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A Pilot Study: High Intensity Intermittent Training to combat chronic stress in the New Zealand Police.

A master's thesis presented in partial fulfilment of the requirements for the degree of Masters of Sport and Exercise.

At Massey University, Wellington, New Zealand

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Abstract

**Background:** Stress is increasingly being reported to be associated with immunosuppression, disease progression and psychological illness (Sergerstrom & Miller, 2004). Police are considered to be employed in a highly stressful occupation and due to this are at a heightened risk for developing negative chronic stress related disorders (de Terte & Stephens, 2014). There are known strategies to combat stress such as exercise. However, in moderately active individuals a more vigorous exercise programme is needed to reduce the effects of stress. High intensity intermittent training (HIIT) could be a potential stress reducing mechanism especially due to its success in treating obesity, weight loss and cardiac issues (Gibala, Little, MacDonald, & Hawley, 2012; Schoenfeld & Dawes, 2009).

**Aim:** To see the effects of HIIT on chronic stress indices in the New Zealand Police.

**Method:** Using three single case studies, this study employed a 10-week HIIT intervention measuring markers of stress such as Perceived Stress Scale scores, cortisol levels and associated blood immune markers at baseline and post-intervention.

**Results:** Chronically stressed police officers displayed high perceived stress scale scores and compromised immune functioning due to decreased cortisol secretion and increased eosinophil count. Post-intervention decreased perceived stress, normalised cortisol levels and reduced immune inflammation markers.

**Conclusion:** High Intensity intermittent training decreases perceived chronic stress while also providing further evidence for the relationship between systemic inflammation and mental disease.
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Approval for this study was via the Massey University Ethics Committee for the experiments carried out and described in this thesis.
# Contents

Abstract ................................................................................................................... ii
Acknowledgments ................................................................................................... iii
Contents .................................................................................................................. iv
List of Figures and Tables ...................................................................................... vi

## Chapter One Introduction ................................................................................. 8

### Chapter Two Literature Review ..................................................................... 10
  1. What is chronic stress ................................................................................. 10
  2. Stress Response Systems ........................................................................... 14
  3. Hormones associated with the stress response system ......................... 17
  4. Bodily responses to stress ........................................................................ 20
  5. How exercise has worked for mental illness such as depression and anxiety 25
  6. The use of HIIT to decrease effects of chronic stress and normalise immune function? 33
  7. High Intensity Intermittent Training (HIIT) .............................................. 34

## Chapter Three Method ................................................................................. 41
  1. Design ........................................................................................................ 41
  2. Hypothesis .................................................................................................. 42
  3. Ethics ......................................................................................................... 42
  4. Participants ............................................................................................... 42
  5. Measures ................................................................................................... 45
  6. Study Protocol .......................................................................................... 49
  7. High Intensity Intermittent Training Intervention ...................................... 51
  8. Data Analysis ............................................................................................. 53

## Chapter Four Results .................................................................................... 56
  1. Anthropometric Data .............................................................................. 56
  2. Perceived Stress Scale ............................................................................. 57
  3. Cortisol ..................................................................................................... 60
  4. Full Blood Count and Lymphocyte Subset analysis .................................. 63
  5. Summary of results ................................................................................. 65

## Chapter Five Discussion ............................................................................... 67
  1. General Discussion ................................................................................... 67
  2. Limitations ............................................................................................... 79
  3. Future Directions ..................................................................................... 81

## Chapter Six Conclusion ............................................................................... 83

## Chapter Seven References .......................................................................... 84

## Chapter Eight Appendices ........................................................................... 100
  Appendix A: Participant Information Sheet .................................................. 101
  Appendix B: Consent Form ........................................................................... 106
  Appendix C: Personal Letter of Invite ............................................................ 107
  Appendix D: Perceived Stress Scale ................................................................. 108
  Appendix E: Revised Mini Nutritional Assessment ......................................... 110
  Appendix F: International Physical Activity Questionnaire (IPAQ) ................. 111
  Appendix G: Methodology Flow Dig .............................................................. 114
  Appendix H: Exercise Intervention sample of day one of weeks one, five and ten. 115
  Appendix I: Exercise Demonstration Sheet .................................................... 118
Appendix J: Heart Rate recording sheet ...................................................... 120
Appendix K: Participant Information Consent Form for Participants Section ................. 121
List of Figures and Tables

Figure 1: The neuroendocrine response to psychological stress. Adapted from Foley and Kirschbaum (2010). .................................................................................................................. 16

Figure 2. Perceived Stress scale scores and VO_{2}\text{\_max} changes over the course of baseline, mid- and post-intervention testing .......................................................................................................................... 58

Figure 3. Brinley plots comparing participant averages of PSS scores at baseline and post-intervention. .......................................................................................................................... 60

Figure 4. Cortisol slope measures for all three participants calculated from averages at baseline, mid- and post-intervention. ................................................................. 62

Figure 5. Model of stress and inflammation. .................................................................................................................. 78

Table 1. Anthropometric data for participants one to three from baseline to post-intervention testing. .......................................................................................................................... 56

Table 2. Calculations and observations made at the completion of the Beep Test, in order to determine the HR and VO_{2}\text{\_max} ranges for each participant. ................................................. 57

Table 3. Mean scores for each of the three participants PSS questionnaires at baseline and post-intervention .................................................................................................................. 59

Table 4. Cortisol levels (nmol/L) of all three participants at both morning (08.30am) and afternoon (15.30pm) at baseline, mid- and post-intervention. .................................. 60

Table 5. Baseline, mid intervention and post testing cortisol level(nmol/L) averages at both morning (8.30am) and afternoon (15.30pm). ......................................................... 61

Table 6. Immune Protein Blood markers at baseline, mid- and post-intervention for each of the three participants. ..................................................................................................... 63

Table 7. Eosinophil count (x10^{9}/L) of all three participants from baseline, mid- and post-intervention. .................................................................................................................. 65
Chapter One Introduction

Police officers due to the nature of their work are considered to be involved with potentially stressful and traumatic situations (de Terte & Stephens, 2014). Negative aspects of the work such as boredom, lack of respect from members of the public, excessive paperwork, contact with the public that is sometimes negative and confrontational, shift work, threats of violence and the militaristic nature of policing structure are among some of the work stressors police may find themselves faced with (Burke, 1998). Researchers have long argued that job performance can be affected when officers experience chronic stress (He, Jihong, & Archbold, 2002). As a result of these stressors symptoms and reactions may present themselves by lowering an individual’s work performance, increasing absenteeism, in addition to adverse physiological states such as burnout, fatigue, depression, anger and psychosomatic and physical conditions such as headaches and ulcers (Burke, 1998; He et al., 2002). Due to the compelling evidence that demonstrates how at risk police officers are this study decided to use the New Zealand Police to trial a 10-week high intensity intermittent training (HIIT) intervention in its ability to reduce perceived stress.

For this particular study we asked to work within one unit of the New Zealand Police to try and minimise their stress through HIIT. As we already know that exercise can help people who are depressed and stressed. The issue here is instead what exercise protocol can we institute in people who already perceive themselves to be moderately fit. Therefore, a more vigorous exercise approach was utilised, as it had seen benefits in other areas such as weight loss and improving cardiovascular health. This project was also influenced by emerging research that systematic inflammation either causes or is influenced by mental health disorder (Chrousos, 2009; Dantzer, O’Connor, Freund, Johnson, & Kelley, 2008). Therefore the stress hormone cortisol was measured due to its immunosuppressive role (Cohen et al., 2012; Glaser & Kiecolt-Glaser, 2005; Herbert & Cohen, 1993; McEwen, 1998). In addition, key blood inflammation markers were measured to show either heightened inflammation or suppressed immune function as part of the chronic stress negative spiral.
This study therefore examines the direct effect HIIT has on individuals perceived stress scale scores, cortisol levels and immune markers before, during and after a 10-week HIIT intervention. The focus of this pilot study was to discover whether HIIT training could successfully be used to decrease police stress levels. The study aims to show through the use of a series of single case studies that HIIT can decrease the symptomology associated with chronic stress and reduce perceived stress. Furthermore, it will aim to investigate the association of perceived stress score reductions, cortisol levels and immune responses.
Chapter Two Literature Review

i. What is chronic stress
Stress is often defined as any actions or situations that place physical or psychological demands on a person, where their equilibrium becomes unbalanced due to either good or bad, pleasant or unpleasant situations (National Institute of Mental Health, 2016; Nieman, 1998; Selye, 1978). Selye (1978) stated that no matter whether the demands are good or bad the body undergoes virtually the same nonspecific responses to the varying positive or negative stimuli that are acting on the body (Jackson, Morrow, Hill, & Dishman, 2004; Nieman, 1998; Selye, 1978; & Selye, 1956). For the purpose of this study stress will be defined as the:

Real or perceived imbalance between the environmental demands required for survival and the individuals capacity to adapt and respond to these requirements (Davis, Sherbourne, Marshall, & Grant, 1999; Selye, 1978).

