies in the thesis have been presented in each of the study discussions, and have also been interspersed throughout the general discussion. It has been emphasised throughout the general discussion that both noise sensitivity and health are multifaceted and complex concepts that may be influenced by a variety of factors. The results of the overall thesis should be considered in light of the limitations of this research programme. The main limitations of the thesis are summarised below.

First, the two studies of the thesis were cross-sectional, which, in turn, limits the extent to which definitive conclusions can be drawn about the direction of relationships among study variables. Second, although there is a large body of research confirming the negative impact of stress on health and well-being (e.g., Chida et al., 2008; Cohen et al., 2007), the potential bias in self-report studies must be acknowledged.

Finally, the CAR was chosen as an outcome variable for assessing functioning of the HPA axis. While the CAR is becoming a popular method of measuring neuroendocrine activity, especially in relation to health and psychological variables (e.g., Chida & Steptoe, 2009b; Clow, Hucklebridge, Stalder, et al., 2010; Dedovic et al., 2010; Merswolken, Deter,

Siebenhuener, Orth-Gomer, & Weber, 2012), the precise neurological basis for the CAR is unknown. This limits the strength of the conclusions for the null findings between global noise sensitivity and CAR in Study 2. Nonetheless, with the number of research centres using the CAR across a variety of studies (e.g., Adam et al., 2010; Heaney, Phillips, & Carroll, 2010; Mangold et al., 2012; van Santen et al., 2011; Zoccola et al., 2011), its precise role in health problems, well-being and daily functioning (e.g., the awakening process) will hopefully be clarified in the near future.

Despite the limitations of this research, this thesis helps fill a gap in the literature through the investigation of stress-related factors in the relationship between noise sensitivity and diminished health. While this relationship has been reported in previous studies (Heinonen-Guzejev et al., 2004; Kishikawa et al., 2009; Nivison & Endresen, 1993), some dating back over 25 years (Kelly, 1986; Stansfeld et al., 1985a), the mechanisms involved have still not been adequately explained. This research involved both a large community-based study (Study 1) as well as a physiological study (Study 2). This allowed for the testing of associations between variables using a large community-based sample ($n > 1000$). Perceived stress was a significant mediator of the association between noise sensitivity and health complaints, even after controlling for the influence of neuroticism. The results also implicated sleep problems as an intermediary factor in the relationship between noise sensitivity and diminished health, thus confirming previous laboratory research (e.g., Marks & Griefahn, 2007; Öhrström & Björkman, 1988).

The physiological study, Study 2, involved a measure of HPA axis activity, which indicated that the association between noise sensitivity and diminished health may not be due to dysregulation of the HPA axis. This evidence then points researchers to study other potential physiological underpinnings of the relationship between noise sensitivity and

diminished health through assessment of other allostatic load parameters (e.g., immune system biomarkers, blood pressure, adrenaline, noradrenaline, cholesterol).

7.7. Future Directions in Research on Noise Sensitivity and Diminished Health

Future directions for the study of the association between noise sensitivity and diminished health have been highlighted throughout the thesis. Broadly, they fall into three areas of study: (1) psychological stress in relation to coping and recovery, (2) the measurement of allostatic load and health outcomes, and (3) clarification of the nature of noise sensitivity and its association with diminished health through longitudinal research.

7.7.1. Psychological stress. Psychological stress (i.e., perceived stress) appears to play an important role in the link between noise sensitivity and health complaints. Therefore, further understanding the role of stress in relation to noise sensitivity offers an interesting and relevant avenue for future study. As previously discussed, because noise sensitivity is largely a stable attitudinal trait (Ellermeier et al., 2001; Smith, 2003; Stansfeld, 1992), the coping mechanisms employed by the highly noise sensitive might provide some insight into how noise sensitive individuals perceive and cope with noise and other stressors.

Psychological stress may be improved through access to restorative physical (e.g., greenspace; Li, Chau, & Tang, 2010; van den Berg et al., 2010; Yang, Bao, & Zhu, 2011) and acoustic (e.g., access to a quiet room in the house; Öhrström et al., 2006) environments. Therefore, although noise sensitivity largely complicates the findings of public health research concerning noise by suggesting that individuals are not equally impacted by noise, it alludes that the interventions to protect the highly noise sensitive should involve considering the types of local acoustic and physical environments that may improve psychological stress.

7.7.2. Allostatic load. This thesis also investigated the relationship between noise sensitivity and HPA axis activity using the CAR. HPA axis regulation is only one physiological parameter that provides insight into the association between psychosocial factors and health. Although noise sensitivity was not strongly associated with the CAR, the stress vulnerability component of noise sensitivity may be reflected in other physiological outcomes. Composite measures of allostatic load (see Juster et al., 2010), previously discussed in Chapter 2 (p. 18), may provide a more thorough view of the physiological impact of noise sensitivity on the body (e.g., immune system biomarkers, blood pressure, cholesterol). The CAR was chosen as the most relevant physiological parameter for the overall aim of the thesis for practical, financial, and popular reasons. Nonetheless, where research funds allow, using other allostatic load biomarkers in relation to noise sensitivity may be valuable in determining the physiological mechanisms possibly underlying the relationship between noise sensitivity and diminished health.

7.7.3. Health outcomes. Future research on the association between noise sensitivity and diminished health would benefit from using various health outcome measures ranging from the subjective to the objective. Subjective measures that can be completed within minutes, such as the Subjective Health Complaints Inventory, offer the advantage of being useful in large community-based studies (such as Study 1). Such subjective measures are relatively easy for the participant to complete (e.g., in comparison to undergoing a physician examination), thus decreasing participant burden, and in turn, help in encouraging participation (Galea & Tracy, 2007). However, self-report measures are inevitably susceptible to bias because they are based on the participant's own subjective perceptions.

In order to continue to build upon this avenue of research, comparison among subjective health and well-being measures (e.g., health complaints, HRQOL) in relation to objective health outcomes (e.g., laboratory-confirmed medical conditions, mortality) in noise sensitivity research will be helpful in placing the body of research on the relationship between noise sensitivity and diminished health in context. Heinonen-Guzejev et al. (2004, 2007) have used objective outcomes including cardiovascular disease incidence and mortality in studying the relationship between noise sensitivity and diminished health. However, beyond these studies on the Finnish Twin Cohort, there has been limited research on the relationship between noise sensitivity and objective health outcomes.

Further, it must be emphasised that *health* is a broad concept, involving physical, psychological, and social components (Jadad & O'Grady, 2008). Therefore, this thesis, in focusing on self-reported health complaints and regulation of the HPA axis, assessed diminished health rather than encompassing other aspects of well-being. Noise sensitivity appears to be related to subjective health complaints, as well as perceived stress and sleep problems. Testing other health outcomes that fall under the broad concept of health, such as quality of life, social well-being, and disease incidence, will appropriately expand this area of research.

7.7.4. Longitudinal research. Much of the research conducted on the topic of noise sensitivity has been cross-sectional (e.g., Fyhri & Klæboe, 2009; Kishikawa et al., 2009; Nivison & Endresen, 1993; Schreckenberget al., 2010a). As discussed, this limits the extent to which conclusions can be made about causal relationships between variables. The model proposed in this thesis was that noise sensitivity leads to health problems via stress-related factors. Although Study 1 provided support for this model, the study design was

nonetheless cross-sectional. Therefore, it is possible that health problems contribute to stress perceptions and noise sensitivity.

Experimental studies are regarded as the gold standard in psychological research. They allow experimental manipulation of independent variables, and in turn, conclusions about causation can be inferred from the results. However, given that this thesis attempted to study noise sensitivity in relation to health problems, experimental manipulation of either of these primary variables was not feasible. Thus, this thesis relied on naturalistic cross-sectional studies. In future research, quality longitudinal studies would offer a better understanding of the nature of noise sensitivity, as well as a clearer picture of the direction of the relationship among noise sensitivity, stress and health problems.

7.8. Conclusion

In conclusion, this thesis provides considerable insight into the association between noise sensitivity and diminished health. Perceived stress and sleep problems mediated the association between noise sensitivity and subjective health complaints, while neuroticism accounted for the relationship between noise sensitivity and mental health complaints. Exposure to road traffic noise did not impact these relationships. Noise sensitivity was not associated with HPA axis activity, as measured with the CAR. This indicates that other physiological parameters may provide clearer information about the biological relevance of the relationship between noise sensitivity and diminished health. However, gender largely influenced the CAR levels in Study 2, thereby possibly occluding any potential conclusions about the association between psychosocial variables and the CAR.

Importantly, this research has addressed an important gap that exists in the noise and health literature through the examination of the under-studied relationship between noise sensitivity and diminished health (Heinonen-Guzejev et al., 2007; Kishikawa et al.,

2009; Schreckenberg et al., 2010a; van Kamp et al., 2004). With the increase in environmental noise exposure across the globe (Berglund et al., 1999; Prasher, 2009; World Health Organization, 2009), noise sensitivity will likely remain an important variable in noise-related public health research. Further, because of its association with perceived stress and health complaints, this research has demonstrated that noise sensitivity is an important personal trait to consider in health psychology research. In sum, this research provides empirical evidence for the role of perceived stress and sleep problems in the relationship between noise sensitivity and diminished health, which may aid public health researchers and health psychologists in understanding psychosocial factors that negatively impact health.

Summary of Thesis Findings

Findings Relevant to the Research Question

- The relationship between noise sensitivity and health complaints was significantly mediated by perceived stress and sleep problems.
- The relationship between noise sensitivity and mental health complaints (anxiety, depression) was accounted for by neuroticism.
- There was no evidence of an association between noise sensitivity and self-reported hypertension.
- Gender and road traffic noise exposure did not influence the association between noise sensitivity and mediators (perceived stress and sleep problems).
- Noise sensitivity was not associated with the CAR.

Additional Findings

- Gender significantly influenced CAR levels, with females exhibiting greater cortisol secretion across the awakening period (CAR_{auc}).
- Across both studies, females reported greater noise sensitivity and health complaints.

References

- Aasvang, G. M., Moum, T., & Engdahl, B. (2008). Self-reported sleep disturbances due to railway noise: Exposure-response relationships for nighttime equivalent and maximum noise levels. *Journal of the Acoustical Society of America*, 124(1), 257-268. doi:10.1121/1.2932074
- Aasvang, G. M., Overland, B., Ursin, R., & Moum, T. (2011). A field study of effects of road traffic and railway noise on polysomnographic sleep parameters. *Journal of the Acoustical Society of America*, 129, 3716-3726.
- Abel, S. M., Krever, E. M., & Alberti, P. W. (1990). Auditory detection, discrimination and speech processing in ageing, noise-sensitive and hearing-impaired listeners. *Scandinavian Audiology*, 19, 43-54.
- Abey-Wickrama, I., A'Brook, M. F., Gattoni, F. E. G., & Herridge, C. F. (1969). Mental hospital admissions and aircraft noise. *The Lancet ii*, 1275-1277. doi:10.1016/S0140-6736(69)90810-1
- Abrorelius, L., Owens, M. J., Plotsky, P. M., & Nemeroff, C. B. (1999). The role of corticotropin-releasing factor in depression and anxiety disorders. *Journal of Endocrinology*, 160, 1-12. doi:10.1677/joe.0.1600001
- Adam, E. K., Doane, L. D., Zinbag, R. E., Mineka, S., Craske, M. G., & Griffith, J. W. (2010). Prospective prediction of major depressive disorder from cortisol awakening responses in adolescence. *Psychoneuroendocrinology*, 35(6), 921-931. doi:10.1016/j.psyneuen.2009.12.007
- Adler, N. E., & Newman, K. (2002). Socioeconomic disparities in health: Pathways and policies. *Health Affairs*, 21(2), 60-76. doi:10.1377/hlthaff.21.2.60
- Al-Mutairi, N. Z., Al-Attar, M. A., & Al-Rukaibi, F. S. (2011). Traffic-generated noise pollution: Exposure of road users and populations in Metropolitan Kuwait. *Environmental Monitoring and Assessment*, 183(1-4), 65-75. doi:10.1007/s10661-011-1906-0
- Alimohammadi, I., Nassiri, P., Azkhosh, M., & Hoseini, M. (2010). Factors affecting road traffic noise annoyance among white-collar employees working in Tehran. *Iranian Journal of Environmental Health Science & Engineering*, 7(1), 25-34.
- Almeida, D. M., & Horn, M. C. (2004). Is daily life more stressful during middle adulthood? In O. G. Brim, C. D. Ryff, & R. C. Kessler (Eds.), *How healthy are we? A national study of well-being at midlife* (pp. 425-451). Chicago, IL: University of Chicago Press.
- Almeida, D. M., Piazza, J. R., & Stawski, R. S. (2009). Interindividual differences and intraindividual variability in the cortisol awakening response: An examination of age and gender. *Psychology and Aging*, 24(4), 819-827. doi:10.1037/a0017910
- Amann, E., Lercher, P., Weichbold, V., & Eisenmann, A. (2007, August). *The effects of measuring noise sensitivity with a single or several rating scale on the population prevalence and distribution of this personality trait*. Paper presented at the meeting of the Inter-Noise, Istanbul, Turkey.
- Ana, G. R., Shendell, D. G., Brown, G. E., & Sridhar, M. K. (2009). Assessment of noise and associated health impacts at selected secondary schools in Ibadan, Nigeria. *Journal of Environmental and Public Health*, 2009, 1-6. doi:10.1155/2009/739502
- Anderson, C. M. B. (1971). *The measurement of attitude to noise and noises*. Middlesex, UK: National Physical Laboratory Acoustics Report, Ac 52.

- Andersson, L., Johansson, A., Millqvist, E., Nordin, S., & Bende, M. (2008). Prevalence and risk factors for chemical sensitivity and sensory hyperreactivity in teenagers. *International Journal of Hygiene and Environmental Health*, 211(5-6), 690-697. doi:10.1016/j.ijheh.2008.02.002
- Aniansson, G., Pettersson, K., & Peterson, Y. (1983). Traffic noise annoyance and noise sensitivity in persons with normal and impaired hearing. *Journal of Sound and Vibration*, 88(1), 85-97. doi:10.1016/0022-460X(83)90680-6
- Arber, S., Bote, M., & Meadows, R. (2009). Gender and socio-economic patterning of self-reported sleep problems in Britain. *Social Science & Medicine*, 68(2), 281-289. doi:10.1016/j.socscimed.2008.10.016
- Aron, E. N., & Aron, A. (1997). Sensory-processing sensitivity and its relation to introversion and emotionality. *Journal of Personality and Social Psychology*, 73(2), 345-368. doi:10.1037/0022-3514.73.2.345
- Aron, E. N., Aron, A., & Davies, K. M. (2005). Adult shyness: The interaction of temperamental sensitivity and an adverse childhood environment. *Personality & Social Psychology Bulletin*, 31(2), 181-197. doi:10.1177/0146167204271419
- Aydin, Y., & Kaltenbach, M. (2007). Noise perception, heart rate and blood pressure in relation to aircraft noise in the vicinity of the Frankfurt airport. *Clinical Research in Cardiology*, 96(6), 347-358. doi:10.1007/s00392-007-0507-y
- Babisch, W. (2000). Traffic noise and cardiovascular disease: Epidemiological review and synthesis. *Noise & Health*, 2(8), 9-32.
- Babisch, W. (2002). The noise/stress concept, risk assessment and research needs. *Noise & Health*, 4(16), 1-11.
- Babisch, W. (2003). Stress hormones in research on cardiovascular effects of noise. *Noise & Health*, 5(18), 1-10.
- Babisch, W. (2006). Transportation noise and cardiovascular risk: Updated review and synthesis of epidemiological studies indicate that the evidence has increased. *Noise & Health*, 8, 1-28.
- Babisch, W. (2010, June). *Noise sensitivity in cardiovascular noise studies*. Paper presented at the meeting of the Inter-Noise, Lisbon, Portugal.
- Babisch, W. (2011). Cardiovascular effects of noise. *Noise & Health*, 13(52), 201-204. doi:10.4103/1463-1741.80148
- Babisch, W., Beule, B., Schust, M., Kersten, N., & Ising, H. (2005). Traffic noise and risk of myocardial infarction. *Epidemiology*, 16(1), 33-40. doi:10.1097/01.ede.0000147104.84424.24
- Babisch, W., Fromme, H., Breyer, A., & Ising, H. (2001). Increased catecholamine levels in urine in subjects exposed to road traffic noise: The role of stress hormones in noise research. *Environment International*, 26, 475-481. doi:10.1016/S0160-4120(01)00030-7
- Babisch, W., Houthuijs, D., Pershagen, G., Cadum, E., Katsouyanni, K., Velonakis, M., ... Jarup, L. (2009). Annoyance due to aircraft noise has increased over the years--results of the HYENA study. *Environment International*, 35(8), 1169-1176. doi:10.1016/j.envint.2009.07.012
- Babisch, W., Ising, H., Gallacher, J. E., Sweetnam, P. M., & Elwood, P. C. (1999). Traffic noise and cardiovascular risk: The Caerphilly and Speedwell studies, third phase--10-year follow up. *Archives of Environmental Health*, 54(3), 210-216. doi:10.1080/00039899909602261

- Babisch, W., Schulz, C., Seiwert, M., & Conrad, A. (2010). Noise annoyance as reported by 8- to 14-year-old children. *Environment and Behavior*, 44(1), 68-86. doi:10.1177/0013916510387400
- Barnes, P. J., & Adcock, I. M. (2009). Glucocorticoid resistance in inflammatory diseases. *The Lancet*, 373, 1905-1917. doi:10.1016/S0140-6736(09)60326-3
- Barnett, V., & Lewis, T. (1994). *Outliers in statistical data*. Chichester, UK: Wiley & Sons.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51, 1173-1182.
- Barozzi, S., Soi, D., Comiotto, E., Borghi, A., Gavioli, C., Spreafico, E., ... Brambilla, D. (2012). Audiological findings in Williams syndrome: A study of 69 patients. *American Journal of Medical Genetics Part A*, 158A(4), 759-771. doi:10.1002/ajmg.a.35241
- Barregard, L. (2011). Traffic noise and hypertension. *Environmental Research*, 111(1), 186-187. doi:10.1016/j.envres.2010.10.008
- Barregard, L., Bonde, E., & Öhrström, E. (2009). Risk of hypertension from exposure to road traffic noise in a population-based sample. *Occupational and Environmental Medicine*, 66(6), 410-415. doi:10.1136/oem.2008.042804
- Basner, M., Samel, A., & Isermann, U. (2006). Aircraft noise effects on sleep: Application of the results of a large polysomnographic field study. *Journal of the Acoustical Society of America*, 119(5), 2772-2784. doi:10.1121/1.2184247
- Baum, A. (1990). Stress, intrusive imagery, and chronic distress. *Health Psychology*, 9(6), 653-675.
- Baum, A., Singer, J. E., & Baum, C. S. (1984). Stress and the environment. *Journal of Social Issues*, 37, 4-35.
- Becker, K. D., & Chorpita, B. F. (2008). Negative affect. In A. R. Eisen (Ed.), *Treating childhood behavioral and emotional problems: A step-by-Step, evidence-based approach* (pp. 212-265). New York, NY: The Guilford Press.
- Beelen, R., Hoek, G., Houthuijs, D., van den Brandt, P. A., Goldbohm, R. A., Fischer, P., ... Brunekreef, B. (2009). The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. *Occupational and Environmental Medicine*, 66(4), 243-250. doi:10.1136/oem.2008.042358
- Bell, I. R., Hardin, E. E., Baldwin, C. M., & Schwartz, G. E. (1995). Increased limbic system symptomatology and sensitizability of young adults with chemical and noise sensitivities. *Environmental Research*, 70, 84-97. doi:10.1006/enrs.1995.1052
- Belojević, G., & Jakovljević, B. (2001). Factors influencing subjective noise sensitivity in an urban population. *Noise & Health*, 4(13), 17-24.
- Belojević, G., Jakovljević, B., & Aleksić, O. (1997). Subjective reactions to traffic noise with regard to some personality traits. *Environment International*, 23(2), 221-226.
- Belojević, G., Jakovljević, B., & Slepcević, V. (2003). Noise and mental performance: Personality attributes and noise sensitivity. *Noise & Health*, 6(21), 77-89.
- Belojević, G., Jakovljević, B., Stojanov, V., Paunovic, K., & Ilic, J. (2008). Urban road-traffic noise and blood pressure and heart rate in preschool children. *Environment International*, 34(2), 226-231. doi:10.1016/j.envint.2007.08.003
- Belojević, G., Jakovljević, B., Stojanov, V. J., Slepcević, V. Z., & Paunovic, K. Z. (2008). Nighttime road-traffic noise and arterial hypertension in an urban population. *Hypertension Research*, 31, 775-781. doi:10.1291/hypres.31.775

- Belojević, G., Öhrström, E., & Rylander, R. (1992). Effects of noise on mental performance with regard to subjective noise sensitivity. *International Archives of Occupational and Environmental Health*, 64, 293-301.
- Belojević, G., & Saric-Tanaskovic, M. (2002). Prevalence of arterial hypertension and myocardial infarction in relation to subjective ratings of traffic noise exposure. *Noise & Health*, 4, 33-37.
- Bendokiene, I., Grazuleviciene, R., & Dedele, A. (2011). Risk of hypertension related to road traffic noise among reproductive-age women. *Noise & Health*, 13(55), 371-377. doi:10.4103/1463-1741.90288
- Benfield, J. A., Nurse, G. A., Jakubowski, R., Gibson, A. W., Taff, B. D., Newman, P., & Bell, P. A. (2012). Testing noise in the field: A brief measure of individual noise sensitivity. *Environment and Behavior*. doi:10.1177/0013916512454430
- Benham, G. (2006). The Highly Sensitive Person: Stress and physical symptom reports. *Personality and Individual Differences*, 40(7), 1433-1440. doi:10.1016/j.paid.2005.11.021
- Berg-Beckhoff, G., Blettner, M., Kowall, B., Breckenkamp, J., Schlehofer, B., Schmiedel, S., ... Schuz, J. (2009). Mobile phone base stations and adverse health effects: Phase 2 of a cross-sectional study with measured radio frequency electromagnetic fields. *Occupational and Environmental Medicine*, 66(2), 124-130. doi:10.1136/oem.2008.039834
- Berglund, B., Hassmen, P., & Soames Job, R. F. (1996). Sources and effects of low-frequency noise. *Journal of the Acoustical Society of America*, 99(5), 2985-3002. doi:10.1121/1.414863
- Berglund, B., & Lindvall, T. (1995). *Community Noise*. Stockholm, Sweden: Stockholm University and Karolinska Institutet.
- Berglund, B., Lindvall, T., & Schwela, D. H. (1999). *Guidelines for community noise*. Geneva, Switzerland: World Health Organization.
- Bhattacharyya, M. R., Molloy, G. J., & Steptoe, A. (2008). Depression is associated with flatter cortisol rhythms in patients with coronary artery disease. *Journal of Psychosomatic Research*, 65(2), 107-113. doi:10.1016/j.jpsychores.2008.03.012
- Binder, E. B., & Nemeroff, C. B. (2010). The CRF system, stress, depression and anxiety—insights from human genetic studies. *Molecular Psychiatry*, 15, 574-588. doi:10.1038/mp.2009.141
- Birk, M., Ivina, O., von Klot, S., Babisch, W., & Heinrich, J. (2011). Road traffic noise: Self-reported noise annoyance versus GIS modelled road traffic noise exposure. *Journal of Environmental Monitoring*, 13(11), 3237-3245. doi:10.1039/c1em10347d
- Black, D., Black, J., Issarayangyun, T., & Samuels, S. (2007). Aircraft noise exposure and resident's stress and hypertension: A public health perspective for airport environmental management. *Journal of Air Transport Management*, 13(5), 264-276. doi:10.1016/j.jairtraman.2007.04.003
- Bluhm, G., Berglind, N., Nordling, E., & Rosenlund, M. (2007). Road traffic noise and hypertension. *Occupational and Environmental Medicine*, 64(2), 122-126. doi:10.1136/oem.2005.025866
- Bluhm, G., Nordling, E., & Berglind, N. (2004). Road traffic noise and annoyance—an increasing environmental health problem. *Noise & Health*, 6(24), 43-49.
- Bobak, M., Pikhart, H., Hertzmann, C., Rose, R., & Marmot, M. (1998). Socioeconomic factors, perceived control and self-reported health in Russia. A cross-sectional

- survey. *Social Science & Medicine*, 47, 269-279. doi:10.1016/S0277-9536(98)00095-1
- Bodin, T., Albin, M., Ardo, J., Stroh, E., Ostergren, P. O., & Bjork, J. (2009). Road traffic noise and hypertension: Results from a cross-sectional public health survey in southern Sweden. *Environmental Health*, 8, 38. doi:10.1186/1476-069X-8-38
- Bodin, T., Bjork, J., Öhrström, E., Ardo, J., & Albin, M. (2012). Survey context and question wording affects self reported annoyance due to road traffic noise: A comparison between two cross-sectional studies. *Environmental Health*, 11(1), 14. doi:10.1186/1476-069X-11-14
- Bolin, K., Bluhm, G., Eriksson, G., & Nilsson, M. E. (2011). Infrasound and low frequency noise from wind turbines: Exposure and health effects. *Environmental Research Letters*, 6(3), 035103. doi:10.1088/1748-9326/6/3/035103
- Boman, E., & Enmarker, I. (2004). Factors affecting pupils' noise annoyance in schools: The building and testing of models. *Environment & Behavior*, 36(2), 207-228. doi:10.1177/0013916503256644
- Bonnet, M. H. (2005). Acute sleep deprivation. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (pp. 51-66). Philadelphia, PA: Elsevier, Inc.
- Booi, H., & van den Berg, F. (2012). Quiet areas and the need for quietness in Amsterdam. *International Journal of Environmental Research and Public Health*, 9(4), 1030-1050. doi:10.3390/ijerph9041030
- Borg, G. (1998). *Borg's perceived exertion and pain scales*. Champaign, IL: Human Kinetics.
- Bouchard, G., & Poirier, L. (2011). Neuroticism and well-being among employed new parents: The role of the work-family conflict. *Personality and Individual Differences*, 50(5), 657-661. doi:10.1016/j.paid.2010.12.012
- Bouma, E. M., Riese, H., Ormel, J., Verhulst, F. C., & Oldehinkel, A. J. (2009). Adolescents' cortisol responses to awakening and social stress; effects of gender, menstrual phase and oral contraceptives. The TRAILS study. *Psychoneuroendocrinology*, 34(6), 884-893. doi:10.1016/j.psyneuen.2009.01.003
- Bowsher, J. M., Johnson, D. R., & Robinson, D. W. (1966). A further experiment on judging the noisiness of aircraft flight. *Acustica*, 17, 245-267.
- Boyce, W. T., & Ellis, B. J. (2004). Biological sensitivity to context: I. An evolutionary-developmental theory of origins and functions of stress reactivity. *Development and Psychopathology*, 17, 271-301. doi:10.1017/S0954579405050145
- Brant, H., Wetherell, M. A., Lightman, S., Crown, A., & Vedhara, K. (2010). An exploration into physiological and self-report measures of stress in pre-registration doctors at the beginning and end of a clinical rotation. *Stress*, 13(2), 155-162. doi:10.3109/10253890903093778
- Brink, M. (2011). Parameters of well-being and subjective health and their relationship with residential traffic noise exposure — A representative evaluation in Switzerland. *Environment International*, 37(4), 723-733. doi:10.1016/j.envint.2011.02.011
- Broderick, J. E., Arnold, D., Kudielka, B. M., & Kirschbaum, C. (2004). Salivary cortisol sampling compliance: Comparison of patients and healthy volunteers. *Psychoneuroendocrinology*, 29(5), 636-650. doi:10.1016/s0306-4530(03)00093-3
- Broner, N. (1978). The effects of low frequency noise on people — A review. *Journal of Sound and Vibration*, 58(4), 483-500.

- Brooker, P. (2010). Aircraft noise annoyance estimation: UK time-pattern effects. *Applied Acoustics*, 71(7), 661-667. doi:10.1016/j.apacoust.2010.01.010
- Brophy, K. M., Scarlett-Ferguson, H., & Webbers, K. S. (2010). *Clinical drug therapy for Canadian practice*. Philadelphia, PA: Lippincott Williams & Wilkins.
- Bruehl, H., Wolf, O. T., & Convit, A. (2009). A blunted cortisol awakening response and hippocampal atrophy in type 2 diabetes mellitus. *Psychoneuroendocrinology*, 34(6), 815-821. doi:10.1016/j.psyneuen.2008.12.010
- Buchanan, T. W., Tranel, D., & Kirschbaum, C. (2009). Hippocampal damage abolishes the cortisol response to psychosocial stress in humans. *Hormones and Behavior*, 56(1), 44-50. doi:10.1016/j.yhbeh.2009.02.011
- Buford, T. W., & Willoughby, D. S. (2008). Impact of DHEA(S) and cortisol on immune function in aging: A brief review. *Applied Physiology, Nutrition, and Metabolism*, 33(3), 429-433. doi:10.1139/H08-013
- Bugental, D. B., & Cortez, V. L. (1988). Physiological reactivity to responsive and unresponsive children as moderated by perceived control. *Child Development*, 59(3), 686-693.
- Bullinger, M., Hygge, S., Evans, G. W., & von Mackensen, S. (1999). The psychological cost of aircraft noise for children. *International Journal of Hygiene and Environmental Health*, 202(2-4), 127-138.
- Buyse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, 28, 193-213.
- Campbell, J. B. (1992). Extraversion and noise sensitivity: A replication of Dornic and Ekehammar's study. *Personality and Individual Differences*, 13(8), 953-955. doi:10.1016/0191-8869(92)90015-H
- Campbell, J. M. (1983). Ambient stressors. *Environment and Behavior*, 15(3), 355-380. doi:10.1177/0013916583153005
- Cannon, W. B. (1929). Organization for physiological homeostasis. *Physiological Reviews*, IX(3), 399-431.
- Carlsson, F., Persson, R., Karlson, B., Österberg, K., Hansen, Å. M., Garde, A. H., & Ørbæk, P. (2006). Salivary cortisol and self-reported stress among persons with environmental annoyance. *Scandinavian Journal of Work, Environment & Health*, 32(2), 109-120. doi:10.5271/sjweh.986
- Carrion, V. G., Weems, C. F., & Reiss, A. L. (2007). Stress predicts brain changes in children: A pilot longitudinal study on youth stress, posttraumatic stress disorder, and the hippocampus. *Pediatrics*, 119(3), 509-516. doi:10.1542/peds.2006-2028
- Catlin, F. I. (1965). Noise and emotional stress. *Journal of Chronic Diseases*, 18, 509-518.
- Cavatorta, A., Falzoi, M., Romanelli, A., Cigala, F., Ricco, M., Bruschi, G., ... Borghetti, A. (1987). Adrenal response in the pathogenesis of arterial hypertension in workers exposed to high noise levels. *Journal of Hypertension*, 5(suppl 5), S463-S466.
- Chang, T. Y., Lai, Y. A., Hsieh, H. H., Lai, J. S., & Liu, C. S. (2009). Effects of environmental noise exposure on ambulatory blood pressure in young adults. *Environmental Research*, 109(7), 900-905. doi:10.1016/j.envres.2009.05.008
- Charmandari, E., Tsigos, C., & Chrousos, G. (2005). Endocrinology of the stress response. *Annual Review of Physiology*, 67, 259-284. doi:10.1146/annurev.physiol.67.040403.120816

- Chida, Y., Hamer, M., Wardle, J., & Steptoe, A. (2008). Do stress-related psychosocial factors contribute to cancer incidence and survival? *Nature Reviews Clinical Oncology*, 5, 466-476. doi:10.1038/nrponc1134
- Chida, Y., & Steptoe, A. (2008). Positive psychological well-being and mortality: A quantitative review of prospective observational studies. *Psychosomatic Medicine*, 70, 741-756. doi:10.1097/PSY.0b013e31818105ba
- Chida, Y., & Steptoe, A. (2009a). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology*, 53(11), 936-946. doi:10.1016/j.jacc.2008.11.044
- Chida, Y., & Steptoe, A. (2009b). Cortisol awakening response and psychosocial factors: a systematic review and meta-analysis. *Biological Psychology*, 80(3), 265-278. doi:10.1016/j.biopsycho.2008.10.004
- Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews. Endocrinology*, 5(7), 374-381. doi:10.1038/nrendo.2009.106
- Clark, C., Martin, R., van Kempen, E., Alfred, T., Head, J., Davies, H. W., ... Stansfeld, S. A. (2006). Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: The RANCH project. *American Journal of Epidemiology*, 163(1), 27-37. doi:10.1093/aje/kwj001
- Clark, C., & Stansfeld, S. A. (2007). The effect of transportation noise on health and cognitive development: A review of recent evidence. *International Journal of Comparative Psychology*, 20, 145-158.
- Clausen, T., Christensen, K. B., Lund, T., & Kristiansen, J. (2009). Self-reported noise exposure as a risk factor for long-term sickness absence. *Noise & Health*, 11, 93-97.
- Clow, A., Hucklebridge, F., Stalder, T., Evans, P., & Thorn, L. (2010). The cortisol awakening response: More than a measure of HPA axis function. *Neuroscience and Biobehavioral Reviews*, 35(1), 97-103. doi:10.1016/j.neubiorev.2009.12.011
- Clow, A., Hucklebridge, F., & Thorn, L. (2010). The cortisol awakening response in context. *International Review of Neurology*, 93, 153-175. doi:10.1016/S0074-7742(10)93007-9
- Clow, A., Thorn, L., Evans, P., & Hucklebridge, F. (2004). The awakening cortisol response: Methodological issues and significance. *Stress*, 7(1), 29-37. doi:10.1080/10253890410001667205
- Cohen, S. (1985). Cognitive processes as determinants of environmental stress. *Issues in Mental Health Nursing*, 7(1-4), 85-81. doi:10.3109/01612848509009450
- Cohen, S., Evans, G. W., Krantz, D. S., & Stokols, D. (1980). Physiological, motivational, and cognitive effects of aircraft noise on children. *American Psychologist*, 35(1), 231-243.
- Cohen, S., Evans, G. W., Krantz, D. S., Stokols, D., & Kelly, S. (1981). Aircraft noise and children: Longitudinal evidence on adaptation to noise and the effectiveness of noise abatement. *Journal of Personality and Social Psychology*, 40(2), 331-345.
- Cohen, S., Evans, G. W., Stokols, D., & Krantz, D. (1986). *Behavior, health, and environmental stress*. New York, NY: Plenum Press.
- Cohen, S., Glass, D. C., & Phillips, S. (1979). Environment and health. In H. Freeman, S. Levine, & L. G. Reeder (Eds.), *Handbook of medical sociology* (pp. 134-149). Cliffs, NJ: Prentice-Hall.

- Cohen, S., Glass, D. C., & Singer, J. E. (1973). Apartment noise, auditory discrimination, and reading ability in children. *Journal of Experimental Social Psychology*, 9, 407-422.
- Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *Journal of the American Medical Association*, 298(14), 1685-1687. doi:10.1001/jama.298.14.1685
- Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, 24, 385-396.
- Cohen, S., Tyrrell, D. A. J., & Smith, A. P. (1991). Psychological stress and susceptibility to the common cold. *New England Journal of Medicine*, 325, 606-612.
- Cohen, S., & Weinstein, N. (1981). Nonauditory effects of noise on behavior and health. *Journal of Social Issues*, 37(1), 36-70. doi:10.1111/j.1540-4560.1981.tb01057.x
- Cohen, S., & Williamson, G. M. (1988). Perceived stress in a probability sample of the United States. In S. Spacaman & S. Oskamp (Eds.), *The social psychology of health* (pp. 31-67). Newbury Park, CA: Sage.
- Collins, F. L., Sorrocco, K. H., Halaa, K. R., Miller, B. I., & Lovallo, W. R. (2003). Stress and health. In L. E. Cohen, C. E. McChargue, & F. L. Collins (Eds.), *The health psychology handbook: Practical issues for the behavioral specialist* (pp. 169-186). London, UK: Sage.
- Cuijpers, P., Smith, F., Penninx, B. W., de Graaf, R., ten Have, M., & Beekman, A. T. F. (2010). Economic costs of neuroticism. *Archives of General Psychiatry*, 67(1086-1093).
- Curtis, B. M., & O'Keefe, J. H. (2002). Autonomic tone as a cardiovascular risk factor: The dangers of chronic fight or flight. *Mayo Clinic Proceedings*, 77(1), 45-54. doi:10.4065/77.1.45
- Darbonne, A., Uchino, B. N., & Ong, A. D. (2012). What mediates links Between age and well-being? A test of social support and interpersonal conflict as potential interpersonal pathways. *Journal of Happiness Studies*. doi:10.1007/s10902-012-9363-1
- Daruna, J. H. (2004). *Introduction to psychoneuroimmunology*. Oxford, UK: Elsevier, Inc.
- Davis, R. R., Kozel, P., & Erway, L. C. (2003). Genetic influences in individual susceptibility to noise: A review. *Noise & Health*, 5, 19-28.
- de Jong, R. G. (1990). Community response to noise: A review of recent developments. *Environment International*, 16, 515-522. doi:10.1016/0160-4120(90)90020-7
- de Kloet, C. S., Vermetten, E., Heijnen, C. J., Geuze, E., Lentjes, E. G., & Westenberg, H. G. (2007). Enhanced cortisol suppression in response to dexamethasone administration in traumatized veterans with and without posttraumatic stress disorder. *Psychoneuroendocrinology*, 32(3), 215-226. doi:10.1016/j.psyneuen.2006.12.009
- de Kloet, E. R., Joels, M., & Holsboer, F. (2005). Stress and the brain: From adaptation to disease. *Nature Reviews. Neuroscience*, 6(6), 463-475. doi:10.1038/nrn1683
- de Kloet, E. R., Vreugdenhil, E., Oitzil, M. S., & Joels, M. (1998). Brain corticosteroid receptor balance in health and disease. *Endocrine Reviews*, 19(3), 269-301.
- de Kluizenaar, Y., Gansevoort, R., Miedema, H. M. E., & de Jong, P. E. (2007). Hypertension and road traffic noise exposure. *Journal of Occupational and Environmental Medicine*, 49(5), 484-492. doi:10.1097/JOM.0b013e318058a9ff
- de Kluizenaar, Y., Janssen, S. A., van Lenthe, F. J., Miedema, H. M. E., & Mackenbach, J. P. (2009). Long-term road traffic noise exposure is associated with increase in

- morning tiredness. *Journal of the Acoustical Society of America*, 126(2), 626-633. doi:10.1121/1.3158834
- De Vente, W., Olff, M., Van Amsterdam, J. G. C., Kamphuis, J. H., & Emmelkamp, P. M. G. (2003). Physiological differences between burnout patients and healthy controls: Blood pressure, heart rate, and cortisol responses. *Occupational and Environmental Medicine*, 60, 54i-61. doi:10.1136/oem.60.suppl_1.i54
- Dedovic, K., Duchesne, A., Andrews, J., Engert, V., & Pruessner, J. C. (2009). The brain and the stress axis: The neural correlates of cortisol regulation in response to stress. *NeuroImage*, 47(3), 864-871. doi:10.1016/j.neuroimage.2009.05.074
- Dedovic, K., Engert, V., Duchesne, A., Lue, S. D., Andrews, J., Efanov, S. I., ... Pruessner, J. C. (2010). Cortisol awakening response and hippocampal volume: Vulnerability for major depressive disorder? *Biological Psychiatry*, 68(9), 847-853. doi:10.1016/j.biopsych.2010.07.025
- Denollet, J. (1994). Health complaints and outcome assessment in coronary heart disease. *Journal of Psychosomatic Research*, 56(5), 463-474.
- Di Nisi, J., Muzet, A., & Weber, L. D. (1987). Cardiovascular responses to noise: Effects of self-estimated sensitivity to noise, sex, and time of the day. *Journal of Sound and Vibration*, 114(2), 271-279. doi:10.1016/S0022-460X(87)80153-0
- Dickerson, S. S., & Kemeny, M. E. (2002). Acute stressors and cortisol reactivity: A meta-analytic review. *Psychosomatic Medicine*, 64, 105-123.
- Dijk, D. J. (2008). Slow-wave sleep, diabetes, and the sympathetic nervous system. *Proceedings of the National Academy of Sciences of the United States of America*, 105(4), 1107-1108. doi:10.1073/pnas.0711635105
- Dornic, S., & Ekehammar, B. (1990). Extraversion, neuroticism, and noise sensitivity. *Personality and Individual Differences*, 11(9), 989-992. doi:10.1016/0191-8869(90)90283-W
- Dratva, J., Phuleria, H. C., Foraster, M., Gaspoz, J. M., Keidel, D., Kunzli, N., ... Schindler, C. (2012). Transportation noise and blood pressure in a population-based sample of adults. *Environmental Health Perspectives*, 120(1), 50-55. doi:10.1289/ehp.1103448
- Dratva, J., Zemp, E., Felber Dietrich, D., Bridevaux, P. O., Rochat, T., Schindler, C., & Gerbase, M. W. (2010). Impact of road traffic noise annoyance on health-related quality of life: Results from a population-based study. *Quality of Life Research*, 19(1), 37-46. doi:10.1007/s11136-009-9571-2
- Eberhardt, J. L. (1988). The influence of road traffic on sleep. *Journal of Sound and Vibration*, 127(3), 449-455. doi:10.1016/0022-460X(88)90369-0
- Eberhardt, J. L., Strale, L.-O., & Berlin, M. H. (1987). The influence of continuous and intermittent traffic noise on sleep. *Journal of Sound and Vibration*, 116(3), 445-464. doi:10.1016/S0022-460X(87)81376-7
- Ebrecht, M., Hextall, J., Kirtley, L.-G., Taylor, A., Dyson, M., & Weinman, J. (2004). Perceived stress and cortisol levels predict speed of wound healing in healthy male adults. *Psychoneuroendocrinology*, 29(6), 798-809. doi:10.1016/s0306-4530(03)00144-6
- Eek, F., Karlson, B., Garde, A. H., Hansen, A. M., & Orbaek, P. (2012). Cortisol, sleep, and recovery - Some gender differences but no straight associations. *Psychoneuroendocrinology*, 37(1), 56-64. doi:10.1016/j.psyneuen.2011.05.003
- Ekehammar, B., & Dornic, S. (1990). Weinstein's Noise Sensitivity Scale: Reliability and construct validity. *Perceptual and Motor Skills*, 70, 129-130.