As the definition portrays stress encompasses a wide range of demands that could be placed on the body. Due to one’s personal reactions to stress are numerous. An individual’s stress response is primarily dependent on two factors. These being how an individual perceives and interprets the situation if as a threat, then behavioural and physiological responses occur; if the situation is not perceived as a threat then the response either does not occur or the response is quite different and more benign (McEwen, 1998). The second individual difference that occurs concerns the body itself. For example in people who are relatively fit and active, they can handle strenuous exercise far better than those who are not, hence the bodies response to the stressor will be different for the physically fit person as opposed to the other (McEwen, 1998).

Stress can fall into a variety of different classifications, all of which create a physiological and psychological response within the body. These stressors can be from routine stress at work, family and daily responsibility, stress brought on by negative life events such as losing a job, traumatic stress brought on as a result of accidents, natural disaster, death or war (Powers & Dood, 2009; Mental Health Foundation Victoria, 2014). Moreover, as can be seen for any type of event or
performance in a person life there will be some level of stress. So for every individual there is an optimal level of stress needed to be successful. As there is an optimal stress level there must be good and bad stress. Eustress is good stress that motivates and inspires a person and is where we optimally function (Nieman, 1998; Powers & Dood, 2009; Selye, 1978). Distress however, is considered bad stress and can be acute; where it is intense but disappears quickly or chronic; where it may not be as intense but lasts for a prolonged period of time (Nieman, 1998; Powers & Dood, 2009; Selye, 1978). When stress is at this point a person’s health and performance decreases due too mental, emotional and physiological limitations.

Although some stress is necessary in a person’s life, stress that occurs for a prolonged period of time can often lead to ill health (Jackson et al., 2004; Krantz, Thorn, & Klecrolt-Glaser, 2013; Sergerstrom & Miller, 2004). This kind of prolonged stress is often termed chronic Stress. Chronic stress results from repeated exposure to situations that leads to the release of the body’s stress hormones (Krantz et al., 2013). Chronic stressors tend to be open ended, usually using all of the body’s resources to cope but not showing any sign of resolution (Wheaton, 1994, 1997). Most generally chronic stress occurs due to an array of problems that people may constantly face in life such as: work, family, deadlines, conflict, decisions, uncertainty and illness. What makes chronic stress so different to event stress, is that event stress has an end point. Event stress occurs suddenly without warning having a clear and defined onset, followed by a short length and an eventual resolution. An example of this kind of event could would be a car accident. The opposite of this is chronic stress, the onset of chronic stress is relatively slow and gradually over time continues with no end point. This type of chronic reoccurring stress causes wear and tear on the body and the mind. When stressors are always present and a person feels that they are constantly under attack the stress response remains activated due to the on-going- open ended problem (Wheaton & Montazer, 2010).

Exposure to chronic stress may cause physical, behavioural or emotional manifestations such as; anxiety, depression, cardiovascular dysfunction; such as hypotension, metabolic disorders; such as obesity, diabetes, metabolic syndrome
and neurovascular diseases such as; osteoporosis in addition to sleep disorders, fatigue, fibromyalgia and increased risk of catching infectious diseases (Chrousos, 2009; Sheldon Cohen et al., 2012; Glaser & Kiecolt-Glaser, 2005; Miller, Chen, & Zhou, 2007; Puterman et al., 2010)

Wheaton (1997) and Wheaton and Montazer (2010) explain that there are seven basic themes that reflect the fundamental forms of chronic stress. These seven forms are: (1) threats (2) demands, (3) structural constraints, (4) under reward (5) complexity, (6) uncertainty, (7) conflict. These are discussed in detail by Wheaton (1997) and the descriptions below are based off Wehaton (1997) and Wheaton and Montazer’s (2010) outline for each theme.

**Threat:** Often chronic stress can be caused by the continuing possibility or expectation of potential harm and threat. This such stressor is one that can be faced by people in relationships of abuse, life in areas of high crime and life in a war zone. The stress is not the issue of actual danger it is rather the constant presence of the possibility of danger that cannot be avoided or controlled. Threat also exists in the form of pressure, when the number of demands on a person across a role begin to compromise with their ability to perform in any of their roles. For example, when a person is overloaded due to excessive demands, there is a threat to the normal operation of their social role which is essential to their well-being. In these cases, the threat operates apart from the actual fulfilment of the threat and this is the fundamental part of what is stressful about the situation.

**Demands:** Demands and the inability to keep up with the demands placed upon a person is a common term used for defining the nature of stress. Often a person feels there is too much to do and that they are being pulled by a multitude of demands that cannot be ignored. Excessive demand is often at the heart of a stressful situation and is often in conjunction with other features of chronic stress.

**Structural Constraint:** There are problems posed by the consequences of a person’s structural environment that can leave an individual with reduced opportunities, choices and alternatives. This theme is key as it is important to realise that stressors can also come for a variety of structural constraints. These constraints can come in many forms, from rules and regulations that prevent a
person from dealing with a problem quickly to social disadvantage due to inequality.

*Under reward:* Under reward is when the reduced outputs form a relationship relative to the inputs. Often under reward can happen at work, where a person may feel that they are not being paid enough for the time and effort they put into their role, or being paid less than someone who has the same qualifications due to discrimination. Under reward can also occur in relationships, where a person may feel that they contribute significantly more than the other and that they are now not getting what they deserve.

*Complexity:* Complexity can represent a form of excessive demand, or direct conflict of responsibility across roles. Complexity of routines across social roles is one form of chronic stress. For example, when one is a parent there are a range of issues involved in every day such as child care arrangements, work demands and unpredictable event occurring these all begin to make a person’s life very complex. As a result, a larger amount of time out of a person’s day is spent arranging and making plans to ensure that every issue and task is handled. Over recent years’ complexity has become an increasing source of stress due to social changes. An important part of complexity is that it is a set of daily hassles that cannot be scheduled for.

*Uncertainty:* Closely related to complexity is the stressor of uncertainty. Complexity and uncertainty feed off each other in the idea that uncertainty multiples complexity and that complexity multiples the effect of uncertainty. Uncertainty is a common part of life and becomes a stressor when a person desires or needs a resolution of an ongoing issue. Uncertainty is a feature in many social situations and social relations and at times can become a defining feature. Uncertainty also involves unwanted waiting for an outcome. Often in these cases the decision is out of the person’s hands and no action on their behalf can change the outcome. If there is no time period of an expected outcome uncertainty can progressively get worse.

*Conflict:* Conflict at work, in relationships and families are a normal part of life and can often be regular, due to differences in viewpoints, values, goals or desires.
However, these conflicts become long term due to the inability to agree. Often the fear of bringing the conflict up and the constant enactment of the conflict become a source of stress. The lack of resolution and the basic fundamental differences of goals and values can lead to this becoming a chronic stressor.

These seven categories have been conceptualised again more recently to fall into fours broader areas; (a) persistent life difficulties or chronically stressful situations; (b) role strain, including the strain within a role as well as the strain of multiple roles; (c) chronic strains that derive from societal responses to characteristic of a person and (d) chronic community wide strains for example living in a high crime neighbourhood (Wheaton and Montazer, 2012).

\[ \text{ii. Stress Response Systems} \]

An individual’s stress response system is complex. When an individual is experiencing a stressful situation or life event various stress response systems are activated. Two key mediators in the body’s stress response system are the Hypothalamic-Pituitary-Adrenal Axis and the Sympathetic Adrenal Medullary Axis. Both of these axes play a key role in an individual body’s response to stress (Foley & Kirschbaum, 2010; Tsigos & Chrousos, 2002.).

\[ \text{Hypothalamic-Pituitary-Adrenal (HPA) Axis and Sympathetic Adrenal Medullary Axis (SAM).} \]

Stress triggers the activation of the hypothalamic-pituitary adrenal axis (HPA) and the sympathetic adrenal medullary axis (SAM). The HPA axis is responsible for the neuroendocrine adaptation component of the stress response. (Tsigos & Chrousos, 2002). The HPA axis controls the long term stress response via the secretion of glucocorticoids (Wetherell et al, 2006). The activation of the HPA axis is governed by the secretion for corticotropin-releasing hormone (CRH) and vasopressin (AVP) from the hypothalamus, this in turn stimulates the secretion of glucocorticoids (otherwise known as cortisol in humans) from the adrenal cortex (Juruena, Cleare, & Pariante, 2004; Tsigos & Chrousos, 2002). Glucocorticoids then interact with their receptors in multiple tissue targets including the HPA axis, where they are responsible for the feedback inhibition of the secretion of adrenocorticotropic hormone (ACTH) from the pituitary and CRF from the hypothalamus (Juruena et al., 2004; Padgett & Glaser, 2003).
The sympathetic adrenal medullary axis is a critical element in the integrated physiological response to stress (Foley & Kirschbaum, 2010; Kunz-Ebrecht,
Mohamed-Ali, Feldman, Krischbaum, & Steptoe, 2003; Lundberg, 2005). The Sympathetic Nervous System (SNS) originates in the posterior hypothalamus and innervates the adrenal medulla. Sympathetic activation stimulates the adrenal medulla to release adrenaline. Both the release of adrenaline and cortisol target a number of tissues such as leukocytes that respond by releasing cytokines in times of stress (Padgett & Glaser, 2003). Exposure to stressors is attended by the secretion of epinephrine (EPI) from the adrenal medulla and the release of epinephrine from the sympathetic nerves (Konarska & McCarty, 1989). The release of these hormones promotes the immediate fight or flight response to stress by effecting the cardiovascular system and the release of energy in the form of glucose and free fatty acids (Lundberg, 2005; Wetherell et al., 2006).