- Elenkov, I. J. (2008). Effects of catecholamines on the immune response. In A. del Ray, G. P. Chrousos, & H. Besedovsky (Eds.), *The hypothalamus-pituitary-adrenal axis* (pp. 189-206): Elsevier, Inc.
- Ellermeier, W., Eigenstetter, M., & Zimmer, K. (2001). Psychoacoustic correlates of individual noise sensitivity. *Journal of the Acoustical Society of America*, 109(4), 1464. doi:10.1121/1.1350402
- Ellermeier, W., & Zimmer, K. (1997). Individual differences in susceptibility to the "irrelevant speech effect". *Journal of the Acoustical Society of America*, 102(4), 2191-2199. doi:10.1121/1.419596
- Ellis, B., Jackson, J., & Boyce, W. (2006). The stress response systems: Universality and adaptive individual differences. *Developmental Review*, 26(2), 175-212. doi:10.1016/j.dr.2006.02.004
- Enmarker, I., & Boman, E. (2004). Noise annoyance responses of middle school pupils and teachers. *Journal of Environmental Psychology*, 24(4), 527-536. doi:10.1016/j.jenvp.2004.09.005
- Eriksen, H. R., Ihlebaek, C., & Ursin, H. (1999). A scoring system for subjective health complaints (SHC). *Scandinavian Journal of Public Health*, 27, 63-72. doi:10.1177/14034948990270010401
- Eriksen, H. R., & Ursin, H. (2002). Sensitization and subjective health complaints. *Scandinavian Journal of Psychology*, 43(189-196).
- Eriksen, H. R., & Ursin, H. (2004). Subjective health complaints, sensitization, and sustained cognitive activation (stress). *Journal of Psychosomatic Research*, 56(4), 445-448. doi:10.1016/s0022-3999(03)00629-9
- Eriksson, C., Bluhm, G., Hilding, A., Ostenson, C. G., & Pershagen, G. (2010). Aircraft noise and incidence of hypertension--gender specific effects. *Environmental Research*, 110(8), 764-772. doi:10.1016/j.envres.2010.09.001
- Eriksson, C., Rosenlund, M., Pershagen, G., Hilding, A., Ostenson, C. G., & Bluhm, G. (2007). Aircraft noise and incidence of hypertension. *Epidemiology*, 18(6), 716-721. doi:10.1097/EDE.0b013e3181567e77
- Evans, G. W. (1984). *Environmental stress*. Cambridge, UK: Cambridge University Press.
- Evans, G. W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology*, 39(5), 924-933. doi:10.1037/0012-1649.39.5.924
- Evans, G. W., Barer, M. L., & Marmor, T. R. (1994). *Why are some people healthy and others not? The determinants of health of populations*. New York, NY: Walter De Gruyter.
- Evans, G. W., Bullinger, M., & Hygge, S. (1998). Chronic noise exposure and physiological response: A prospective study of children living under environmental stress. *Psychological Science*, 9(1), 75-77.
- Evans, G. W., & Carrere, S. (1991). Traffic congestion, perceived control, and psychophysiological stress among urban bus drivers. *Journal of Applied Psychology*, 76(5), 658-663.
- Evans, G. W., & Cohen, S. (1987). Environmental stress. In D. Stokols & I. Altman (Eds.), *Handbook of environmental psychology, Vol. I.* (pp. 571-610). New York, NY: Wiley.
- Evans, G. W., & Jacobs, S. V. (1981). Air pollution and human behavior. *Journal of Social Issues*, 37(1), 95-125. doi:10.1111/j.1540-4560.1981.tb01059.x

- Evans, G. W., Lercher, P., Meis, M., Ising, H., & Kofler, W. W. (2001). Community noise exposure and stress in children. *Journal of the Acoustical Society of America*, 109(3), 1023. doi:10.1121/1.1340642
- Evans, G. W., & Maxwell, L. (1997). Chronic noise exposure and reading deficits: The mediating effects of language acquisition. *Environment and Behavior*, 29(5), 638-656. doi:10.1177/0013916597295003
- Evans, G. W., & Stecker, R. (2004). Motivational consequences of environmental stress. *Journal of Environmental Psychology*, 24(2), 143-165. doi:10.1016/s0272-4944(03)00076-8
- Eysenck, H. J. (1967). *The biological basis of personality*. Springfield, IL: Thomas.
- Eysenck, H. J., & Eysenck, S. B. G. (1968). *Manual for the Eysenck Personality Inventory*. San Diego, CA: Educational and Industrial Testing Service.
- Farr, L. E. (1967). Medical consequences of environmental home noises. *Journal of the American Medical Association*, 202(3), 171-174.
- Fields, J. M. (1993). Effects of personal and situational variables on noise annoyance in residential areas. *Journal of the Acoustical Society of America*, 93(5), 2753-2763.
- Fink, G. (2009). *Stress science: Neuroendocrinology*. San Diego, CA: Elsevier, Inc.
- Flindell, I. H., & Stallen, P. J. (1999). Non-acoustical factors in environmental noise. *Noise & Health*, 1(3), 11-16.
- Floud, S., Vigna-Taglianti, F., Hansell, A., Blangiardo, M., Houthuijs, D., Breugelmans, O., ... Jarup, L. (2011). Medication use in relation to noise from aircraft and road traffic in six European countries: Results of the HYENA study. *Occupational and Environmental Medicine*, 68(7), 518-524. doi:10.1136/oem.2010.058586
- Folkman, S., Lazarus, R. S., Dunkel-Schetter, C., DeLongis, A., & Gruen, R. J. (1986). Dynamics of a stressful encounter: Cognitive appraisal, coping, and encounter outcomes. *Journal of Personality and Social Psychology*, 50(5), 992-1003.
- Fooladi, M. M. (2012). Involuntary and persistent environmental noise influences health and hearing in Beirut, Lebanon. *Journal of Environmental and Public Health*, 2012, 235618. doi:10.1155/2012/235618
- Franssen, E. A. M., van Wiechen, C. M. A. G., Nagelkerke, N. J. D., & Lebre, E. (2004). Aircraft noise around a large international airport and its impact on general health and medication use. *Occupational and Environmental Medicine*, 61(5), 405-413. doi:10.1136/oem.2002.005488
- Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology*, 72(1), 67-73. doi:10.1016/j.ijpsycho.2008.03.014
- Fung, M. M., Peters, K., Redline, S., Ziegler, M. G., Ancoli-Israel, S., Barrett-Connor, E., & Stone, K. L. (2011). Decreased slow wave sleep increases risk of developing hypertension in elderly men. *Hypertension*, 58(4), 596-603. doi:10.1161/HYPERTENSIONAHA.111.174409
- Fyhri, A., & Aasvang, G. M. (2010). Noise, sleep and poor health: Modeling the relationship between road traffic noise and cardiovascular problems. *Science of the Total Environment*, 408(21), 4935-4942. doi:10.1016/j.scitotenv.2010.06.057
- Fyhri, A., & Klæboe, R. (2006). Direct, indirect influences of income on road traffic noise annoyance. *Journal of Environmental Psychology*, 26(1), 27-37. doi:10.1016/j.jenvp.2006.04.001

- Fyhri, A., & Klæboe, R. (2009). Road traffic noise, sensitivity, annoyance and self-reported health--a structural equation model exercise. *Environment International*, 35(1), 91-97. doi:10.1016/j.envint.2008.08.006
- Galea, S., & Tracy, M. (2007). Participation rates in epidemiologic studies. *Annals of Epidemiology*, 17(9), 643-653. doi:10.1016/j.annepidem.2007.03.013
- Galesic, M., & Bosnjak, M. (2009). Effects of questionnaire length on participation and indicators of response quality in a web survey. *Public Opinion Quarterly*, 73(2), 349-360. doi:10.1093/poq/nfp031
- Gan, W. Q., Davies, H. W., Koehoorn, M., & Brauer, M. (2012). Association of long-term exposure to community noise and traffic-related air pollution with coronary heart disease mortality. *American Journal of Epidemiology*. doi:10.1093/aje/kwr424
- Garrido, P., de Blas, M., Del Arco, A., Segovia, G., & Mora, F. (2012). Aging increases basal but not stress-induced levels of corticosterone in the brain of the awake rat. *Neurobiology of Aging*, 33(2), 375-382. doi:10.1016/j.neurobiolaging.2010.02.015
- Geen, R. G., McCown, E. J., & Broyles, J. W. (1985). Effects of noise on sensitivity of introverts and extraverts to signals in a vigilance task. *Personality and Individual Differences*, 6(2), 237-241.
- Gidlöf-Gunnarsson, A., & Öhrström, E. (2007). Noise and well-being in urban residential environments: The potential role of perceived availability to nearby green areas. *Landscape and Urban Planning*, 83(2-3), 115-126. doi:10.1016/j.landurbplan.2007.03.003
- Gitanjali, B., & Ananth, R. (2003). Effect of acute exposure to loud occupational noise during daytime on the nocturnal sleep architecture, heart rate, and cortisol secretion in healthy volunteers. *Journal of Occupational Health*, 45, 146-152.
- Glaser, R., Kiecolt-Glaser, J. K., Malarkey, W. B., & Sheridan, J. F. (1998). The influence of psychological stress on the immune response to vaccines. *Annals of the New York Academy of Sciences*, 840, 649-655. doi:10.1111/j.1749-6632.1998.tb09603.x
- Glass, D. C., & Singer, J. E. (1972a). Behavioral aftereffects of unpredictable and uncontrollable aversive events. *American Scientist*, 6(4), 457-465.
- Glass, D. C., & Singer, J. E. (1972b). *Urban stress*. New York, NY: Academic Press.
- Glass, D. C., Singer, J. E., Leonard, H. S., Krantz, D., & Cohen, S. (1973). Perceived control of aversive stimulation and the reduction of stress responses. *Journal of Personality and Social Psychology*, 41(4), 577-595.
- Gobina, I., Valimaa, R., Tynjala, J., Villberg, J., Villerusa, A., Iannotti, R. J., ... Kuntsche, E. (2011). The medicine use and corresponding subjective health complaints among adolescents, a cross-national survey. *Pharmacoepidemiology and Drug Safety*, 20(4), 424-431. doi:10.1002/pds.2102
- Goines, L., & Hagler, L. (2007). Noise pollution: A modern plague. *Southern Medical Journal*, 100(3), 287-294.
- Goldberg, D. P. (1972). *The detection of psychiatric illness by questionnaire*. London, UK: Oxford University Press.
- Gouin, J. P., Hantsoo, L., & Kiecolt-Glaser, J. K. (2008). Immune dysregulation and chronic stress among older adults: A review. *Neuroimmunomodulation*, 15(4-6), 251-259. doi:10.1159/000156468
- Gow, R., Thomson, S., Rieder, M., Van Uum, S., & Koren, G. (2010). An assessment of cortisol analysis in hair and its clinical applications. *Forensic Science International*, 196(1-3), 32-37. doi:10.1016/j.forsciint.2009.12.040

- Goyal, S., Gupta, V., & Walia, L. (2010). Effect of noise stress on autonomic function tests. *Noise & Health*, 12(48), 182-186. doi:10.4103/1463-1741.64976
- Grady, C. L., Springer, M. V., Hongwanishkul, D., McIntosh, A. R., & Winocur, G. (2006). Age-related changes in brain activity across the adult lifespan. *Journal of Cognitive Neuroscience*, 18(2), 227-241.
- Graeven, D. (1974). The effects of airplane noise on health: The examination of 3 hypotheses. *Journal of Health and Social Behavior*, 15, 336-343.
- Greaves-Lord, K., Ferdinand, R. F., Oldehinkel, A. J., Sondeijker, F. E., Ormel, J., & Verhulst, F. C. (2007). Higher cortisol awakening response in young adolescents with persistent anxiety problems. *Acta Psychiatrica Scandinavica*, 116(2), 137-144. doi:10.1111/j.1600-0447.2007.01001.x
- Green, S. B. (1991). How many subjects does it take to do a regression analysis? *Multivariate Behavioral Research*, 26, 299-510. doi:10.1207/s15327906mbr2603_7
- Griefahn, B. (2002). Sleep disturbances related to environmental noise. *Noise & Health*, 4(15), 57-60.
- Griefahn, B., Brode, P., Marks, A., & Basner, M. (2008). Autonomic arousals related to traffic noise during sleep. *Sleep*, 31(4), 569-577.
- Griefahn, B., & Di Nisi, J. (1992). Mood and cardiovascular functions during noise, related to sensitivity, type of noise and sound pressure level. *Journal of Sound and Vibration*, 155(1), 111-123.
- Griefahn, B., Marks, A., & Robens, S. (2006). Noise emitted from road, rail and air traffic and their effects on sleep. *Journal of Sound and Vibration*, 295(1-2), 129-140. doi:10.1016/j.jsv.2005.12.052
- Griefahn, B., & Robens, S. (2010). Experimental studies on the effects of nocturnal noise on cortisol awakening response. *Noise & Health*, 12(47), 129-136. doi:10.4103/1463-1741.63215
- Griefahn, B., & Robens, S. (2011). Cortisol awakening response - Are sampling delays of 15 minutes acceptable? *International Journal of Psychophysiology*, 82(2), 202-205. doi:10.1016/j.ijpsycho.2011.08.005
- Griffiths, I. D., & Delauzan, F. R. (1977). Individual differences in sensitivity to traffic noise: An empirical study. *Journal of Sound and Vibration*, 55(1), 93-107. doi:10.1016/0022-460X(77)90585-5
- Griffiths, I. D., & Langdon, F. J. (1968). Subjective response to road traffic noise. *Journal of Sound and Vibration*, 8(1), 16-32.
- Griffiths, I. D., & Raw, H. J. (1986). Community and individual response to changes in traffic noise exposure. *Journal of Sound and Vibration*, 111(2), 209-217.
- Grzywacz, J. G., Almeida, D. M., Neupert, S. D., & Ettner, S. L. (2004). Socioeconomic status and health: A micro-level analysis of exposure and vulnerability to daily stressors. *Journal of Health and Social Behavior*, 45(1), 1-16. doi:10.1177/002214650404500101
- Gu, J. W., Halpin, C. F., Nam, E. C., Levine, R. A., & Melcher, J. R. (2010). Tinnitus, diminished sound-level tolerance, and elevated auditory activity in humans with clinically normal hearing sensitivity. *Journal of Neurophysiology*, 104(6), 3361-3370. doi:10.1152/jn.00226.2010
- Gurung, R. A. R. (2010). *Health psychology: A cultural approach*. Belmont, CA: Wadsworth.
- Guski, R. (1999). Personal and social variables as co-determinants of noise annoyance. *Noise & Health*, 3, 45-56.

- Haines, M. M., Stansfeld, S. A., Brentnall, S., Head, J., Berry, B., Jiggins, M., & Hygge, S. (2001). The West London Schools Study: The effects of chronic aircraft noise exposure on child health. *Psychological Medicine*, 31(8). doi:10.1017/s003329170100469x
- Haines, M. M., Stansfeld, S. A., Job, R. F. S., Berglund, B., & Head, J. (2001a). Chronic aircraft noise exposure, stress responses, mental health and cognitive performance in school children. *Psychological Medicine*, 31, 265-277.
- Haines, M. M., Stansfeld, S. A., Job, R. F. S., Berglund, B., & Head, J. (2001b). A follow-up study of effects of chronic aircraft noise exposure on child stress responses and cognition. *International Journal of Epidemiology*, 30, 839-845.
- Hall, F. L. (1984). Community response to noise: Is all noise the same? *Journal of the Acoustical Society of America*, 76(4), 1161-1168. doi:10.1121/1.391408
- Haralabidis, A. S., Dimakopoulou, K., Vigna-Taglianti, F., Giampaolo, M., Borgini, A., Dudley, M. L., ... Jarup, L. (2008). Acute effects of night-time noise exposure on blood pressure in populations living near airports. *European Heart Journal*, 29(5), 658-664. doi:10.1093/eurheartj/ehn013
- Hardoy, M. C., Carta, M. G., Marci, A. R., Carbone, F., Cadeddu, M., Kovess, V., ... Carpinello, B. (2005). Exposure to aircraft noise and risk of psychiatric disorders: The Elmas survey--aircraft noise and psychiatric disorders. *Social Psychiatry and Psychiatric Epidemiology*, 40(1), 24-26. doi:10.1007/s00127-005-0837-x
- Harris, A., Ursin, H., Murison, R., & Eriksen, H. R. (2007). Coffee, stress and cortisol in nursing staff. *Psychoneuroendocrinology*, 32(4), 322-330. doi:10.1016/j.psyneuen.2007.01.003
- Hatfield, J., Job, R. F. S., Carter, N. L., Peploe, P., Taylor, R., & Morrell, S. (2001). The influence of psychological factors on self-reported physiological effects of noise. *Noise & Health*, 3(10), 1-13.
- Hatfield, J., Job, R. F. S., Hede, A. H., Carter, N. L., Peploe, P., Taylor, R., & Morrell, S. (2002). Human response to environmental noise: The role of perceived control. *International Journal of Behavioral Medicine*, 9(4), 341-359.
- Hathaway, S. R., & McKinley, J. C. (1951). *MMPI manual*. New York, NY: Psychological Corporation.
- Hawkey, L. C., Lavelle, L. A., Berntson, G. G., & Cacioppo, J. T. (2011). Mediators of the relationship between socioeconomic status and allostatic load in the Chicago Health, Aging, and Social Relations Study (CHASRS). *Psychophysiology*, 48(8), 1134-1145. doi:10.1111/j.1469-8986.2011.01185.x
- Hayes, A. F. (2009). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication Monographs*, 76(4), 408-420. doi:10.1080/03637750903310360
- Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling [Manuscript submitted for publication]. Retrieved from <http://www.afhayes.com/public/process2012.pdf>.
- Hayes, A. F., & Preacher, K. J. (2010). Quantifying and testing indirect effects in simple mediation models when the constituent paths are nonlinear. *Multivariate Behavioral Research*, 45(4), 627-660. doi:10.1080/00273171.2010.498290
- Heaney, J. L., Phillips, A. C., & Carroll, D. (2010). Ageing, depression, anxiety, social support and the diurnal rhythm and awakening response of salivary cortisol. *International Journal of Psychophysiology*, 78(3), 201-208. doi:10.1016/j.ijpsycho.2010.07.009

- Hebert, S., & Lupien, S. J. (2009). Salivary cortisol levels, subjective stress, and tinnitus intensity in tinnitus sufferers during noise exposure in the laboratory. *International Journal of Hygiene and Environmental Health*, 212(1), 37-44. doi:10.1016/j.ijheh.2007.11.005
- Heim, C., & Binder, E. B. (2012). Current research trends in early life stress and depression: Review of human studies on sensitive periods, gene–environment interactions, and epigenetics. *Experimental Neurology*, 233, 102-111. doi:10.1016/j.expneurol.2011.10.032
- Heinonen-Guzejev, M. (2009). *Noise sensitivity - medical, psychological and genetic aspects*. University of Helsinki, Helsinki.
- Heinonen-Guzejev, M., Jauhiainen, T., Vuorinen, H., Viljanen, A., Rantanen, T., Koskenvuo, M., ... Kaprio, J. (2011). Noise sensitivity and hearing disability. *Noise & Health*, 13(50), 51-58.
- Heinonen-Guzejev, M., Vuorinen, H. S., Kaprio, J., Heikkilä, K., Mussalo-Rauhamaa, H., & Koskenvuo, M. (2000). Self-report of transportation noise exposure, annoyance and noise sensitivity in relation to noise map information. *Journal of Sound and Vibration*, 234(2), 191-206. doi:10.1006/jsvi.1999.2885
- Heinonen-Guzejev, M., Vuorinen, H. S., Mussalo-Rauhamaa, H., Heikkilä, K., Koskenvuo, M., & Kaprio, J. (2004). Somatic and psychological characteristics of noise-sensitive adults in Finland. *Archives of Environmental Health*, 59(8), 410-417. doi:10.3200/AEOH.59.8.410-417
- Heinonen-Guzejev, M., Vuorinen, H. S., Mussalo-Rauhamaa, H., Heikkilä, K., Koskenvuo, M., & Kaprio, J. (2007). The association of noise sensitivity with coronary heart and cardiovascular mortality among Finnish adults. *Science of the Total Environment*, 372(2-3), 406-412. doi:10.1016/j.scitotenv.2006.08.048
- Heinonen-Guzejev, M., Vuorinen, H. S., Mussalo-Rauhamaa, H., Heikkilä, K., Koskenvuo, M., & Kaprio, J. (2005). Genetic component of noise sensitivity. *Twin Research and Human Genetics*, 8(3), 245-249. doi:10.1375/twin.8.3.245
- Hellhammer, D. H., Wüst, S., & Kudielka, B. M. (2009). Salivary cortisol as a biomarker in stress research. *Psychoneuroendocrinology*, 34(2), 163-171. doi:10.1016/j.psyneuen.2008.10.026
- Hellhammer, J., Fries, E., Schweisthal, O. W., Schlotz, W., Stone, A. A., & Hagemann, D. (2007). Several daily measurements are necessary to reliably assess the cortisol rise after awakening: State- and trait components. *Psychoneuroendocrinology*, 32(1), 80-86. doi:10.1016/j.psyneuen.2006.10.005
- Hellhammer, J., Schlotz, W., Stone, A. A., Pirke, K. M., & Hellhammer, D. H. (2004). Allostatic load, perceived stress, and health – A prospective study in two age groups. *Annals of the New York Academy of Sciences*, 1032, 8-13. doi:10.1196/annals.1314.002
- Henry, J. D., & Crawford, J. R. (2005). The short-form version of the Depression Anxiety Stress Scales (DASS-21): Construct validity and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, 44, 227-239.
- Hill, E. M., Shepherd, D., Welch, D., Dirks, K. N., & McBride, D. (2012). Perceptions of neighborhood problems and health-related quality of life. *Journal of Community Psychology*, 40(7), 814-827. doi:10.1002/jcop.21490
- Huber, T. J., Issa, K., Schik, G., & Wolf, O. T. (2006). The cortisol awakening response is blunted in psychotherapy inpatients suffering from depression. *Psychoneuroendocrinology*, 31(7), 900-904. doi:10.1016/j.psyneuen.2006.03.005

- Hume, K. (2010). Sleep disturbance due to noise: Current issues and future research. *Noise & Health*, 12(47), 70-76. doi:10.4103/1463-1741.63206
- Hygge, S., Evans, G. W., & Bullinger, M. (2002). A prospective study of some effects of aircraft noise on cognitive performance in schoolchildren. *Psychological Science*, 13(5), 469-474.
- Ihlebaek, C., Eriksen, H. R., & Ursin, H. (2002). Prevalence of subjective health complaints (SHC) in Norway. *Scandinavian Journal of Public Health*, 30(1), 20-29. doi:10.1177/14034948020300010701
- Indregard, A. M., Ihlebaek, C. M., & Eriksen, H. R. (2012). Modern health worries, subjective health complaints, health care utilization, and sick leave in the Norwegian working population. *International Journal of Behavioral Medicine*. doi:10.1007/s12529-012-9246-1
- Irish, L., Kobayashi, I., & Delahanty, D. L. (2010). Long-term physical health consequences of childhood sexual abuse: A meta-analytic review. *Journal of Pediatric Psychology*, 35(5), 450-461. doi:10.1093/jpepsy/jsp118
- Irwin, M. R., Daniels, M., Risch, S. C., Bloom, E., & Weiner, H. (1988). Plasma cortisol and natural killer cell activity during bereavement. *Biological Psychiatry*, 24, 173-178.
- Ising, H., Babisch, W., & Kruppa, B. (1999). Noise-induced endocrine effects and cardiovascular risk. *Noise & Health*, 1(4), 37-48.
- Ising, H., Babisch, W., Kruppa, B., Lindthammer, A., & Wiens, D. (1997). Subjective work noise: A major risk factor for myocardial infarction. *Sozial- und Preventivmedizin*, 42, 216-222.
- Ising, H., & Braun, C. (2000). Acute and chronic endocrine effects of noise: Review of the research conducted at the Institute for water, soil and air hygiene. *Noise & Health*, 2(7), 7-24.
- Ising, H., Dienel, D., Gunther, T., & Markert, B. (1980). Health effects of traffic noise. *International Archives of Occupational and Environmental Health*, 47, 179-190.
- Ising, H., & Ising, M. (2002). Chronic cortisol increases in the first half of the night caused by road traffic noise. *Noise & Health*, 4(16), 13-21.
- Ising, H., & Kruppa, B. (2004). Health effects caused by noise: Evidence in the literature from the past 25 years. *Noise & Health*, 6(22), 5-13.
- Issarayangyun, T., Black, D., Black, J., & Samuels, S. (2005). Aircraft noise and methods for the study of community health and well-being. *Journal of the Eastern Asia Society for Transportation Studies*, 6, 3293-3308.
- Iwata, O. (1981). Relationships of noise sensitivity to sex and sound-associated responses. *Journal of Gakugei, Tokushima University (Educational Science)*, 30, 41-45.
- Iwata, O. (1984). The relationship of noise sensitivity to health and personality. *Japanese Psychological Research*, 26(2), 75-81.
- Jacko, J. A., & Sears, A. (2003). *The human-computer interaction handbook: Fundamentals, evolving technologies, and emerging applications*. New York, NY: Routledge.
- Jadad, A. R., & O'Grady, L. (2008). How should health be defined? *British Medical Journal*, 337, a2900. doi:10.1136/bmj.a2900
- Jakovljević, B., Paunovic, K., & Belojević, G. (2009). Road-traffic noise and factors influencing noise annoyance in an urban population. *Environment International*, 35(3), 552-556. doi:10.1016/j.envint.2008.10.001

- Jarup, L., Babisch, W., Houthuijs, D., Pershagen, G., Katsouyanni, K., Cadum, E., ... Vigna-Taglianti, F. (2008). Hypertension and exposure to noise near airports: The HYENA study. *Environmental Health Perspectives*, 116(3), 329-333. doi:10.1289/ehp.10775
- Jastreboff, P. J., & Jastreboff, M. M. (2010). Decreased sound tolerance. In J. B. Snow (Ed.), *Tinnitus: Theory and management*. USA: People's Medical Publishing House.
- Jenkins, D. C., Stanton, B. A., Niemcryk, S. J., & Rose, R. M. (1988). A scale for the estimation of sleep problems in clinical research. *Journal of Clinical Epidemiology*, 41(4), 313-321. doi:10.1016/0895-4356(88)90138-2
- Jenkins, L. M., Tarnopolsky, A., Hand, D. J., & Barker, S. M. (1979). Comparison of three studies of aircraft noise and psychiatric hospital admissions conducted in the same area. *Psychological Medicine*, 9, 681-693. doi:10.1017/S0033291700033997
- Job, R. F. S. (1988). Community response to noise: A review of factors influencing the relationship between noise exposure and reaction. *Journal of the Acoustical Society of America*, 83(3), 991-1001.
- Job, R. F. S. (1996). The influence of subjective reactions to noise on health effects of the noise. *Environment International*, 22(1), 93-104. doi:10.1016/0160-4120(95)00107-7
- Job, R. F. S. (1999). Noise sensitivity as a factor influencing human reaction to noise. *Noise & Health*, 1, 57-68.
- John, O. P., Donahue, E. M., & Kentle, R. L. (1991). *The Big Five Inventory—Versions 4a and 54*. Berkeley, CA: University of California, Berkeley, Institute of Personality and Social Research.
- Johnston-Brooks, C. H., Lewis, M. A., Evans, G. W., & Whalen, C. K. (1998). Chronic stress and illness in children: The role of allostatic load. *Psychological Medicine*, 60, 597-603.
- Johnston, D. W., Propper, C., & Shields, M. A. (2009). Comparing subjective and objective measures of health: Evidence from hypertension for the income/health gradient. *Journal of Health Economics*, 28(3), 540-552. doi:10.1016/j.jhealeco.2009.02.010
- Jonsson, A., & Hansson, L. (1977). Prolonged exposure to a stressful stimulus (noise) as a cause of raised blood-pressure in man. *The Lancet*, 309(8002), 86-87. doi:10.1016/S0140-6736(77)91093-5
- Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, 35(1), 2-16. doi:10.1016/j.neubiorev.2009.10.002
- Kageyama, T., Kabuto, M., Nitta, H., Kurokawa, Y., Taira, K., Suzuki, S., & Takemoto, T. (1997). A population study on risk factors for insomnia among adult Japanese women: A possible effect of road traffic volume. *Sleep*, 20, 962-971.
- Kalat, J. W. (2007). *Biological psychology*. Belmont, CA: Thompson Learning.
- Karlen, J., Ludvigsson, J., Frostell, A., Theodorsson, E., & Faresjö, T. (2011). Cortisol in hair measured in young adults - a biomarker of major life stressors? *BMC Clinical Pathology*, 11(1), 12. doi:10.1186/1472-6890-11-12
- Karlson, B., Eek, F., Hansen, Å. M., Garde, A. H., & Ørbaek, P. (2011). Cortisol variability and self-reports in the measurement of work-related stress. *Stress and Health*, 27(2), e11-e24. doi:10.1002/smi.1330
- Kawada, T. (2011). Noise and health - Sleep disturbance in adults. *Journal of Occupational Health*, 53, 413-416.

- Kelly, S. A. (1986). *Effects of aircraft noise and noise sensitivity on the physical and mental well-being of older adults*. University of California, Irvine.
- Kemeny, M. E. (2003). The psychobiology of stress. *Current Directions in Psychological Science*, 12(4), 124-129. doi:10.1111/1467-8721.01246
- Kiecolt-Glaser, J. K., Loving, T. J., Stowell, J. R., Malarkey, W. B., Lemeshow, S., Dickinson, S. L., & Glaser, R. (2005). Hostile marital interactions, proinflammatory cytokine production, and wound healing. *Archives of General Psychiatry*, 62, 1377-1384.
- Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Psychoneuroimmunology: Psychological influences on immune function and health. *Journal of Consulting and Clinical Psychology*, 70(3), 537-547. doi:10.1037/0022-006X.70.3.53
- Kiecolt-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W. B., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences*, 100(15), 9090-9095. doi:10.1073/pnas.1531903100
- Killgore, W. D. (2010). Effects of sleep deprivation on cognition. *Progress in Brain Research*, 185, 105-129.
- King, R. P., & Davis, J. R. (2003). Community noise: Health effects and management. *International Journal of Hygiene and Environmental Health*, 206(2), 123-131. doi:10.1078/1438-4639-00202
- Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology*, 19(4), 313-333.
- Kirschbaum, C., & Hellhammer, D. H. (1999). Noise and stress - salivary cortisol as a non-invasive measure of allostatic load. *Noise & Health*, 4, 57-65.
- Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, 61, 154-162.
- Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28, 76-81. doi:10.1159/000119004
- Kishikawa, H., Matsui, T., Uchiyama, I., Miyakawa, M., Hiramatsu, K., & Stansfeld, S. A. (2006). The development of Weinstein's noise sensitivity scale. *Noise & Health*, 8(33), 154-160.
- Kishikawa, H., Matsui, T., Uchiyama, I., Miyakawa, M., Hiramatsu, K., & Stansfeld, S. A. (2009). Noise sensitivity and subjective health: Questionnaire study conducted along trunk roads in Kusatsu, Japan. *Noise & Health*, 11, 111-117.
- Kjellberg, A., Landstrom, U., Tesarz, M., Soderberg, L., & Akerlund, E. (1996). The effects of nonphysical noise characteristics, ongoing task and noise sensitivity on annoyance and distraction due to noise at work. *Journal of Environmental Psychology*, 16, 123-136.
- Klæboe, R. (2007). Are adverse impacts of neighbourhood noisy areas the flip side of quiet area benefits? *Applied Acoustics*, 68(5), 557-575. doi:10.1016/j.apacoust.2005.05.007

- Klæboe, R., Amundsen, A. H., Fyhri, A., & Solberg, S. (2004). Road traffic noise – The relationship between noise exposure and noise annoyance in Norway. *Applied Acoustics*, 65(9), 893-912. doi:10.1016/j.apacoust.2004.04.001
- Klok, M. D., Vreeburg, S. A., Penninx, B. W., Zitman, F. G., de Kloet, E. R., & DeRijk, R. H. (2011). Common functional mineralocorticoid receptor polymorphisms modulate the cortisol awakening response: Interaction with SSRIs. *Psychoneuroendocrinology*, 36(4), 484-494. doi:10.1016/j.psyneuen.2010.07.024
- Kolassa, I. T., Eckart, C., Ruf, M., Neuner, F., de Quervain, D. J., & Elbert, T. (2007). Lack of cortisol response in patients with posttraumatic stress disorder (PTSD) undergoing a diagnostic interview. *BMC Psychiatry*, 7, 54. doi:10.1186/1471-244X-7-54
- Koopmans, G. T., & Lamers, L. M. (2007). Gender and health care utilization: The role of mental distress and help-seeking propensity. *Social Science & Medicine*, 64(6), 1216-1230. doi:10.1016/j.socscimed.2006.11.018
- Korte, S. M., Koolhaas, J. M., Wingfield, J. C., & McEwen, B. S. (2005). The Darwinian concept of stress: Benefits of allostasis and costs of allostatic load and the trade-offs in health and disease. *Neuroscience and Biobehavioral Reviews*, 29, 3-28. doi:10.1016/j.neubiorev.2004.08.009
- Kroesen, M., Molin, E. J. E., & Van Wee, B. (2010). Determining the direction of causality between psychological factors and aircraft noise annoyance. *Noise & Health*, 12(46), 17-25.
- Kroesen, M., Molin, E. J. E., & van Wee, B. (2011). Policy, personal dispositions and the evaluation of aircraft noise. *Journal of Environmental Psychology*, 31(2), 147-157. doi:10.1016/j.jenvp.2010.09.004
- Krog, N. H., Engdahl, B., & Tambs, K. (2010). The association between tinnitus and mental health in a general population sample: Results from the HUNT Study. *Journal of Psychosomatic Research*, 69(3), 289-298. doi:10.1016/j.jpsychores.2010.03.008
- Kudielka, B. M. (2003). Compliance with saliva sampling protocols: Electronic monitoring reveals invalid cortisol daytime profiles in noncompliant subjects. *Psychosomatic Medicine*, 65(2), 313-319. doi:10.1097/01.psy.0000058374.50240.bf
- Kudielka, B. M., & Kirschbaum, C. (2003). Awakening cortisol response are influenced by health status and awakening time but not by menstrual cycle phase. *Psychoneuroendocrinology*, 28, 35-47. doi:10.1016/S0306-4530(02)00008-2
- Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: A review. *Biological psychology*, 69(1), 113-132. doi:10.1016/j.biopsycho.2004.11.009
- Kudielka, B. M., & Wüst, S. (2010). Human models in acute and chronic stress: Assessing determinants of individual hypothalamus–pituitary–adrenal axis activity and reactivity. *Stress*, 13(1), 1-14. doi:10.3109/10253890902874913
- Kuehner, C., Holzhauser, S., & Huffziger, S. (2007). Decreased cortisol response to awakening is associated with cognitive vulnerability to depression in a nonclinical sample of young adults. *Psychoneuroendocrinology*, 32(2), 199-209. doi:10.1016/j.psyneuen.2006.12.007
- Kumsta, R., Entringer, S., Koper, J. W., van Rossum, E. F., Hellhammer, D. H., & Wüst, S. (2007). Sex specific associations between common glucocorticoid receptor gene variants and hypothalamus-pituitary-adrenal axis responses to psychosocial stress. *Biological Psychiatry*, 62(8), 863-869. doi:10.1016/j.biopsycho.2007.04.013

- Kunz-Ebrecht, S. R., Kirschbaum, C., Marmot, M., & Steptoe, A. (2004). Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology*, 29(4), 516-528. doi:10.1016/s0306-4530(03)00072-6
- Kyrou, I., & Tsigos, C. (2009). Stress hormones: Physiological stress and regulation of metabolism. *Current Opinion in Pharmacology*, 9(6), 787-793. doi:10.1016/j.coph.2009.08.007
- Lahey, B. B. (2009). Public health significance of neuroticism. *The American Psychologist*, 64(4), 241-256. doi:10.1037/a0015309
- Lai, J. C., & Wan, K. K. (2009). *Trait anxiety accentuates the cortisol awakening response*. Paper presented at the meeting of the Bioinformatics and Biomedical Engineering, Beijing, China. doi:10.1109/ICBBE.2009.5163695
- Lam, K.-C., Chan, P.-K., Chan, T.-C., Au, W.-H., & Hui, W.-C. (2009). Annoyance response to mixed transportation noise in Hong Kong. *Applied Acoustics*, 70(1), 1-10. doi:10.1016/j.apacoust.2008.02.005
- Langdon, F. J. (1976). Noise nuisance caused by road traffic in residential areas: Part I. *Journal of Sound and Vibration*, 47(2), 243-263.
- Langdon, F. J., & Buller, I. B. (1977). Road traffic noise and disturbance to sleep. *Journal of Sound and Vibration*, 50(1), 13-28. doi:10.1016/0022-460X(77)90548-X
- Lauc, G., Zvonar, K., Vuksic-Mihaljevic, Z., & Flögel, M. (2004). Post-awakening changes in salivary cortisol in veterans with and without PTSD. *Stress and Health*, 20(2), 99-102. doi:10.1002/smi.1001
- Lawrence, M., Gonzalez, G., & Hawkins, J. E. (1967). Some physiological factors in noise-induced hearing loss. *American Industrial Hygiene Association Journal*, 28(5), 425-430. doi:10.1080/00028896709342661
- Lazarus, R. S., DeLongis, A., Folkman, S., & Gruen, R. (1985). Stress and adaptational outcomes: The problem of confounded measures. *American Psychologist*, 40(7), 770-785.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York, NY: Springer.
- Lercher, P., Botteldooren, D., Widmann, U., Uhrner, U., & Kammeringer, E. (2011). Cardiovascular effects of environmental noise: Research in Austria. *Noise & Health*, 13(52), 234-250.
- Lercher, P., Brink, M., Rudisser, J., Van Renterghem, T., Botteldooren, D., Baulac, M., & Defrance, J. (2010). The effects of railway noise on sleep medication intake: Results from the ALPNAP-study. *Noise & Health*, 12(47), 110-119.
- Lercher, P., Evans, G. W., Meis, M., & Kofler, W. W. (2002). Ambient neighbourhood noise and children's mental health. *Occupational and Environmental Medicine*, 59, 380-386.
- Lercher, P., & Kofler, W. W. (1996). Behavioral and health responses associated with road traffic noise exposure along alpine through-traffic routes. *Science of the Total Environment*, 189/190, 85-89.
- Leventhall, G. (2009). Low frequency noise. What we know, what we do not know, and what we would like to know. *Journal of Low Frequency Noise, Vibration and Active Control*, 28(2), 79-104.
- Leventhall, H. G. (2004). Low frequency noise and annoyance. *Noise & Health*, 6, 59-72.