These systems have been extensively described by Foley and Kirschbaum (2010); Gupta, Aslakson, Gurbaxani and Vernon (2002); Mccarty, Horwatt and Konarska (1988), Miller, Chen, Zhou (2007) and Raison and Miller (2003). Due to the complexity and breadth of these systems they will not be discussed at length here, for a more in-depth look at these systems however a large amount of literature exists and should be referred to.

The hormonal response system (HPA and SAM axes) to stress can be activated by a number of stressors. In order to activate these system neurons in the paraventricular nucleus of the hypothalamus secrete the CRH. This hormone then travels to the anterior pituitary gland, which responds to its presence by secreting ACTH. The ACTH then travels through the adrenal glands to synthesize and release glucocorticoids. Cortisol is a key hormone of the stress response due to its widespread regulatory processes.
**Figure 1:** The neuroendocrine response to psychological stress. Adapted from Foley and Kirschbaum (2010).

Figure one shows how the hypothalamic pituitary axis and the sympathetic adrenal medulla axis both originate in the hypothalamus. The medial hypothalamus is in control of the secretion of glucocorticoids (i.e. cortisol), through the release of CRF, which stimulates the release of ACTH into the systemic circulation (Foley & Kirschbaum, 2010; Miller et al., 2007). The adrenal cortex then responds to the ACTH by releasing cortisol. The SNS originates from the posterior hypothalamus and innervates the adrenal medulla to release adrenaline (Foley & Kirschbaum, 2010). Both cortisol and adrenaline then target various cells and tissues in the body (Miller et al., 2007). Cortisol for example influences the
metabolism in the cells, the fat disruption and the immune system, whereas adrenaline in involved in increasing heart rate and blood pressure (Lundberg, 2005).

**iii. Hormones associated with the stress response system**

A key hormone associated with these HPA axis and SAM is cortisol. This hormone plays a key role in the regulation and maintenance of an individual’s stress response. Without cortisol the body cannot effectively regulate the inflammatory response through the suppression of pro inflammatory cytokines such as tumour necrosis factor alpha, interleukin-1 and interleukin-6 (Fries, Hesse, Hellhammer, & Hellhammer, 2005). This allows inflammatory process to take over.

a. Cortisol

Cortisol is a steroid hormone that is commonly referred to as the “stress hormone”, and is secreted by the adrenal glands, to repair tissue and maintain blood glucose levels (Stone et al., 2001). Cortisol is a glucocorticoid hormone synthesized from cholesterol by enzymes in the adrenal cortex, and is regulated by the HPA axis. Cortisol is secreted on a diurnal rhythm (Chung, Son, & Kim, 2011). More specifically, cortisol, has 10-15 secretory episodes over the 24hr period with the strongest activity shortly after awakening with a steady decreased thereafter (Ockenfels et al., 1995; Stone et al., 2001). The trough of cortisol secretion is reached around midnight (Ockenfels et al., 1995; Stone et al., 2001). This pattern is typically robust and in non-ill individuals experiencing typical daily life there should be little variation from the peak to trough profiles (Stone et al., 2001). However, in individuals where they are subjected to environmental extremes such as torture extreme physical distress, major alteration in diet, shift work and psychological stress there may be alterations such as lowered morning cortisol peaks, flattened daily profiles reduced difference between morning to evening cortisol levels and heightened cortisol levels (Kotozaki et al., 2012; Stone et al., 2001). Absence of the diurnal cycle due to flattened cortisol cycles can be indicative of a dysregulation of the HPA axis due to either associated physiological and psychological distress and or altered environmental circumstances (Stone et al., 2001).
Cortisol plays a major role in the functioning of the immune systems and its secretion is controlled by the HPA axis. When the HPA axis is functioning normally cortisol will return to normal levels once the hypothalamus and the limbic system have sensed that enough cortisol was released (Kunz-Ebrecht et al., 2003). When a person in depressed, anxious or chronically stress cortisol levels can become abnormal resulting in unusually high or low levels (Fries et al., 2005; Miller et al., 2007; Tops, Riese, Oldehinkel, Rijsdijk, & Ormel, 2008). In someone who are chronically stressed or have mental illness such as depression the important feedback loop between cytokines and cortisol is affected (Kunz-Ebrecht et al., 2003).

Normally pro inflammatory cytokines such as interleukin-1 and interleukin-6 are potent activators of the HPA axis (Kunz-Ebrecht et al., 2003). Cortisol in turn negatively controls cytokine production and by this exact mechanism it is able to minimise the inflammatory process (Chrousos, 2009; Raison & Miller, 2003; Sergerstrom & Miller, 2004). In patients with depression often high levels of interleukin-1 and interleukin-6 are presented as a results of higher levels of cortisol (Kunz-Ebrecht et al., 2003). However, blunted cortisol response to stress can also occur. This can be due to the persistent or inhibited secretion of cortisol in those who are stressed (Sergerstrom & Miller, 2004; Tops et al., 2008). Prolonged cortisol secretion leads white blood cells to mount a counter regulatory response by downregulating cortisol receptors and therefore decreasing their sensitivity (Kunz-Ebrecht et al., 2003; Sergerstrom & Miller, 2004). The downregulation reduces the capacity of cells to respond to anti-inflammatory signals and instead allows the cytokine, mediated inflammatory processes to take hold (Sergerstrom & Miller, 2004).

As an example of this in studies of those with stress related disorders such major depression have reported reduced glucocorticoid responsiveness and therefore reduced glucocorticoid response (Holsboer, 2000; Pariante & Miller, 2001; Raison & Miller, 2003). As glucocorticoids are the most potent anti-inflammatory hormones recued response means that the suppression and stimulation of pro and inflammatory mediators is decreased (Fries et al., 2005). While glucocorticoids promote Th2 development by enhancing interleukin-4 and interleukin-10
secretion by macrophages and Th2 cells, they inhibit inflammatory responses and suppress the production and release of pro inflammatory cytokines thus restraining the inflammatory action (Fries et al., 2005; McEwen et al., 1997). Major depressive disorders have shown blunted stress reactivity. Depressed individuals exhibit relatively flat and unresponsive patterns of cortisol secretion (Burke, Davis, Otte, & Mohr, 2005). There are three reasons as to why this may occur 1) down regulation secondary to persistent hypocortisolism, 2) primary alteration in the genetic structure to glucocorticoid receptors and 3) a decrease in glucocorticoid receptor function (Pariante & Miller, 2001). It was concluded by Pariante and Miller, (2001) that given the importance of glucocorticoids and the glucocorticoid receptors in the regulation of the stress induced HPA axis that it was logical to hypothesis that disruption of glucocorticoid action through altered function of the glucocorticoid receptors may contribute to psychological disease expression.

Although there is a lot of research surrounding the effects of suppressed cortisol in depression when looking specifically at chronic stress many studies focus on Post Traumatic Stress Disorder (PTSD) patients. Yehuda, Bolsonedu, Lowry and Giller (1995), showed that combat veterans with PTSD suppressed cortisol to a greater extent in comparisons with normal controls. The experiment used a dose of dexamethasone to examine the glucocorticoid receptor and cortisol response. Dexamethasone is an anti-inflammatory and immunosuppressive steroid (MedSafe, 2011). The experiment results displayed dose related hypo-suppression of cortisol as a response to the administration of dexamethasone (Yehuda, Boisoneau, Lowy, & Giller, 1995). This data indicates that patients with PTSD suppressed cortisol to a greater extent than combat veterans without PTSD (Yehuda, Boisoneau, et al., 1995). They also noted low baseline levels of basal plasma cortisol taken at 8am, which were consistent with low urinary and plasma cortisol levels in patients with PTSD (Yehuda, Boisoneau, et al., 1995).