- Levin, K., Inchley, J., Currie, D., & Currie, C. (2012). Subjective health and mental well-being of adolescents and the health promoting school: A cross-sectional multilevel analysis. *Health Education, 112*(2), 170-184. doi:10.1108/09654281211203439
- Li, H. N., Chau, C. K., & Tang, S. K. (2010). Can surrounding greenery reduce noise annoyance at home? *Science of the Total Environment, 408*(20), 4376-4384. doi:10.1016/j.scitotenv.2010.06.025
- Liss, M., Mailloux, J., & Erchull, M. J. (2008). The relationships between sensory processing sensitivity, alexithymia, autism, depression, and anxiety. *Personality and Individual Differences, 45*(3), 255-259. doi:10.1016/j.paid.2008.04.009
- Liss, M., Timmel, L., Baxley, K., & Killingsworth, P. (2005). Sensory processing sensitivity and its relation to parental bonding, anxiety, and depression. *Personality and Individual Differences, 39*(8), 1429-1439. doi:10.1016/j.paid.2005.05.007
- Little, R. J. A., & Rubin, D. B. (1987). *Statistical analysis with missing data*. New York, NY: Wiley.
- Ljungberg, J. K., & Neely, G. (2007). Stress, subjective experience and cognitive performance during exposure to noise and vibration. *Journal of Environmental Psychology, 27*(1), 44-54. doi:10.1016/j.jenvp.2006.12.003
- Lockley, S. W. (2010). Principles of sleep-wake regulation. In F. P. Cappuccio, M. A. Miller, & S. W. Lockley (Eds.), *Sleep, health, and society: From aetiology to public health*. New York, NY: Oxford University Press.
- Lovallo, W. R. (2005). *Stress and health: Biological and psychology interactions*. Thousand Oaks, CA: Sage.
- Lovallo, W. R. (2011). Do low levels of stress reactivity signal poor states of health? *Biological Psychology, 86*(2), 121-128. doi:10.1016/j.biopsycho.2010.01.006
- Lovell, B., Moss, M., & Wetherell, M. A. (2011). Perceived stress, common health complaints and diurnal patterns of cortisol secretion in young, otherwise healthy individuals. *Hormones and Behavior, 60*(3), 301-305. doi:10.1016/j.yhbeh.2011.06.007
- Lundberg, U. (1999). Coping with stress: Neuroendocrine reactions and implications for health. *Noise & Health, 1*(4), 67-74.
- Lupien, S. J., de Leon, M., de Santi, S., Convit, A., Tarshish, C., Nair, N. P. V., ... Meaney, M. J. (1998). Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nature Neuroscience, 1*(1), 69-73. doi:10.1038/271
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews. Neuroscience, 10*(6), 434-445. doi:10.1038/nrn2639
- Luppi, P. H., Clement, O., Sapin, E., Peyron, C., Gervasoni, D., Leger, L., & Fort, P. (2012). Brainstem mechanisms of paradoxical (REM) sleep generation. *Pflügers Archiv: European Journal of Physiology, 463*(1), 43-52. doi:10.1007/s00424-011-1054-y
- Maas, J., van Dillen, S. M., Verheij, R. A., & Groenewegen, P. P. (2009). Social contacts as a possible mechanism behind the relation between green space and health. *Health & Place, 15*(2), 586-595. doi:10.1016/j.healthplace.2008.09.006
- MacKinnon, D. P., & Luecken, L. J. (2008). How and for whom? Mediation and moderation in health psychology. *Health Psychology, 27*(2 Suppl), S99. doi:10.1037/0278-6133.27.2(Suppl.).S99

- Maina, G., Bovenzi, M., Palmas, A., Rossi, F., & Filon, F. L. (2011). Psychosocial environment and health: Methodological variability of the salivary cortisol measurements. *Toxicology Letters*. doi:10.1016/j.toxlet.2011.08.019
- Mair, C. A., Cutchin, M. P., & Kristen Peek, M. (2011). Allostatic load in an environmental riskscape: The role of stressors and gender. *Health & Place*, 17(4), 978-987. doi:10.1016/j.healthplace.2011.03.009
- Malmusi, D., Artazcoz, L., Benach, J., & Borrell, C. (2011). Perception or real illness? How chronic conditions contribute to gender inequalities in self-rated health. *European Journal of Public Health*. doi:10.1093/eurpub/ckr184
- Malouff, J. M., Thorsteinsson, E. B., & Schütte, N. S. (2005). The relationship between the five-factor model of personality and symptoms of clinical disorders: A meta-analysis. *Journal of Psychopathology and Behavioral Assessment*, 27(2), 101-114. doi:10.1007/s10862-005-5384-y
- Mangold, D., Mintz, J., Javors, M., & Marino, E. (2012). Neuroticism, acculturation and the cortisol awakening response in Mexican American adults. *Hormones and Behavior*, 61(1), 23-30. doi:10.1016/j.yhbeh.2011.09.009
- Manuck, S. B., Cohen, S., Rabin, B. S., Muldoon, M. F., & Bachen, E. A. (2001). Individual differences in cellular immune response to stress. *Psychological Science*, 2(2), 111-115.
- Maris, E. (2008). *The social side of noise annoyance*. University Leiden, Meiden, The Netherlands.
- Maris, E., Stallen, P. J., Vermunt, R., & Steensma, H. (2007). Evaluating noise in social context: The effect of procedural unfairness on noise annoyance judgments. *Journal of the Acoustical Society of America*, 122(6), 3483-3494. doi:10.1121/1.2799901
- Marks, A., & Griefahn, B. (2007). Associations between noise sensitivity and sleep, subjectively evaluated sleep quality, annoyance, and performance after exposure to nocturnal traffic noise. *Noise & Health*, 9(34), 1-7.
- Marshall, N. S., & Stranges, S. (2010). Sleep duration: risk factor or risk marker for ill-health? In F. P. Cappuccio, M. A. Miller, & S. W. Lockley (Eds.), *Sleep, health, and society: From aetiology to public health*. New York, NY: Oxford University Press.
- Marucha, P. T., Kiecolt-Glaser, J. K., & Favagehi, M. (1998). Mucosal wound healing is impaired by examination stress. *Psychosomatic Medicine*, 60, 362-365.
- Maschke, C., Rupp, T., & Hecht, K. (2000). The influence of stressors on biochemical reactions - a review of present scientific findings with noise. *International Journal of Hygiene and Environmental Health*, 203(1), 45-53. doi:10.1078/s1438-4639(04)70007-3
- Matheson, M. P., Stansfeld, S. A., & Haines, M. M. (2003). The effects of chronic aircraft noise exposure on children's cognition and health: 3 field studies. *Noise & Health*, 5, 31-40.
- Matsui, T., Stansfeld, S. A., Haines, M., & Head, J. (2004). Children's cognition and aircraft noise exposure at home-the West London Schools Study. *Noise & Health*, 7, 49-57.
- Matsumura, Y., & Rylander, R. (1991). Noise sensitivity and road traffic annoyance in a population sample. *Journal of Sound and Vibration*, 151(3), 415-419.
- Matud, M. P. (2004). Gender differences in stress and coping styles. *Personality and Individual Differences*, 37(7), 1401-1415. doi:10.1016/j.paid.2004.01.010

- Mayer, E. (2000). The neurobiology of stress and gastrointestinal disease. *Gut*, 47(6), 861-869. doi:10.1136/gut.47.6.861
- McCrae, R. R. (1990). Controlling neuroticism in the measurement of stress. *Stress Medicine*, 6, 237-241.
- McCrae, R. R., & Costa, P. T. (1987). Validation of the five-factor model of personality across instruments and observers. *Journal of Personality and Social Psychology*, 49, 81-90.
- McDonough, P., & Walters, V. (2001). Gender and health: Reassessing patterns and explorations. *Social Science & Medicine*, 52, 547-559.
- McEwen, B. S. (1998a). Protective and damaging effects of stress mediators. *The New England Journal of Medicine*, 338, 171-179. doi:10.1056/nejm199801153380307
- McEwen, B. S. (1998b). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 840, 33-44. doi:10.1111/j.1749-6632.1998.tb09546.x
- McEwen, B. S. (2001). Plasticity of the hippocampus: Adaptation to chronic stress and allostatic load. *Annals of the New York Academy of Sciences*, 993, 265-277. doi:10.1111/j.1749-6632.2001.tb05830.x
- McEwen, B. S. (2006). Sleep deprivation as a neurobiologic and physiologic stressor: Allostasis and allostatic load. *Metabolism: Clinical and Experimental*, 55(Suppl 2), S20-23. doi:10.1016/j.metabol.2006.07.008
- McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87(3), 873-904. doi:10.1152/physrev.00041.2006.-The
- McEwen, B. S. (2008). Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology*, 583, 174-185. doi:10.1016/j.ejphar.2007.11.071
- McEwen, B. S., & Gianaros, P. J. (2011). Stress- and allostasis-induced brain plasticity. *Annual Review of Medicine*, 62, 431-445. doi:10.1146/annurev-med-052209-100430
- McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 896, 30-47. doi:10.1111/j.1749-6632.1999.tb08103.x
- McEwen, B. S., & Stellar, E. (1993). Stress and the individual. *Archives of Internal Medicine*, 153, 2093-2101.
- McFadden, D. (1998). Sex differences in the auditory system. *Developmental Neuropsychology*, 14(2-3), 261-298.
- McKennell, A. C. (1963). *Aircraft noise annoyance around London (Heathrow) airport*. London, UK.
- Meecham, W. C., & Smith, H. G. (1977). Effects of jet aircraft noise and mental hospital admissions. *British Journal of Audiology*, 11, 81-85.
- Meerlo, P., Sgoifo, A., & Suchecki, D. (2008). Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Medicine Reviews*, 12(3), 197-210. doi:10.1016/j.smrv.2007.07.007
- Meewisse, M. L., Reitsma, J. B., de Vries, G. J., Gersons, B. P., & Olf, M. (2007). Cortisol and post-traumatic stress disorder in adults: Systematic review and meta-analysis. *The British Journal of Psychiatry*, 191, 387-392. doi:10.1192/bjp.bp.106.024877

- Meijer, H., Knipschild, P., & Salle, H. (1985). Road traffic noise annoyance in Amsterdam. *International Archives of Occupational and Environmental Health*, 56, 285-297.
- Meister, E. A., & Donatelle, R. J. (2000). The impact of commercial-aircraft noise on human health: A neighborhood study in metropolitan Minnesota. *Environmental Health*, 63(4), 9-15.
- Melamed, S., & Bruhis, S. (1996). The effects of chronic industrial noise exposure on urinary cortisol, fatigue, and irritability. *Journal of Occupational and Environmental Medicine*, 38(3), 252-256. doi:10.1097/00043764-199603000-00009
- Merswolken, M., Deter, H. C., Siebenhuener, S., Orth-Gomer, K., & Weber, C. S. (2012). Anxiety as predictor of the cortisol awakening response in patients with coronary heart disease. *International Journal of Behavioral Medicine*, Epub ahead of print.
- Michaud, D. S., Keith, S. E., & McMurchy, D. (2005). Noise annoyance in Canada. *Noise & Health*, 7, 39-47.
- Michaud, D. S., Keith, S. E., & McMurchy, D. (2008). Annoyance and disturbance of daily activities from road traffic noise in Canada. *Journal of the Acoustical Society of America*, 123(2), 784-792. doi:10.1121/1.2821984
- Miedema, H. M. E. (2007). Annoyance caused by environmental noise: Elements for evidence-based noise policies. *Journal of Social Issues*, 63(1), 41-57.
- Miedema, H. M. E., & Vos, H. (1999). Demographic and attitudinal factors that modify annoyance from transportation noise. *Journal of the Acoustical Society of America*, 105(6), 3336-3344.
- Miedema, H. M. E., & Vos, H. (2003). Noise sensitivity and reactions to noise and other environmental conditions. *Journal of the Acoustical Society of America*, 113(3), 1492-1504. doi:10.1121/1.1547437
- Miedema, H. M. E., & Vos, H. (2007). Associations between self-reported sleep disturbance and environmental noise based on reanalyses of pooled data from 24 studies. *Behavioral Sleep Medicine*, 5(1), 1-20.
- Mignot, E. (2008). Why we sleep: The temporal organization of recovery. *PLoS Biology*, 6(4), 661-669. doi:10.1371/journal.pbio.0060106
- Mikolajczak, M., Quoidbach, J., Vanootighem, V., Lambert, F., Lahaye, M., Fillée, C., & de Timary, P. (2010). Cortisol awakening response (CAR)'s flexibility leads to larger and more consistent associations with psychological factors than CAR magnitude. *Psychoneuroendocrinology*, 35(5), 752-757. doi:10.1016/j.psyneuen.2009.11.003
- Miles, R., Coutts, C., & Mohamadi, A. (2012). Neighborhood urban form, social environment, and depression. *Journal of Urban Health*, 89(1), 1-18. doi:10.1007/s11524-011-9621-2
- Miller, D. J., Vachon, D. D., & Lynam, D. R. (2009). Neuroticism, negative affect, and negative affect instability: Establishing convergent and discriminant validity using ecological momentary assessment. *Personality and Individual Differences*, 47(8), 873-877. doi:10.1016/j.paid.2009.07.007
- Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*, 60, 501-524. doi:10.1146/annurev.psych.60.110707.163551
- Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, 133(1), 25-45. doi:10.1037/0033-2909.133.1.25

- Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulations of pro-inflammatory cytokines: A glucocorticoid-resistance model. *Health Psychology, 21*, 531-541.
- Ministry of Health. (2010). *Mortality and demographic data 2007*. Wellington, New Zealand.
- Ministry of Social Development. (2010). *The Social Report 2010: Health*. Wellington, New Zealand.
- Mirowsky, J., & Ross, C. E. (1995). Sex differences in distress: Real or artifact? *American Sociological Review, 60*(3), 449-468.
- Moreira, N. M., & Bryan, M. E. (1972). Noise annoyance susceptibility. *Journal of Sound and Vibration, 21*(4), 449-462.
- Morell, S., Taylor, R., & Lyle, D. (1997). A review of the health effects of aircraft noise. *Australian and New Zealand Journal of Public Health, 21*(2), 221-236.
- Moudon, A. V. (2009). Real noise from the urban environment: How ambient community noise affects health and what can be done about it. *American Journal of Preventive Medicine, 37*(2), 167-171. doi:10.1016/j.amepre.2009.03.019
- Muhtz, C., Zyriax, B. C., Bondy, B., Windler, E., & Otte, C. (2011). Association of a common mineralocorticoid receptor gene polymorphism with salivary cortisol in healthy adults. *Psychoneuroendocrinology, 36*(2), 298-301. doi:10.1016/j.psyneuen.2010.08.003
- Munafò, M. R., Lee, L., Ayres, R., Flint, J., Goodwin, G., & Harmer, C. J. (2006). Early morning salivary cortisol is not associated with extraversion. *Personality and Individual Differences, 40*(2), 395-400. doi:10.1016/j.paid.2005.06.028
- Murata, M., & Sakamoto, H. (1995). A case of high noise sensitivity. *Journal of Sound and Vibration, 186*(4), 617-621. doi:10.1006/jsvi.1995.0473
- Muzet, A. (2007). Environmental noise, sleep and health. *Sleep Medicine Reviews, 11*(2), 135-142. doi:10.1016/j.smrv.2006.09.001
- Nater, U. M., Maloney, E., Boneva, R. S., Gurbaxani, B. M., Lin, J. M., Jones, J. F., ... Heim, C. (2008). Attenuated morning salivary cortisol concentrations in a population-based study of persons with chronic fatigue syndrome and well controls. *The Journal of Clinical Endocrinology and Metabolism, 93*(3), 703-709. doi:10.1210/jc.2007-1747
- Ndrepepa, A., & Twardella, D. (2011). Relationship between noise annoyance from road traffic noise and cardiovascular diseases: A meta-analysis. *Noise & Health, 13*(52), 251-259.
- Neus, H., & Boikat, U. (2000). Evaluation of traffic noise-related cardiovascular risk. *Noise & Health, 2*(7), 65-77.
- Neus, H., Ruddel, H., & Schulte, W. (1983). Traffic noise and hypertension: An epidemiological study on the role of subjective reactions. *International Archives of Occupational and Environmental Health, 51*, 223-229.
- Newman, D. A. (2003). Longitudinal modeling with randomly and systematically missing data: A simulation of ad hoc, maximum likelihood, and multiple imputation techniques. *Organizational Research Methods, 6*, 328-362.
- Neylan, T. C., Brunet, A., Pole, N., Best, S. R., Metzler, T. J., Yehuda, R., & Marmar, C. R. (2005). PTSD symptoms predict waking salivary cortisol levels in police officers. *Psychoneuroendocrinology, 30*(4), 373-381. doi:10.1016/j.psyneuen.2004.10.005

- Niemann, H., Bonnefoy, X., Braubach, M., Hecht, K., Maschke, C., Rodrigues, C., & Robbel, N. (2006). Noise-induced annoyance and morbidity results from the pan-European LARES study. *Noise & Health, 8*(31), 63-79.
- Nijland, H. A., Hartemink, S., van Kamp, I., & van Wee, B. (2007). The influence of sensitivity for road traffic noise on residential location: Does it trigger a process of spatial selection? *Journal of the Acoustical Society of America, 122*(3), 1595-1601. doi:10.1121/1.2756970
- Nivison, M. E., & Endresen, I. M. (1993). An analysis of relationships among environmental noise, annoyance and sensitivity to noise, and the consequences for health and sleep. *Journal of Behavioral Medicine, 16*(3), 257-276. doi:10.1007/BF00844759
- Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality and Social Psychology, 77*(5), 1061-1072.
- Nordin, S., Millqvist, E., Löwhagen, O., & Bende, M. (2003). The Chemical Sensitivity Scale: Psychometric properties and comparison with the noise sensitivity scale. *Journal of Environmental Psychology, 23*(4), 359-367. doi:10.1016/s0272-4944(03)00002-1
- Nordstrom, C. K., Dwyer, K. M., Merz, N. B., Shicore, A., & Dwyer, J. H. (2001). Work-related stress and early atherosclerosis. *Epidemiology, 12*(2), 180-185.
- Noto, Y., Sato, T., Kudo, M., Kurata, K., & Hirota, K. (2005). The relationship between salivary biomarkers and state-trait anxiety inventory score under mental arithmetic stress: A pilot study. *Anesthesia and Analgesia, 101*(6), 1873-1876. doi:10.1213/01.ANE.0000184196.60838.8D
- Novak, C. C., La Lopa, J., & Novak, R. E. (2010). Effects of sound pressure levels and sensitivity to noise on mood and behavioral intent in a controlled fine dining restaurant environment. *Journal of Culinary Science & Technology, 8*(4), 191-218. doi:10.1080/15428052.2010.535756
- O'Connor, T. M., O'Halloran, D. J., & Shanahan, F. (2000). The stress response and the hypothalamic-pituitary-adrenal axis: From molecule to melancholia. *QJM: An International Journal of Medicine, 93*, 323-333.
- Ogden, J. (2007). *Health psychology: A textbook*. New York, NY: Open University Press.
- Öhrström, E. (1991). Psycho-social effects of traffic noise exposure. *Journal of Sound and Vibration, 151*(3), 513-517.
- Öhrström, E. (1995). Effects of low levels of road traffic noise during the night: A laboratory study on number of events, maximum noise levels and noise sensitivity. *Journal of Sound and Vibration, 179*(4), 603-615.
- Öhrström, E. (2004). Longitudinal surveys on effects of changes in road traffic noise—annoyance, activity disturbances, and psycho-social well-being. *Journal of the Acoustical Society of America, 115*(2), 719-729. doi:10.1121/1.1639333
- Öhrström, E., & Björkman, M. (1988). Effects of noise-disturbed sleep—A laboratory study on habituation and subjective noise sensitivity. *Journal of Sound and Vibration, 122*(2), 277-290.
- Öhrström, E., Björkman, M., & Rylander, R. (1988). Noise annoyance with regard to neurophysiological sensitivity, subjective noise sensitivity and personality variables. *Psychological Medicine, 18*, 605-613.

- Öhrström, E., Björkman, M., & Rylander, R. (1990). Effects of noise during sleep with reference to noise sensitivity and habituation. *Environment International*, 16, 477-482.
- Öhrström, E., Rylander, R., & Björkman, M. (1988). Effects of night time road traffic noise—An overview of laboratory and field studies on noise dose and subjective noise sensitivity. *Journal of Sound and Vibration*, 127(3), 441-448.
- Öhrström, E., Skanberg, A., Svensson, H., & Gidlof-Gunnarsson, A. (2006). Effects of road traffic noise and the benefit of access to quietness. *Journal of Sound and Vibration*, 295(1-2), 40-59. doi:10.1016/j.jsv.2005.11.034
- Oldehinkel, A. J., Ormel, J., Bosch, N. M., Bouma, E. M., Van Roon, A. M., Rosmalen, J. G., & Riese, H. (2011). Stressed out? Associations between perceived and physiological stress responses in adolescents: The TRAILS study. *Psychophysiology*, 48(4), 441-452. doi:10.1111/j.1469-8986.2010.01118.x
- Olsen Widen, S. E., & Erlandsson, S. I. (2004). Self-reported tinnitus and noise sensitivity among adolescents in Sweden. *Noise & Health*, 7, 29-40.
- Ong, A. D., Fuller-Rowell, T. E., Bonanno, G. A., & Almeida, D. M. (2011). Spousal loss predicts alterations in diurnal cortisol activity through prospective changes in positive emotion. *Health Psychology*, 30(2), 220-227. doi:10.1037/a0022262
- Oskis, A., Loveday, C., Hucklebridge, F., Thorn, L., & Clow, A. (2009). Diurnal patterns of salivary cortisol across the adolescent period in healthy females. *Psychoneuroendocrinology*, 34(3), 307-316. doi:10.1016/j.psyneuen.2008.09.009
- Oswald, L. M., Zandi, P., Nestadt, G., Potash, J. B., Kalaydjian, A. E., & Wand, G. S. (2006). Relationship between cortisol responses to stress and personality. *Neuropsychopharmacology*, 31(7), 1583-1591. doi:10.1038/sj.npp.1301012
- Ouis, D. (2001). Annoyance from road traffic noise: A review. *Journal of Environmental Psychology*, 21(1), 101-120. doi:10.1006/jevp.2000.0187
- Ouis, D. (2002). Annoyance caused by exposure to road traffic noise: An update. *Noise & Health*, 4(15), 69-79.
- Owens, I. P. (2002). Ecology and evolution. Sex differences in mortality rate. *Science*, 297(5589), 2008-2009. doi:10.1126/science.1076813
- Padgett, D. A., & Glaser, R. (2003). How stress influences the immune response. *Trends in Immunology*, 24(8), 444-448. doi:10.1016/s1471-4906(03)00173-x
- Padungtod, C., Ekpanyaskul, C., Nuchongsai, P., Laemun, N., Matsui, T., & Hiramatsu, K. (2011). Aircraft noise exposure and its effects on quality of life and cognitive function among Thai residents. *Epidemiology*, 22(1), S259.
- Pasch, L. A., Bradbury, T. N., & Davila, J. (1997). Gender, negative affectivity, and observed social support behavior in marital interaction. *Personal Relationships*, 4, 361-378.
- Passchier-Vermeer, W., & Passchier, W. F. (2000). Noise exposure and public health. *Environmental Health Perspectives*, 108, 123-130. doi:10.2307/3454637
- Paunovic, K., Jakovljević, B., & Belojević, G. (2009). Predictors of noise annoyance in noisy and quiet urban streets. *Science of the Total Environment*, 407(12), 3707-3711. doi:10.1016/j.scitotenv.2009.02.033
- Paunovic, K., Stansfeld, S. A., Clark, C., & Belojević, G. (2011). Epidemiological studies on noise and blood pressure in children: Observations and suggestions. *Environment International*, 37(5), 1030-1041. doi:10.1016/j.envint.2011.03.017
- Pearson, J. D., Morrell, C. H., Gordon-Salant, S., Brant, L. J., Metter, E. J., Klein, L. K., & Fozard, J. L. (1995). Gender differences in a longitudinal study of age-associated

- hearing loss. *Journal of the Acoustical Society of America*, 97(2), 1195-1205.
doi:10.1121/1.412231
- Pearsons, K. S., Barber, D. S., Tabachnick, B. G., & Fidell, S. (1995). Predicting noise-induced sleep disturbance. *Journal of the Acoustical Society of America*, 97(1), 331-338.
- Pedersen, E., Hallberg, L.-M., & Waye, K. P. (2007). Living in the vicinity of wind turbines — A grounded theory study. *Qualitative Research in Psychology*, 4(1-2), 49-63. doi:10.1080/14780880701473409
- Pedersen, E., & Persson Waye, K. (2004). Perception and annoyance due to wind turbine noise—a dose-response relationship. *Journal of the Acoustical Society of America*, 116(6), 3460-3470. doi:10.1121/1.1815091
- Pedersen, E., & Persson Waye, K. (2007). Wind turbine noise, annoyance and self-reported health and well-being in different living environments. *Occupational and Environmental Medicine*, 64(7), 480-486. doi:10.1136/oem.2006.031039
- Pedersen, E., & Persson Waye, K. (2008). Wind turbines—low level noise sources interfering with restoration? *Environmental Research Letters*, 3(1), 1-5.
doi:10.1088/1748-9326/3/1/015002
- Pelle, A. J., Pedersen, S. S., Szabo, B. M., & Denollet, J. (2009). Beyond Type D personality: Reduced positive affect (anhedonia) predicts impaired health status in chronic heart failure. *Quality of Life Research*, 18(6), 689-698. doi:10.1007/s11136-009-9485-z
- Persson, K., & Rylander, R. (1988). Disturbance from low-frequency noise in the environment: A survey among the local environmental health authorities in Sweden. *Journal of Sound and Vibration*, 121(2), 339-345.
- Persson, R., Osterberg, K., Garde, A. H., Hansen, A. M., Orbaek, P., & Karlson, B. (2010). Seasonal variation in self-reported arousal and subjective health complaints. *Psychology, Health & Medicine*, 15(4), 434-444.
doi:10.1080/13548506.2010.482140
- Persson Waye, K., Agge, A., Clow, A., & Hucklebridge, F. (2004). Cortisol response and subjective sleep disturbance after low-frequency noise exposure. *Journal of Sound and Vibration*, 277(3), 453-457. doi:10.1016/j.jsv.2004.03.004
- Persson Waye, K., Bengtsson, J., Kjellberg, A., & Benton, S. (2001). Low frequency noise "pollution" interferes with performance. *Noise & Health*, 4(13), 33-49.
- Persson Waye, K., Bengtsson, J., Rylander, R., Hucklebridge, F., Evans, P., & Clow, A. (2002). Low frequency noise enhances cortisol among noise sensitive subjects during work performance. *Life Sciences*, 70, 745-758.
- Persson Waye, K., Clow, A., Edwards, S., Hucklebridge, F., & Rylander, R. (2003). Effects of nighttime low frequency noise on the cortisol response to awakening and subjective sleep quality. *Life Sciences*, 72, 863-875.
- Persson Waye, K., & Öhrström, E. (2002). Psycho-acoustic characters of relevance for annoyance of wind turbine noise. *Journal of Sound and Vibration*, 250(1), 65-73.
doi:10.1006/jsvi.2001.3905
- Pervin, L. A., & John, O. P. (2001). *Personality: Theory and research (8th ed.)*. New York, NY: John Wiley & Sons.
- Piccinelli, M., & Wilkinson, G. (2000). Gender differences in depression: Critical review. *The British Journal of Psychiatry*, 177(6), 486-492. doi:10.1192/bjp.177.6.486

- Pickett, K. E., & Pearl, M. (2001). Multilevel analyses of neighbourhood socioeconomic context and health outcomes: A critical review. *Journal of Epidemiology and Community Health*, 55(2), 111-122. doi:10.1136/jech.55.2.111
- Pierrette, M., Marquis-Favre, C., Morel, J., Rioux, L., Vallet, M., Viollon, S., & Moch, A. (2012). Noise annoyance from industrial and road traffic combined noises: A survey and a total annoyance model comparison. *Journal of Environmental Psychology*, 32(2), 178-186. doi:10.1016/j.jenvp.2012.01.006
- Pirrerera, S., De Valck, E., & Cluydts, R. (2010). Nocturnal road traffic noise: A review on its assessment and consequences on sleep and health. *Environment International*, 36(5), 492-498. doi:10.1016/j.envint.2010.03.007
- Polk, D. E., Cohen, S., Doyle, W. J., Skoner, D. P., & Kirschbaum, C. (2005). State and trait affect as predictors of salivary cortisol in healthy adults. *Psychoneuroendocrinology*, 30(3), 261-272. doi:10.1016/j.psyneuen.2004.08.004
- Portella, M. J., Harmer, C. J., Flint, J., Cowen, J., Goodwin, P., & Guy, M. (2005). Enhanced early morning salivary cortisol in neuroticism. *The American Journal of Psychiatry*, 162(4), 807-809.
- Prasher, D. (2009). Is there evidence that environmental noise is immunotoxic? *Noise & Health*, 11(44), 151-155.
- Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, 40(3), 879-891. doi:10.3758/brm.40.3.879
- Preacher, K. J., Rucker, D. H., & Hayes, A. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research*, 42(1), 185-227.
- Pruessner, J. C., Dedovic, K., Pruessner, M., Lord, C., Buss, C., Collins, L., ... Lupien, S. J. (2010). Stress regulation in the central nervous system: Evidence from structural and functional neuroimaging studies in human populations - 2008 Curt Richter Award Winner. *Psychoneuroendocrinology*, 35(1), 179-191. doi:10.1016/j.psyneuen.2009.02.016
- Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology*, 28(7), 916-931. doi:10.1016/s0306-4530(02)00108-7
- Pruessner, J. C., Wolf, O. T., von Auer, K., Jobst, S., Kaspers, F., Buske-Kirschbaum, A., ... Kirschbaum, C. (1997). The cortisol response to awakening: Consistency, reliability, and individual differences. *Life Sciences*, 61, 2539-2549.
- Pruessner, M., Hellhammer, D. H., Pruessner, J. C., & Lupien, S. J. (2003). Self-reported depressive symptoms and stress levels in healthy young men: Associations with the cortisol response to awakening. *Psychosomatic Medicine*, 65(1), 92-99. doi:10.1097/01.psy.0000040950.22044.10
- Pulles, M. P. J., Siesiot, W., & Stewart, R. (1990). Adverse effects of environmental noise on health: An interdisciplinary approach. *Environment International*, 16, 437-445. doi:10.1016/0160-4120(90)90012-U
- Quirin, M., Pruessner, J. C., & Kuhl, J. (2008). HPA system regulation and adult attachment anxiety: Individual differences in reactive and awakening cortisol. *Psychoneuroendocrinology*, 33(5), 581-590. doi:10.1016/j.psyneuen.2008.01.013

- Rabat, A., Bouyer, J. J., Aran, J. M., Le Moal, M., & Mayo, W. (2005). Chronic exposure to an environmental noise permanently disturbs sleep in rats: Inter-individual vulnerability. *Brain Research*, 1059(1), 72-82. doi:10.1016/j.brainres.2005.08.015
- Rademaker, A. R., Kleber, R. J., Geuze, E., & Vermetten, E. (2009). Personality dimensions harm avoidance and self-directedness predict the cortisol awakening response in military men. *Biological Psychology*, 81(3), 177-183. doi:10.1016/j.biopsycho.2009.04.002
- Raggam, R. B., Cik, M., Holdrich, R. R., Fallast, K., Gallasch, E., Fend, M., ... Marth, E. (2007). Personal noise ranking of road traffic: Subjective estimation versus physiological parameters under laboratory conditions. *International Journal of Hygiene and Environmental Health*, 210(2), 97-105. doi:10.1016/j.ijheh.2006.08.007
- Ramirez, J. M., Alvarado, J. M., & Santisteban, C. (2004). Individual differences in anger reaction to noise. *Individual Differences Research*, 2(2), 125-136.
- Rammsayer, T. H., & Troche, S. J. (2011). On sex-related differences in auditory and visual sensory functioning. *Archives of Sexual Behavior*, 41(3), 583-590. doi:10.1007/s10508-011-9880-8
- Raw, G. J., & Griffiths, I. D. (1988). Individual differences in response to road traffic noise. *Journal of Sound and Vibration*, 121(3), 463-471.
- Rhudy, J. L., & Meagher, M. W. (2001). Noise stress and human pain thresholds: Divergent effects in men and women. *The Journal of Pain*, 1, 57-64.
- Rippetoe, P. A., & Rogers, R. W. (1987). Effects of components of protection-motivation theory on adaptive and maladaptive coping with a health threat. *Journal of Personality and Social Psychology*, 52(3), 596-604. doi:10.1037/0022-3514.52.3.596
- Riva, R., Mork, P. J., Westgaard, R. H., & Lundberg, U. (2012). Comparison of the cortisol awakening response in women with shoulder and neck pain and women with fibromyalgia. *Psychoneuroendocrinology*, 37(2), 299-306. doi:10.1016/j.psyneuen.2011.06.014
- Roberts, L. E., Eggermont, J. J., Caspary, D. M., Shore, S. E., Melcher, J. R., & Kaltenbach, J. A. (2010). Ringing ears: The neuroscience of tinnitus. *The Journal of Neuroscience*, 30(45), 14972-14979.
- Rogers, D. S., Harkrider, A. W., Burchfield, S. B., & Nabelek, A. K. (2003). The influence of listener's gender on the acceptance of background noise. *Journal of the American Academy of Audiology*, 14(7), 372-382.
- Roozendaal, B., McEwen, B. S., & Chattarji, S. (2009). Stress, memory and the amygdala. *Nature Reviews. Neuroscience*, 10(6), 423-433. doi:10.1038/nrn2651
- Rorschach, H. (1942). *Psychodiagnostics: A diagnostic test based on perception*. New York, NY: Grune & Statton.
- Rosenkranz, J. A., Venheim, E. R., & Padival, M. (2010). Chronic stress causes amygdala hyperexcitability in rodents. *Biological Psychiatry*, 67(12), 1128-1136. doi:10.1016/j.biopsych.2010.02.008
- Rosenlund, M., Berglind, N., Pershagen, G., Jarup, L., & Bluhm, G. (2001). Increased prevalence of hypertension in a population exposed to aircraft noise. *Occupational and Environmental Medicine*, 58, 769-773. doi:10.1136/oem.58.12.769
- Ruff, R. M., Iverson, G. L., Barth, J. T., Bush, S. S., & Broshek, D. K. (2009). Recommendations for diagnosing a mild traumatic brain injury: A National

- Academy of Neuropsychology education paper. *Archives of Clinical Neuropsychology*, 24(1), 3-10. doi:10.1093/arclin/acp006
- Ryckman, R. M. (2007). *Theories of personality*. Belmont, CA: Cengage Learning.
- Rylander, R. (2004). Physiological aspects of noise-induced stress and annoyance. *Journal of Sound and Vibration*, 277(3), 471-478. doi:10.1016/j.jsv.2004.03.008
- Rylander, R. (2006). Noise, stress and annoyance. *Noise & Vibration Worldwide*, 37(6), 9-13.
- Rylander, R., Bjorkman, M., Ahrlin, U., Arntzen, E., & Solberg, S. (1986). Dose-response relationships for traffic noise and annoyance. *Archives of Environmental Health*, 41(1), 7-10.
- Ryu, J. K., & Jeon, J. Y. (2011). Influence of noise sensitivity on annoyance of indoor and outdoor noises in residential buildings. *Applied Acoustics*, 72(6), 336-340. doi:10.1016/j.apacoust.2010.12.005
- Sandrock, S., Schütte, M., & Griefahn, B. (2007). The reliability of the noise sensitivity questionnaire in a cross-national analysis. *Noise & Health*, 9, 8-14.
- Sandrock, S., Schütte, M., & Griefahn, B. (2009). Impairing effects of noise in high and low noise sensitive persons working on different mental tasks. *International Archives of Occupational and Environmental Health*, 82(6), 779-785. doi:10.1007/s00420-008-0379-0
- Sandrock, S., Schütte, M., & Griefahn, B. (2010). Mental strain and annoyance during cognitive performance in different traffic noise conditions. *Ergonomics*, 53(8), 962-971. doi:10.1080/00140139.2010.500401
- Sapolsky, R. M. (2004). *Why zebras don't get ulcers: An updated guide to stress, stress-related diseases and coping* (3rd ed.). New York, NY: Holt.
- Sapolsky, R. M., Krey, L., & McEwen, B. S. (1985). Prolonged glucocorticoid exposure reduces hippocampal neuron number: Implications for aging. *Journal of Neuroscience*, 5(5), 1222-1227.
- Sapolsky, R. M., Krey, L., & McEwen, B. S. (1986). The neuroendocrinology of stress and aging: The glucocorticoid cascade hypothesis. *Endocrine Reviews*, 7, 284-303.
- Sapolsky, R. M., Uno, H., Rebert, C. S., & Finch, C. E. (1990). Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *The Journal of Neuroscience*, 10(9), 2897-2902.
- Sauvé, B., Koren, G., Walsh, G., Tokmakejian, S., & Van Uum, S. H. M. (2007). Measurement of cortisol in human hair as a biomarker of systemic exposure. *Clinical and Investigative Medicine*, 30(5), 83-91.
- Sax, L. (2010). Sex differences in hearing: Implications for best practice in the classroom. *Advances in Gender and Education*, 2, 13-21.
- Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, 7, 147-177.
- Schlotz, W., Hellhammer, J., Schulz, P., & Stone, A. A. (2004). Perceived work overload and chronic worrying predict weekend-weekday differences in the cortisol awakening response. *Psychosomatic Medicine*, 66(2), 207-214. doi:10.1097/01.psy.0000116715.78238.56
- Schmidt-Reinwald, A., Pruessner, J. C., Hellhammer, D. H., Federenko, I., Rohleder, N., Schumeyer, T. H., & Kirschbaum, C. (1999). The cortisol response to awakening in relation to different challenge tests and a 12-hour cortisol rhythm. *Life Sciences*, 64(18), 1653-1660.

- Schrekenberg, D., Griefahn, B., & Meis, M. (2010). The associations between noise sensitivity, reported physical and mental health, perceived environmental quality, and noise annoyance. *Noise & Health*, 12(46), 7-15.
- Schrekenberg, D., Griefahn, B., & Meis, M. (2010a). The associations between noise sensitivity, reported physical and mental health, perceived environmental quality and noise annoyance. *Noise & Health*, 12, 46-54.
- Schrekenberg, D., Meis, M., Kahl, C., Peschel, C., & Eikmann, T. (2010b). Aircraft noise and quality of life around Frankfurt Airport. *International Journal of Environmental Research and Public Health*, 7(9), 3382-3405. doi:10.3390/ijerph7093382
- Schultz, T. J. (1978). Synthesis of social surveys on noise annoyance. *Journal of the Acoustical Society of America*, 64(2), 377-405.
- Schulz, A., & Northridge, M. E. (2004). Social determinants of health: Implications for environmental health promotion. *Health Education & Behavior*, 31(4), 455-471. doi:10.1177/1090198104265598
- Schulz, P., Kirschbaum, C., Pruessner, J. C., & Hellhammer, D. H. (1998). Increased free cortisol secretion after awakening in chronically stressed individuals due to work overload. *Stress Medicine*, 14, 91-97.
- Schütte, M., Marks, A., Wenning, E., & Griefahn, B. (2007). The development of the noise sensitivity questionnaire. *Noise & Health*, 9, 15-34.
- Schütte, M., Sandrock, S., & Griefahn, B. (2007). Factorial validity of the noise sensitivity questionnaire. *Noise & Health*, 9(37), 96-100.
- Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences of the United States of America*, 98(8), 4770-4775. doi:10.1073/pnas.081072698
- Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation—Allostatic load and its health consequences. *Archives of Internal Medicine*, 157, 2259-2268.
- Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, 130(4), 601-630. doi:10.1037/0033-2909.130.4.601
- Seidman, M. D., & Standring, R. T. (2010). Noise and quality of life. *International Journal of Environmental Research and Public Health*, 7(10), 3730-3738. doi:10.3390/ijerph7103730
- Selander, J. (2010). *Traffic noise and cardiovascular disease*. Karolinska Institute, Stockholm.
- Selander, J., Bluhm, G., Theorell, T., Pershagen, G., Babisch, W., Seiffert, I., ... Jarup, L. (2009a). Saliva cortisol and exposure to aircraft noise in six European countries. *Environmental Health Perspectives*, 117(11), 1713-1717. doi:10.1289/ehp.0900933
- Selander, J., Nilsson, M. E., Bluhm, G., Rosenlund, M., Lindqvist, M., Nise, G., & Pershagen, G. (2009b). Long-term exposure to road traffic noise and myocardial infarction. *Epidemiology*, 20(2), 272-279. doi:10.1097/EDE.0b013e31819463bd
- Selgrade, J. F. (2010). Bifurcation analysis of a model for hormonal regulation of the menstrual cycle. *Mathematical Biosciences*, 225(2), 108-114. doi:10.1016/j.mbs.2010.02.004
- Seligman, M. E., Maier, S. F., & Geer, J. H. (1968). Alleviation of learned helplessness in the dog. *Journal of Abnormal Psychology*, 73(3), 256-262.

- Seligman, M. E. P. (1975). *Helplessness: On depression, development, and death*. San Francisco, CA: Freeman.
- Selye, H. (1936). A syndrome produced by diverse nocuous agents. *Nature*, 138, 32. doi:10.1038/138032a0
- Selye, H. (1946). The general adaptation syndrome and the diseases of adaptation. *Journal of Clinical Endocrinology and Metabolism*, 6(2), 117-230. doi:10.1210/jcem-6-2-117
- Shabana, M. I., Selim, M. H., El Refaie, A., El Dessouky, T. M., & Soliman, R. Y. (2011). Assessment of hyperacusis in Egyptian patients: Evaluation of the Arabic version of the Khalfa questionnaire. *Audiological Medicine*, 9(4), 127-134. doi:10.3109/1651386x.2011.624684
- Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nature Reviews. Neuroscience*, 12(3), 154-167. doi:10.1038/nrn2994
- Shea, A. K., Streiner, D. L., Fleming, A., Kamath, M. V., Broad, K., & Steiner, M. (2007). The effect of depression, anxiety and early life trauma on the cortisol awakening response during pregnancy: Preliminary results. *Psychoneuroendocrinology*, 32, 1013-1020. doi:10.1016/j.psyneuen.2007.07.006
- Shepherd, D., McBride, D., Welch, D., Dirks, K. N., & Hill, E. M. (2011). Evaluating the impact of wind turbine noise on health-related quality of life. *Noise & Health*, 13(54), 333-339.
- Shepherd, D., Welch, D., Dirks, K. N., & Mathews, R. (2010). Exploring the relationship between noise sensitivity, annoyance and health-related quality of life in a sample of adults exposed to environmental noise. *International Journal of Environmental Research and Public Health*, 7(10), 3579-3594. doi:10.3390/ijerph7103580
- Sherwood, L. (2012). *Human physiology: From cells to systems*. Belmont, CA: Cengage Learning.
- Shih, J. H., & Eberhart, N. K. (2010). Gender differences in the associations between interpersonal behaviors and stress generation. *Journal of Social and Clinical Psychology*, 29(3), 243-255.
- Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences*, 1071, 67-79. doi:10.1196/annals.1364.007
- Short, A. E., Ahern, N., Holdgate, A., Morris, J., & Sidhu, B. (2010). Using music to reduce noise stress for patients in the emergency department: A pilot study. *Music and Medicine*, 2(4), 201-207. doi:10.1177/1943862110371808
- Siegel, J. M. (2001). The REM sleep-memory consolidation hypothesis. *Science*, 295(5544), 1058-1063. doi:10.1126/science.1063049
- Siegel, J. M. (2005). Clues to the functions of mammalian sleep. *Nature*, 437(7063), 1264-1271. doi:10.1038/nature04285
- Smith, A. (1989). A review of the effects of noise on human performance. *Scandinavian Journal of Psychology*, 30, 185-206.
- Smith, A. (2003). The concept of noise sensitivity: Implications for noise control. *Noise & Health*, 5(18), 57-59.
- Smith, A., Nutt, D., Wilson, S., Rich, N., Hayward, S., & Heatherley, S. (2002). *Noise and insomnia: A study of community noise exposure, sleep disturbance, noise sensitivity*

- and subjective reports of health. Cardiff, UK: Report to the UK Department of Health and Department of Environment, Transport and the Regions.
- Smith, A., & Stansfeld, S. A. (1986). Aircraft noise exposure, noise sensitivity, and everyday errors. *Environment and Behavior*, 18(2), 214-226. doi:10.1177/0013916586182004
- Smolewska, K. A., McCabe, S. B., & Woody, E. Z. (2006). A psychometric evaluation of the Highly Sensitive Person Scale: The components of sensory-processing sensitivity and their relation to the BIS/BAS and "Big Five". *Personality and Individual Differences*, 40(6), 1269-1279. doi:10.1016/j.paid.2005.09.022
- Sobotova, L., Jurkovicova, J., Stefanikova, Z., Sevcikova, L., & Aghova, L. (2010). Community response to environmental noise and the impact on cardiovascular risk score. *Science of the Total Environment*, 408(6), 1264-1270. doi:10.1016/j.scitotenv.2009.12.033
- Son, J., Erno, A., Shea, D. G., Femia, E. E., Zarit, S. H., & Stephens, M. A. (2007). The caregiver stress process and health outcomes. *Journal of Aging and Health*, 19(6), 871-887. doi:10.1177/0898264307308568
- Sorensen, M., Hvidberg, M., Hoffmann, B., Andersen, Z. J., Nordsborg, R. B., Lillelund, K. G., ... Raaschou-Nielsen, O. (2011). Exposure to road traffic and railway noise and associations with blood pressure and self-reported hypertension: A cohort study. *Environmental Health*, 10, 92-102. doi:10.1186/1476-069X-10-92
- Spencer, R. L., Kim, P. L., Kalman, B. A., & Cole, M. A. (1998). Evidence for mineralocorticoid receptor facilitation of glucocorticoid receptor-dependent regulation of hypothalamic-pituitary-adrenal axis activity. *Endocrinology*, 139(6), 2718-2726.
- Spreng, M. (2000a). Central nervous system activation by noise. *Noise & Health*, 2, 49-57.
- Spreng, M. (2000b). Possible health effects of noise induced cortisol increase. *Noise & Health*, 2(7), 59-63.
- Spreng, M. (2004). Noise induced nocturnal cortisol secretion and tolerable overheard flights. *Noise & Health*, 6(22), 35-47.
- Stalder, T., Hucklebridge, F., Evans, P., & Clow, A. (2009). Use of a single case study design to examine state variation in the cortisol awakening response: Relationship with time of awakening. *Psychoneuroendocrinology*, 34(4), 607-614. doi:10.1016/j.psyneuen.2008.10.023
- Stallen, P. J. M. (1999). A theoretical framework for environmental noise annoyance. *Noise & Health*, 3, 69-79.
- Stansfeld, S. A. (1992). Noise, noise sensitivity and psychiatric disorder – epidemiological and psychophysiological studies. *Psychological Medicine, Monograph Supplement*, 22, 1-44.
- Stansfeld, S. A., Clark, C., Cameron, R. M., Alfred, T., Head, J., Haines, M. M., ... Lopez-Barrio, I. (2009). Aircraft and road traffic noise exposure and children's mental health. *Journal of Environmental Psychology*, 29(2), 203-207. doi:10.1016/j.jenvp.2009.01.002
- Stansfeld, S. A., Clark, C. R., Jenkins, L. M., & Tarnopolsky, A. (1985a). Sensitivity to noise in a community sample: I. Measurement of psychiatric disorder and personality. *Psychological Medicine*, 15, 243-254. doi:10.1017/S0033291700023539
- Stansfeld, S. A., Clark, C. R., Turpin, L., Jenkins, M., & Tarnopolsky, A. (1985b). Sensitivity to noise in a community sample: II. Measurement of

- psychophysiological indices. *Psychological Medicine*, 15, 255-263.
doi:10.1017/S0033291700023539
- Stansfeld, S. A., & Crombie, R. (2011). Cardiovascular effects of environmental noise: Research in the United Kingdom. *Noise & Health*, 13(52), 229-233.
- Stansfeld, S. A., Gallacher, J., Babisch, W., & Shipley, M. (1996). Road traffic noise and psychiatric disorder: Prospective findings from the Caerphilly study. *British Medical Journal*, 313, 266-267.
- Stansfeld, S. A., Haines, M., & Brown, B. (2000). Noise and health in the urban environment. *Reviews on Environmental Health*, 15, 43-82.
- Stansfeld, S. A., Haines, M. M., Burr, M., Berry, B., & Lercher, P. (2000). A review of environmental noise and mental health. *Noise & Health*, 2(8), 1-8.
- Stansfeld, S. A., Head, J., Clark, C., van Kamp, I., & Barrio, I. L. (2005). Aircraft and road traffic noise and children's cognition – Authors' reply. *The Lancet*, 366(9487), 716.
doi:10.1016/s0140-6736(05)67175-9
- Stansfeld, S. A., & Matheson, M. (2003). Noise pollution: Non-auditory effects on health. *British Medical Bulletin*, 68(1), 243-257. doi:10.1093/bmb/ldg033
- Stansfeld, S. A., Sharp, D. S., Gallacher, J., & Babisch, W. (1993). Road traffic noise, noise sensitivity and psychological disorder. *Psychological Medicine*, 23, 977-985.
- Stansfeld, S. A., & Shine, P. (1993). Noise sensitivity and psychophysiological responses to noise in the laboratory. In M. Vallet (Chair), Symposium conducted at the meeting of the 6th International Congress of Noise as a Public Health Problem, Arcueil Cedex, France.
- Staples, S. L. (1996). Human response to environmental noise. *American Psychologist*, 51(2), 143-150.
- Staples, S. L. (1997). Public policy and environmental noise: Modeling exposure or understanding effects. *American Journal of Public Health*, 87, 2063-2067.
- Staples, S. L., Cornelius, R. R., & Gibbs, M. S. (1999). Noise disturbance from a developing airport: Perceived risk or general annoyance? *Environment and Behavior*, 31(5), 692-710. doi:10.1177/00139169921972308
- Statistics New Zealand. (2006a). *QuickStats about Auckland region*. Retrieved 21 May, 2012, from <http://www.stats.govt.nz/>
- Statistics New Zealand. (2006b). *QuickStats about culture and identity*. Retrieved 16 April, 2012, from <http://www.stats.govt.nz>
- Statistics New Zealand. (2006c). *QuickStats about education and training*. Retrieved 16 April, 2012, from <http://www.stats.govt.nz>
- Steptoe, A., Brydon, L., & Kunz-Ebrecht, S. (2005). Changes in financial strain over three years, ambulatory blood pressure, and cortisol responses to awakening. *Psychosomatic Medicine*, 67, 281-287. doi:10.1097/01.psy.0000156932.96261.d2
- Steptoe, A., Cropley, M., Griffith, J., & Kirschbaum, C. (2000). Job strain and anger expression predict early morning elevations in salivary cortisol. *Psychosomatic Medicine*, 62, 286-292.
- Steptoe, A., & Willemsen, G. (2004). The influence of low job control on ambulatory blood pressure and perceived stress over the working day in men and women from the Whitehall II Cohort. *Journal of Hypertension*, 22, 915-920.
doi:10.1097/01.hjh.0000098299.36684.76
- Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher & J. Reason (Eds.), *Handbook of life stress, cognition, and health* (pp. 629-649). Chichester, UK: John Wiley & Sons.

- Stickgold, R. (2011). The role of REM sleep in memory consolidation, enhancement, and integration. In B. N. Mallick, S. R. Pandi-Perumal, R. W. McCarley, & A. R. Morrison (Eds.), *REM sleep: Regulation and function* (pp. 328-338). Cambridge, UK: Cambridge University Press.
- Stiegler, L. N., & Davis, R. (2010). Understanding sound sensitivity in individuals with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 25(2), 67-75. doi:10.1177/1088357610364530
- Stokols, D. (1992). Establishing and maintaining healthy environments: Toward a social ecology of health promotion. *American Psychologist*, 47(1), 6-22.
- Stošić, L., & Blagojević, L. (2011). Environmental noise and mental disturbances in urban population. *Acta Medica Medianae*, 34-39. doi:10.5633/amm.2011.0306
- Stroud, L. R., Papandonatos, G. D., Williamson, D. E., & Dahl, R. E. (2011). Sex differences in cortisol response to corticotropin releasing hormone challenge over puberty: Pittsburgh Pediatric Neurobehavioral Studies. *Psychoneuroendocrinology*, 36(8), 1226-1238. doi:10.1016/j.psyneuen.2011.02.017
- Sun, W., Deng, A., Jayaram, A., & Gibson, B. (2012). Noise exposure enhances auditory cortex responses related to hyperacusis behavior. *Brain Research*. doi:10.1016/j.brainres.2012.02.008
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics (5th ed.)*. Boston, MA: Allyn and Bacon.
- Tamres, L. K., Janicki, D., & Helgeson, V. S. (2002). Sex differences in coping behavior: A meta-analytic review and an examination of relative coping. *Personality and Social Psychology Review*, 6(1), 2-30. doi:10.1207/s15327957pspr0601_1
- Tarnopolsky, A., Barker, S. M., Wiggins, R. D., & McLean, E. K. (1978). The effect of aircraft noise on the mental health of a community sample: A pilot study. *Psychological Medicine*, 8, 219-233.
- Tarnopolsky, A., & Morton-Williams, J. (1980). *Aircraft Noise and Prevalence of Psychiatric Disorders, Research Report*. London, UK.
- Tarnopolsky, A., Watkins, G., & Hand, D. J. (1980). Aircraft noise and mental health: I. Prevalence of individual symptoms. *Psychological Medicine*, 10, 683-698.
- Tasali, E., Leproult, R., Ehrmann, D. A., & Van Cauter, E. (2008). Slow-wave sleep and the risk of type 2 diabetes in humans. *Proceedings of the National Academy of Sciences of the United States of America*, 105(3), 1044-1049. doi:10.1073/pnas.0706446105
- Taylor, S. E. (2010). The impact of early adversity on health. In *Handbook of behavioral medicine: Methods and applications* (pp. 559-570). New York, NY: Springer.
- Taylor, S. E., & Repetti, R. L. (1997). Health psychology: What is an unhealthy environment and how does it get under the skin? *Annual Review of Psychology*, 48, 411-447.
- Tennant, C. (2001). Work-related stress and depressive disorders. *Journal of Psychosomatic Research*, 51, 697-704.
- Thayer, J. F., & Sternberg, E. (2006). Beyond heart rate variability: Vagal regulation of allostatic systems. *Annals of the New York Academy of Sciences*, 1088, 361-372. doi:10.1196/annals.1366.014
- Therrien, F., Drapeau, V., Lupien, S. J., Beaulieu, S., Dore, J., Tremblay, A., & Richard, D. (2008). Awakening cortisol response in relation to psychosocial profiles and eating behaviors. *Physiology & Behavior*, 93(1-2), 282-288. doi:10.1016/j.physbeh.2007.08.019

- Thomas, J. R., & Jones, D. M. (1982). Individual differences in noise annoyance and the uncomfortable loudness level. *Journal of Sound and Vibration*, 82(2), 289-304.
- Thorn, L., Hucklebridge, F., Esgate, A., Evans, P., & Clow, A. (2004). The effect of dawn simulation on the cortisol response to awakening in healthy participants. *Psychoneuroendocrinology*, 29(7), 925-930. doi:10.1016/j.psyneuen.2003.08.005
- Thorn, L., Hucklebridge, F., Evans, P., & Clow, A. (2006). Suspected non-adherence and weekend versus week day differences in the awakening cortisol response. *Psychoneuroendocrinology*, 31(8), 1009-1018. doi:10.1016/j.psyneuen.2006.05.012
- Thorn, L., Hucklebridge, F., Evans, P., & Clow, A. (2009). The cortisol awakening response, seasonality, stress and arousal: A study of trait and state influences. *Psychoneuroendocrinology*, 34(3), 299-306. doi:10.1016/j.psyneuen.2008.11.005
- Topf, M. (1985). Noise-induced stress in hospital patients: Coping and nonauditory health outcomes. *Journal of Human Stress*, 1(3), 125-134.
- Topf, M. (1989). Sensitivity to noise, personality hardiness, and noise-induced stress in critical care nurses. *Environment and Behavior*, 21(6), 717-733. doi:10.1177/0013916589216004
- Topf, M. (1994). Theoretical considerations for research on environmental stress and health. *Journal of Nursing Scholarship*, 26(4), 289-293. doi:10.1111/j.1547-5069.1994.tb00336.x
- Topf, M., & Thompson, S. (2001). Interactive relationships between hospital patients' noise-induced stress and other stress with sleep. *Heart & Lung: The Journal of Acute and Critical Care*, 30(4), 237-243. doi:10.1067/mhl.2001.116592
- Ulrich, R. S., Simons, R. F., Losito, B. D., Fiorito, E., Miles, M. A., & Zelson, M. (1991). Stress recovery during exposure to natural and urban environments. *Journal of Environmental Psychology*, 11, 201-230.
- Ursin, H., Endresen, I. M., & Ursin, H. (1988). Psychological factors and self-reports of muscle pain. *European Journal of Applied Physiology*, 57, 282-290.
- Ursin, H., & Eriksen, H. R. (2004). The cognitive activation theory of stress. *Psychoneuroendocrinology*, 29(5), 567-592. doi:10.1016/s0306-4530(03)00091-x
- Ursin, H., & Eriksen, H. R. (2010). Cognitive activation theory of stress (CATS). *Neuroscience and Biobehavioral Reviews*, 34(6), 877-881. doi:10.1016/j.neubiorev.2009.03.001
- Vallet, M., Gagneux, J. M., Blanchet, V., Favre, B., & Labiale, G. (1983). Long term sleep disturbance due to traffic noise. *Journal of Sound and Vibration*, 90(2), 173-191.
- Vallet, M., Maurin, M., Page, M. A., Favre, B., & Pachiaudi, G. (1978). Annoyance from habituation to road traffic noise from urban expressways. *Journal of Sound and Vibration*, 60(3), 423-440.
- van den Berg, A. E., Maas, J., Verheij, R. A., & Groenewegen, P. P. (2010). Green space as a buffer between stressful life events and health. *Social Science & Medicine*, 70(8), 1203-1210. doi:10.1016/j.socscimed.2010.01.002
- Van Gerven, P. W. M., Vos, H., Van Boxtel, M. P., Janssen, S. A., & Miedema, H. M. (2009). Annoyance from environmental noise across the lifespan. *Journal of the Acoustical Society of America*, 126(1), 187-194. doi:10.1121/1.3147510
- van Kamp, I., & Davies, H. (2008). *Environmental noise and mental health: Five year review and future directions*. Paper presented at the meeting of the 9th International Congress on Noise as a Public Health Problem (ICBEN), Foxwoods, CT.
- van Kamp, I., Job, R. F. S., Hatfield, J., Haines, M., Stellato, R. K., & Stansfeld, S. A. (2004). The role of noise sensitivity in the noise-response relation: A comparison of

- three international airport studies. *Journal of the Acoustical Society of America*, 116(6), 3471-3479.
- van Kempen, E., & Babisch, W. (2012). The quantitative relationship between road traffic noise and hypertension: A meta-analysis. *Journal of Hypertension*. doi:10.1097/HJH.0b013e328352ac54
- van Kempen, E., Kruize, H., Boshuizen, H. C., Ameling, C. B., Staatsen, B. A. M., & Hollander, A. E. M. (2002). The association between noise exposure and blood pressure and ischemic heart disease: A meta-analysis. *Environmental Health Perspectives*, 110(3), 307-317.
- van Kempen, E., van Kamp, I., Fischer, P., Davies, H., Houthuijs, D., Stellato, R., ... Stansfeld, S. A. (2006). Noise exposure and children's blood pressure and heart rate: The RANCH project. *Occupational and Environmental Medicine*, 63(9), 632-639. doi:10.1136/oem.2006.026831
- van Kempen, E., van Kamp, I., Nilsson, M., Lammers, J., Emmen, H., Clark, C., & Stansfeld, S. (2010). The role of annoyance in the relation between transportation noise and children's health and cognition. *Journal of the Acoustical Society of America*, 128(5), 2817-2828. doi:10.1121/1.3483737
- van Kempen, E., Van Kamp, I., Stellato, R. K., Lopez Barrio, I., Haines, M. M., Nilsson, M. E., ... Stansfeld, S. A. (2009). Children's annoyance reactions to aircraft and road traffic noise. *Journal of the Acoustical Society of America*, 125(2), 895-904. doi:10.1121/1.3058635
- van Leeuwen, N., Kumsta, R., Entringer, S., de Kloet, E. R., Zitman, F. G., DeRijk, R. H., & Wüst, S. (2010). Functional mineralocorticoid receptor (MR) gene variation influences the cortisol awakening response after dexamethasone. *Psychoneuroendocrinology*, 35(3), 339-349. doi:10.1016/j.psyneuen.2009.07.006
- van Raaj, M. T. M., Dobbe, C. J. G., Timmerman, A., Schenk, E., Oortgiesen, M., & Wiegant, V. M. (1997). Hormonal status and the neuroendocrine response to novel heterotypic stressor involving subchronic noise exposure. *Neuroendocrinology*, 65(3), 200-209.
- van Santen, A., Vreeburg, S. A., Van der Does, A. J., Spinhoven, P., Zitman, F. G., & Penninx, B. W. (2011). Psychological traits and the cortisol awakening response: Results from the Netherlands Study of Depression and Anxiety. *Psychoneuroendocrinology*, 36(2), 240-248. doi:10.1016/j.psyneuen.2010.07.014
- Van Uum, S. H., Sauve, B., Fraser, L. A., Morley-Forster, P., Paul, T. L., & Koren, G. (2008). Elevated content of cortisol in hair of patients with severe chronic pain: A novel biomarker for stress. *Stress*, 11(6), 483-488. doi:10.1080/10253890801887388
- Vasan, R. S., Larson, M. G., Leip, E. P., Evans, J. C., O'Donnell, C. J., Kannel, W. B., & Levy, D. (2001). Impact of high-normal blood pressure on the risk of cardiovascular disease. *New England Journal of Medicine*, 345(18), 1291-1297.
- Velle, M. (1987). Sex differences in sensory functions. *Perspectives in Biology and Medicine*, 30, 490-522.
- Vreeburg, S. A., Zitman, F. G., van Pelt, J., DeRijk, R. H., Verhagen, J. C. M., van Dyck, R., ... Penninx, B. W. J. H. (2010). Salivary cortisol levels in persons with and without difference anxiety disorders. *Psychosomatic Medicine*, 72(4), 340-347. doi:10.1097/PSY.0b013e3283181d2f0c8
- Vythilingam, M., Gill, J. M., Luckenbaugh, D. A., Gold, P. W., Collin, C., Bonne, O., ... Charney, D. (2010). Low early morning plasma cortisol in posttraumatic stress

- disorder is associated with co-morbid depression but not with enhanced glucocorticoid feedback inhibition. *Psychoneuroendocrinology*, 35(3), 442-450. doi:10.1016/j.psyneuen.2009.08.006
- Wahbeh, H., Kishiyama, S. S., Zajdel, D., & Oken, B. S. (2008). Salivary cortisol awakening response in mild Alzheimer Disease, caregivers, and noncaregivers. *Alzheimer Disease and Associated Diseases*, 22(2), 181-183.
- Walker, S., O'Connor, D. B., Schaefer, A., Talbot, D., & Hendrickx, H. (2011). The cortisol awakening response: Associations with trait anxiety and stress reactivity. *Personality and Individual Differences*, 51(2), 123-127. doi:10.1016/j.paid.2011.03.026
- Wallenius, M. A. (2004). The interaction of noise stress and personal project stress on subjective health. *Journal of Environmental Psychology*, 24(2), 167-177. doi:10.1016/j.jenvp.2003.12.002
- Ware, J. E., & Sherbourne, C. D. (1992). The MOS 36-item shortform general health survey (SF-36): Conceptual framework and item selection. *Medical Care*, 30, 473-483.
- Watkins, G., Tarnopolsky, A., & Jenkins, L. M. (1981). Aircraft noise and mental health: II. Use of medicines and health care services. *Psychological Medicine*, 11, 155-168.
- Watson, D., Clark, C., & Carey, G. (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of Abnormal Psychology*, 97(3), 346-353.
- Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review*, 96(2), 234-254.
- Weekes, N. Y., Lewis, R. S., Goto, S. G., Garrison-Jakel, J., Patel, F., & Lupien, S. (2008). The effect of an environmental stressor on gender differences on the awakening cortisol response. *Psychoneuroendocrinology*, 33(6), 766-772. doi:10.1016/j.psyneuen.2008.03.003
- Weinmann, T., Ehrenstein, V., von Kries, R., Nowak, D., & Radon, K. (2011). Subjective and objective personal noise exposure and hypertension: An epidemiologic approach. *International Archives of Occupational and Environmental Health*, 85(4), 363-371. doi:10.1007/s00420-011-0679-7
- Weinstein, N. D. (1978). Individual differences in reactions to noise: A longitudinal study in a college dormitory. *Journal of Applied Psychology*, 63(4), 458-466.
- Weinstein, N. D. (1980). Individual differences in critical tendencies and noise annoyance. *Journal of Sound and Vibration*, 68(2), 241-248.
- Weinstein, N. D. (1982). Community noise problems: Evidence against adaptation. *Journal of Environmental Psychology*, 2, 87-97.
- Wessa, M., Rohleder, N., Kirschbaum, C., & Flor, H. (2006). Altered cortisol awakening response in posttraumatic stress disorder. *Psychoneuroendocrinology*, 31(2), 209-215. doi:10.1016/j.psyneuen.2005.06.010
- Westman, J. C., & Walters, J. R. (1981). Noise and stress: A comprehensive approach. *Environmental Health Perspectives*, 41, 291-309.
- Whitehead, D. L., Perkins-Porras, L., Strike, P. C., Magid, K., & Steptoe, A. (2007). Cortisol awakening response is elevated in acute coronary syndrome patients with type-D personality. *Journal of Psychosomatic Research*, 62(4), 419-425. doi:10.1016/j.jpsychores.2006.11.005

- WHOQOL Group. (1995). The World Health Organization quality of life assessment (WHOQOL): Position paper from the World Health Organization. *Social Science & Medicine*, 41(10), 1403-1409. doi:10.1016/0277-9536(95)00112-K
- WHOQOL Group. (1998). The World Health Organization quality of life assessment (WHOQOL): Development and general psychometric properties. *Social Science & Medicine*, 46, 1569-1585.
- Widen, S. E., Bohlin, M., & Johansson, I. (2011). Gender perspectives in psychometrics related to leisure time noise exposure and use of hearing protection. *Noise & Health*, 13(55), 407-414.
- Widen, S. E., Holmes, A. E., & Erlandsson, S. I. (2006). Reported hearing protection use in young adults from Sweden and the USA: Effects of attitude and gender. *International Journal of Audiology*, 45(5), 273-280. doi:10.1080/14992020500485676
- Wilhelm, I., Born, J., Kudielka, B. M., Schlotz, W., & Wüst, S. (2007). Is the cortisol awakening rise a response to awakening? *Psychoneuroendocrinology*, 32(4), 358-366. doi:10.1016/j.psyneuen.2007.01.008
- Wilkinson, R. T. (1984). Disturbance of sleep by noise: Individual differences. *Journal of Sound and Vibration*, 95, 55-63.
- Williams, E., Magid, K., & Steptoe, A. (2005). The impact of time of waking and concurrent subjective stress on the cortisol response to awakening. *Psychoneuroendocrinology*, 30(2), 139-148. doi:10.1016/j.psyneuen.2004.06.006
- Willich, S. N., Wegscheider, K., Stallmann, M., & Keil, T. (2006). Noise burden and the risk of myocardial infarction. *European Heart Journal*, 27(3), 276-282. doi:10.1093/eurheartj/ehi658
- Winneke, G., & N., M. (1992). Psychological response to sensory stimulation by environmental stressors: Trait or state? *Applied Psychology: An International Review*, 41(3), 257-267. doi:10.1111/j.1464-0597.1992.tb00704.x
- Witvliet, M. I., Kunst, A. E., Stronks, K., & Arah, O. A. (2012). Variations between world regions in individual health: A multilevel analysis of the role of socio-economic factors. *European Journal of Public Health*, 22(2), 284-289. doi:10.1093/eurpub/ckr001
- Wolfram, M., Bellingrath, S., & Kudielka, B. M. (2011). The cortisol awakening response (CAR) across the female menstrual cycle. *Psychoneuroendocrinology*, 36(6), 905-912. doi:10.1016/j.psyneuen.2010.12.006
- Wood, J. M., & Lilienfeld, S. O. (1999). The Rorschach Inkblot Test: A case of overstatement? *Assessment*, 6(4), 341-351. doi:10.1177/107319119900600405
- World Health Organization. (2009). *Night noise guidelines for Europe*. Copenhagen, Denmark: WHO Regional Office for Europe.
- World Health Organization. (2012). *The top 10 causes of death*. Retrieved 11 April, 2012, from <http://www.who.int/mediacentre/factsheets/fs310/en/index.html>
- Wright, C. E., Erblich, J., Valdimarsdottir, H. B., & Bovbjerg, D. H. (2007). Poor sleep the night before an experimental stressor predicts reduced NK cell mobilization and slowed recovery in healthy women. *Brain, Behavior, and Immunity*, 21(3), 358-363. doi:10.1016/j.bbi.2006.08.007
- Wright, C. E., & Steptoe, A. (2005). Subjective socioeconomic position, gender and cortisol responses to waking in an elderly population. *Psychoneuroendocrinology*, 30(6), 582-590. doi:10.1016/j.psyneuen.2005.01.007

- Wüst, S., Federenko, I., Hellhammer, D. H., & Kirschbaum, C. (2000a). Genetic factors, perceived chronic stress, and the free cortisol response to awakening. *Psychoneuroendocrinology*, 25, 707-720.
- Wüst, S., Wolf, J., Hellhammer, D. H., Federenko, I., Schommer, N., & Kirschbaum, C. (2000b). The cortisol awakening response - normal values and confounds. *Noise & Health*, 7, 79-88.
- Yablon, N. (2007). Echoes of the city: Spacing sound, sounding space, 1888-1916. *American Literary History*, 19(3), 629-660. doi:10.1093/alh/ajm026
- Yang, F., Bao, Z. Y., & Zhu, Z. J. (2011). An assessment of psychological noise reduction by landscape plants. *International Journal of Environmental Research and Public Health*, 8(4), 1032-1048. doi:10.3390/ijerph8041032
- Yehuda, R. (2006). Advances in understanding neuroendocrine alterations in PTSD and their therapeutic implications. *Annals of the New York Academy of Sciences*, 1071, 137-166. doi:10.1196/annals.1364.012
- Yoshida, T., Osada, Y., Kawaguchi, T., Hoshiyama, Y., Yoshida, K., & Yamamoto, K. (1997). Effects of road traffic noise on inhabitants of Tokyo. *Journal of Sound and Vibration*, 205(4), 517-522.
- Zaharna, M., & Guilleminault, C. (2010). Sleep, noise and health: Review. *Noise & Health*, 12(47), 64-69.
- Zimmer, K., & Ellermeier, W. (1998). Konstruktion und evaluation eines fragebogens zur erfassung der individuellen lärmempfindlichkeit. *Diagnostica*, 44, 11-20.
- Zimmer, K., & Ellermeier, W. (1999). Psychometric properties of four measures of noise sensitivity: A comparison. *Journal of Environmental Psychology*, 19, 295-302.
- Zoccola, P. M., Dickerson, S. S., & Yim, I. S. (2011). Trait and state perseverative cognition and the cortisol awakening response. *Psychoneuroendocrinology*, 36(4), 592-595. doi:10.1016/j.psyneuen.2010.10.004

Glossary

<u>Term</u>	<u>Definition</u>
<i>Allostatic Load</i>	The long-term physiological or health damage to the body as a result of a sustained exposure to stressors or a continuous stress response (see p. 14).
<i>Allostasis</i>	The process by which the body attempts to maintain homeostasis in face of a stressor. It refers to short-term physiological changes (see p. 14).
<i>Chronic Stress</i>	A broad term to describe stress that is not acute. Chronic stress may occur due to situational factors (e.g., caregiving for a loved one, environmental noise) or could be brought on by negative psychological traits (see p. 12).
<i>Cortisol</i>	Stress hormone released from the adrenal cortex as a primary output of the HPA axis (see p. 10).
<i>Cortisol Awakening Response</i>	The pattern of morning cortisol output whereby cortisol levels increase 50-100% within the first 30-45 minutes of awakening. This response can be dysregulated as a result of chronic stress or health problems (see p. 18).
<i>Diminished Health</i>	Broad term used to describe increased health problems (e.g., greater health complaints, greater incidence of disease). Typically used in reference to noise sensitivity studies indicating a relationship between noise sensitivity and poor

health (e.g., Kelly, 1986; Fyhri & Klæboe, 2009; Nivision & Endresen, 1993).

Dysregulation

The regular functioning of the physiological process has been altered. For example, dysregulation of the HPA axis has been implicated in health and psychological problems (see p. 12).

Environmental Noise

Broad concept that often refers to transportation-induced noise (e.g., aircraft, railway, road traffic; see p. 33).

Occupational noise (noise occurring in the workplace) is usually considered separately from environmental noise (Berglund et al., 1999).

Hypothalamic-Pituitary-Adrenal Axis

The major stress axis of the neuroendocrine system. The output of this axis is cortisol in humans (see p. 10).

Negative Affect

A negative emotional state (e.g., anger, nervousness, guilt).

Negative Affectivity

The tendency to experience negative emotions (see p. 90).

Neuroticism

One of the big five personality dimensions; refers to the tendency to experience anxiety and negative affect (see p. 89). The Neuroticism Scale of the Big Five Inventory was used to assess negative affectivity in this thesis (see p. 108).

Noise Annoyance

Negative reaction to noise when it interferes with feelings, thoughts or actions (see p. 41). Noise annoyance increases

with noise exposure at the population level.

Noise Sensitivity

A personal trait that increases an individual's vulnerability to noise and other stressors; regarded as independent of noise exposure (see p. 64).

Psychological Stress

The perception of stress; measured in this thesis with the 10-item Perceived Stress Scale (Cohen & Williamson, 1988; see p. 51)

Stress

Broadly refers the psychological and physiological reaction to a stressor. Negative emotions (e.g., fear) and physiological changes (e.g., activation of the HPA and SAM axes) occur (see p.7).

Stressor

An external agent that causes a stress response (e.g., noise; see p. 8).

Sympathetic-Adrenal-Medullary Axis

The autonomic nervous system stress axis that initiates the secretion of adrenaline and noradrenaline (see p. 9).

Appendices

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Appendix A: AUTECH Approval Letter for Study 1



MEMORANDUM

Auckland University of Technology Ethics Committee (AUTECH)

To: Daniel Shepherd
 From: **Dr Rosemary Godbold and Madeline Banda** Executive Secretary, AUTECH
 Date: 16 May 2011
 Subject: Ethics Application Number 10/271 **Noise, health and environmental perceptions among Auckland residents.**

Dear Daniel

Thank you for providing written evidence as requested. We are pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTECH) at their meeting on 8 November 2010 and that on 18 February 2011, we approved your ethics application. This delegated approval is made in accordance with section 5.3.2.3 of AUTECH's *Applying for Ethics Approval: Guidelines and Procedures* and is subject to endorsement at AUTECH's meeting on 13 June 2011.

Your ethics application is approved for a period of three years until 18 February 2014.

We advise that as part of the ethics approval process, you are required to submit the following to AUTECH:

- A brief annual progress report using form EA2, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 18 February 2014;
- A brief report on the status of the project using form EA3, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. This report is to be submitted either when the approval expires on 18 February 2014 or on completion of the project, whichever comes sooner;

It is a condition of approval that AUTECH is notified of any adverse events or if the research does not commence. AUTECH approval needs to be sought for any alteration to the research, including any alteration of or addition to any documents that are provided to participants. You are reminded that, as applicant, you are responsible for ensuring that research undertaken under this approval occurs within the parameters outlined in the approved application.

Please note that AUTECH grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

When communicating with us about this application, we ask that you use the application number and study title to enable us to provide you with prompt service. Should you have any further

enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at ethics@aut.ac.nz or by telephone on 921 9999 at extension 8860.

On behalf of AUTECH and ourselves, we wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Dr Rosemary Godbold and Madeline Banda
Executive Secretary
Auckland University of Technology Ethics Committee

Cc: Erin Hill Erin.hill@aut.ac.nz

Participant Information Sheet



Date Information Sheet Produced:

20/10/2010

Project Title

Noise, Health and Environmental Perceptions among Auckland Residents

An Invitation

My name is Erin Hill, and I am currently a PhD student at the Auckland University of Technology (AUT) studying the relationship among environmental stressors, personality and health. I would like to take this opportunity to invite you to participate in my Auckland-based community health study.

What is the purpose of this research?

The objective of this study is to investigate the relationship among noise exposure, environmental perceptions, personality and health and well-being. It is hoped that the findings of the study will provide information about the influence a person's environment and personality can have on their health and well-being.

How was I identified and why am I being invited to participate in this research?

You are invited to participate in this research because you are currently residing in a relatively noisy area of Auckland or a relatively quiet area. There are no specific criteria applied for participating in this research other than potential participants should be residing in the selected household and aged 18 years or older.

What will happen in this research?

If you agree to participate, this will involve completing a questionnaire asking you about health complaints in the last month, environmental noise, day-to-day hassles, and individual characteristics. It should take approximately 5-10 minutes to complete.

What are the discomforts and risks?

Some of the questions in the survey are personal in nature, but please be reassured that all responses will remain anonymous and will be used for research purposes only.

What are the benefits?

Because this research focuses on two very important issues in our modern world – a form of environmental pollution (i.e., noise) and health, it is hoped that this research will provide significant information that could contribute to policy development and guide practice in the area.

How will my privacy be protected?

The questionnaire is anonymous and you are asked not to provide any distinguishing/identifying information.

What are the costs of participating in this research?

There are no costs to you other than your time. The questionnaire should take 5-10 minutes to complete.

What opportunity do I have to consider this invitation?

There is no obligation to complete this questionnaire. It is completely voluntary. If you decide to participate, please complete the questionnaire and return it as soon as possible in the pre-paid envelope provided to you.

How do I agree to participate in this research?

By completing the questionnaire and returning it to the researcher in the pre-paid envelope, you are giving your consent to participate in this research.

Will I receive feedback on the results of this research?

If you would like feedback on this project, it can be provided upon request.

What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, *Dr. Daniel Shepherd*, daniel.shepherd@aut.ac.nz, 921 9999 ext 7238.

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTECH, Madeline Banda, madeline.banda@aut.ac.nz, 921 9999 ext 8044.

Whom do I contact for further information about this research?***Researcher Contact Details:***

Erin Hill, PhD Student – e-mail: erin.hill@aut.ac.nz

Project Supervisor Contact Details:

Dr. Daniel Shepherd (Project Supervisor) – e-mail: daniel.shepherd@aut.ac.nz ph: 921 9999 ext 7238

Approved by the Auckland University of Technology Ethics Committee on 18 February, 2011, AUTECH Reference number 10/271.

Used in Study 1

Appendix C: 3-Item Noise Sensitivity Scale

Noise Sensitivity Scale (3-NS; Amann et al., 2007)

For the following questions **please circle** the response that best describes you.

	Completely Agree				Completely Disagree
1. I think I am less noise sensitive than others.	1	2	3	4	5
2. I think I can cope with noise better than others.	1	2	3	4	5
3. I often feel completely overwhelmed by noise.	1	2	3	4	5

Used in Study 1 and Study 2

Appendix D: Subjective Health Complaints Inventory

Subjective Health Complaints (SHCI; Ursin et al., 1988; Eriksen et al., 1999)

In the next section, you will find some ordinary health problems and complaints. We want you to look at each and every one of them and report **to what extent you have been affected** during last month, and the **number of days** you have been suffering from the problem.

Example: If you feel you have been suffering *some* with the cold/flu last month, and the duration was *7 days*, this is recorded the following way:

Circle the suitable number.

		Not at all	A little	Some	Serious	Number of days
1.	Cold/flu.....	0	1	2	3	7

It is important that you record both to what extent you have been suffering from the problem and the approximate number of days it last month.

		Not at all	A little	Some	Seriously	Number of days (e.g. 2, 7)
1.	Cold/flu.....	0	1	2	3	
2.	Cough/bronchitis.....	0	1	2	3	
3.	Asthma.....	0	1	2	3	
4.	Headache.....	0	1	2	3	
5.	Neck pain.....	0	1	2	3	
6.	Pain – upper part of back..	0	1	2	3	
7.	Pain – lower part of back..	0	1	2	3	
8.	Pain in arms.....	0	1	2	3	
9.	Pain in shoulders.....	0	1	2	3	
10.	Migraine.....	0	1	2	3	
11.	Extra heart beats.....	0	1	2	3	
12.	Chest pain.....	0	1	2	3	
13.	Breathing difficulties.....	0	1	2	3	
14.	Pain in feet during exercise	0	1	2	3	
15.	Heart-burn.....	0	1	2	3	
16.	Stomach discomfort.....	0	1	2	3	
17.	Gastritis.....	0	1	2	3	
18.	Stomach pains.....	0	1	2	3	
19.	Gas.....	0	1	2	3	
20.	Diarrhoea.....	0	1	2	3	
21.	Constipation.....	0	1	2	3	
22.	Eczema.....	0	1	2	3	
23.	Allergic skin problems.....	0	1	2	3	
24.	“Flushes”/Heat Sensations..	0	1	2	3	
25.	Sleep Problems.....	0	1	2	3	
26.	Tiredness.....	0	1	2	3	
27.	Dizziness.....	0	1	2	3	
28.	Anxiety.....	0	1	2	3	
29.	Sad/Depressed.....	0	1	2	3	

Used in Study 1 and Study 2

Appendix E: Perceived Stress Scale

Perceived Stress Scale (PSS; Cohen & Williamson, 1988)

For each question, please circle the number that corresponds to how often you felt or thought this way ***in the last month.***

In the last month, how often have you...

	Never	Almost never	Some- times	Fairly often	Very often
1. Been upset because of something that happened unexpectedly?	0	1	2	3	4
2. Felt that you were unable to control the important things in your life?	0	1	2	3	4
3. Felt nervous or “stressed”?	0	1	2	3	4
4. Felt confident about your ability to handle your personal problems?	0	1	2	3	4
5. Felt that things were going your way?	0	1	2	3	4
6. Found that you could not cope with all the things that you had to do?	0	1	2	3	4
7. Been able to control irritations in your life?	0	1	2	3	4
8. Felt that you were on top of things?	0	1	2	3	4
9. Been angered because of things that were outside of your control?	0	1	2	3	4
10. Felt difficulties were piling up so high that you could not overcome them?	0	1	2	3	4

Used in Study 1 and Study 2

Appendix F: Big Five Inventory – Neuroticism Scale

Big Five Inventory – Neuroticism Scale (John et al., 1991)

For each of the characteristics listed below, **please circle** how descriptive each characteristic is of you using the scale from 1 to 5.

I see myself as someone who...