Additionally evidence examining cortisol levels in holocaust survivors with PTSD, showed decreased urinary cortisol excretion in comparison with two control groups and that low cortisol levels were an indication of current and chronic PTSD (Yehuda, Kahana, Binder-Brynes, & Southwick, 1995). Aerni and colleagues (2004), showed that the administration of a low dose of cortisol in patients experiencing
traumatic PTSD symptoms decreased the cardinal symptoms of their PTSD thus indicating the prominent role cortisol has in controlling one's psychological response to stress. When facing a lowered cortisol and glucocorticoid receptor sensitivity cortisol cannot complete its necessary immunological and psychology buffering processes (Aerni et al., 2004). This study by Aerni (2004), indicates that with an increase of cortisol, patients' symptomology especially in relation to traumatic memories is decreased.

Evidence has also been presented in the way of a more normalised population sample that has not faced trauma or major traumatic experiences. Decreased cortisol at awakening has been seen in patients with chronic fatigue, fibromyalgia, hypertension, rheumatoid arthritis and asthma as a direct result of chronic inflammation caused due to the prolonged chronic stress their body is facing (Chida & Steptoe, 2009; Fries et al., 2005). Yang and colleagues (2001) examined stress and cortisol levels in emergency and general nurses. Emergency department workers showed significantly lower urinary cortisol at morning testing in comparison to general ward nurses. With a negative correlation between self-perceived stress scores and cortisol levels. Emergency ward nurses saw themselves as experiencing greater perceived stress and directly correlated with this was lowered cortisol levels (Yang et al., 2001). It is suggested that cortisol levels are decreased in chronically stressed individuals due to reduced glucocorticoid receptor response (Cohen et al., 2012). Stress-related disorders lead to insufficient glucocorticoid signalling due to decreased hormone production (hypocortisolism) and/or decreased glucocorticoid responsiveness (Raison & Miller, 2003). The down regulation in cortisol receptor sensitivity (glucocorticoids responsiveness) puts an individual at the risk of developing autoimmune diseases, coronary heart diseases and rheumatoid arthritis due to excessive and chronic nonspecific inflammation (Sergerstrom & Miller, 2004).

iv. Bodily responses to stress

Stress has many physical effects, that while not measured as part of this study, are worthy of note to show the far-reaching physiological effects of severe and/or chronic stress. With the activation of an individual’s stress response, there are a variety of physiological changes that occur within the body. These changes usually
only occur for a short period of time. However, when one is chronically stressed the consistent activation of the stress systems and therefore the body's physiological response can create long term irreversible changes to the body.

a. Immunological effect

Pre modern times the ability for an individual to respond to environmental stressors enhanced the chances of survival. These responses included increased oxygen and glucose to the heart and large skeletal muscles as a result of the “fight or flight” response. It is thought that immune responses may have been a necessary part of this stress response. In addition to the risk of the stressful situation fighting or fleeing used to carry the risk of injury and therefore the susceptibility for an individual to pick up infectious agents into the bloodstream or develop infectious skin wounds. Therefore, stress induced changes in the immune system that could accelerate wound repair and prevent infections from taking hold would be necessary (Sergerstrom & Miller, 2004). However, in the modern world, it is not often that an individual finds itself in a situation that would evoke the evolutionary fight or flight response that their ancestors needed. The trouble is however that the physiological response continues to be the same despite the changes in human environments and the stressors they face (Sergerstrom & Miller, 2004).

Therefore, stress has a still effects the immune systems due to its evolutionary response. Both glucocorticoids (cortisol) and catecholamine’s (adrenaline and dopamine) influence trafficking and or the functions of leukocytes as well as suppressing the secretion of pro inflammatory cytokines (Chrousos, 2009). Both hormone families induce a systematic switch from Th1 response (this is cellular immunity) to Th2 response (this is hormonal immunity) (Chrousos, 2009). The pro inflammatory cytokines stimulate the stress system, also at multiple levels in both the central and peripheral nervous system including the hypothalamus, pituitary glands and adrenal glands (Chrousos, 2009). This stimulation of the central and peripheral nervous system leads to the secretion of glucocorticoids and consequently suppresses the inflammatory reaction (Chrousos, 2009). All of these actions form the negative feedback loop that minimise the immune system from an
exacerbation of its inflammatory processes, therefore protecting the body (Chrousos, 2009).

As discussed the HPA plays a key role in the stress response, therefore it is also crucial to the immune mediated inflammatory reaction (Raison & Miller, 2003). Glucocorticoids that are released as part of the HPA axis are invaluable anti-inflammatory and immunosuppressive agents (Miller et al., 2007; Raison & Miller, 2003). Glucocorticoids act on lymphocytes in order to induce the production of Th2 cytokines and decrease the production of Th1 cytokines (Murali, Hanson, & Chen, 2007). Specifically by suppressing interleukin-2 production thus preventing the presence of cytokines necessary for TH1 development and reducing the ability of interleukin-12 to stimulate the production of other cytokines (Murali et al., 2007). By suppressing the immune activation of these cells glucocorticoids inhibit the production of cytokines and other mediators of inflammation and also cause resistance to cytokines (Raison & Miller, 2003). Glucocorticoids affects subgroups of T lymphocytes these being: type 1 helper lymphocytes, and stimulate apoptosis (cell death) of eosinophils. This means glucocorticoids are key mediators in eosinophil counts (Fulkerson & Rothenberg, 2013). Activated glucocorticoid receptors also inhibit pro inflammatory activity of many growth factor cytokines by blocking the transcription necessary for the expression of or actions of these substances (Xavier, Anunciato, Rosenstock, & Glezer, 2016). With a decrease in glucocorticoids and in glucocorticoid receptors due to prevailing stress the responses discussed above are altered therefore placing the individual at a heightened risk for infection of disease.

Eosinophils are another key mediator of the immune system. They are a bone-marrow derived, peripheral blood and tissue granulocyte prominent in allergic and inflammatory responses (Davis & Rothenberg, 2014). Eosinophils rarely increase in activity and number in healthy individuals (Kita & Bochner, 2013). The life cycle of an eosinophil is divided into bone marrow, blood and tissue phases (Kay & Corrigan, 1992; Kita & Bochner, 2013). Although formed in the peripheral circulation eosinophils are primarily tissue dwelling cells. The normal range for eosinophils is 0-0.5x10^3/mm^3 and exhibits a diurnal rhythm in humans (Kita & Bochner, 2013). The lowest and highest levels are seen in the morning and
evenings respectively (Eng & DeFelice, 2016). Eosinophils are a source of a number of pro inflammatory cytokines. For example, eosinophils produce cytokines that affect cells and those that influence other immune cells. In those that influence immune cells, eosinophils modulate their functions. Eosinophil production and recruitment is due to Th2 lymphocyte stimulation, with the help of cytokines IL-3, IL-4, IL-5, IL-13 and granulocyte macrophages colony stimulating factors (Eng & DeFelice, 2016; Rosenberg, 2016). Interlukin-4 and Interleukin-13 stimulate immunoglobulin E production and promote eosinophil recruitment and IL-5 mediates enhanced eosinophil production. IL-5 has an impact on eosinophils biology. IL-5 is produced primarily by activated Th2 lymphocytes, mast cells and natural killer cells and by eosinophils themselves. IL-5 promotes eosinophil mediated activation and recruitment into tissues and acute inflammatory responses (Rosenberg, 2016).

In order for eosinophils to traffic to inflamed tissues CC chemokine receptors (or beta chemokine receptors) are essentials. CC chemokine receptors are integral membrane proteins that bind to cytokine, they are necessary for trafficking eosinophils due to the fact that eosinophils express a receptor that binds eosinophil-specific chemokines (Eng & DeFelice, 2016). In a normal healthy individual mucosal surfaces of the lower gastrointestinal tract, thymus and uterus are major targets for eosinophil tissue migration (Eng & DeFelice, 2016; Rosenberg, 2016). However, when inflammation regulated by Th2 cells occurs eosinophils will move to other areas including the lungs and the skin (Eng & DeFelice, 2016). Once there the eosinophil attaches to extracellular matrix proteins which bind to specific tissues. The eosinophils then receive a signal to degranulation and release its preformed components. The cytokines and chemokines that are released promote longevity of eosinophils in tissues which aids in their signalling, activation and survival. Additionally, these proteins target any foreign antigens, promote inflammation to the area and cause significant damage to surrounding structures. Eosinophils are immunomodulatory mediators both for health and in response to dysfunction and diseases (Rosenberg, 2016). While the presence of eosinophils is not uncommon in healthy individuals these granulocytes play a central role in inflammation and allergic processes. Higher levels of eosinophils in the peripheral blood or certain tissue types typically
correlates with the protective mechanisms of the body against asthma, allergies, atopic dermatitis, parasitic infections, gastrointestinal disorders and more (Eng & DeFelice, 2016).