	<i>Strongly disagree 1</i>	<i>Disagree a little 2</i>	<i>Neither agree or disagree 3</i>	<i>Agree a little 4</i>	<i>Strongly agree 5</i>
1. Is depressed...	1	2	3	4	5
2. Is relaxed, handles stress well...	1	2	3	4	5
3. Can be tense...	1	2	3	4	5
4. Worries a lot...	1	2	3	4	5
5. Is emotionally stable, not easily upset...	1	2	3	4	5
6. Can be moody...	1	2	3	4	5
7. Remains calm in tense situations...	1	2	3	4	5
8. Gets nervous easily	1	2	3	4	5

Used in Study 1

Appendix G: Noise Perception and Residence Questions

1. How **noisy** is your home environment? (e.g., children playing, road traffic noise that you can hear inside the house, dogs barking)

Not at all noisy 1	Not especially noisy 2	Somewhat noisy 3	Quite noisy 4	Very noisy 5
--------------------------	------------------------------	------------------------	------------------	-----------------

2. How much are you able to control the noise in your home environment? (e.g., closing the windows to lessen road traffic noise; children have a playroom that lessens noise in quieter parts of the house, etc.)

No control 1	Very little 2	Some control 3	A lot of control 4
-----------------	------------------	-------------------	-----------------------

3. How **annoyed** are you by the noise in your home environment?

Not at all annoyed 1	Not especially annoyed 2	Somewhat annoyed 3	Quite annoyed 4	Very annoyed 5
----------------------------	--------------------------------	--------------------------	--------------------	-------------------

4. How **noisy** is your work environment? (e.g., industrial noise, classroom/office noise, office building situated by a motorway or road, etc.)

Not at all noisy 1	Not especially noisy 2	Somewhat noisy 3	Quite noisy 4	Very noisy 5
--------------------------	------------------------------	------------------------	------------------	-----------------

5. How much are you able to control the noise in your work environment? (e.g., closing your office door, moving to work in a quieter room, etc.)

No control 1	Very little 2	Some control 3	A lot of control 4
-----------------	------------------	-------------------	-----------------------

6. How **annoyed** are you by the noise in your work environment?

Not at all annoyed 1	Not especially annoyed 2	Somewhat annoyed 3	Quite annoyed 4	Very annoyed 5
----------------------------	--------------------------------	--------------------------	--------------------	-------------------

7. How long have you lived in your current residence? _____ years

8. Approximately how many hours per day do you spend in your home (including sleeping hours)? _____

9. Approximately how many hours per day do you spend at your workplace?

10. How long have you been working at your current workplace? _____ years

Used in Study 1

Appendix H: Demographic and Lifestyle Questions for Study 1

1. Are you (please tick): ☐ Male ☐ Female
2. What is your age? _____
3. Which ethnic group do you belong to? Tick the option or options that most apply to you.

☐ New Zealand European	☐ Māori	☐ Samoan
☐ Cook Island Māori	☐ Tongan	☐ Niuean
☐ Chinese	☐ Indian	☐ European
☐ North American (Canada, US)	☐ Middle Eastern	☐ African
☐ Australian	☐ Central or South American	☐ Asian
☐ Other, please state: _____		
4. What is the highest level of education you have completed?

☐ Secondary School	☐ Technical Institute	☐ University Degree
---	--	--
5. What is your current employment status?

☐ Full-time work	☐ Part-time work	☐ Retired	☐ Student	☐ Unemployed
☐ On leave or sick-leave	☐ Own household work	☐ Other_____		
6. Do you have hypertension (high blood pressure)? ☐ Yes ☐ No
7. What is your current smoking status?

☐ Non-smoker	☐ Ex-smoker	☐ Occasional smoker	☐ Daily smoker
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Used in Study 2

Appendix I: Demographics and Sleep-related Questions for Study 2

1. Are you (please tick): ☐ Male ☐ Female
2. What is your age? _____
3. Which ethnic group do you belong to? Tick the option or options that most apply to you.

☐ New Zealand European	☐ Māori	☐ Samoan
☐ Cook Island Māori	☐ Tongan	☐ Niuean
☐ Chinese	☐ Indian	☐ European
☐ North American (Canada, US)	☐ Middle Eastern	☐ African
☐ Australian	☐ Central or South American	☐ Asian
☐ Other, please state: _____		
4. What is the highest level of education you have completed?

☐ Secondary school	☐ Technical College	☐ University Degree
---	--	--
5. What is your current employment status?

☐ Full-time work	☐ Part-time work	☐ Retired	☐ Student
☐ Unemployed	☐ On leave or sick-leave	☐ Own household work	
☐ Other: _____			
6. Do you have hypertension (high blood pressure)? ☐ Yes ☐ No
7. Do you have cardiovascular disease (heart disease)? ☐ Yes ☐ No

Saliva Collection Day Information
(please complete when you collect your samples)
 Collection of saliva is to be done on a weekday

8. Date (e.g., Monday, 2 June): _____
9. Time of Awakening (e.g., 6:40am): _____
10. Number of hours slept (e.g., 6.5 hours): _____
11. Sleep quality during the night (please tick the appropriate box)

☐ well	☐ fairly well	☐ fairly poorly	☐ poorly
-------------------------------	--------------------------------------	--	---------------------------------
12. Did you use an alarm to wake you up? ☐ Yes ☐ No

Used in Study 2

Appendix J: Noise Sensitivity Questionnaire (NoiSeQ)

Schütte et al. (2007)

In the following questionnaire your opinion is asked concerning a variety of sounds. Please try to imagine the situation presented in each statement, and indicate to which extent you agree or disagree with it. It is your personal assessment of the topics presented here that is of interest, so there is no right or wrong answer, only your opinion. If you are unsure as to which option to mark, please choose that option which comes closest in reflecting your opinion.

		Strongly disagree				Strongly agree
1.	I find it hard to relax in a noisy environment.	1	2	3	4	5
2.	I need peace and quiet to do difficult work.	1	2	3	4	5
3.	For a quiet place to live, I would accept other disadvantages.	1	2	3	4	5
4.	I am very sensitive to neighbourhood noise.	1	2	3	4	5
5.	I find it hard to communicate while it is noisy.	1	2	3	4	5
6.	I have no problems to do routine work in a noisy environment.	1	2	3	4	5
7.	I become very agitated if I can hear someone talking when I am trying to fall asleep.	1	2	3	4	5
8.	When I am absorbed in conversation I do not notice if it is noisy around me.	1	2	3	4	5
9.	I can fall asleep even when it is noisy.	1	2	3	4	5
10.	My performance is much worse in noisy places.	1	2	3	4	5
11.	Listening to loud music helps me relax after work.	1	2	3	4	5
12.	In a restaurant I cannot concentrate well on my conversation when people are taking loudly at other tables.	1	2	3	4	5
13.	When I am at home, I become accustomed to noise quickly.	1	2	3	4	5
14.	When people around are noisy I don't get on with my work.	1	2	3	4	5
15.	I need an absolutely quiet environment to get a good night's sleep.	1	2	3	4	5

16.	Even the slightest noise can prevent me from falling asleep.	1	2	3	4	5
17.	I need quiet surrounds to be able to work on new tasks.	1	2	3	4	5
18.	It would not bother me to live on a noisy street.	1	2	3	4	5
19.	If I'm dancing I don't mind how loud the music is.	1	2	3	4	5
20.	If my workplace was noisy I would try to find a way for me to change this.	1	2	3	4	5
21.	I find it hard to follow a conversation when the radio is playing.	1	2	3	4	5
22.	I think music interferes with conversations.	1	2	3	4	5
23.	In the cinema I am annoyed by other people whispering and by rustling paper.	1	2	3	4	5
24.	When other people's children are noisy I would prefer that they not play in front of my house.	1	2	3	4	5
25.	On weekends I prefer quiet surroundings.	1	2	3	4	5
26.	I do not feel well-rested if there has been a lot of noise the night before.	1	2	3	4	5
27.	The sound of thunder does not usually wake me up.	1	2	3	4	5
28.	Loud music in a restaurant makes me stop my conversation.	1	2	3	4	5
29.	I can do complicated work even while background music is playing.	1	2	3	4	5
30.	I wake up at the slightest noise.	1	2	3	4	5
31.	I avoid leisure activities that are loud.	1	2	3	4	5
32.	I don't like noisy activities in my residential area.	1	2	3	4	5
33.	Noise from neighbours can be extremely disturbing.	1	2	3	4	5
34.	When I am at home I find it uncomfortable if the radio or TV is left on in the background.	1	2	3	4	5
35.	High noise levels make it hard for me to concentrate on my conversation.	1	2	3	4	5

Used in Study 2

Appendix K: Pittsburgh Sleep Quality Index (PSQI)

(Buysse et al., 1989)

Instructions:

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?

BED TIME _____

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

NUMBER OF MINUTES _____

3. During the past month, what time have you usually gotten up in the morning?

GETTING UP TIME _____

4. During the past month, how many hours of actual sleep did you get at night (This may be different than the number of hours you spent in bed.)

HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you....

- a) Cannot get to sleep within 30 minutes

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	-------------------------------	-------------------------------------

- b) Wake up in the middle of the night or early morning

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	-------------------------------	-------------------------------------

c) Have to get up to use the bathroom

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

d) Cannot breathe comfortably

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

e) Cough or snore loudly

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

f) Feel too cold

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

g) Feel too hot

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

h) Had bad dreams

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

i) Have pain

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

Other reason(s), please describe

6. During the past month, how would you rate your sleep quality overall?

Very Good _____ Fairly Good _____ Fairly Bad _____ Very Bad _____

7. During the past month, how often have you taken medicine to help you sleep (prescribed or “over the counter”)?

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	-------------------------------	--

8. During the past month, how often have you had trouble staying awake while driving, eating meals or engaging in social activity?

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	----------------------------------	--

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

Not problem at all _____	Only a very slight problem _____	Somewhat of a problem _____	A very big problem _____
-----------------------------	--	--------------------------------	--------------------------------

10. Do you have a bed partner or roommate?

No bed partner or roommate _____	Partner/ roommate in other room _____	Partner in same room, but not same bed _____	Partner in same bed _____
-------------------------------------	---	--	------------------------------

If you have a roommate or bed partner, ask him/her how often in the past month you have had...

a) Loud snoring

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	----------------------------------	--

b) Long pauses between breaths while asleep

Not during the past month _____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
------------------------------------	--------------------------------	----------------------------------	--

c) Leg twitching or jerking while you sleep

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

d) Episodes of disorientation or confusion during sleep

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

e) Other restlessness while you sleep, please describe

Not during the past month_____	Less than once a week _____	Once or twice a week _____	Three or more times a week _____
-----------------------------------	--------------------------------	----------------------------------	--

Appendix L: Data Screening and Preliminary Analyses for Study 1

Treatment of Missing Data and Missing Value Analyses

For the 3-NS, 14 cases had missing data, and therefore noise sensitivity scores were not calculated for these participants. That is, because of the short length of the scale used in the present study (3 items), MVA was not conducted; any participant with a missing item on the scale would be missing at least 33% of the data, beyond the 30% cut-off for the appropriate use of EM – the method used to estimate missing values in this thesis (Little & Rubin, 1987; Newman, 2003; Schafer & Graham, 2002). This approach uses the available data and parameter estimates to estimate values for the missing cases.

The scoring system for the Subjective Health Complaints Inventory (SHCI; Eriksen et al., 1999) accounts for missing data in the calculation of the total severity rating and therefore MVA was applied to the scale. However, 14 participants did not complete the scale and therefore no scores were estimated. The sleep problems variable and the depression complaints variable (single items from the SHCI) each had 14 missing values. The anxiety complaints variable (single item from the SHCI) had 16 missing values. No missing values were estimated for the sleep problems, depression and anxiety complaints variables as these variables were assessed with a single item.

For the PSS, 14 cases were removed (30% or more of the scale missing), leaving 1087 cases for the MVA. Of the 1087 cases, 18 had missing data on less than 30% of the scale. The MVA revealed that the missing values of the PSS were not ‘missing completely at random’ ($\chi^2(64) = 142.85, p = .003$). Ideally, missing data would be ‘missing completely at random’ (as indicated by a non-significant Little’s MCAR test), that is to say not missing in a systematic way (Tabachnick & Fidell, 2007).

Therefore, to further investigate the missing values of the PSS, t-tests were conducted to compare participants with missing data to participants without missing data. Participants were not significantly different across any of the primary independent and dependent variables of the study – noise sensitivity ($U = 11,309.5$, $p = .335$), health complaints ($U = 16,057.0$, $p = .074$), anxiety complaints ($U = 14,388.0$, $p = .402$) and depression complaints ($U = 14,548.0$, $p = .355$). It was therefore assumed that the data was ‘missing at random’, and the Expectation Maximisation (EM) algorithm was applied to estimate the missing data on the remaining cases of the PSS in this study (18 cases).

In cleaning the Neuroticism Scale (BFI), 28 cases were removed because of missing data on more than 30% of the scale. Another 25 cases had missing data on less than 30% of the scale. The MVA for the remaining cases indicated that the values were missing completely at random ($\chi^2 (64) = 66.79$, $p = .381$), and therefore again, EM was used to estimate missing values for the scale. Missing values were not estimated for age (27 missing values) or the noise perception items: noise at home (4 missing values), control over noise at home (2 missing values), noise annoyance at home (3 missing values), noise at work (2 missing values), control over noise at work (1 missing value), noise annoyance at work (1 missing value).

Normality

Normality was assessed using histograms, skewness ratios, kurtosis ratios and a visual assessment of Q-Q plots for the following variables: noise sensitivity (3-NS), subjective health complaints (SHCI), anxiety complaints (SHCI single item severity rating), depression complaints (SHCI single item severity rating), sleep problems (SHCI single item severity rating), perceived stress (PSS), neuroticism (BFI neuroticism scale), age, noise at home (5-point Likert scale), noise annoyance at home (5-point Likert scale), noise

control at home (5-point Likert scale), noise at work (5-point Likert scale), noise annoyance at work (5-point Likert scale), and noise control at work (5-point Likert scale). Skewness ratios (skewness statistic/standard error of skewness statistic) and kurtosis ratios (kurtosis statistic/standard error of kurtosis statistic) were calculated for each of the variables to further explore the shape of the distribution (see Table L.1., p. 301). A value greater than ± 3.0 was considered an indicator of skewness or kurtosis of the data (Tabachnick & Fidell, 2007). Histograms for each of the variables are presented in Figure L.1. to Figure L.14. (p. 271-284). The data of the following variables significantly deviated from a normal distribution: subjective health complaints, sleep problems, anxiety complaints, depression complaints, noise at home, noise annoyance at home, noise at work, noise control at work, and noise annoyance at work. Non-parametric statistics were therefore applied when dealing with these variables.

Table L.1. *Skewness and Kurtosis Ratios for Study 1 Variables*

Variable	Skewness Ratio	Kurtosis Ratio
Noise Sensitivity (3-NS)	1.06	-1.26
Subjective Health Complaints	23.68	46.90
Anxiety Complaints	15.76	4.12
Depression Complaints	14.49	3.26
Sleep Problems	9.73	-3.89
Perceived Stress	2.55	-.70
Neuroticism	2.57	-1.21
Age	2.41	-4.07
Noise at Home	2.92	-4.22
Noise Annoyance at Home	7.68	1.13
Noise Control at Home	.44	-1.98
Noise at Work	5.24	-3.52
Noise Annoyance at Work	7.60	2.13
Noise Control at Work	2.37	-4.22

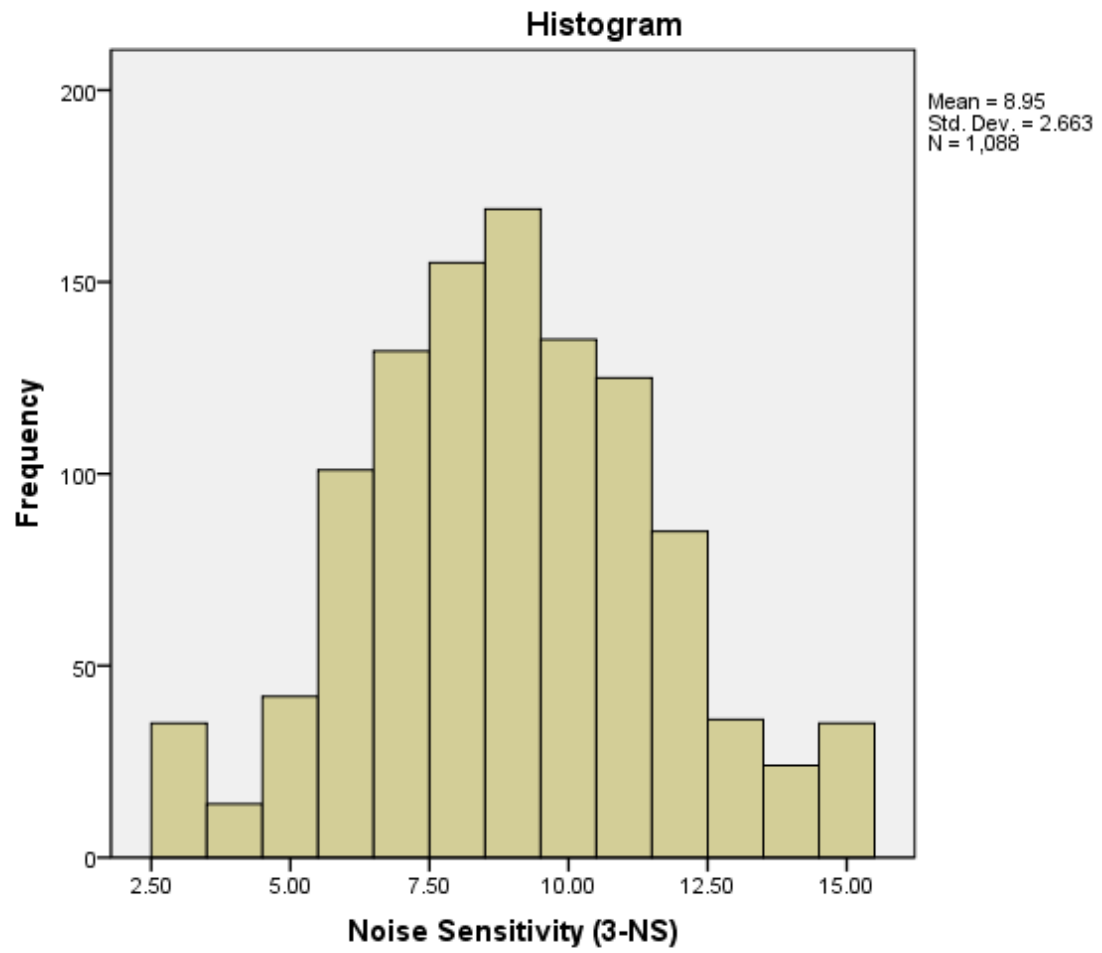


Figure L.1. Histogram of 3-NS scores.

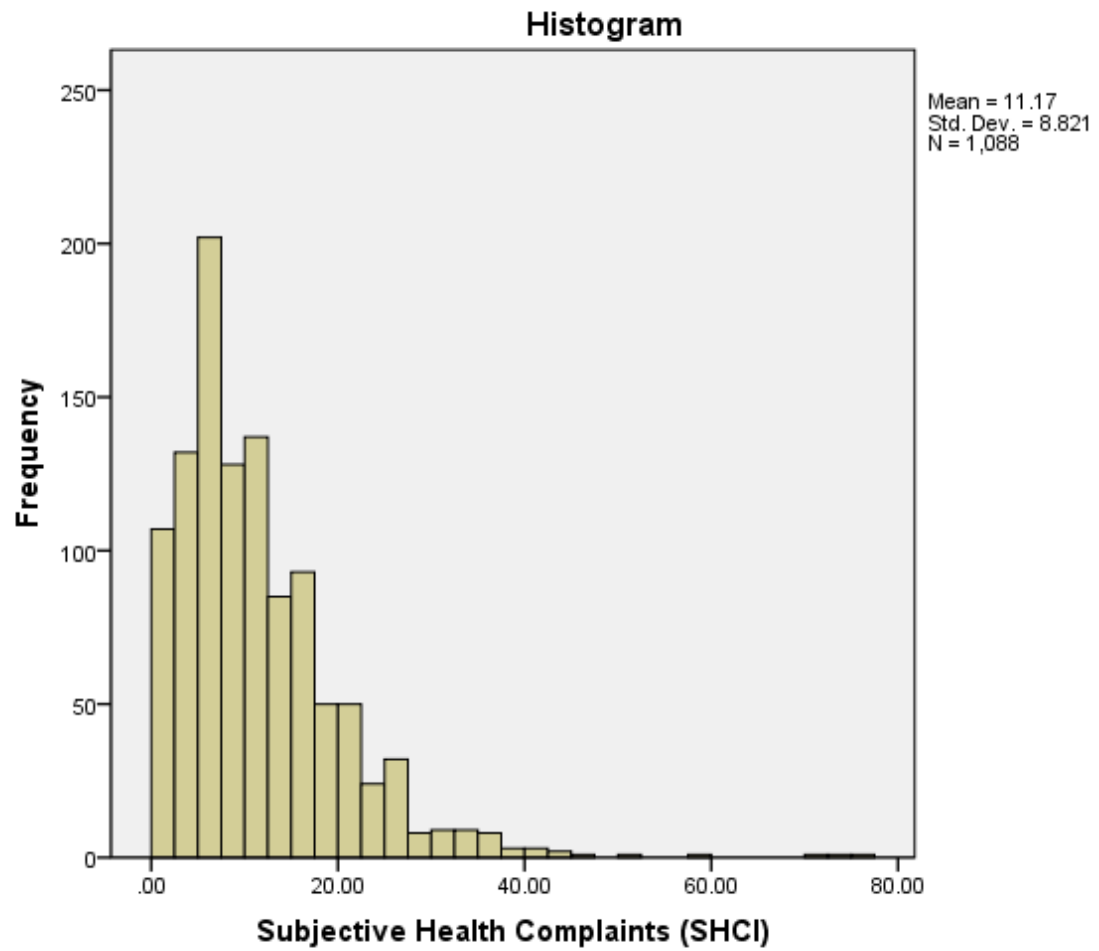


Figure L.2. Histogram of SHCI composite scores (Study 1).

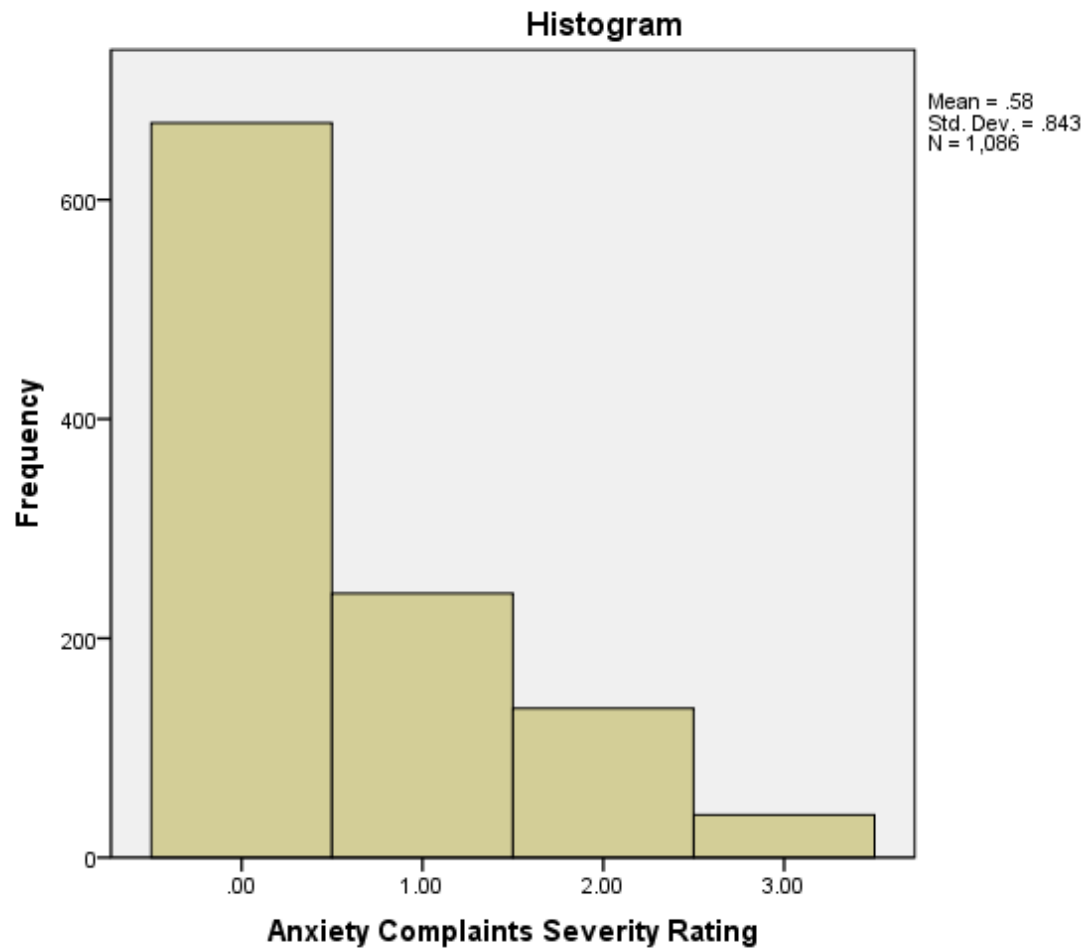


Figure L.3. Histogram of anxiety complaints severity ratings.

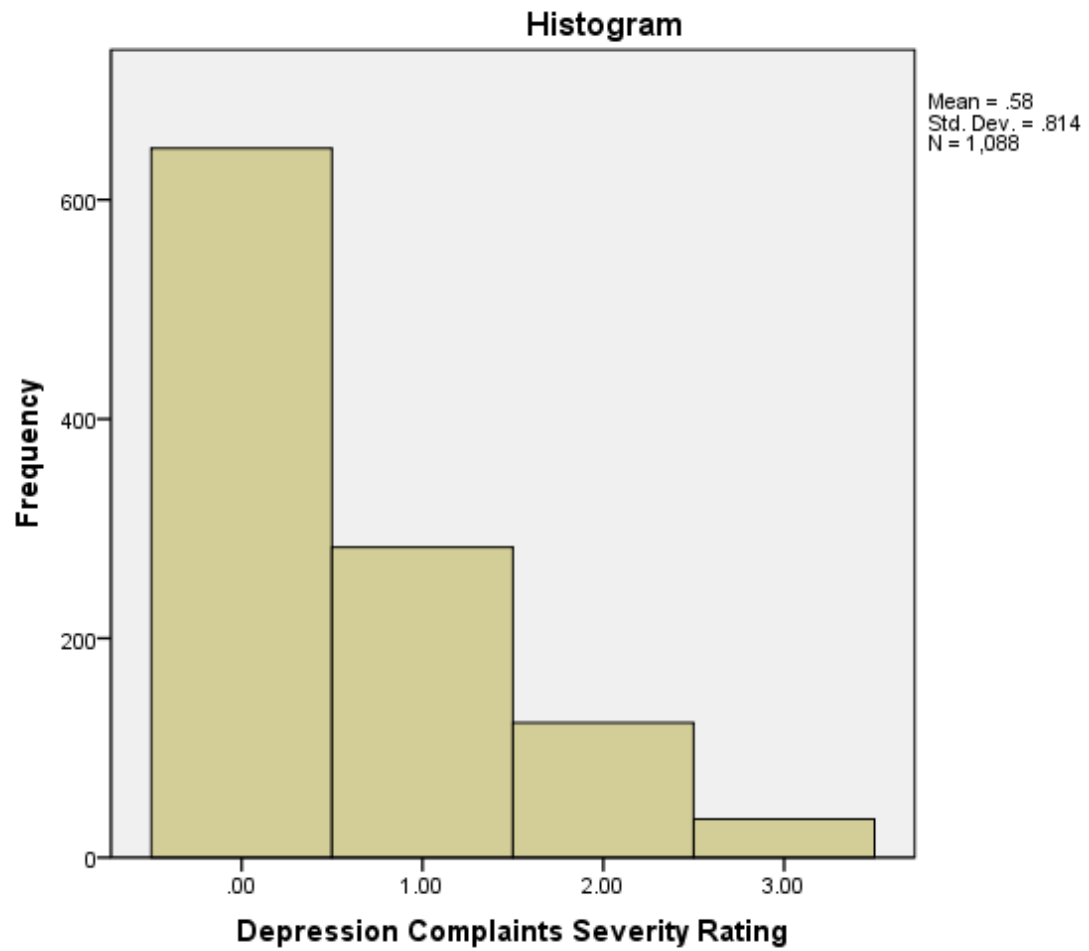


Figure L.4. Histogram of depression complaints severity ratings.

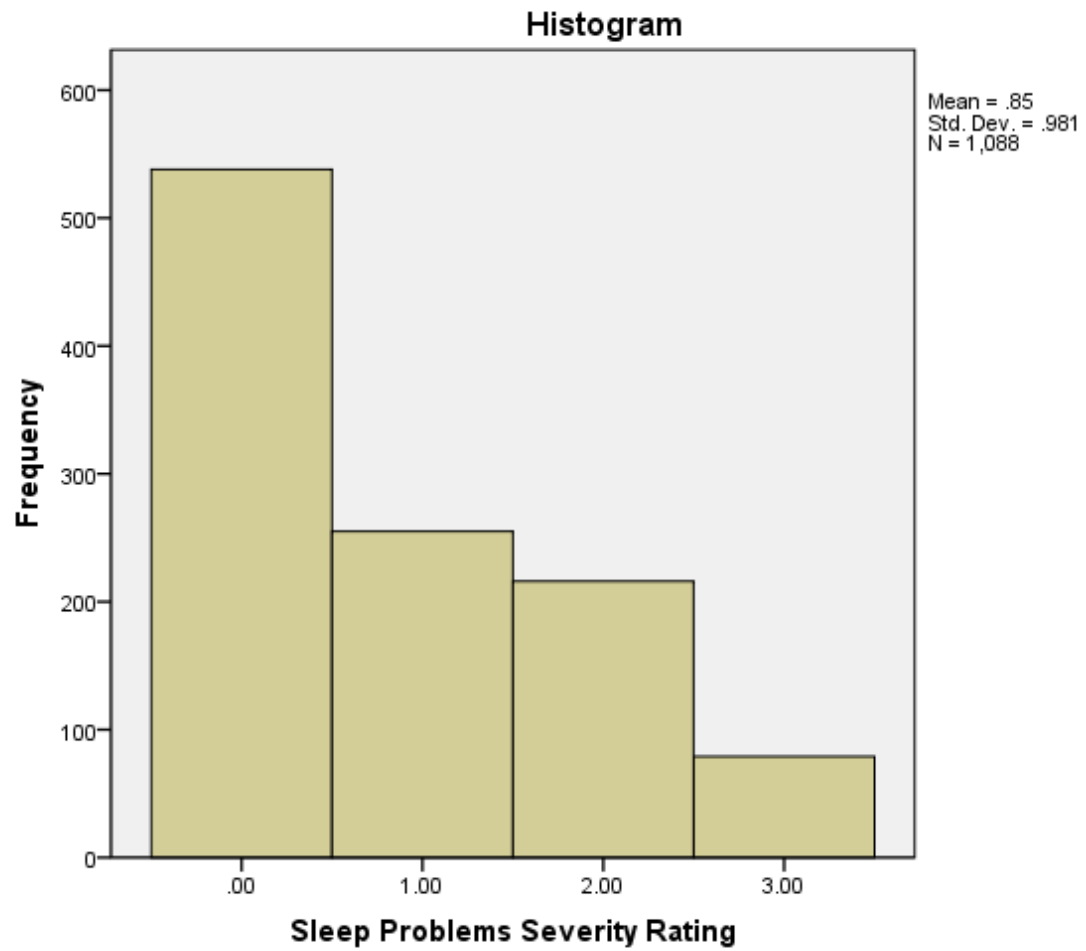


Figure L.5. Histogram of sleep problems severity ratings.

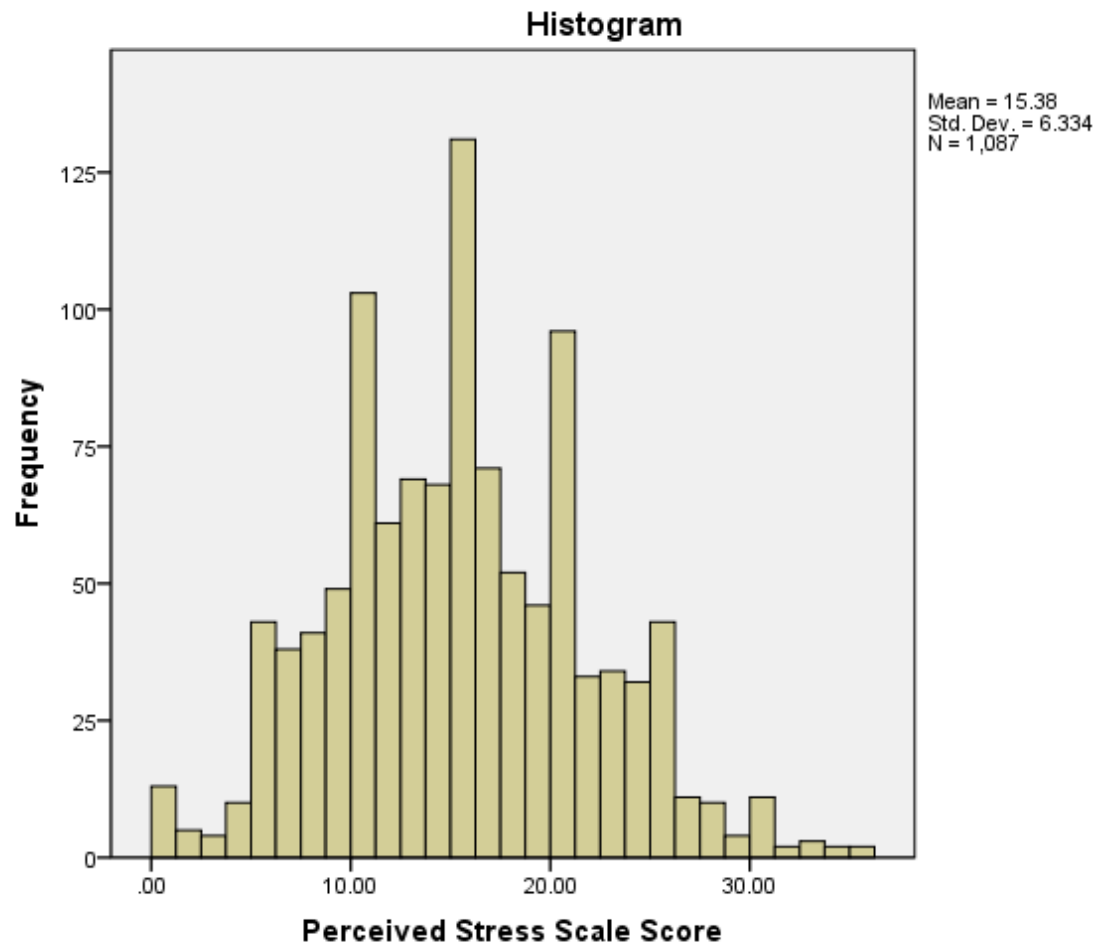


Figure L.6. Histogram of the PSS scores (Study 1).

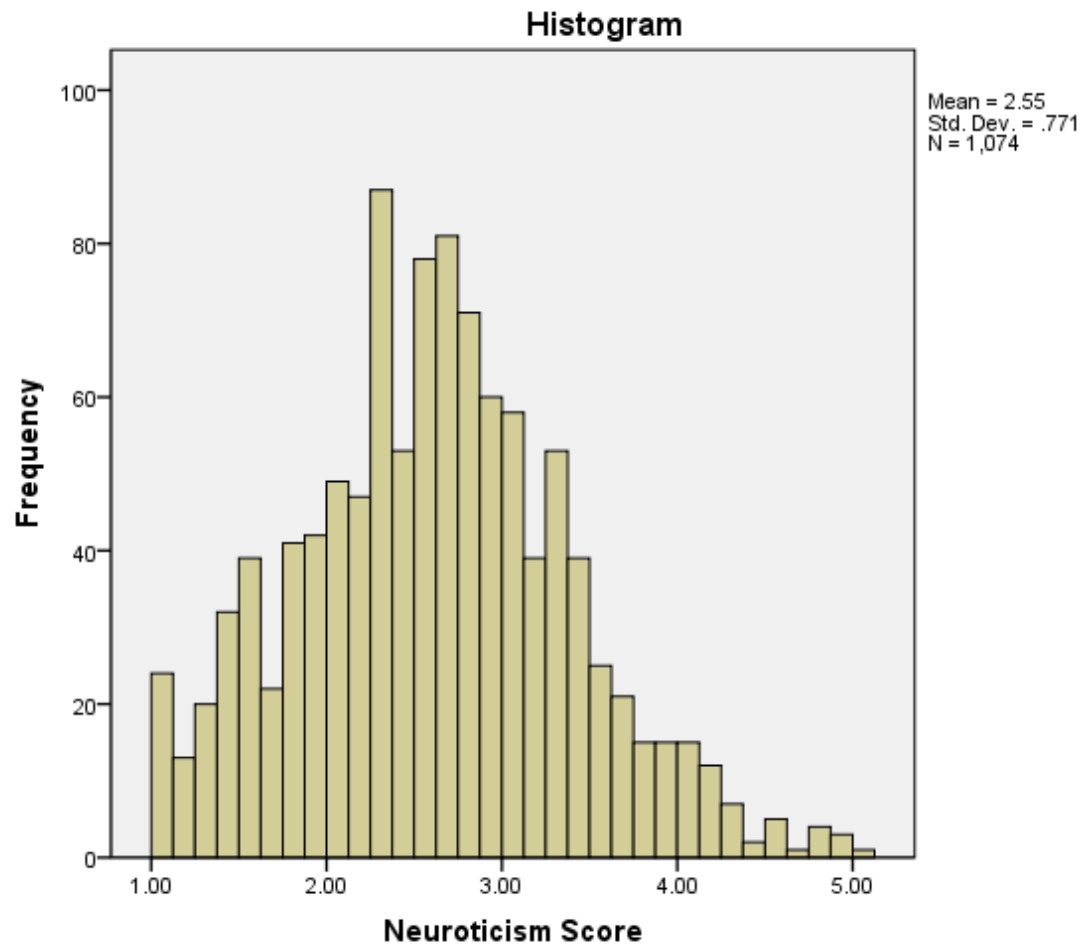


Figure L.7. Histogram of Neuroticism Scale scores (Study 1).

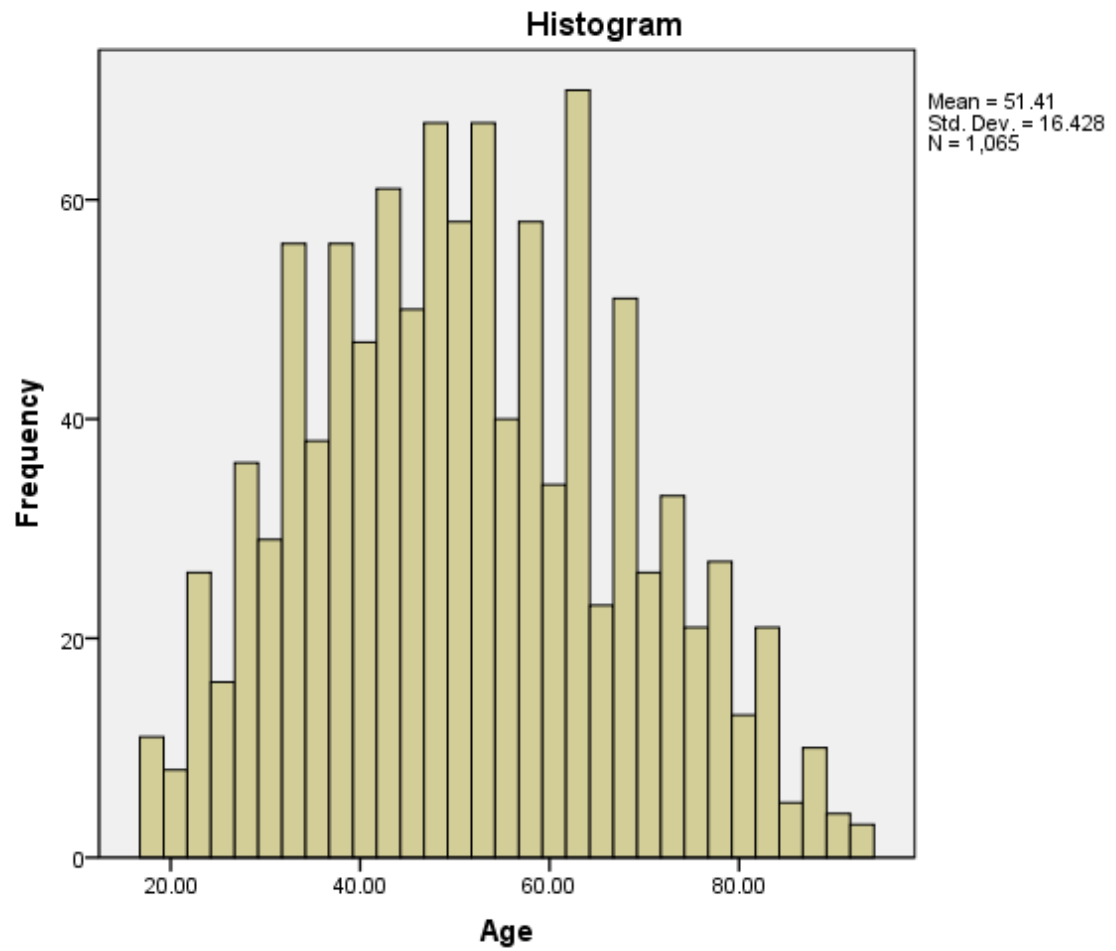


Figure L.8. Histogram of age of participants (Study 1).

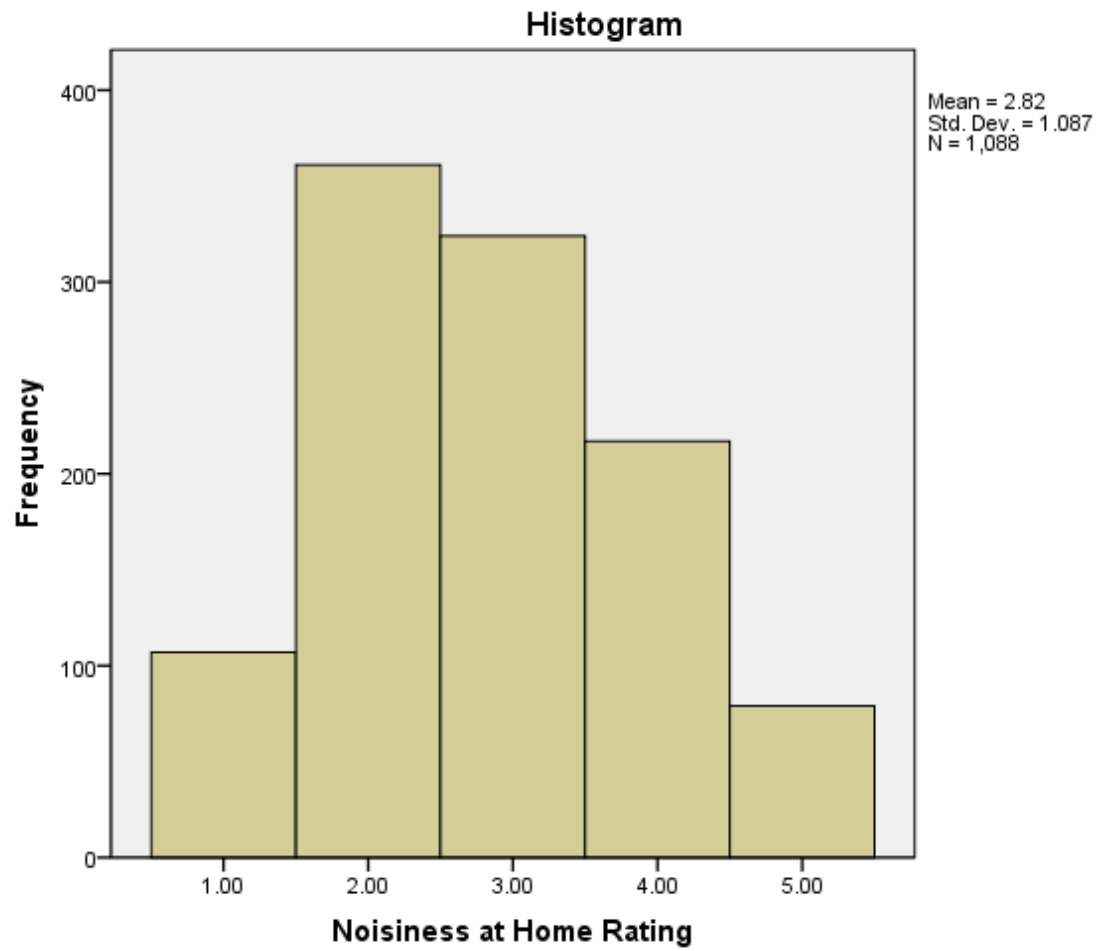


Figure L.9. Histogram of noisiness at home ratings.

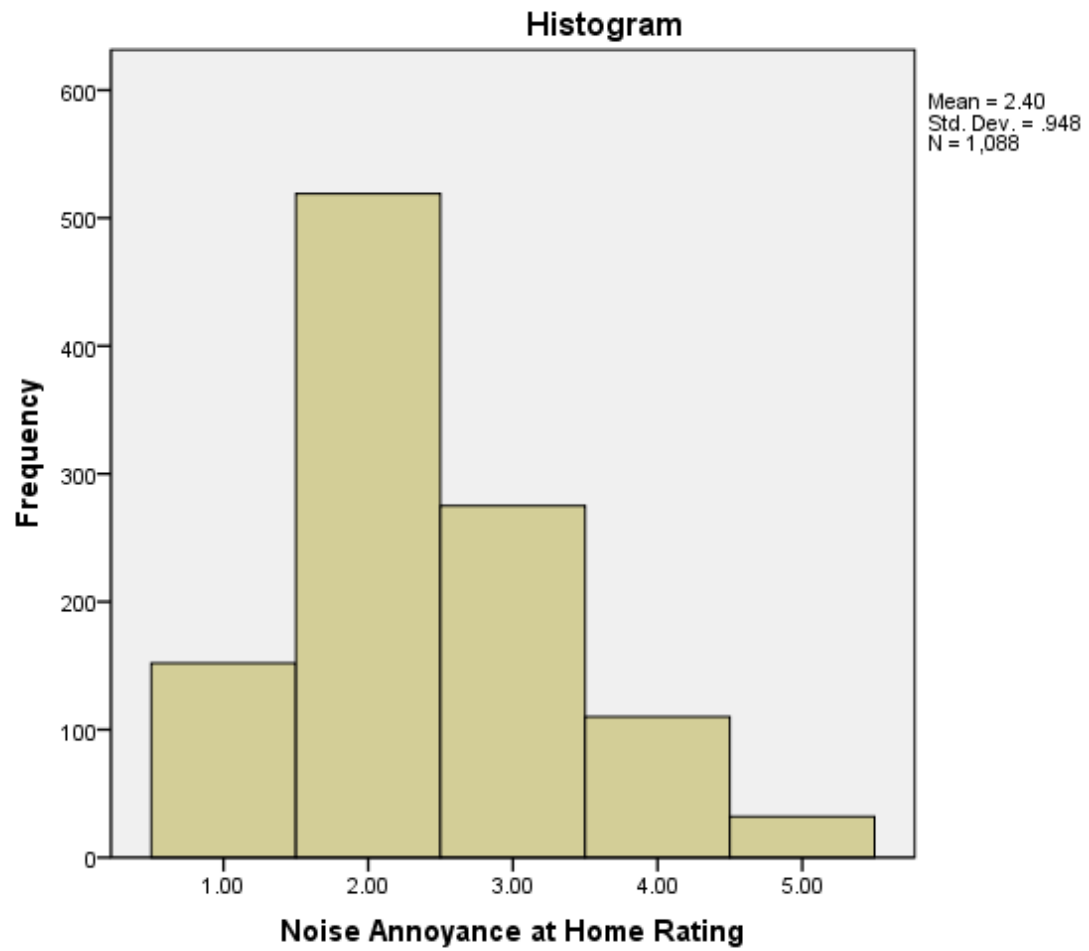


Figure L.10. Histogram of noise annoyance at home ratings.

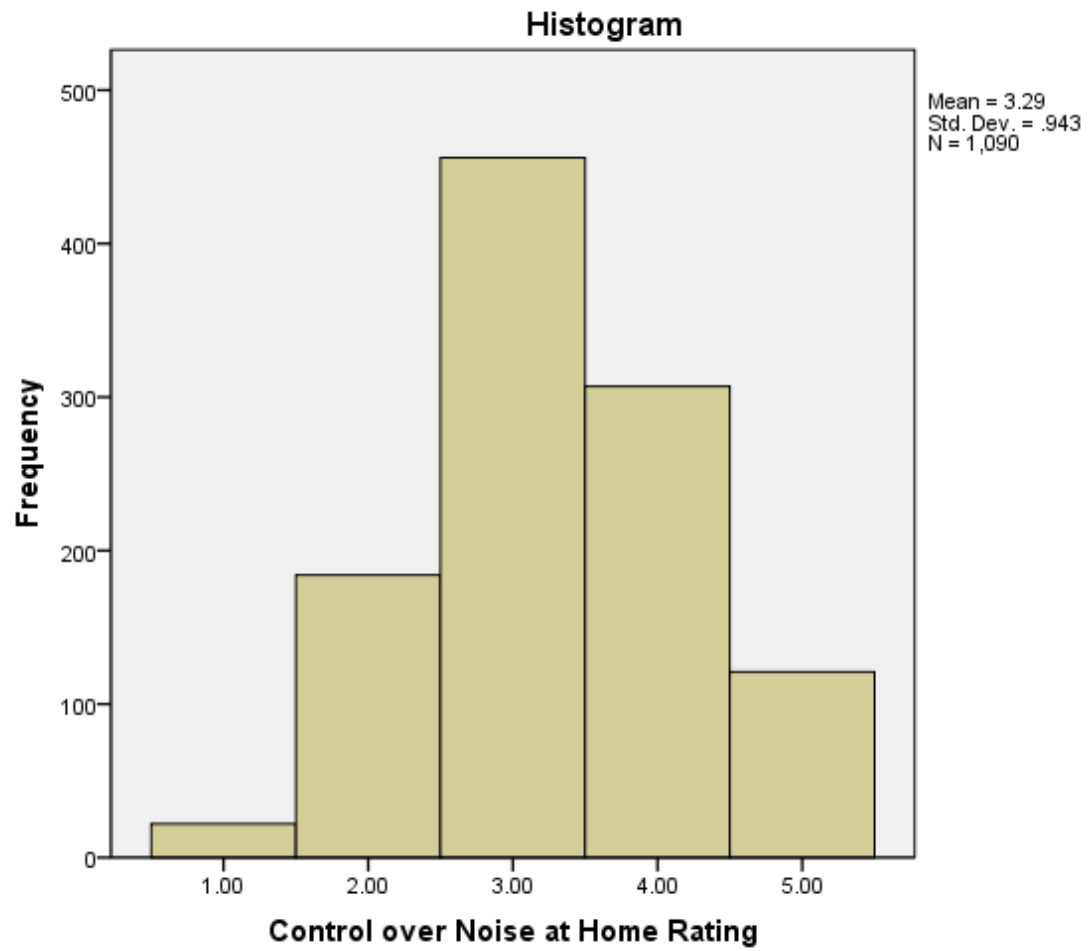


Figure L.11. Histogram of control over noise at home ratings.

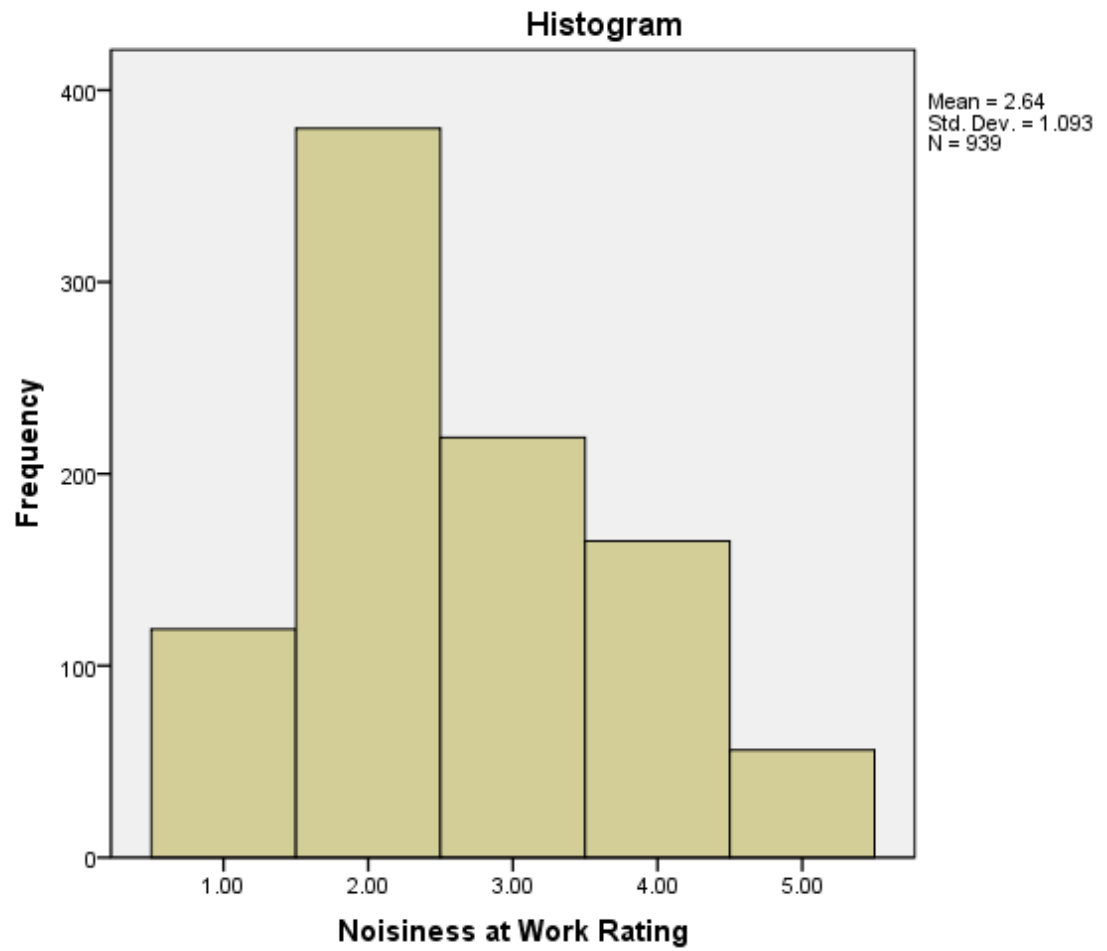


Figure L.12. Histogram of noisiness at work ratings.

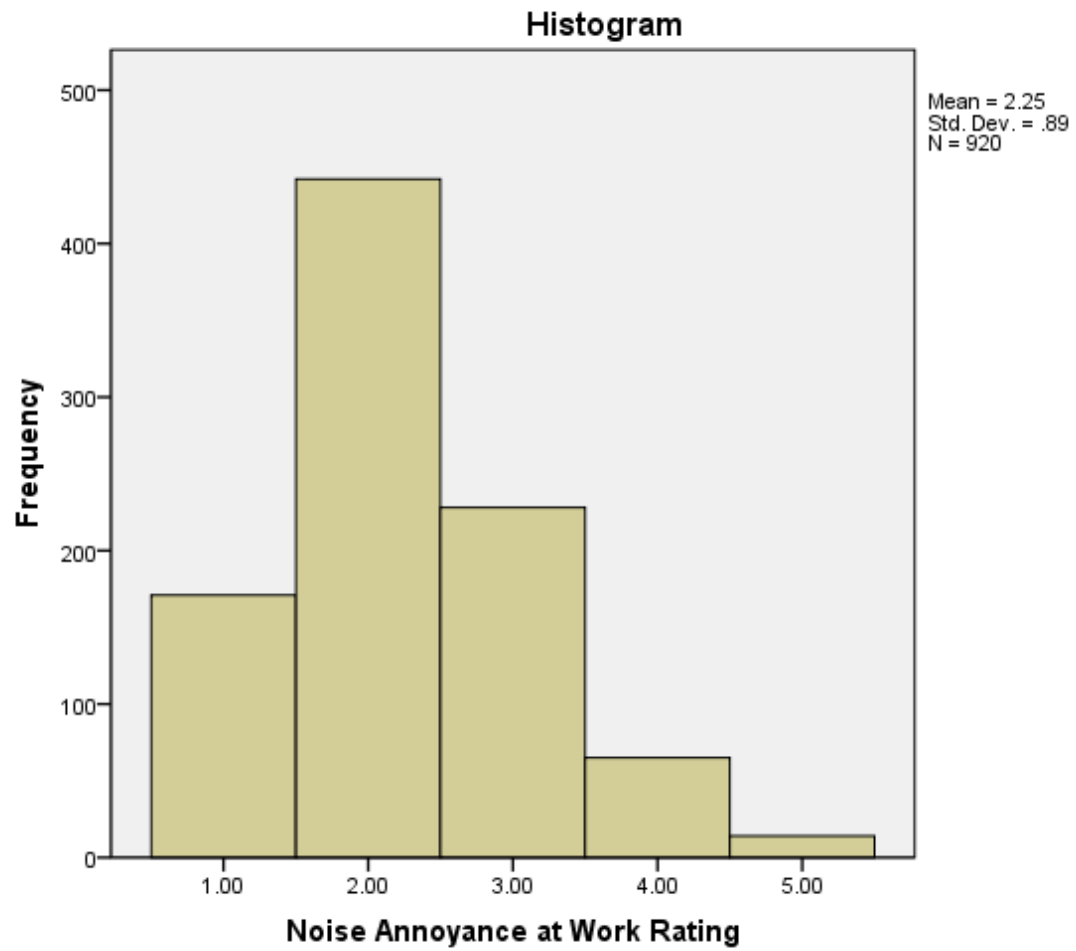


Figure L.13. Histogram of noise annoyance at work ratings.

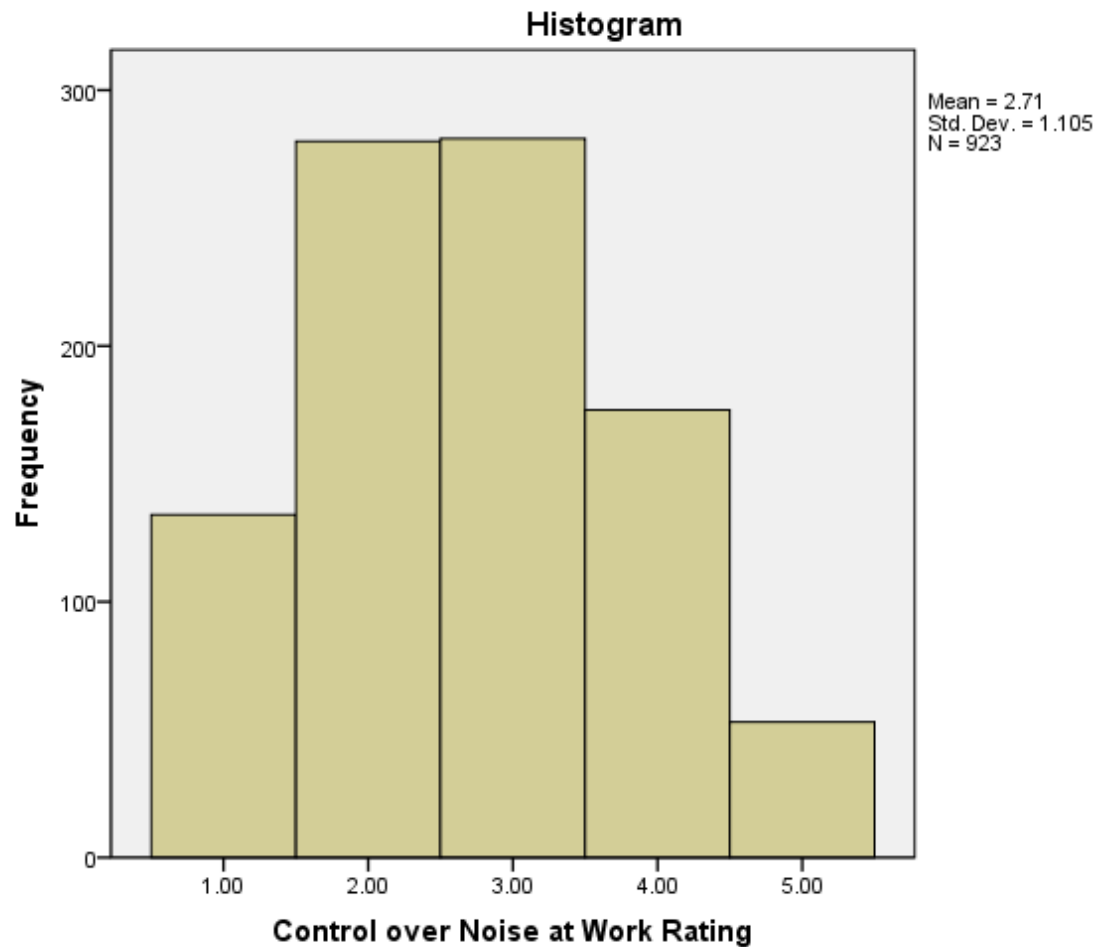


Figure L.14. Histogram of control over noise at work ratings.

Univariate and Multivariate Outliers

Standardised z-scores were computed to assess for univariate outliers for the Study 1 variables. The Perceived Stress Scale (PSS) had one univariate outlier; one participant had the highest score possible on the PSS (40). To treat the outlier, the score was censored (by taking the second highest score (35) and adding 1 = 36), a recommended method for dealing with univariate outliers (Barnett & Lewis, 1994; Tabachnick & Fidell, 2007). Univariate outliers (3) were identified in the Subjective Health Complaints Inventory (SHCI); all outliers were treated with censorship. No outliers were identified for the remaining variables and scales.

Mahalanobis distance was calculated to determine whether multivariate outliers were present among the main variables of the study. The Mahalanobis distance for 4 cases exceeded the critical value, $\chi^2(14) = 36.1, p = .001$. Due to the robust sample size ($n > 1000$), and the problematic nature of multivariate outliers (Tabachnick & Fidell, 2007), the 4 cases were removed from the analyses leaving the final sample size as 1102.

Multicollinearity and Singularity

Multicollinearity and singularity were assessed with correlations, tolerance and variance inflation factors (VIF). The highest correlation was between perceived stress and neuroticism ($r = .657, p < .001$). All correlations were below .8 and therefore multicollinearity was not suspected. Variance inflation factors and tolerance also confirmed no issues with multicollinearity; no values for VIF approached 10, and no tolerance values were less than .2 (Tabachnick & Fidell, 2007).

Preliminary Analyses: Examining Possible Effects of Season

An unforeseen issue that arose during data collection was that the questionnaires delivered as part of the study were distributed at two different times of the year. The large majority of the questionnaires (5000) were delivered in autumn (April – May, 2011), while the remaining questionnaires (2500) were delivered in late winter (August 2011). While Auckland, New Zealand has a subtropical climate with relatively mild winters, there was a concern of the influence of season on the outcome variables in the study. Indeed, a recent Norwegian study by Persson et al. (2010) found that ratings of stress and some health complaints were higher during winter and early spring, highlighting the importance of exploring this issue prior to conducting the main analyses.

Using the Mann-Whitney U test for non-parametric contrasts, the autumn-delivered questionnaires were compared to the winter-delivered questionnaires across the predictor

and outcome variables of interest in the study. Because noise exposure was an important moderator in the study, comparisons were made only across equivalent noise-exposure levels (e.g., autumn-delivered questionnaires distributed along the noisiest streets (> 30,000 vehicles/24 hour) were only compared to winter-delivered questionnaires distributed along streets with the same traffic density).

A Bonferroni correction was applied due to the large number of comparisons conducted (16 per noise-exposure level). The new critical alpha level calculated was .001 (48/.05). Among the participants in the highest noise-exposure group (traffic density > 30,000 vehicles/24 hour), the only significant difference found between the autumn-delivered questionnaires and winter-delivered questionnaires was on the 'noise exposure at home variable' ($U = 3991, p < .001$). Participants in the autumn-delivery group were more likely to report greater noise exposure in the home environment than participants in the winter-delivery group ($M = 4.91, SD = .69$ vs. $M = 3.86, SD = .1.05$). No differences were found across the autumn-delivered questionnaires and winter-delivered questionnaires in the 10,000-20,000 vehicles/24 hour noise-exposure groups.

Among participants in the quietest areas (traffic density less than 10,000 vehicles/24 hour), those in the winter-delivery group had significantly lower ratings of noise exposure ($U = 15615, p < .001, M = 3.86, SD = .1.05$ vs. $M = 4.91, SD = .69$) and significantly higher ratings of perceived control over noise in the home environment ($U = 15615, p < .001, M = 4.91, SD = .69$ vs. $M = 3.86, SD = .1.05$) compared to the autumn-delivery participants. Overall, the autumn-delivery participants did not significantly differ from the winter-delivery participants on any stress or health variables. Therefore, while the groups did differ according to some perceptions of noise exposure and noise control (which is an interesting finding in and of itself relating to noise-related public health research), because

there did not seem to be any effect of season on health or stress, participants were merged for the main analyses.

Appendix M: Moderated Mediation Model with Perceived Noise Exposure as a Moderator

A moderated mediation model testing the influence of noise exposure (assessed with road traffic volume as a proxy measure) on the relationship between noise sensitivity and perceived stress and sleep problems was previously discussed in the Results section of Study 1 (see Figure 6, p. 127). There was no significant interaction between environmental noise exposure (assessed with road traffic volume as a proxy) and noise sensitivity (see Table 10, p. 128). As a result, the analysis was repeated with perceived noise exposure (5-point Likert scale rating; see Appendix G. p. 258) as the moderator variable in order to assess the role of *perceived* noise exposure, in addition to objective environmental noise exposure.

The overall model was significant, $F(8, 1008) = 114.57, p < .001, R^2 = .476$. However, the moderated regression results revealed that there was no moderating effect of perceived noise exposure in the relationship between noise sensitivity and perceived stress, or noise sensitivity and sleep problems (interaction p values $> .05$; see Table M.1., p. 289).

Table M.1. *Moderated Regression Results for the Effect of Gender and Perceived Noise Exposure on the Relationship between Noise Sensitivity and Perceived Stress and Sleep Problems*

	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>
Moderated Multiple Regression of Noise Sensitivity on Perceived Stress				
Noise Sensitivity	.455	.230	.974	.049
Perc. Noise Exposure	1.258	.455	2.764	.006
Perc. Noise Exposure x Noise Sens	-.068	.048	-1.422	.155
Gender	-.036	1.051	-.034	.973
Gender x Noise Sens.	-.071	.115	-.617	.537
Neuroticism	5.046	.198	25.444	<.001
Age	-.041	.010	-4.237	<.001
Education	-.162	.176	-.919	.358
$F(8, 1008) = 114.57, p < .001, R^2 = .476$				
Moderated Multiple Regression of Noise Sensitivity on Sleep Problems				
Noise Sensitivity	-.030	.047	-.635	.526
Perceived Noise Exposure	-.045	.092	-.495	.621
Perc. Noise Exposure x Noise Sens.	.009	.010	.931	.352
Gender	-.106	.212	-.501	.617
Gender x Noise Sens.	.021	.023	.908	.364
Neuroticism	.377	.040	9.418	<.001
Age	.008	.002	3.955	<.001
Education	-.020	.036	-.561	.575
$F(8, 1008) = 17.66, p < .001, R^2 = .123$				

Note. Perc. Noise Exposure = Perceived noise exposure; Noise Sens. = Noise sensitivity; $n = 1017$

Appendix N: Study 2 Advertisement



Can our personality and environment alter our stress hormone levels?



We are currently seeking individuals (18 years of age or older) for our study on the influence of personality, noise sensitivity and the environment (i.e., noise exposure) on stress hormones.

Please note: Participants will be non-smokers who are not taking steroid medication, hormone replacement therapy or estrogen-based contraceptives (e.g., the pill, needle).

For more information please contact Erin Hill at: erin.hill@aut.ac.nz

Stress Study
erin.hill@aut.ac.nz

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erin.hill@aut.ac.nz

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Appendix O: North Shore Times Article about Noise Sensitivity Research Programme

This image has been removed by the author for copyright reasons.

Figure O.1. Article in the North Shore Times newspaper on the noise sensitivity research programme.

Note: The journalist's details of the study procedure were not accurate. When participants contacted me about the study, the procedure was clarified.

Appendix P: AUTECH Approval Letter for Study 2



MEMORANDUM

Auckland University of Technology Ethics Committee (AUTECH)

To: Daniel Shepherd
 From: **Madeline Banda** Executive Secretary, AUTECH
 Date: 6 May 2011
 Subject: Ethics Application Number 10/270 **Personality, health and environmental influences on the cortisol awakening response.**

Dear Daniel

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTECH) at their meeting on 8 November 2010 and that on 18 February 2011, I approved your ethics application. This delegated approval is made in accordance with section 5.3.2.3 of AUTECH's *Applying for Ethics Approval: Guidelines and Procedures* and is subject to endorsement at AUTECH's meeting on 23 May 2011.

Your ethics application is approved for a period of three years until 18 February 2014.

I advise that as part of the ethics approval process, you are required to submit the following to AUTECH:

- A brief annual progress report using form EA2, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 18 February 2014;
- A brief report on the status of the project using form EA3, which is available online through <http://www.aut.ac.nz/research/research-ethics/ethics>. This report is to be submitted either when the approval expires on 18 February 2014 or on completion of the project, whichever comes sooner;

It is a condition of approval that AUTECH is notified of any adverse events or if the research does not commence. AUTECH approval needs to be sought for any alteration to the research, including any alteration of or addition to any documents that are provided to participants. You are reminded that, as applicant, you are responsible for ensuring that research undertaken under this approval occurs within the parameters outlined in the approved application.

Please note that AUTECH grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

When communicating with us about this application, we ask that you use the application number and study title to enable us to provide you with prompt service. Should you have any further enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at ethics@aut.ac.nz or by telephone on 921 9999 at extension 8860.

On behalf of AUTECH and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

A handwritten signature in black ink, appearing to read 'M. Banda', with a stylized flourish at the end.

Madeline Banda
Executive Secretary
Auckland University of Technology Ethics Committee

Cc: Erin Hill Erin.hill@aut.ac.nz

Appendix Q: Participant Information Sheet for Study 2

Participant Information Sheet

**Date Information Sheet Produced:**

20/10/2010

Project Title

Personality, Health and Environmental Influences on the Cortisol Awakening Response

An Invitation

My name is Erin Hill and I am currently a PhD student at the Auckland University of Technology in the School of Public Health and Psychosocial Studies. Thank you for your interest in my PhD research programme on the interaction among environment, personality, health and cortisol levels.

What is the purpose of this research?

The objective of the present research study is to understand the relationship among environmental stress, personality, health and cortisol levels. Morning cortisol levels will provide information about your nervous system's response to stress, therefore it is important to study in relation to health, personality and your environment.

How was I identified and why am I being invited to participate in this research?

You are invited to participate in this research because you are 18 years of age or older, and have expressed interest in my study – one which involves a relatively novel physiological measurement (the collection of morning saliva cortisol levels).

Exclusion criteria for the study are as follows: individuals on hormone therapy or estrogen-based medication (e.g., oral contraceptives, the needle), individuals who smoke, and individuals on steroid medication.

What will happen in this research?

This study involves two parts.

1) Collection of Morning Cortisol Levels

If you choose to participate in this study, you will be asked to choose one regular work day (e.g., Monday-Friday) to collect 3 samples of saliva. You will be provided with 3 small test tubes (called salivettes), which will be used for the saliva sample collection. It is important to note that on the morning you choose to collect your saliva samples, you are asked not to eat, drink or brush your teeth until all samples have been collected.

You will be asked to collect 3 saliva samples:

Sample 1: As soon as you wake up (this can be the regular time you wake up on a weekday morning)

Sample 2: 30 minutes after awakening

Sample 3: 60 minutes after awakening

To collect your saliva, please place the cotton piece (found in the salivette) in your mouth and chew softly on it for about 45 seconds or until the cotton ball is fully saturated (completely soaked). Then take the cotton piece out of your mouth and place it back inside the salivette.

**Please record the time and date of collection on the label of each salivette.*

Please place your saliva samples in your freezer until they are returned to the researcher.

Information for Females: Cortisol levels are sensitive to menstrual status. Therefore, we ask that you collect your saliva samples anytime within 10 days of your expected period.

2) Completion of the Questionnaire Booklet

This part of the study involves the completion of a set of questionnaires that will ask you information about your stress, health, personality, noise exposure and sleep quality. Please note that Section 2 (first page of questionnaire) asks questions pertaining to the dates of your saliva collection.

What are the discomforts and risks?

There are no physical risks to participating in the study. The collection of salivary cortisol levels is widely regarded as an accurate and non-invasive physiological measure. Some of the questions in the questionnaire booklet are personal in nature, but please be reassured that all responses will remain confidential and will be used for research purposes only.

What opportunity do I have to consider this invitation?

There is no obligation to participate in this study – it is completely voluntary.

What are the benefits?

Participation in this research programme involves the use of a relatively novel form of analysis – the measurement of morning salivary cortisol levels. Not only will you be able to learn about your own cortisol awakening response in this study, but you will be contributing to a research programme that aims to identify environmental and individual factors that influence a person's physiology. It is hoped that results from the study will be widely disseminated to other health and academic professionals in the field.

How will my privacy be protected?

Your questionnaire responses will be kept completely confidential. Your saliva samples will be coded and therefore your identity will not be known by technicians performing the cortisol analysis.

What are the costs of participating in this research?

There are no costs to you other than your time. The questionnaire booklet should take approximately 30 minutes to complete. The collection of each saliva sample should take no more than 2 minutes each. However, please note that you are not to eat, drink or brush your teeth within 60 minutes of awakening on the day you decide to collect your saliva samples.

How do I agree to participate in this research?

If you are interested in participating in the research programme, please contact the primary researcher, Erin Hill (erin.hill@aut.ac.nz) and she will provide you with a consent form. Erin will also provide you with all the materials needed for participation in the study.

Will I receive feedback on the results of this research?

On the consent form you will be asked if you would like to receive information regarding your morning cortisol levels. If you would like to receive any other feedback regarding the study, please do not hesitate to contact the primary researcher, Erin Hill (erin.hill@aut.ac.nz).

What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, *Dr. Daniel Shepherd*, *daniel.shepherd@aut.ac.nz*, 921 9999 ext 7238.

Concerns regarding the conduct of the research should be notified to the Executive Secretary, ATEC, Madeline Banda, *madeline.banda@aut.ac.nz*, 921 9999 ext 8044.

Whom do I contact for further information about this research?***Researcher Contact Details:***

Erin Hill, PhD Student – *e-mail*: *erin.hill@aut.ac.nz*

Project Supervisor Contact Details:

Dr. Daniel Shepherd (Project Supervisor) – *e-mail*: *daniel.shepherd@aut.ac.nz* *ph*: 9 921 9999 extension 7238

Approved by the Auckland University of Technology Ethics Committee on *18 February 2011*, ATEC Reference number 10/270.

Appendix R: Consent Form for Study 2

Consent Form	 AUT UNIVERSITY TE WĀNANGA ARONUI O TAMAKI MAKAU RAU
-----------------------	---

Project title: Personality, Health and Environmental Influences on the Cortisol Awakening Response

Project Supervisor: **Dr Daniel Shepherd**

Researcher: **Erin Hill, PhD Student**

- ☐ I have read and understood the information provided about this research project in the Information Sheet dated 20 October 2010.
 - ☐ I have had an opportunity to ask questions and to have them answered.
 - ☐ I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
 - ☐ I am not suffering from any illness or injury that impairs my involvement in the research study.
 - ☐ I agree to provide saliva samples.
 - ☐ I agree to take part in this research.
 - ☐ I wish to receive a copy of the report from the research (please tick one): ☐ Yes ☐ No
 - ☐ The saliva you collect will be analysed in a laboratory in order to assess your cortisol levels.
Would you like the results of the cortisol analysis returned to you? ☐ Yes ☐ No
- Please provide your e-mail address if you would like to receive information regarding your cortisol levels.

Participant's signature:.....

Participant's name:.....

Participant's Contact Details (if appropriate):

.....

.....

.....

Date: **Approved by the Auckland University of Technology Ethics Committee on 18 February 2011** AUTEK Reference number **10/270**

Note: The Participant should retain a copy of this form.

Appendix S: Data Screening for Study 2

Missing Data

One hundred and seven individuals participated in Study 2. For the cortisol analyses, 15 participants were excluded due to non-adherence to protocol or issues with cortisol analyses. Of these 15 participants, seven failed to adhere to the study protocol (i.e., they did not collect the samples at the appropriate times), and the cortisol levels of eight participants were either too low to be recorded or there was not enough saliva for the cortisol levels to be read (leaving 92 cases with usable cortisol data).

MVA was conducted on scales with missing data. For the NoiSeQ, nine cases had missing values. The values were determined to be missing at random, $\chi^2(270) = 296.652$, $p = .127$, and therefore the EM algorithm was applied to estimate the missing values (Newman, 2003; Schafer & Graham, 2002). For the PSS, two participants did not complete the scale, and a remaining two cases had one missing value. For the two cases, which were missing at random, $\chi^2(18) = 17.433$, $p = .494$, EM was applied.

For the PSQI, five participants did not provide information about the time they get up in the morning or the number of hours slept, therefore the sleep efficiency subscale and the total PSQI score could not be calculated for those participants (leaving 102 cases with data for the full PSQI scale and the sleep efficiency subscale, 106 for the sleep duration subscale, and 107 for the remaining PSQI subscales). No cases had missing data for the Neuroticism Scale of the BFI. The scoring method for the SHCI addresses issues of non-adherence through its calculation of the composite score (see Eirksen et al., 1999).

Normality, Outliers, and Multicollinearity

Normality for Study 2 variables was assessed through a visual assessment of histograms and Q-Q plots, as well as skewness and kurtosis ratios. The histograms of the study variables are displayed in Figure S.1. through Figure S.24. (p. 302-325). A skew or kurtosis ratio (statistic/standard error of statistic) greater than ± 3 indicated that the scale/score required further investigation and possible transformation (Tabachnick & Fidell, 2007). Skewness and kurtosis ratios are presented in Table S.1. (p. 301) and Table S.2. (p. 302). The SHCI distribution had a significant positive skew (skewness ratio = 4.18) and had a leptokurtic distribution (kurtosis ratio = 3.69). One univariate outlier was identified for the scale (using standardised Z scores). The outlier was treated with censorship (Tabachnick & Fidell, 2007); however this did not improve the skewness enough to meet the assumption of normality. Therefore, a square root transformation was performed, and the distribution was greatly improved (skewness ratio = -.32; kurtosis ratio = .61).

Age was also positively skewed (skewness ratio = 4.63), which was improved slightly following a square root transformation (skewness ratio = 3.81). Because age was a covariate in the hierarchical linear regressions (CARauc and CARi as outcome variables), analyses were conducted using both the untransformed age variable (Table 16, p. 168; Table 17, p. 169) and the transformed age variable (Table S.3., p. 326; Table S.4., p. 327). Results did not differ significantly; therefore, results with the untransformed age variable are presented in the main document of the thesis. The results from the analyses conducted with the transformed variable are presented in this appendix (Table S.3., p. 326; Table S.4., p. 327). Four of the PSQI subscales had non-normal distributions (see Table S.2., p. 302), and therefore non-parametric statistics were applied in analysing the subscales. No other univariate outliers were identified among the scales. Multivariate outliers were assessed

using the Mahalanobis distance. No cases were identified as multivariate outliers, $\chi^2 (16) = 39.25, p < .001$. No issues with multicollinearity were raised; correlations among primary variables were less than .8, variance inflation values were less than 10 and no tolerance values were less than .2 (Tabachnick & Fidell, 2007).

Table S.1. *Skewness and Kurtosis Ratios for Study 2 Variables*

Variable	Skewness Ratio	Kurtosis Ratio
CARauc	1.46	-.01
CARi	-.11	-.15
Time of Awakening (Collection Day)	-.16	.89
Number of hours slept (Collection Day)	-1.8	3.09
Noise Sensitivity (Global)	1.18	-.35
Noise Sensitivity (Leisure)	.91	-.34
Noise Sensitivity (Work)	.49	-.26
Noise Sensitivity (Sleep)	.55	-1.40
Noise Sensitivity (Communication)	.54	-.09
Noise Sensitivity (Habitation)	.28	-.51
Perceived Stress	.99	-.54
Neuroticism	.68	-.01
Sleep Quality (PSQI)	3.21	.59
Age	4.63	1.09
Age ^T	3.81	-.42
Subjective Health Complaints	4.18	3.69
Subjective Health Complaints ^T	-.32	.61

Note. Subjective Health Complaints^T = Total Severity Score of Subjective Health Complaints Inventory (Eriksen et al., 1999; square root transformation applied to address positive skew).