T helper cells are another key part of an individual’s immune response. T-helper cells help the activity of other immune cells by releasing T cell cytokines. These cells specifically help suppress and regulate the immune response. Mature T helper cells express the surface protein CD4 and are referred to as CD4+ T cells. CD4+ T cells have a predefined role as helper T cells within the immune system. Stressful life events are believed to suppress a person’s resistance to infection. When the demands on a person are exceed the person’s ability to cope with the situation, a psychological stress response composed of a variety of psychological and physiological responses occurs (Cohen, Tyrell, & Smith, 1991).

b. Musculoskeletal System

When the body is fronted with stress, muscles tense up. Muscle tension is a reflex reaction to stress and is the body’s way of guarding and protecting it against pain and injury (Tovian, Thorn, Coons, Labott, Burg, Surwit and Bruns, 2016). After the stressful episode the muscles then release their tension. However, in situations where a person has chronic stress the muscles are more or less in a constant state of tension and guardedness. When the muscles in the body are like this for an extended period of time, this may trigger other reactions of the body like tension-type headaches and migraines and headaches associated with constant muscle tensions in the shoulder, neck and head (Tovian et al., 2016).

c. Cardiovascular

The heart and blood vessels are two components of the cardiovascular system that work together to provide the body with nourishment and oxygen to the organs of the body. These two components are also involved in the body’s stress response. In situations where the person faces acute stress that is short term or momentary such as things like meeting deadlines, being late or avoiding accidents there is an increase in heart rate and stronger contractions of the heart muscles (Torpy, Burke, & Glass, 2007). Stress hormones; adrenaline and cortisol act as messengers for these effects (Engler & Engler, 1995). In addition to this,
blood vessels that direct blood to the larger muscles and the heart dilate, this
increases the amount of blood that is pumped to these parts of the body therefore
increasing the person’s blood pressure (Bernardy & Friedman, 2015; Kulkarni,
O’Farrell, Erasi, & Kochar, 1998). This is known as the fight or flight response. The
fight-or-flight response is thought to be a the body’s preparation for muscular
activity in response to a perceived stressor (Everly & Lating, 2013b). The
mechanism allows the individual to fight or flee. This system is described in great
detail by Everly and Lating, (2013b) so only a brief explanation will be made here.

Everly and Lating (2013b) describe the fight-or-flight response as involving the
sympathetic nervous system and with the onset of stress the response is
associated with specific physiological actions. This is primarily caused by release
of adrenaline and epinephrine from the medulla and adrenal glands. The release is
triggered by acetylcholine released from preganglionic sympathetic nerves. These
hormones initiate the immediate physical reactions by increased heart rate,
cardiac output and breathing rate while also constricting certain blood vessels and
tightening the muscles while also increasing stimulation to the skeletal muscle in
preparation for action (Everly & Lating, 2013b). There is additionally decreased
flow to the gastrointestinal system, kidneys and skin (Everly & Lating, 2013b).
Once the acute stress episode has passed, the body returns to its normal state.
However, in people with chronic stress, or a constant stress experienced over a
prolonged period of time it can contribute to long term heart problems and
inflammation in the endothelial of blood vessels (Black & Garbutt, 2002; Everly &
Lating, 2013a). The consistent increase in heart rate and elevated stress hormones
and blood pressure can be detrimental to the body. In the long term this can lead to
hypertension, heart attack or stroke (Everly & Lating, 2013a).

v. How exercise has worked for mental illness such as depression and anxiety.
Over the past decade it has been proven that exercise can have a positive effect on
one’s mood and wellbeing (Moses, Steptoe, Mathews, & Edwards, 1989). Studies
have indicated that exercise may be useful in the treatment of depression and the
reduction of psychological distresses like stress and anxiety (Daley, 2008).

a. Exercise to help treat depression and anxiety
Depression and anxiety are becoming increasingly common in the general population. Between 1990 and 2013, the number of people suffering from depression and anxiety increased by nearly 50% from 416 million to 615 million (World Health Organization, 2016). The treatments for depression and anxiety are multiple and each have their own degree of effectiveness. Exercise is becoming a more frequent treatment choice as it has been shown to effectively decrease symptoms associated with depression and anxiety while also improve physical health and cognitive functioning (Broman-Fulks, Berman, Rabian, & Webster, 2004; Carek, Laibstain, & Carek, 2011; Kvam, Kleppe, Nordhus, & Hovland, 2016).

In recent years studies have indicated that exercise can cause consistent reductions in anxiety and depression (Motta, Kuligowski, & Marino, 2010). There are a variety of different psychological theories as to why exercise works as a form of treatment, some of these include the distraction hypothesis, self-efficacy theory, mastery theory, and the social interaction hypothesis.

The distraction hypothesis implies that exercise is used as an activity of diversion where the person’s attention is drawn away from the unpleasant stimuli they are experiencing (Faulkner and Biddle, 2004). In general, the use of distracting activities like exercise have proven to be more successful in reducing depression and anxiety as opposed to more self-focused activities like journal keeping (Morrow & Nolen-Hoeksema, 1990). Exercise allows distraction away from pain, anxiety and/or depressive thoughts thus leaving the person with a positive affect after exercise (Bahrke & Morgan, 1978). It is for this reason that exercise could be used to distract individuals from the stressful problems they face by providing them with task.

Banduras (1997) proposed self-efficacy theory suggests that individuals who master something through the use of cognitive skills such as effort and persistence in the face of threatening and difficult situations eventually master a skill (Bandura, 1977). Bandura explains how a depressed person can often be inefficacious to bring about positive and desired outcomes in their lives and have a low efficacy to cope with the symptoms they face (Craft & Perna, 2004). This leads to negative ruminations, and negative evaluations of one’s self. It is thought that exercise provides a learning environment through which self-efficacy can be enhanced as a
result of the accomplishment of an exercise experience leading to increased self-confidence, self-efficacy. Exercise is a way of interrupting the maladaptive thought process of depressive and anxious individuals it is though therefore that it may work in a similar way for stress. As exercise increases an individual’s self-efficacy, it is thought that through this increase previously intimidating or stressful tasks no longer elicit such extreme catecholamine reactions (Bandura, 1990). In addition to this, exercise allows an individual to be successful and exercise control over some part of their life (Bandura, 1990).

Finally, the social interaction hypothesis states that exercise while in a group or the personal attention a person gets while exercising, could be the cause behind the antidepressant effect (North, McCullagh & Tran, 1990). Mason and Holt (2012) analysed thirteen different studies covering a range of physical activity methods with a variety of mental health issues. Out of the thirteen studies, nine studies explicitly reported the importance of social interaction and inclusion as part of their involvement in exercise (Mason & Holt, 2012). The 2015 the New Zealand Ministry of Health survey investigating the Green Prescription programme saw that the encouragement, motivation and supportive, helpful support workers was one of the main reasons for satisfaction with the programme. Along with this, 33% of participants exercised with family, 26% with friends and 21% as part of a Green Prescription Programme group. This recent evidence supports the idea that social support could be a key reason as to why people stay involved with such initiatives (Johnson and Woods, 2015).

Physiological explanations of the effect of exercise on anxiety include the biochemical hypothesis which suggests that exercise enhances the brains aminergic synaptic transmissions, leading to increased arousal and attention. The biochemical hypothesis is the idea that two biochemical compounds react when exercising and thus cause an antidepressant effect; these being monoamines and endorphins. Three monoamines neurotransmitters, serotonin, dopamine and epinephrine have been said to have an antidepressant effect when exercising (North et al., 1990). The hypothesis predicts that the underlying basis of depression is a deficiency of noradrenergic and or serotonergic systems (Delgado, 2000). It is hypothesised that depletion of the monoamines; adrenaline and
serotonin along with that the reduced availability of monoamine neurotransmitters leads to depression (Boer, Slaap & Bosker, 1998). The biochemical hypotheses states that exercise leads to an increase in the availability of these brain neurotransmitters (Craft & Perna, 2004). These neurotransmitters have been seen to increase in plasma and urine following exercise. However, whether the same increase occurs in the brain is still unknown and does not take into account the effect of systematic inflammation which is emerging as having a negative effect on mood. Methodological issues have prevented this line of research from advancing due to the invasive methods needed to obtain samples. It has been observed in chronically exercised rats that there is improved emotionality and epinephrine levels (Tharp & Carson, 1975 ). This enhancement might then result in increased perception of self- efficacy and lowered anxiety and depression.

The endorphin hypothesis also endeavours to explain the effect of exercise as an anti-depressant and anxiety management tool. This hypothesis predicts that exercise has a positive effect on depression and anxiety due to the increased release of beta endorphins (North et al, 1990). The association between exercise and the release of endogenous opiates such as beta endorphins consequently improves mood and induces a feeling of well-being (Craft & Perna, 2004; Daley., 2008). Animal research suggests that the increase of blood level endorphins during vigorous exercise may produce a state of euphoria and or elevated mood (Martinsen, 1987 ). However, it is reminded that although both blood levels of endorphins and mood are increased in association with exercise, this does not establish a causal link (Martinsen, 1987 ). Several studies have also shown increases in plasma endorphins following acute and chronic exercise, yet it is still unclear if this increase is directly related to the decrease in depression (Bortz et al., 1981; Markoff, Ryan, & Young, 1982 ). Overall the endorphin hypothesis suggests that beta-endorphins are produced throughout the body during exercise and these endorphins decrease pain and create a euphoric state they may counter the uncomfortable states associated with anxiety and decrease feelings of unhappiness.
De Moor, Deem, Stubble, Boomsma, and De Geus (2006) examined whether anxiety could be reduced through exercise. Their study consisted of participants with anxiety disorder. The study showed that those adolescents and adults between the ages of 14-24 who exercised regularly reduced their level of diagnosed anxiety-based disorders (De Moor, Beem, Stubble, Boomsma, & De Geus, 2006). In a sample of the US population between the ages of 15-54 regular physical activity was associated with significantly decreased prevalence of current major depression and anxiety disorders (Goodwin, 2003). In addition, evidence indicates that habitual exercise correlates with heightened mental health and wellbeing as well as reduced feeling of anxiety. Another study additionally demonstrated that an increase in maximal cardio respiratory fitness and habitual exercise being associated with lower depressive symptoms and greater overall emotional wellbeing (Galper, Trivedi, Barlow, Dunn, & Kampert, 2006).

vi. *The effect chronic stress is having on Police and emergency services.*

Police are the front line of our emergency and disaster services. They often have to respond quickly with limited regard to their personal safety. While they work to keep our communities safe, thought must be given to the constant and unrelenting stress effects on their physiological and psychological wellbeing. The New Zealand Police’s most recent workplace survey showed important results in regards to workplace stress and wellbeing. In 2016 they released a workplace survey using the IBM Kenexa State Sector Benchmarks to evaluate its performance. The benchmark database consisted of 19 state sector organisations that conducted workplace surveys in the past two years using a total of 35 questions. The 2016 survey had participation from 8,445 employees for the New Zealand police contributing to a total response rate of 70.3% (NZ Police, 2016). The data collected by the New Zealand Police suggested that work related job stressed scored 12.7 points below the state set bench mark and that in regards to the wellbeing of its staff the New Zealand Police also scored below the bench mark by 12.9 points (NZ Police, 2016). This data shows that employees within the New Zealand Police are facing considerable stress in relation to their role which is consistent with past literature discussed below indicating that police work in a high stress potentially physiological and psychologically damaging occupation (de Terte & Stephens, 2014; Huddleston, Stephens, & Paton, 2007).
There are two perceptions in regards to what is believed to be the driving force behind stress in police organisations (Buker & Wiecko, 2007). One category stems from police work itself, these job stressors include use of force, exposure to violence and danger, dealing with confusing situations and shift work (Burke & Mikkelsen, 2005). The second category stems from the organisational aspects of police work; these being poor management, inflexible hierarchical structure and roles, lack of communication, organizational changes and career plateauing (Burke & Mikkelsen, 2005; Crank & Caldero, 1991; Martinussen, Richardsen, & Burke, 2007). It is also significant to note that organisational stressors have been seen to impact on the experiences and attitudes of police offers more than the stressor associated with specific task of being a police officer (Kop & Euwema, 2001). Although there is minimal evidence in New Zealand a recent study by Garbarino et al., (2013) tested whether stress variables were predictors of absenteeism in police officers part of a special force unit called on to maintain law and order in all public events that’s take place in Italy. Their study confirmed the existence of an association between stress and sick leave (Garbarino, Cuomo, Chiorri, & Magnavita, 2013). In addition, Garbarino, Cuomo, Chiorri and Magnavita (2013) reported that in law enforcement tasks lower levels of social reward and high levels of effort and over commitment were associated with a higher level of mental health symptoms. These findings strongly indicated that work-related stress may play a role in the development of mental health disorders in police officers (Garbarino et al., 2013).

Police suffer from relatively high rates of diseases such as; obesity, diabetes, heart diseases, depression, anxiety and other symptoms of excessive stress (Jackson & Maslach, 1982). It has been recorded that police have more stress-related physical and psychological complaints and problems than workers in most other professions (Hart, Wearing, & Headey, 1995). The failure to cope effectively with stress results in increased rates of heart disease, alcohol and drug abuse, depression, anxiety and suicide, as compared to other general populations (Lord, Gray, & Pond, 1991). Violanti and colleagues (2008), provided worrying results in regards to depressive symptoms. Their research indicated a relationship between depression and suicidal ideation in what is considered a healthy working population such as the police force. It was discovered that depression and suicidal thought pervasion was higher than that of the normal American population (Violanti et al., 2008). Of considerable
concern was the increased prevalence of depression in police women that lead to conclusions suggesting that police organisations should consider further development programmes to assist officers in successfully dealing with occupational factors that may lead to stress, depression and suicidal ideation (Violanti et al., 2008). Furthermore, Vila (2006) displayed results that suggested that police officers in the United States were overly fatigued due to long and erratic work hours, shift work and insufficient sleep. It was concluded that these factors were likely contributors to the elevated levels of psychological disorders and family dysfunction observed among the police in the study (Vila, 2006). More specifically Vila (2006) also stated that sleep played a mediating link between stress arising from traumatic life threatening experiences and somatic symptoms. This evidence indicates that irregular hours affecting sleep, contribute directly to the development of stress due to lack of energy and fatigue inducing symptoms.

Traumatic police work exposure is another reason for increased stress in the police force. More recently it has been stated that of all “high risk occupations” police work is to be considered as one of the most “trauma sensitive” meaning that police officers are at a higher risk of being exposed to a traumatic event while at work (Ide Terte & Stephens, 2014; de Terte, Stephens, & Huddleston, 2014; Huddleston et al., 2007; Kurtz, Zavala, & Melander, 2015). Often traumatic exposure is not so much in regards to dangerous situations police find themselves in but more to do with the nature of the casework police are dealing with. Much research has focused on the traumatising effect police officers experience when working with sex offenders as relative to other duties these type of cases can be particularly traumatizing (Craun, Bourke, Berie, & Williams, 2014; Follette & Polusny, 1994). In more recent years with the development of additional sub units of the police which focus on investigating specific sex crimes the concern for the effect it is having on police officers has increased (Craun et al., 2014). Evidence form Bourke and Craun (2012), examined more than 600 Internet Crimes Against Children personnel within the United States, they found that participants were (1) thinking about work when they were not intending to, (2) easily irritable, (3) feeling emotionally numb and (4) experiencing sleep difficulties. This sort of data verifies the idea that trauma exists in police officers who are not considered to be working in the “front line” of duty but are still subjected to traumatic and psychological challenging cases. Furthermore,
traumatic experiences that occurred at work were more likely to be related to the development of PTSD, adding to the fact that lower social support was related to higher PTSD scores (Stephens, Long, & Miller, 1997). This indicated the need for ongoing social support in order to help police officers handle the traumatic experiences faced while at work, in an attempt to lower the development of PTSD.

In direct relation to the effect of stress Anderson, Litzenberger and Plecas (2002) found that police officers experienced both psycho-social and physical stress on the job as a relation to the trauma they faced. It was stated that police officers suffered anticipatory stress at the start of each shift, while also dealing with the stress of critical incidents which could occur while on their shift, with officers often not recovering from or appropriately dealing with their work stress before leaving their shift (Anderson, Litzenberger, & Plecas, 2002). Additionally, when both police chiefs and front line duty police officers completed surveys asking questions in regards to time spent by average police officers across different law enforcement duties and a section for rating perceived stress levels. Both groups reported suspect pursuit, witnessing traumatic events and physical altercations as the most stressful part of their job (Korre, Farioli, Varvarigou, Sato, & Kales, 2014). Kurtz, Zavala and Melander (2015) looked deeper into the effects of stress in police officers with a study that examined the effects of prior strain on police officer incident rates and psychological and physiological response to stress. It was found that exposure to youth strain events and work-related critical incident strain was associated with increased stress. This study is important as it demonstrates that police history is significant in predicating officer stress, suggesting the police need to consider programmes that identify and assist officers at greater risk of negative stress responses due to their earlier life experiences (Kurtz et al., 2015).