Table S.2. *Skewness and Kurtosis Ratios for PSQI Subscales*

Variable	Skewness Ratio	Kurtosis Ratio
Sleep Duration	6.91	4.67
Sleep Disturbance	6.03	5.32
Sleep Latency	1.89	-1.73
Day Dysfunction	2.00	1.34
Sleep Efficiency	-3.85	-1.91
Sleep Quality	6.53	4.42
Sleep Medication	.09	-1.28

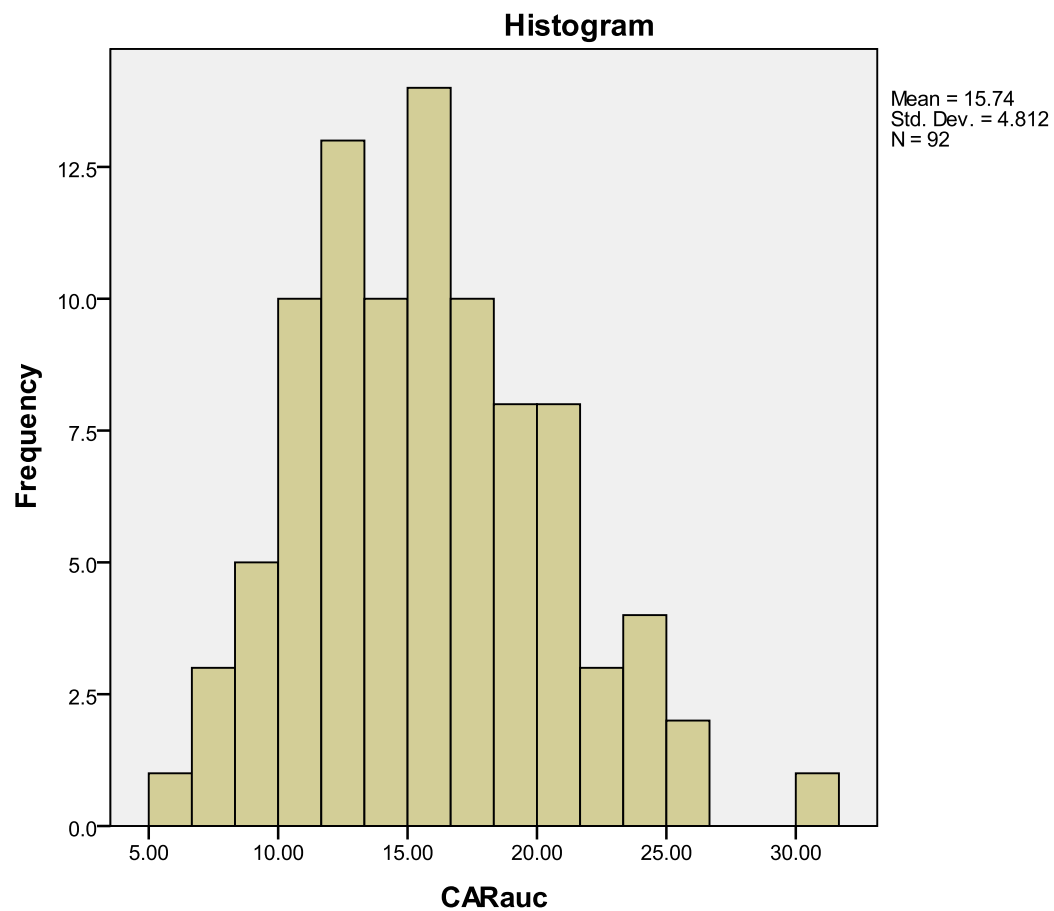


Figure S.1. Histogram of CARauc values.

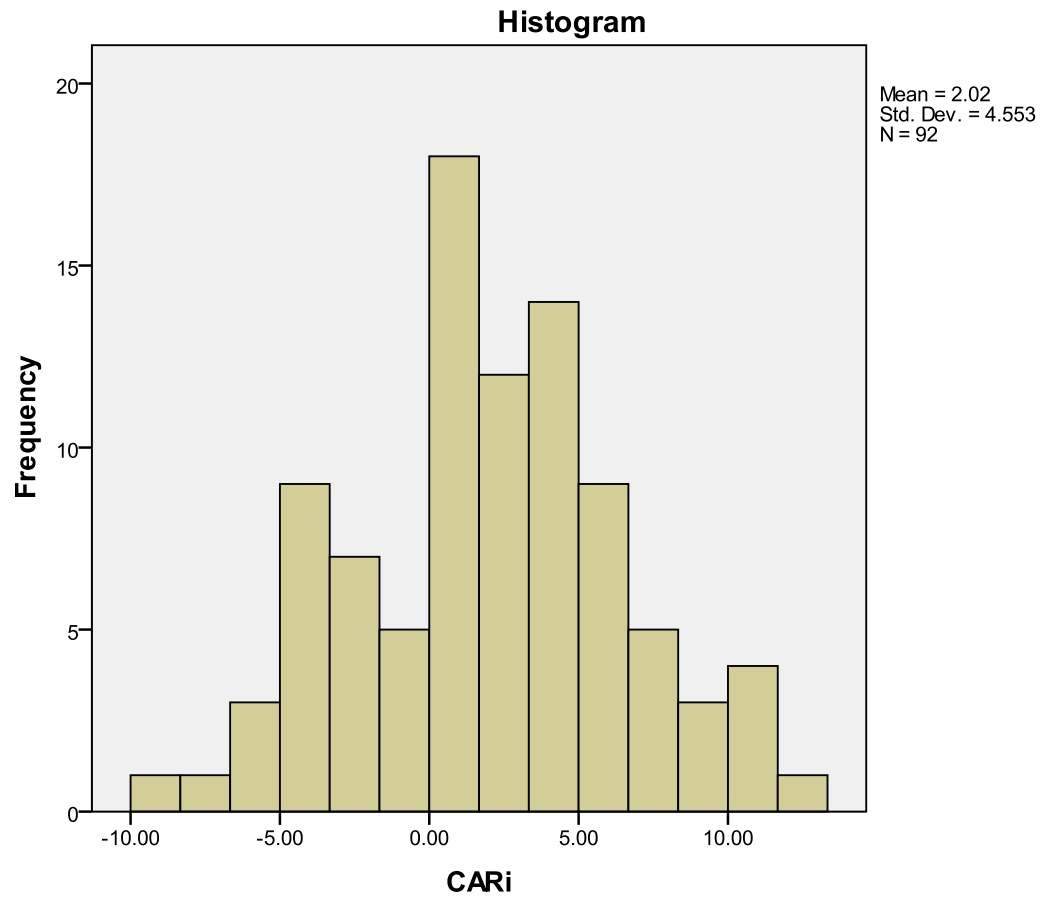


Figure S.2. Histogram of CARi values.

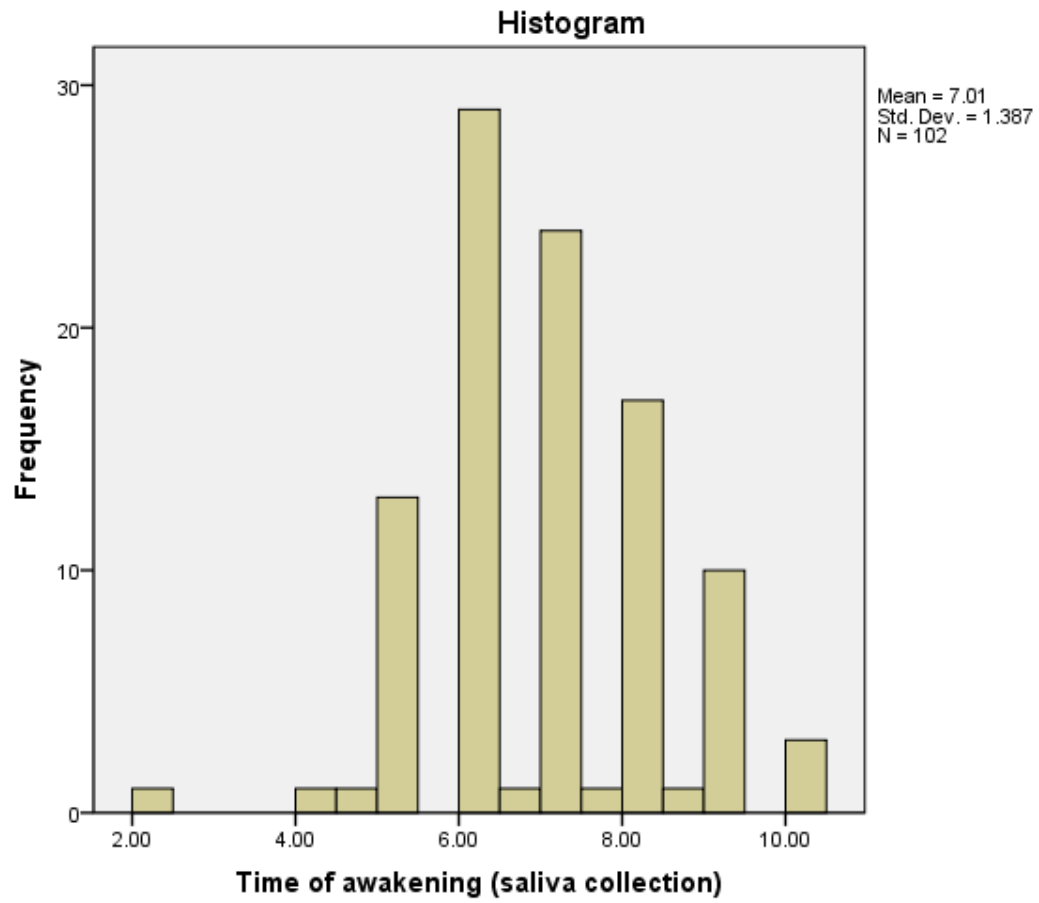


Figure S.3. Histogram of time of awakening (saliva collection day).

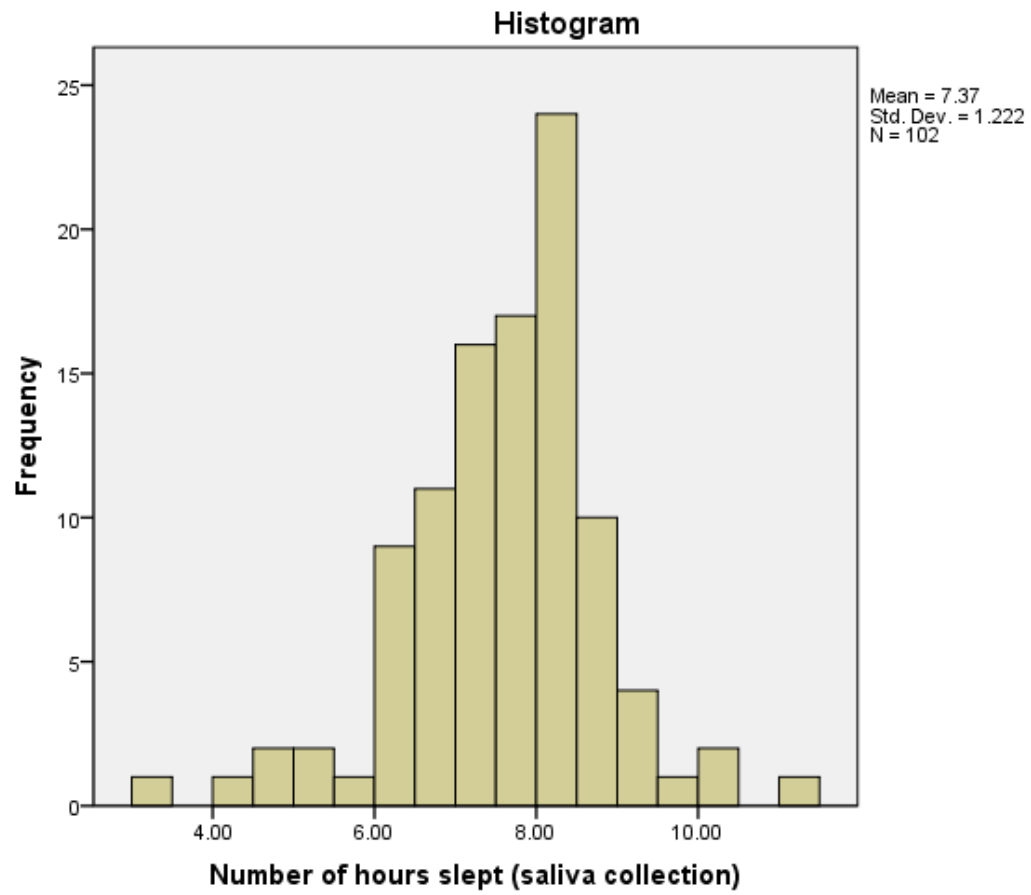


Figure S.4. Histogram of number of hours slept (saliva collection day).

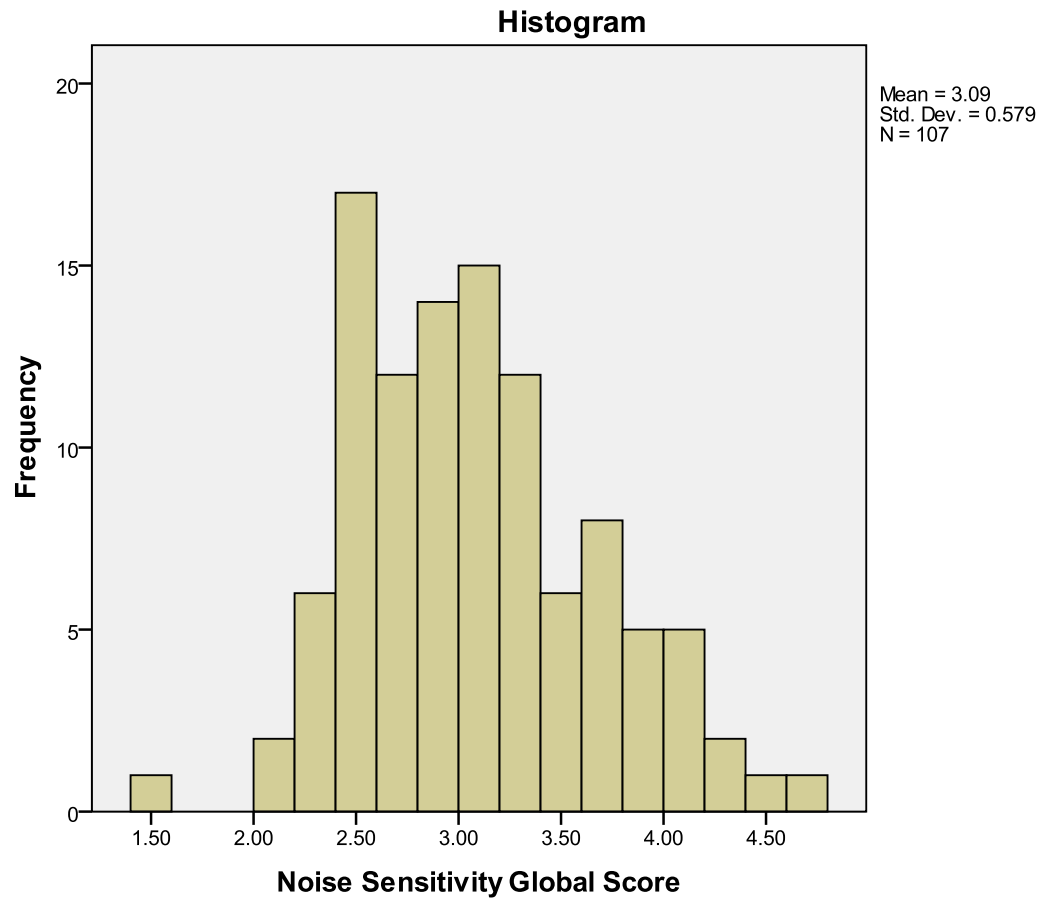


Figure S.5. Histogram of NoiSeQ Global Scale scores.

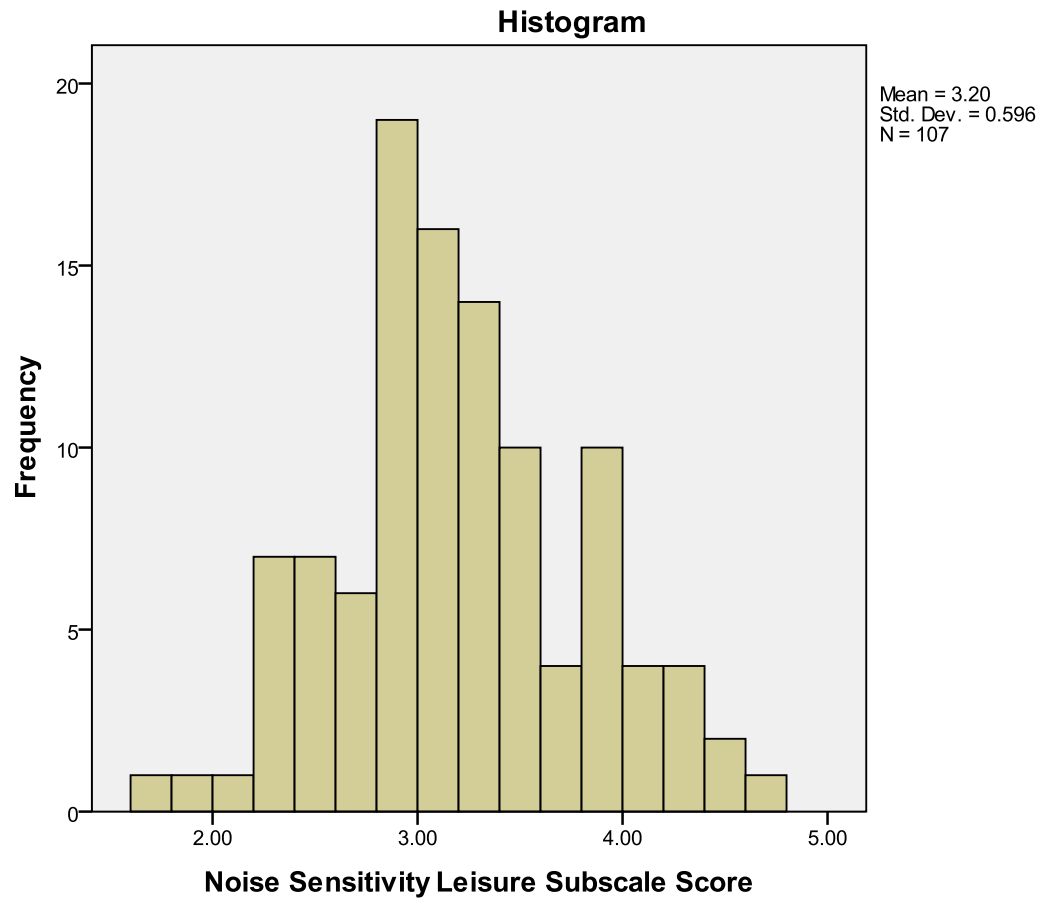


Figure S.6. Histogram of NoiSeQ Leisure Subscale scores.

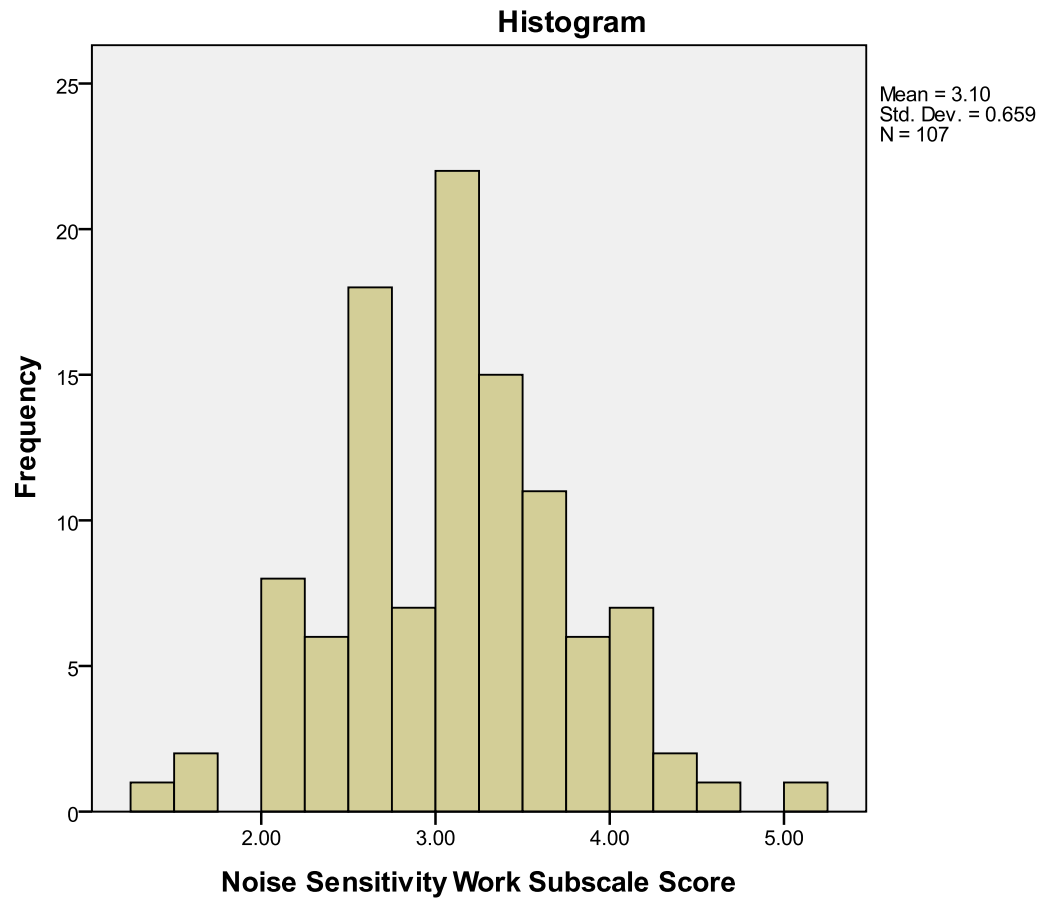


Figure S.7. Histogram of NoiSeQ Work Subscale scores.

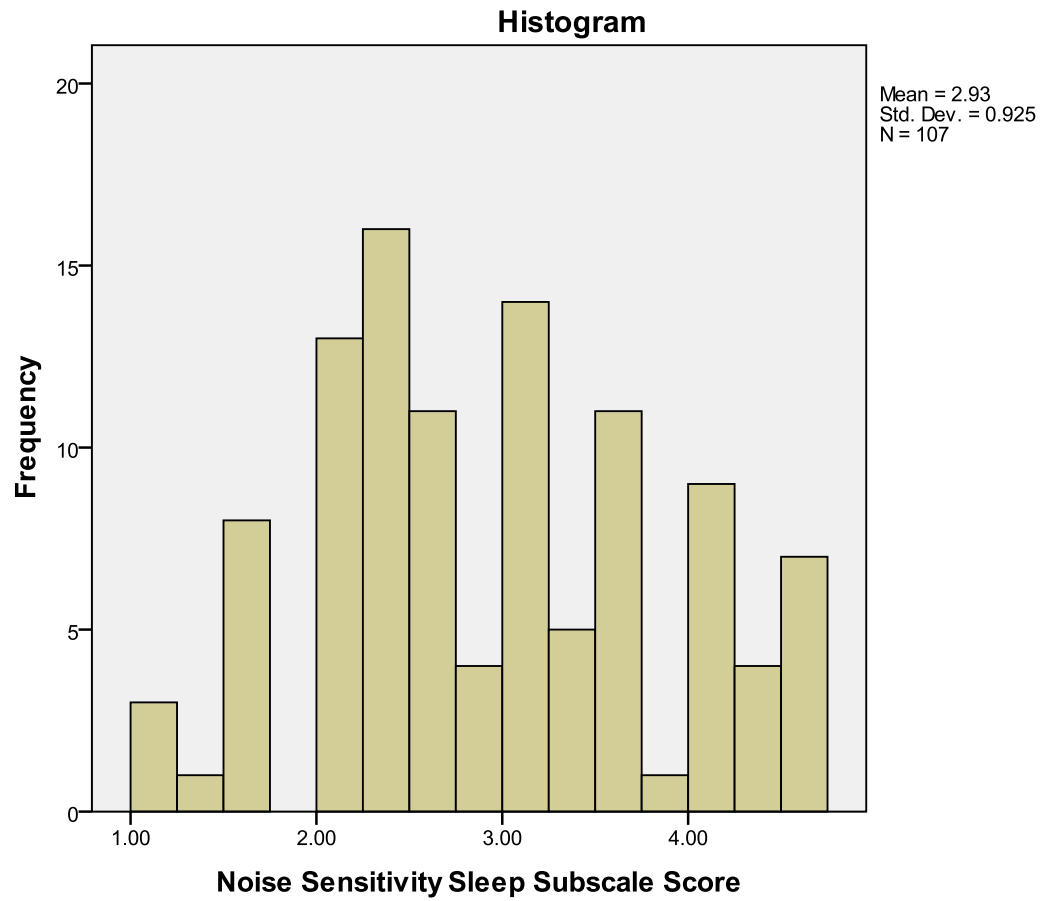


Figure S.8. Histogram of NoiSeQ Sleep Subscale scores.

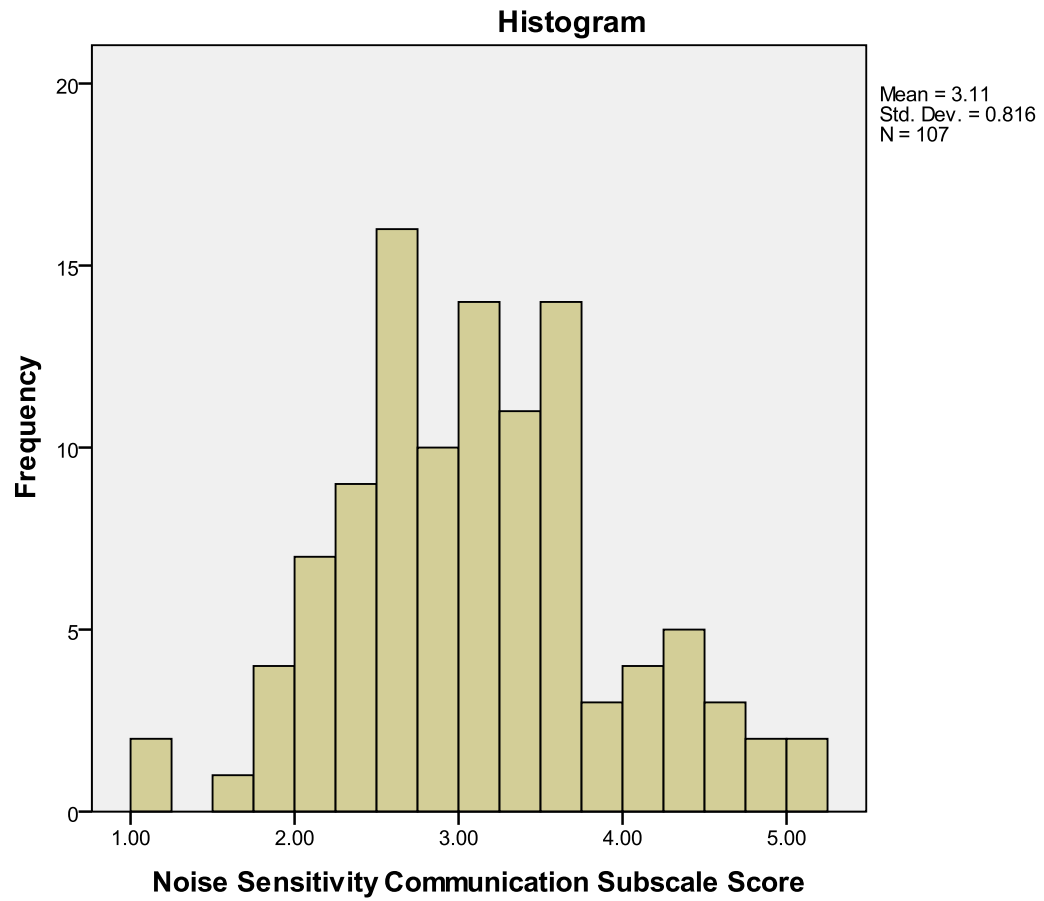


Figure S.9. Histogram of NoiSeQ Communication Subscale scores.

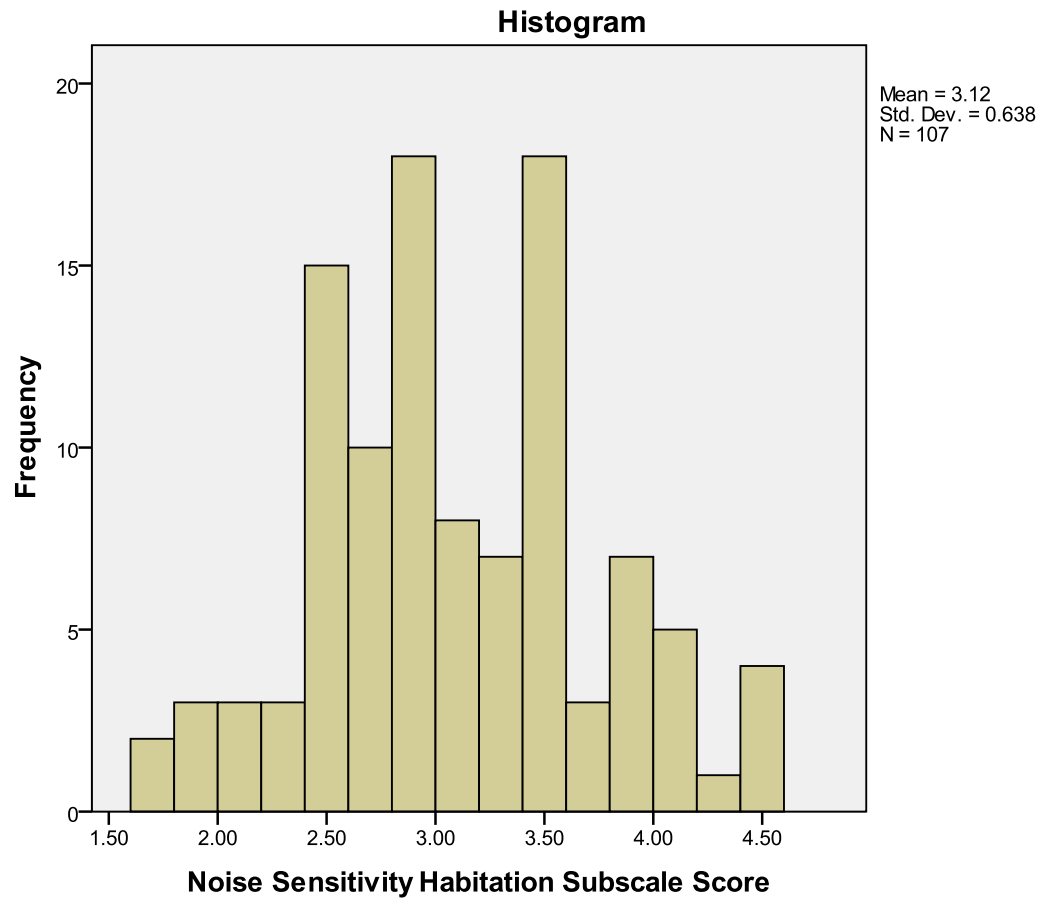


Figure S.10. Histogram of NoiSeQ Habitation Subscale scores.

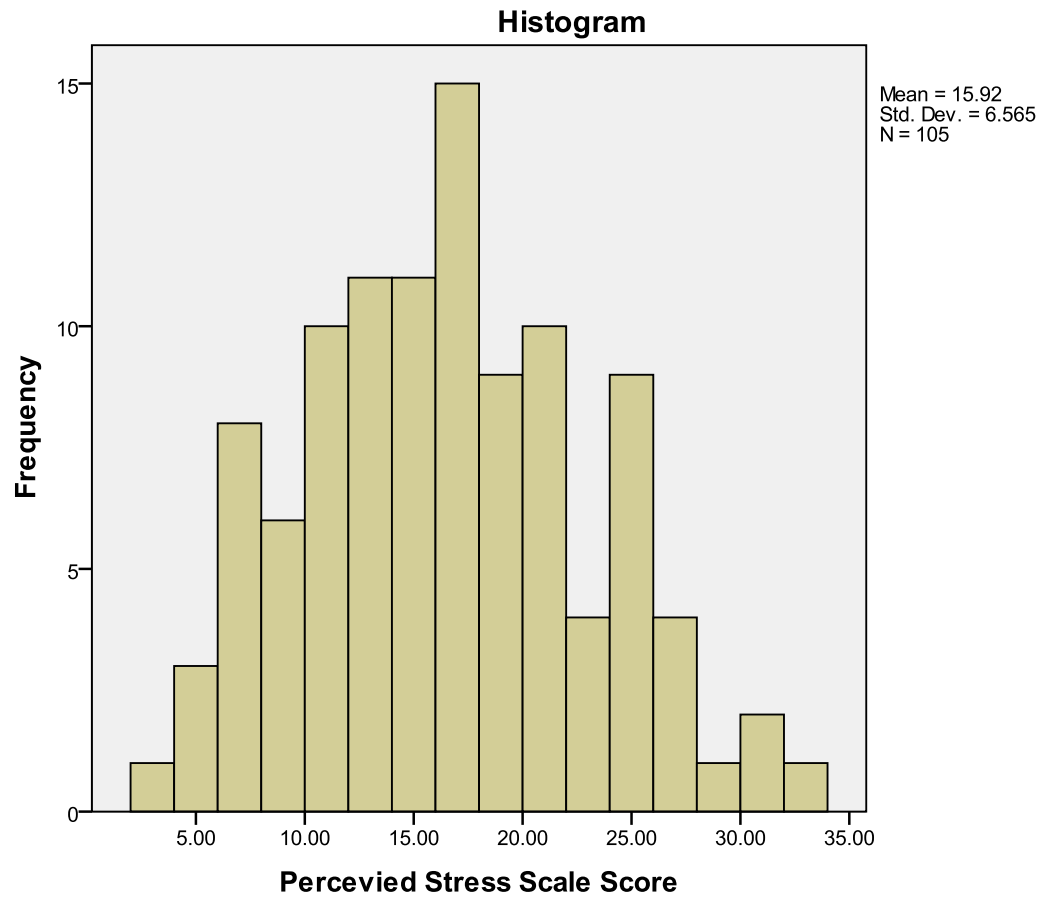


Figure S.11. Histogram of PSS scores (Study 2).

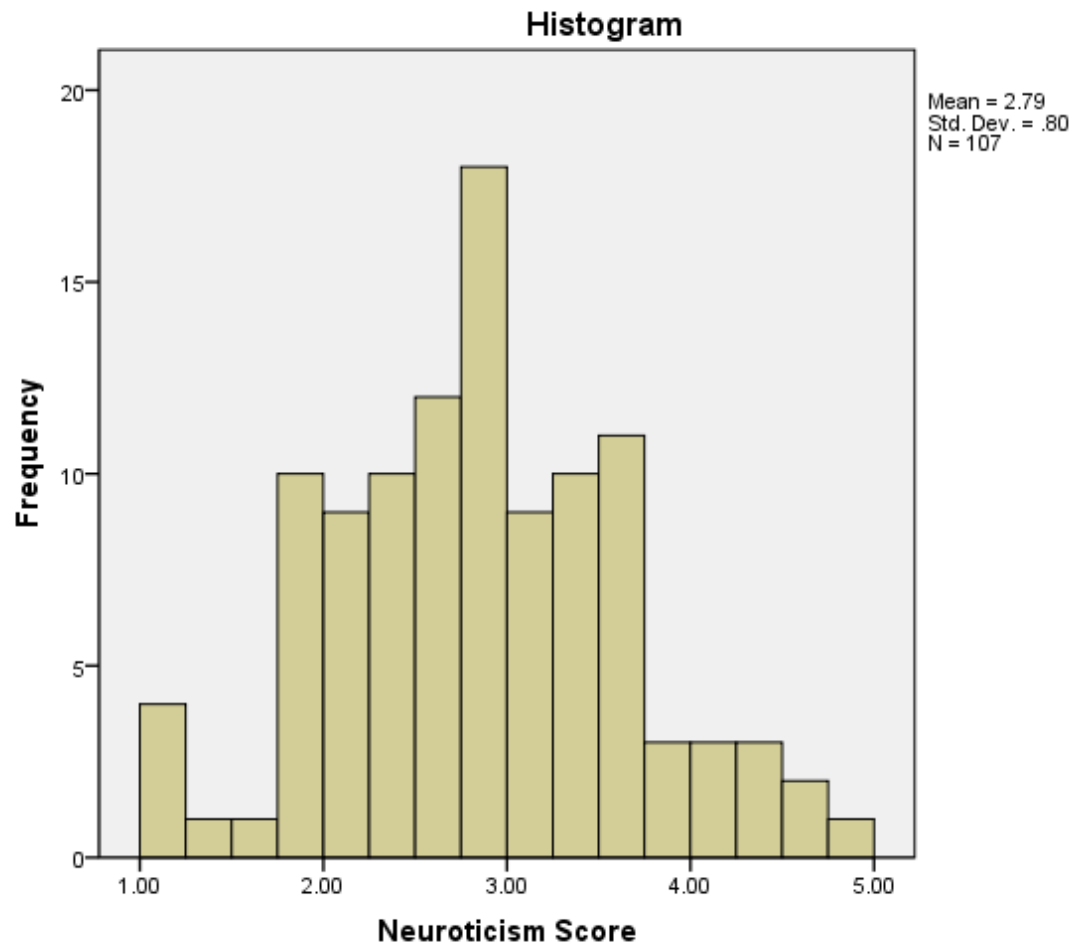


Figure S.12. Histogram of Neuroticism Scale scores (Study 2).

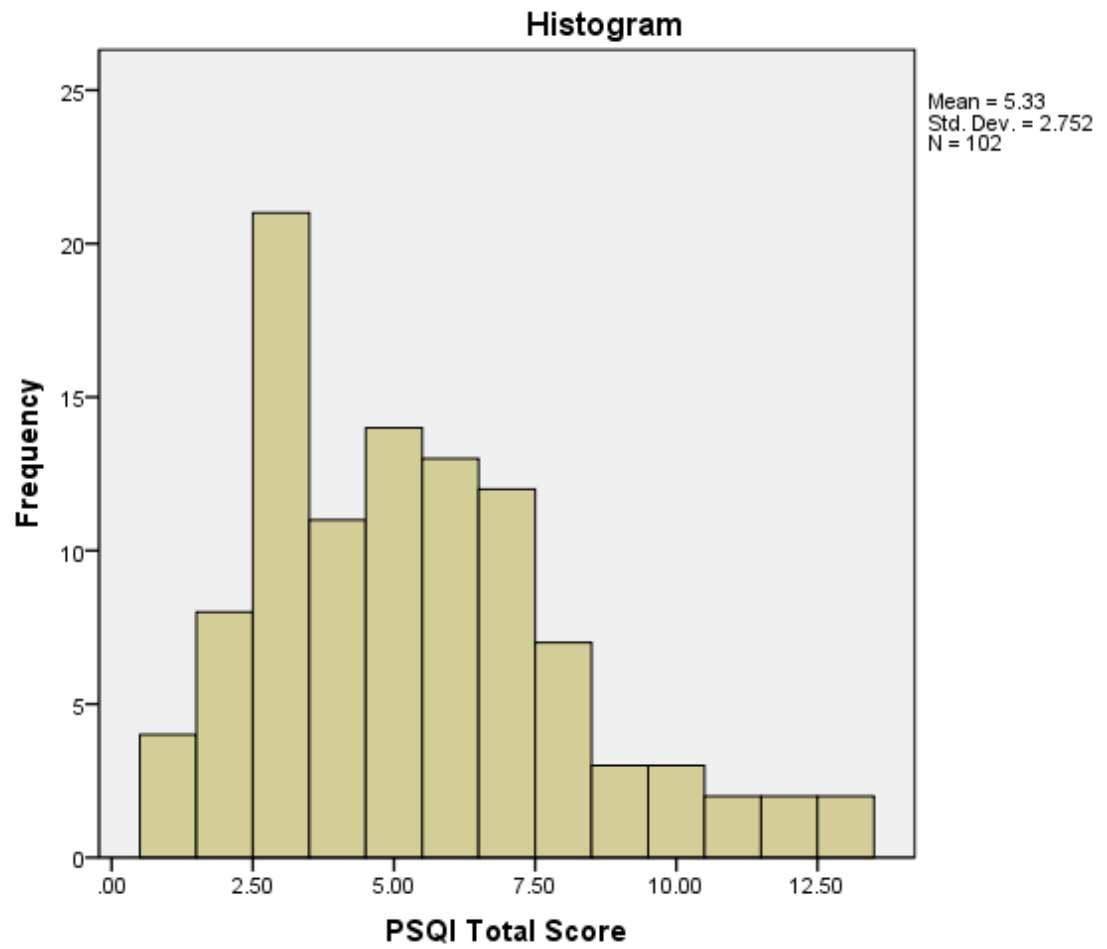


Figure S.13. Histogram of PSQI scores.

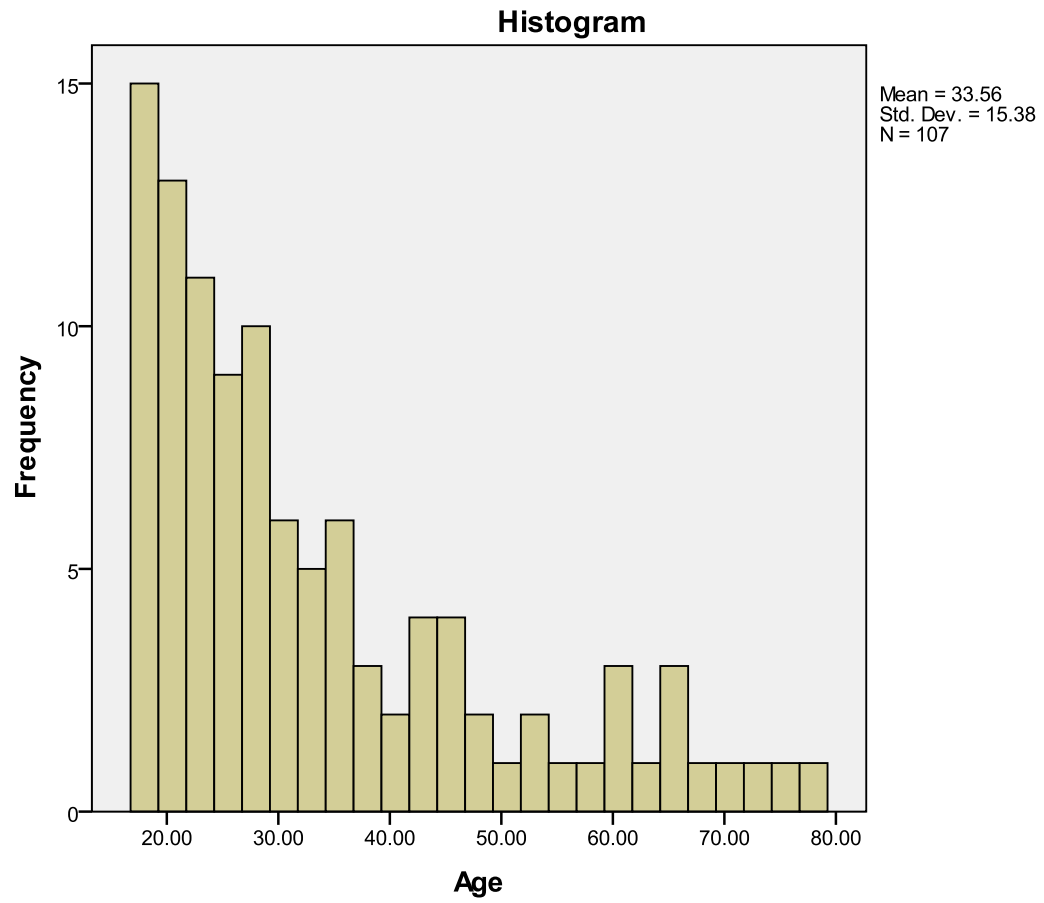


Figure S.14. Histogram of age of participants (Study 2).

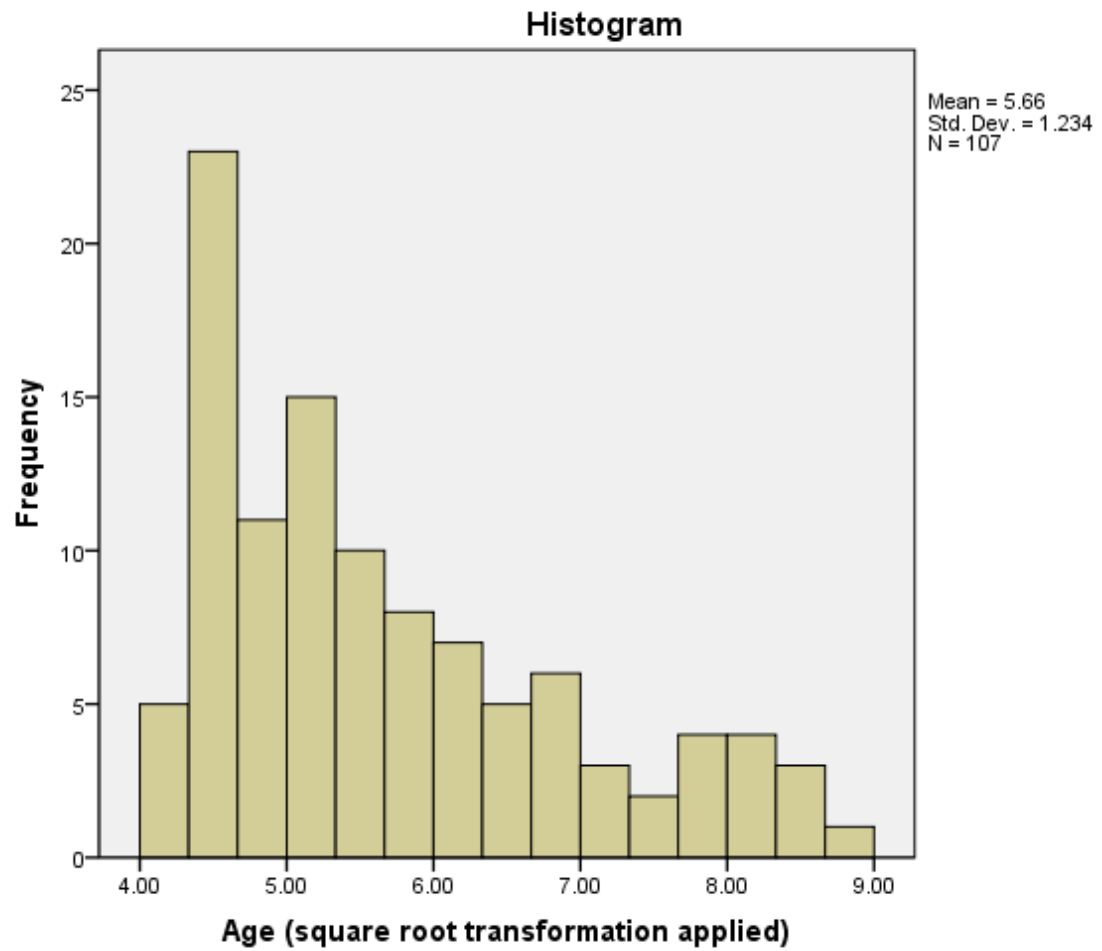


Figure S.15. Histogram of transformed age variable (Study 2).

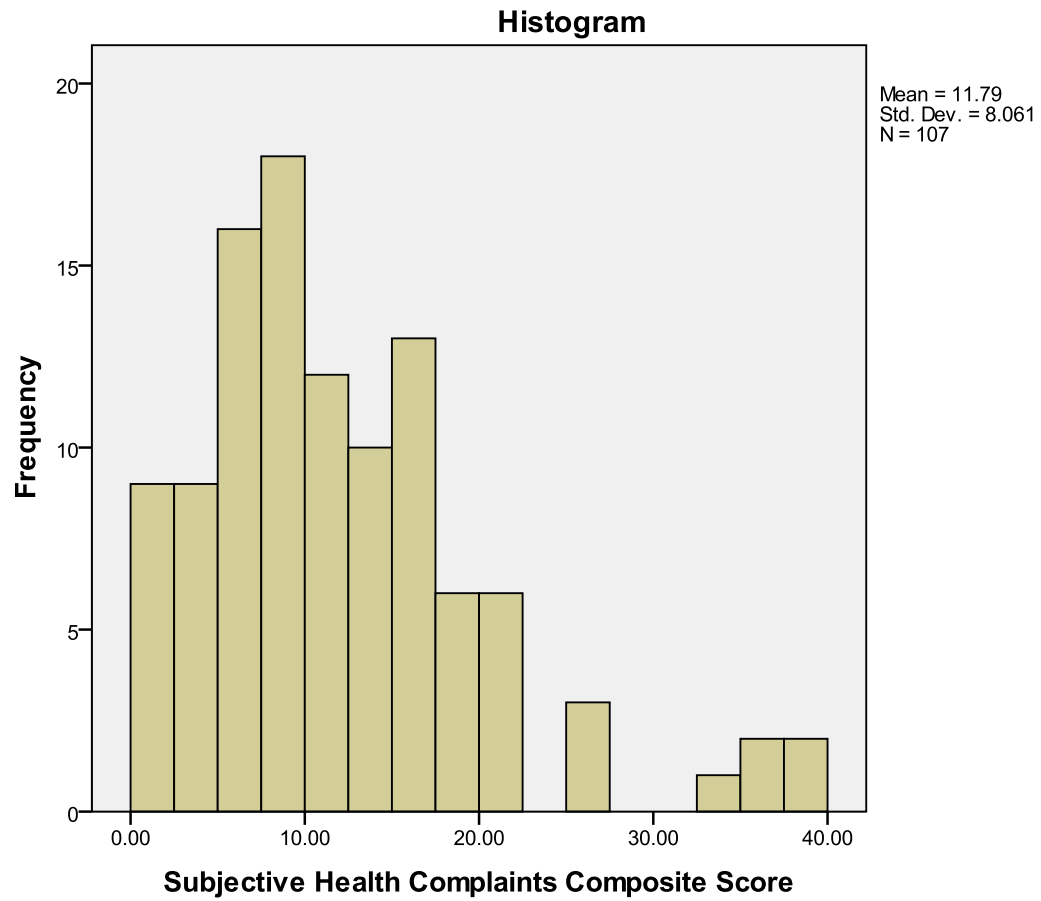


Figure S.16. Histogram of SHCI composite scores (Study 2).

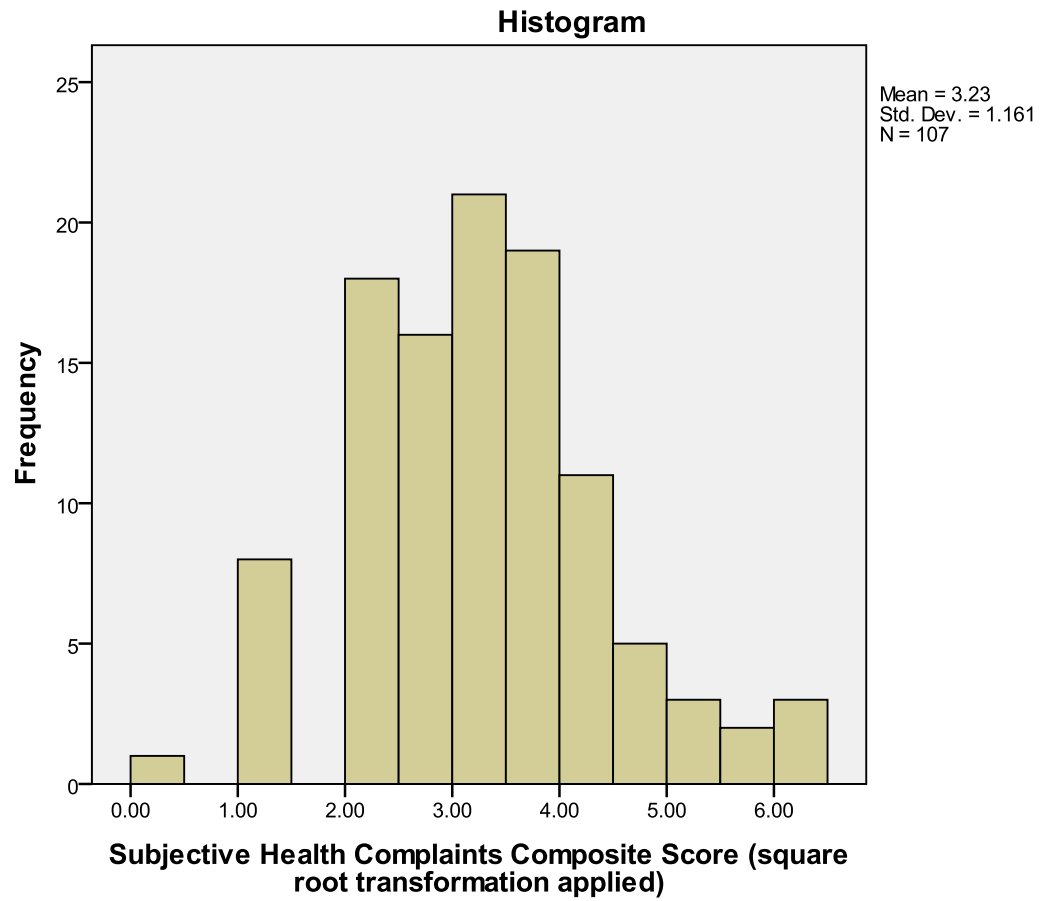


Figure S.17. Histogram of transformed SHCI composite scores (Study 2).

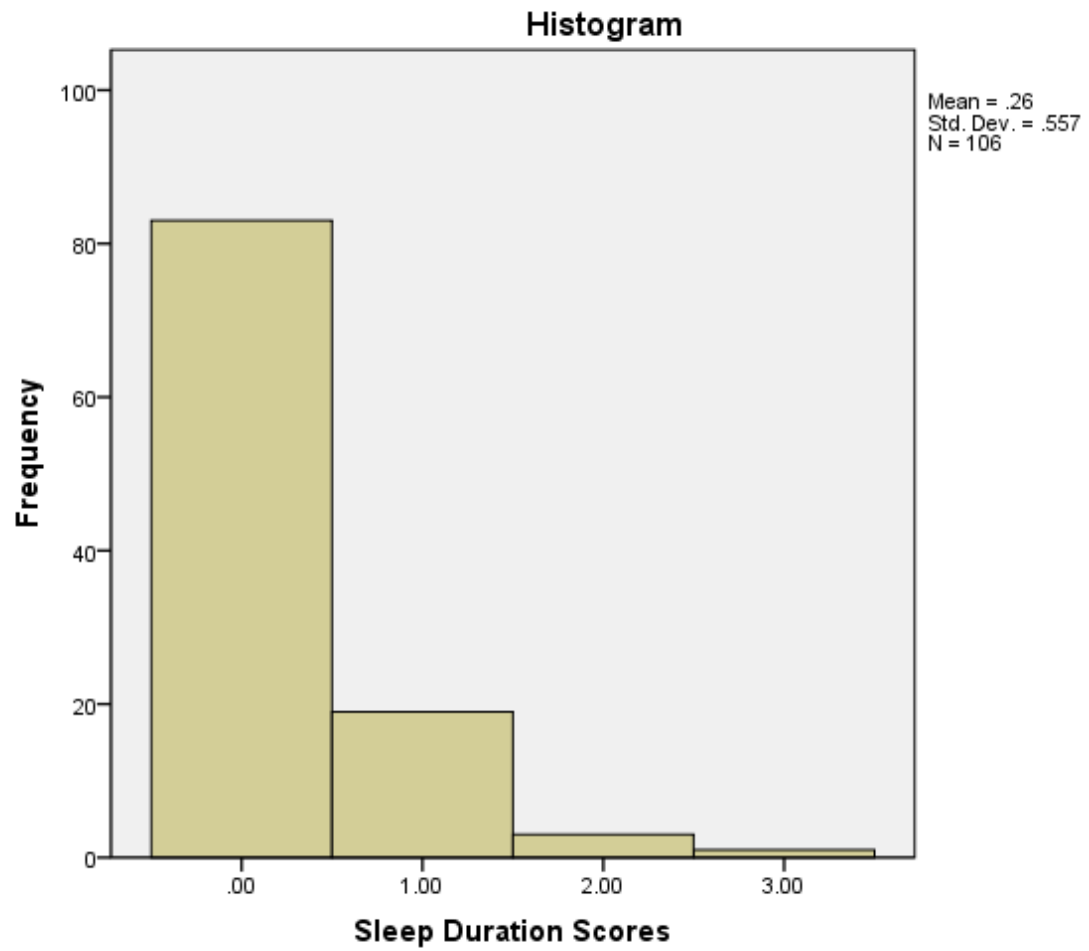


Figure S.18. Histogram of Sleep Duration PSQI Subscale scores.

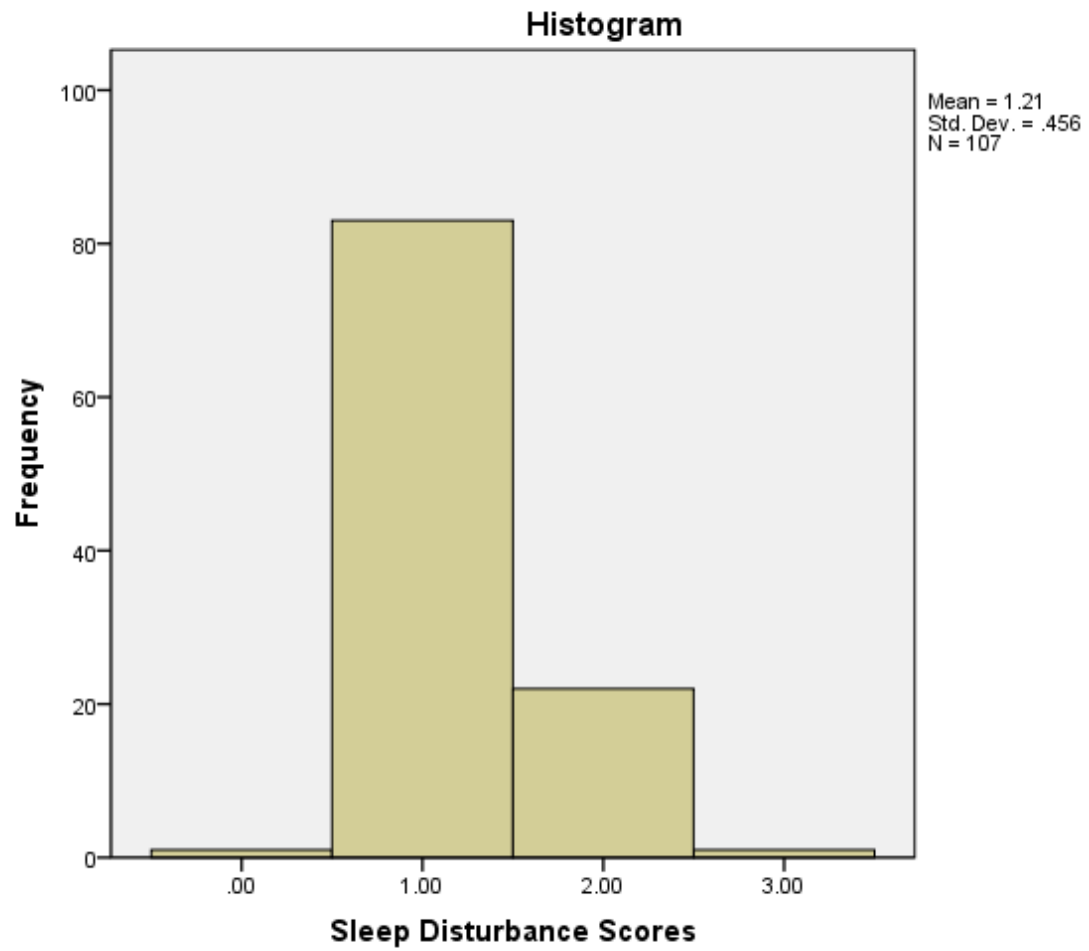


Figure S.19. Histogram of Sleep Disturbance PSQI Subscale scores.

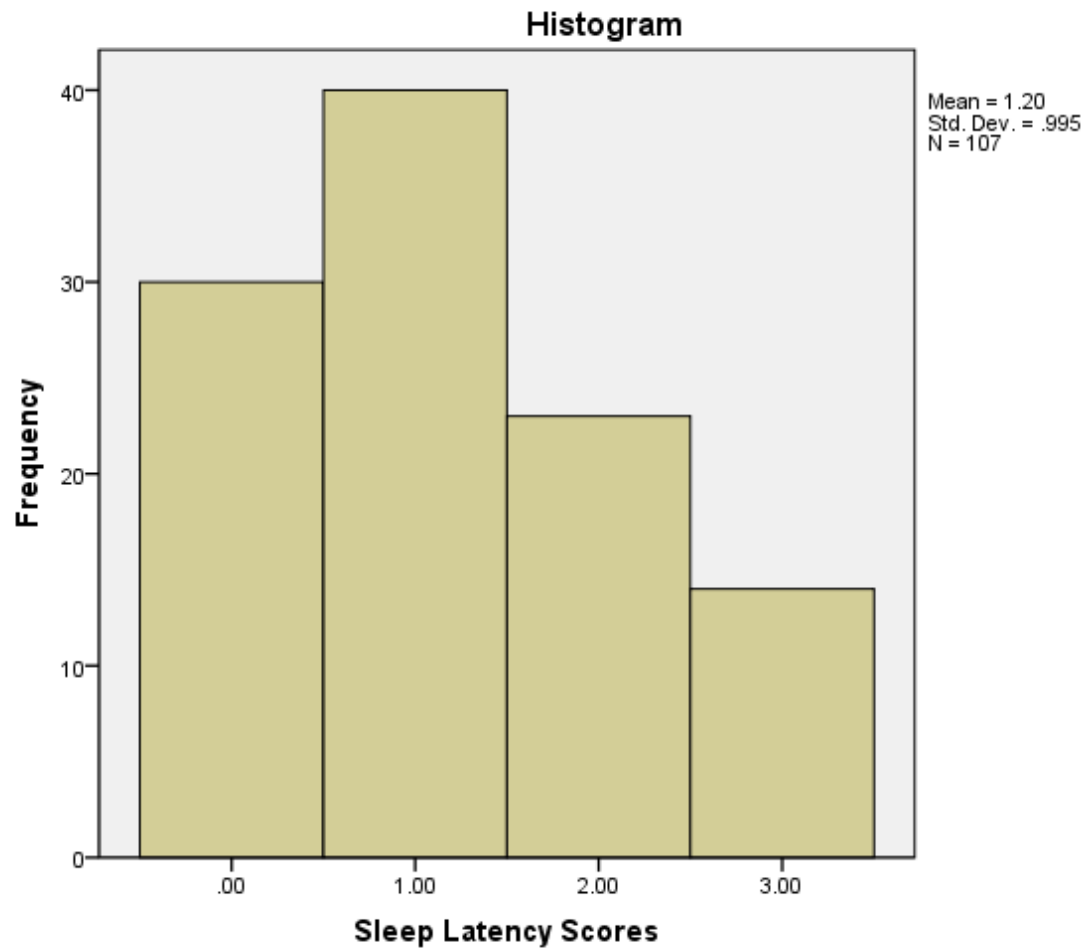


Figure S.20. Histogram of Sleep Latency PSQI Subscale scores.

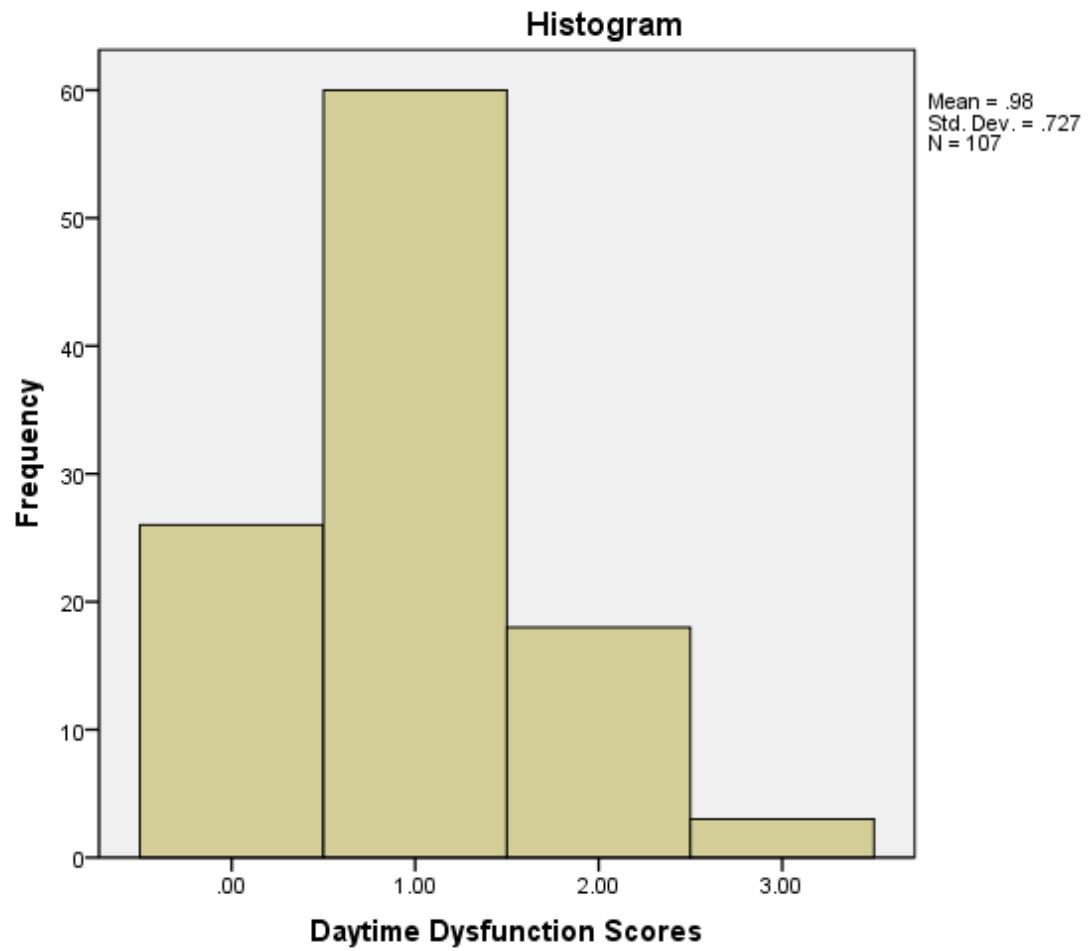


Figure S.21. Histogram of Daytime Dysfunction PSQI Subscale scores.

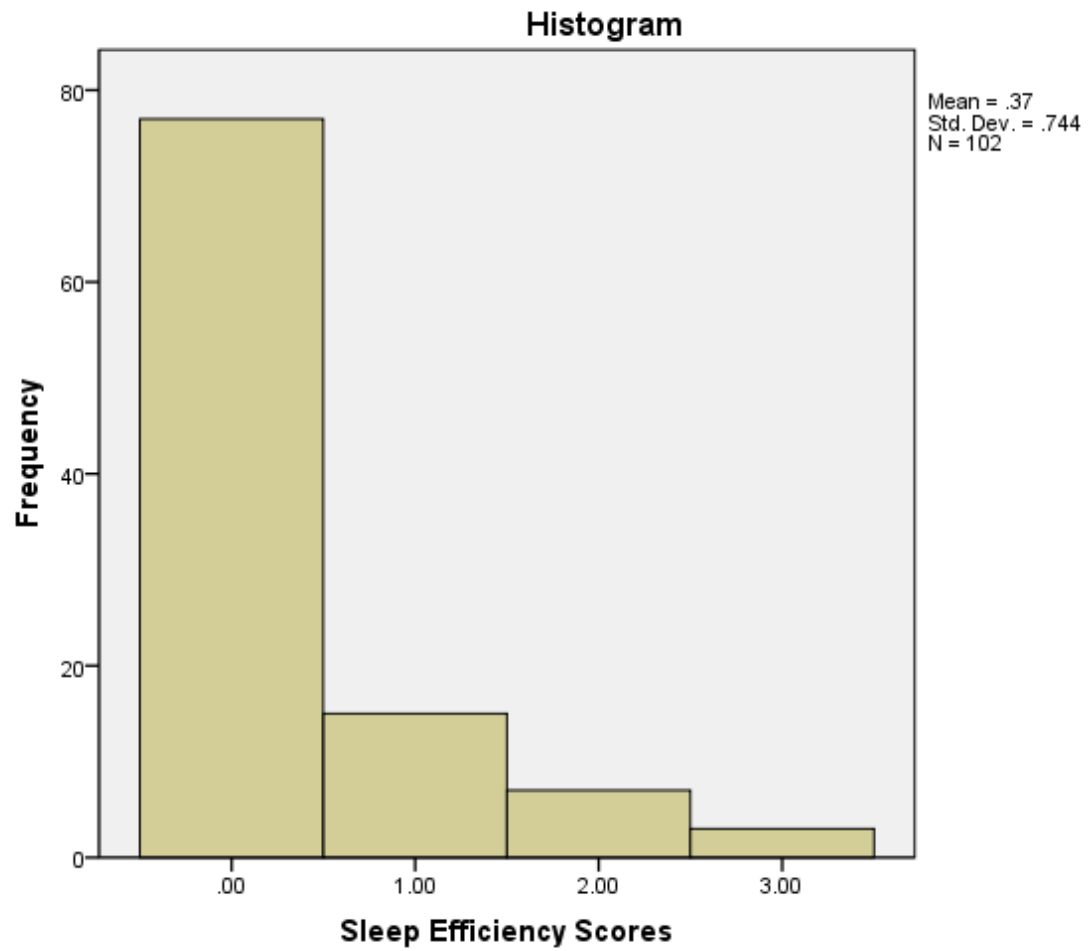


Figure S.22. Histogram of Sleep Efficiency PSQI Subscale scores.

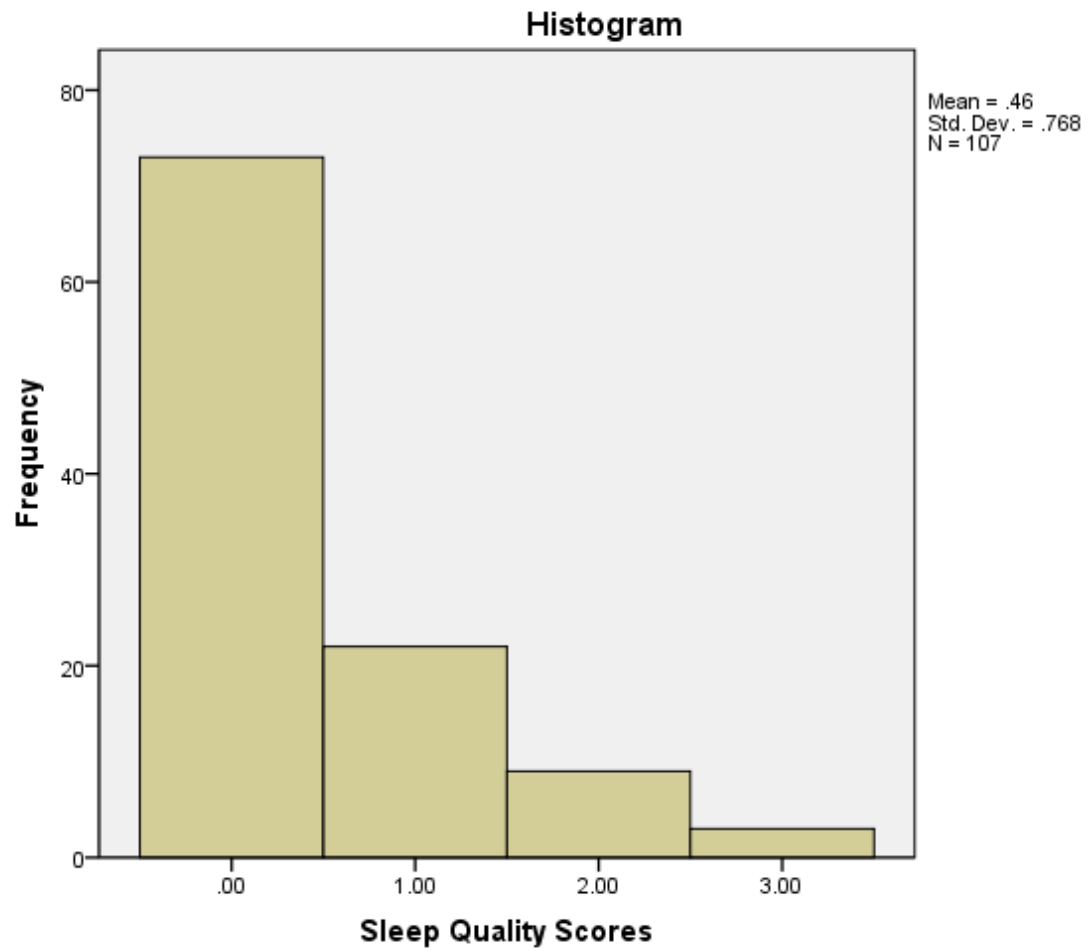


Figure S.23. Histogram of Sleep Quality PSQI Subscale scores.

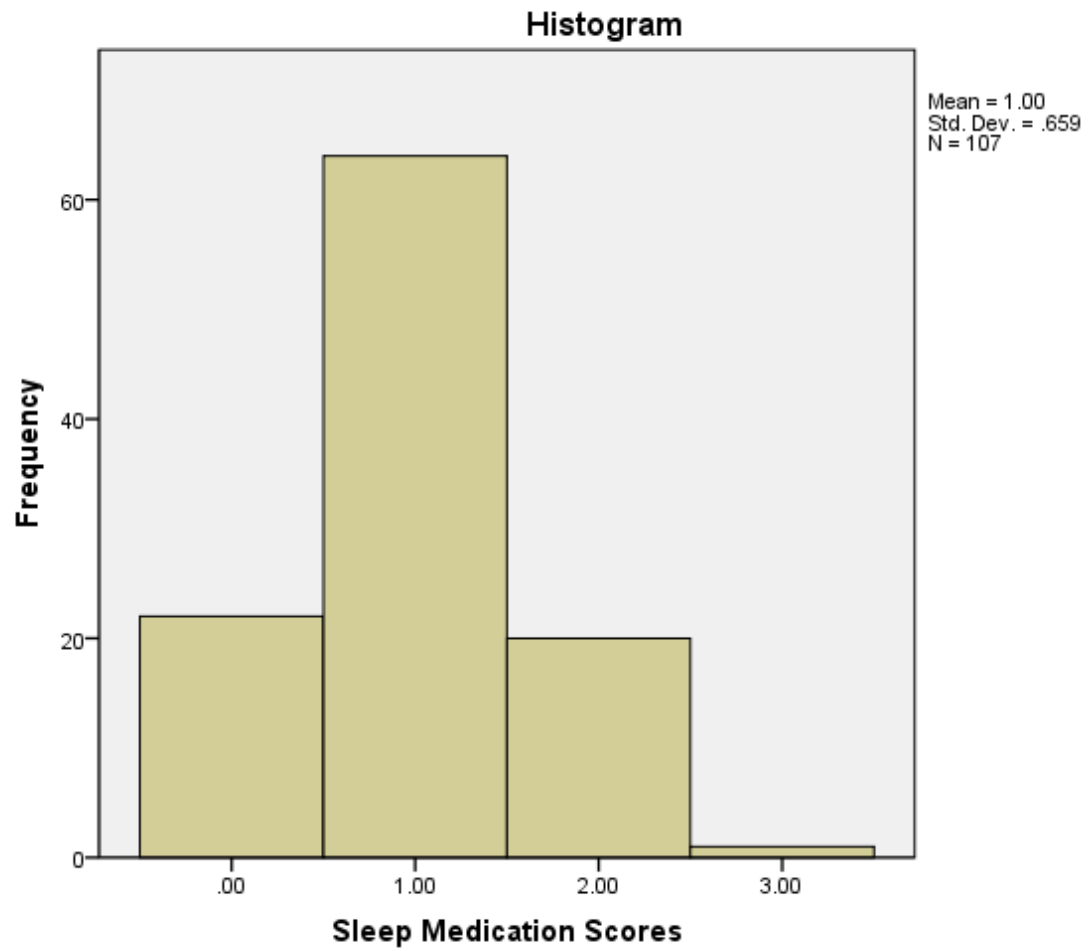


Figure S.24. Histogram of Sleep Medication PSQI Subscale scores.

Table S.4. Hierarchical Linear Regression of Noise Sensitivity and Covariates (including transformed age variable) on CARI

Predictor	β	p	R	R^2	<i>adjusted</i>	F	df	p
				R^2				
<i>Block 1</i>			.219	.048	.016	1.48	3, 88	.225
Gender	.215	.049						
Age ^T	-.062	.559						
Neuroticism	.031	.767						
<i>Block 2</i>			.220	.049	.005	1.11	4, 87	.358
Gender	.222	.053						
Age ^T	-.048	.712						
Neuroticism	.038	.734						
Noise Sensitivity	-.009	.953						

Age^T = square root transformation of age variable

Appendix T: Scatterplots of the Relationship between CAR values and Perceived Stress

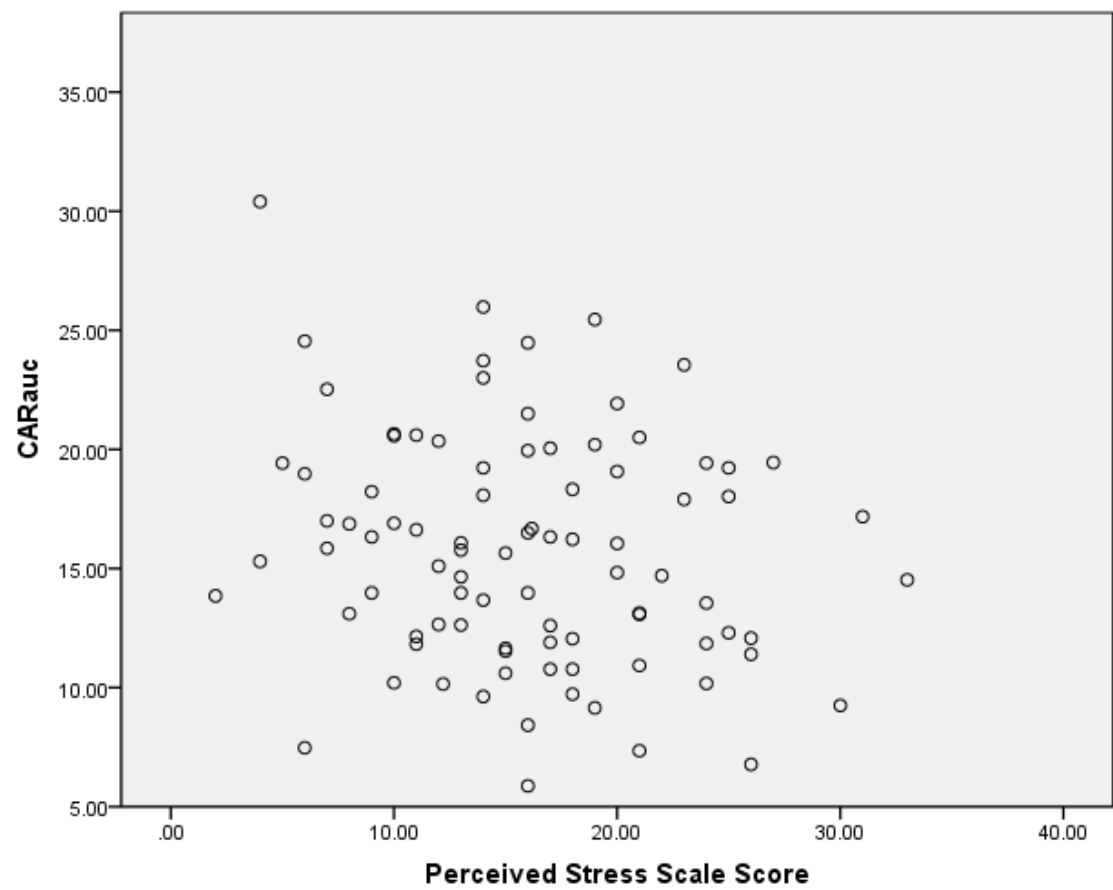


Figure T.1. Scatterplot of the relationship between CARauc and PSS.

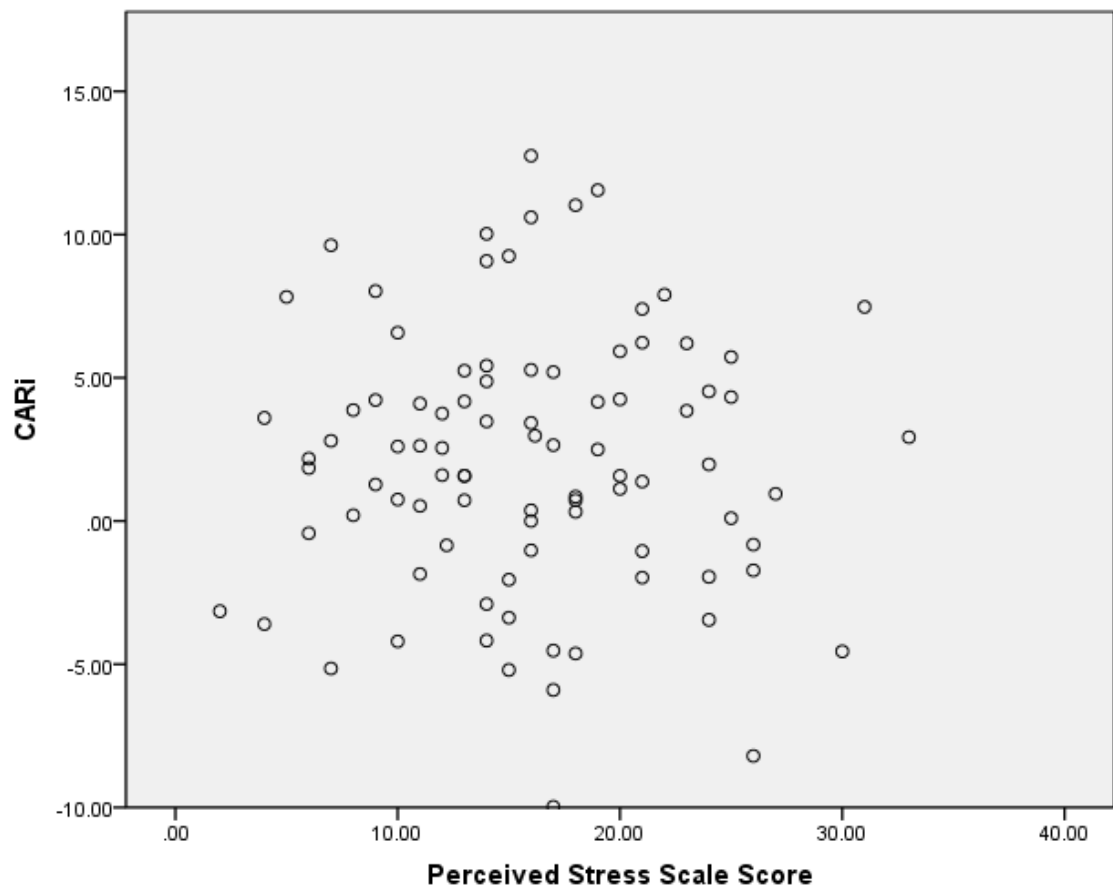


Figure T.2. Scatterplot of the relationship between CARi and PSS.