Additionally, research shows that high levels of job related stress and life stress are significantly related to illness for police officers (Tang & Hammontree, 1992). Tang and Hammontree (1992), reported there was a significant correlation with sickness and absenteeism in police officers who presented with high stress. Results from a study by Burke (1994) showed that in older police officers there was an increase in reported sick days, greater use of medication and increased coffee consumption. Additionally to the information that in police officers with a longer tenure in their
present job, reported more psychosomatic symptoms such as stress, psychological burnout, negative feelings and emotional exhaustion and these officers also reported more sick days (Burke, 1994). A further study by Collins and Gibbs (2003) showed that levels of stress related mental illness were continuing to increase in members of the police. This study indicated that in those who reported feeling stressed reacted with negative/withdrawal behaviours such as working harder, taking work home and keeping things to themselves. These persons were also less likely to exercise to decrease tension, more likely to increase smoking consumption and more likely to take their stress out on others (Collins & Gibbs, 2003). These types of behaviours are considered by some researchers as “sickness behaviours”. Sickness behaviours consist of behavioural changes in regards to a reduction in activity, social interaction and energy levels (Maier & Watkins, 1998). This suggests that in order to manage the demands of stress people sometimes turn to behaviours such as increased alcohol consumption, increased smoking and changes in sleep patterns which could all modify the immune response of a person (Kiecolt-Glaser & Glaser, 1988; Sergerstrom & Miller, 2004).

Inability to cope effectively with stressors results in the underlying psychological and psychological outcomes such as depression, anxiety, chronic stress, burnout, decreased immune function, coronary heart disease and hypertension (Anshel, 2000; Band & Manuele, 1987). The prevention in these stress related problems lies in the police’s stress related coping behaviours. This study believes that high intensity intermittent training can be used as a coping mechanism to control perceived stress in the police resulting in increased fitness, better balanced immune function and increased quality of life.

vii. The use of HIIT to decrease effects of chronic stress and normalise immune function?

Exercise is commonly associated with its anti-depressive and anti-anxiety provoking properties (Asmundson et al., 2013; Cooney, Dwan, & Mead, 2014; Daley, 2008; Jayakody, Gunadasa, & Hosker, 2014). Exercise training offers clinical psychologists with a vehicle for nonspecific therapeutic social and psychological processes that may be particularly effective for patients for whom more conventional psychological interventions such as medication are less acceptable
(Salmon, 2001). Accumulating evidence documents the beneficial effects of regular exercise in preventing the metabolic and psychological issues induced by chronic stress (Tsatsoulis & Fountoulakis, 2006). The benefits derive from the central effect of exercise to reduce sensitivity to stress and also peripheral actions influencing the metabolic function (Tsatsoulis & Fountoulakis, 2006). In a recent study of 32,000 individuals enrolled in a health insurance screening programme, it was reported that less physically active persons where twice as likely to report high stress levels (Taylor, 2000). Similarly another study indicated that active persons were less likely to be reactive to stress (Scully, Kremer, Meade, Graham, & Dudgeon, 1998). All the above mentioned evidence suggests that exercise could be a viable treatment for those who are stressed. However, the problem we face is that the New Zealand Police are already meant to be moderately active in order to pass a required fitness test each year. This leads to the question, that if these individuals are already moderately active but still highly stressed, what type of exercise would be most efficient at reducing stress. The answer could lie in High Intensity Interval Training.

viii. **High Intensity Intermittent Training (HIIT)**

a. What is HIIT

High intensity intermittent training otherwise known as HIIT can be broadly defined as repeated bouts of short to moderate duration exercise (i.e. 10 seconds to 5 minutes) completed at an intensity that is equal to or greater than the individuals anaerobic threshold (Laursen & Jenkins, 2002). The American College of Sports Medicine recommends accumulating 150 minutes of moderate intensity exercise of 75 minutes of vigorous-intensity exercise a week to prevent chronic disease (Garber et al., 2011; Haskell et al., 2007). As a perceived lack of time to exercise is a barrier to engaging, HIIT has become a solution to accumulating the necessary amount of exercise needed in the shortest time possible (Cress, Porcari, & Foster, 2015). Each bout of exercise is then separated by brief periods of low intensity or inactivity that allow for partial but not full recovery (Cress et al., 2015; Laursen & Jenkins, 2002). The purpose of using HIIT is to consistently stress the body’s physiological systems to a greater extent than that of endurance exercise (Laursen & Jenkins, 2002). Using a training prescription such as HIIT is different
to commonly prescribed continuous moderate intensity exercise as it requires individuals to work at a pre-selected heart rate (HR) typically at 80-95% of their HRmax with recovery periods aiming to get participants HR down to 40-50% HRmax (Emtner, Heral, & Stalenheim, 1996; Helgerud et al., 2007; Roy, 2013). An initial VO2max test is also used to determine the intensity of the sessions, whereby participants are expected to work at 80-95% of the VO2max (Bacon, Carter, Ogle, & Joyner, 2013; Boucher, 2011b; Shiraev & Barclay, 2012; Talanian, Galloway, Heigenhauser, Bonen, & Spriet, 2007). Due to the various intervals and intensity levels HIIT offers a break for the monotonous regularity of continuous training. Depending on the individual HIIT can be prescribed via three categories: 1) long intervals of 3-15 minutes, 2) moderate intervals of 1-3 minutes, or 3) short intervals of 10 seconds to 1 minute. It can serve as an effective alternative to continuous endurance training inducing superior changes in a range or physiological and performance health related markers in those who are healthy and those with a range of diseases (Gibala, Little, MacDonald, & Hawley, 2012a).

b. Exercise benefits continuous or otherwise

Exercise provides a variety of physiological and psychological benefits for both the normal population and populations struggling with disease and mental illness. Exercise aids in endothelial function of blood vessels. We now know that cardiovascular and peripheral vascular disease is a result of a disrupted endothelial lining of blood vessels (Sando, van Zanten, Metsios, Carroll, & Kitas, 2010), which is termed endothelial dysfunction. Healthy endothelial cells release nitrous oxide for blood vessel dilation which is synonymous with healthy cardiovascular function. Research studies clearly demonstrated that there is a greater improvement in endothelial function after HIIT compared to continuous exercise (Gibala et al., 2012; Kilpatrick, Jung, & Little, 2014). Ciolac (2012) also demonstrated that HIIT was superior to continuous training for improving cardiorespiratory fitness, endothelial function and markers of insulin sensitivity. In addition to decreasing arterial stiffness in hypertensive and normotensive at high risk for hypertension subjects (Ciolac, 2012). Rakovichuk and colleagues (2008) demonstrate that low dose HIIT programme in healthy men and women increased compliance in peripheral arteries. This protocol also increased endothelial
function that was equivalent to changes observed after continuous moderate intensity exercise however, alteration in central artery distensibility may require a longer training stimuli or possibly greater initial vascular stiffness (Rakobowchuk et al., 2008).

Other factors responsible for physiological improvements are due to skeletal muscle adaptation (Gibala & McGee, 2008). Gibala and McGee (2008) consistently found an increased muscle oxidative capacity ranging from approximately 15% to 35% after only six sessions for HIIT over a two-week period. Burgomaster and colleagues (2005) additionally found that short term sprint interval training also increased muscle oxidative potential and doubled endurance time to fatigue during cycling at ~80% VO_{2peak} in moderately active participants (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005). Other studies have found increased skeletal muscle buffering capacity (Weston, Myburgh, Lindsy, Dennis, Noakes and Hawley,. 1997), increased whole body and skeletal muscle capacity for fatty acid oxidation during exercise (Talanian et al., 2007), as well as decreased lactate accumulation and increased ability to store and utilise glycogen (Burgomaster et al., 2005). The Burgomaster et al., (2006) study consisted of participants completing 4-7 bouts of 30 seconds high intensity cycling followed by 4-minutes low intensity recovery, 3 times a week for two weeks. The study concluded that high intensity intermittent training using short cycle sprints decreased net muscle glycogenolysis and lactate accumulation(Burgomaster, Heigenhauser, & Gibala, 2006; Gibala et al., 2012).

High intensity interval training has also been shown to significantly reduce subcutaneous fat, especially abdominal fat, in additional to total body mass and improved fitness (VO_{2max}). A review by Boutcher (2010) indicated that HIIT could result in modest reductions of subcutaneous fat and abdominal body fat in young normal weight and overweight males and females. In comparison to continuous exercise, HIIT burns more calories and increases post exercise fat oxidation and energy expenditure (Shiraev & Barclay, 2012). Trapp and colleagues (2008) used a 15-week HIIT training intervention using women to determine its effects on subcutaneous fat and insulin resistance. The reported results that of all the three conditions (control, steady state and HIIT), HIIT was the only condition where
women displayed a reduction in total body fat, fat mass and trunk fat in addition to a decreased in fasting plasma insulin levels. This study shows that HIIT, 3xs a week for 15 weeks is successful in decreasing subcutaneous fat and insulin resistance (Trapp, Chisholm, Freund, & Boutcher, 2008). In a similar study by Heydari, Freund and Boutcher (2012) using a 12 week HIIT intervention with overweight young males. Their results indicated that HIIT resulted in significant reductions in total abdominal, trunk and visceral fat as well as significant increases in fat free mass and aerobic power were recorded. The loss of fat mass was attributed to the increased capacity for fat oxidation. Overall there is an increased aerobic and anaerobic fitness, decreased insulin resistance, addition to the increased skeletal muscles capacity for fatty acid oxidation and glycolytic enzyme content (Boutcher, 2011b).

c. HIIT vs Continuous training which method is better?

High intensity Interval training (HIIT) confers several advantages over steady state aerobic training. Research indicates that the body undergoes cardiovascular adaptations of a superior fashion compared to traditional protocols (Gibala et al., 2012; Weston, Wisloff, & Coombes, 2014). HIIT has also shown greater improvements in VO$_2$max, endothelial function, blood pressures, cardiac contractility and insulin signalling over continuous training (Ciocci, 2012; Schoenfeld & Dawes, 2009). One of the key changes as a result of HIIT training seems to be aerobic fitness. For example, Whyte, Gill and Cathcart (2010) carried out a HIIT intervention on obese/overweight sedentary men. The study showed that after only 2 weeks of 3x HIIT sessions per week consisting of four to six Wingate tests of 30-seconds “all out” with a 4-minute recovery, VO$_2$max increased by 7% in the studies untrained and overweight men (Whyte, Gill, & Cathcart, 2005). Helgerud and colleagues (2007) demonstrated in an 8-week training intervention study indicating that aerobic high intensity intervals improve VO$_2$max by 5.5% in a group that completed 15-seconds of 90-95% HR max sprinting with 15-seconds active rest and a 7.2% increase in the group that completed 4 minutes of running at 90-95% HR max followed by active rest of 70% HR max repeated four times. In addition, HIIT training has been used for patients with diabetes and in those undertaking cardiac rehabilitation. These HIIT intervention required
longer Wingate-type intervention programmes that lasted between 12 to 24 weeks. The results indicated large increases in VO\textsubscript{2}max of 41% and 46% respectively in these populations (Mourier, Gautier, De Kerviler, & et al, 1997; Wisloff et al., 2007).

Freyssin, Verkindt, Prieur, Benaich, Maunier and Blanc (2012), demonstrated that compared to an 8-week continuous training programme high intensity intermittent training was more effective at improving submaximal exercise capacity such as VO\textsubscript{2}peak. Additionally, HIIT participants increased their walking distance and oxygen consumption (Freyssin et al., 2012). These results were similar to a study by Tjonna, Lee, Rognmo, Stolen, Bye et al., (2008) that showed in participants with metabolic syndrome, HIIT increased VO\textsubscript{2}max more than continuous training (35% vs 16%). Participants in the HIIT condition also showed an increase in the removal of more risk factors that constitute to metabolic syndrome (Tjønna et al., 2008). In addition to physiological changes, HIIT is also associated with greater enjoyment (Bartlett et al., 2011). Bartlett and colleagues (2011) displayed that high intensity intermittent running showed a higher rate of perceived enjoyment when compared to moderate-intensity continuous running. Collectively these results show that participation in differing HIIT interventions for individuals with varying ages, fitness levels and abilities displays significant changes in physiological and psychological wellbeing.

d. Effects of HIIT on Cortisol

As cortisol is the principle glucocorticoid in humans and plays a major role in immune function it is key to mention here how cortisol may be affected by such high intensity intermittent training. Studies have shown that acute exercise generates changes in plasma cortisol concentrations but the amount is entirely dependent on the type of exercise (McGuigan, Egan, & Foster, 2004). Jacks, Sowash, Anning, McGloughlin and Andres (2002), specifically studied the effects of three different exercise intensities on salivary cortisol levels. Participants in this study completed exercise at intensities of 45.0, 60.0 and 75.0% VO\textsubscript{2}peak. The study found that cortisol concentrations were significantly higher at 59-minutes of high-intensity exercise than it was at 59-minutes of low intensity exercise or at rest. Cortisol concentrations were also higher at 20-minutes recovery from high
intensity exercise than it was at rest or after low intensity exercise (Jacks, Sowash, Anning, Mcgloughlin, & Andres, 2002). McGuigan and colleagues (2004), then measured salivary cortisol response to different intensities of resistance exercise. The high intensity resistance exercise protocol consisted of six, ten-repetition sets using 75% of one repetition maximum (IRM), the low intensity resistance exercise protocol consisted of three, ten-repetitions sets at 30% IRM. Results showed that there was a significant 97% increase in salivary cortisol immediately following the high intensity session (P<0.05) whereas, the low intensity exercise session resulted in no significant changes in cortisol levels (McGuigan et al., 2004). Further studies have also indicated a similar response the change in cortisol level due to high intensity exercise, With each study indicating that exercise between 60-80% VO2max created a greater increase in cortisol over when using high intensity intermittent training over the lower intensity sessions (Guseman, Zack, Battaglini, & Hackney, 2008; Nieman et al., 1994). In these particular studies the cortisol was measured as a post exercise measure this demonstrates that HIIT can influence cortisol on an acute basis. It remains to be seen whether an intervention of HIIT over 10 weeks can create a chronic effect.

e. Effects of HIIT on psychological wellbeing

High Intensity Intermittent training has also been shown to produce changes in one’s psychological wellbeing. Past evidence has suggested that lower doses of activity (i.e. Intensity and duration) are more enjoyable for the average person and thus lead to higher involvement and adherence (Ekkekakis & Petruzzello, 1999). Research has indicated that affect which is an individual’s response that occurs reflexively is no different in HIIT than it is in moderate intensity training and is significantly more positive than continuous vigorous intensity training (Kilpatrick et al., 2014; Martinez, 2013). A study by King, Taylor and Haskell (1993), showed that regardless of the intensity, greater exercise participation was significantly related to less anxiety and fewer depressive symptoms. It was concluded that both high and low intensity exercise had the ability to provide participants with psychological benefits (King, Taylor, & Haskell, 1993). A more recent study by Thum, Parson, Whittle and Astorino, (2017) analysed the difference in enjoyment, affect and perceived exertion between moderate intensity continuous training
(MICT) and high intensity interval training. The results demonstrated that there was a higher enjoyment in response to HIIT, with 11 of the 12 (92%) participants preferring HIIT to MICT. It was concluded that although HIIT was more physically demanding by creating greater increased heart rate and rate of perceived exertion, individuals reported greater enjoyment due to its time efficiency and changing stimulus. Although there used to be concern related to intense exercise and its enjoyment factor, recent data suggests that vigorous intensity exercise practiced in an intermittent fashion is very enjoyable and when compared MICT (Bartlett et al., 2011; Martinez, 2013). These small differences such as time efficiency and enjoyment, may make HIIT a successful treatment for a population that is stressed and already considerably time constrained.

Stress can be both beneficial and detrimental to an individual however, a certain amount of stress is necessary for a person to function optimally (David C Nieman, 1998). When however, stress becomes to prolonged and never ending physiological and psychological issues occur. Chronic stress and its affects directly disturb cortisol production and the body’s ability for the immune system to effectively function. Due to this chronically stressed individuals are more susceptible to developing autoimmune disease, heightened allergic reactions and psychological illness (Chrousos, 2009; Miller et al., 2007; Sergerstrom & Miller, 2004). As can be seen from above, populations such as the police are at increased risk for developing chronic stress due to the heightened risk of their employment (de Terte & Stephens, 2014). It is due to the high levels of stress in Police employees that has led to the investigation into alternative interventions in order to decrease vulnerability and increase resilience to stress. We believe HIIT may be an alternative option to helping New Zealand police manage their stress and its associated symptoms based on its effectiveness with other populations. This study questions whether HIIT is an effective mechanism for normalising markers of immune function and decreasing stress symptoms in already moderately fit police who exhibit negative chronic stress symptoms?
Chapter Three Method

i. Design

This study was based on a series of single case study design with subjective and objective measures. Single case design refers to the participant or group of participants under investigation. Single case study design contrasts experimental group design as single subject experiment research provide their own control data for the purpose of comparison within subjects rather than between subjects (Perone & Hursh, 2013; Smith, 2012). This type of design typically includes baseline measures to serve as comparisons between the subsequent stages of the experiment (Smith, 2012).

The central goal of using a single case study design for this intervention was to determine whether there was a causal relationship between high intensity intermittent exercise and decreased stress. In order to maintain the essence of single case study design none of the measures that were collected repeatedly were combined with those from other participants to produce group averages for data analysis (Smith, 2012). Instead single case study design allowed the researcher to compare the treatment effect between individuals in the study with comparisons being made across the experimental conditions (Lanovaz & Rapp, 2016). The behaviour of a participant in one condition was not compared to that of the other participant, but with his or her behaviour in other phases. This is possibly the only relevant comparison, as individual’s behaviour change is only relevant to his or hers own baseline not that of others in the study (Lanovaz & Rapp, 2016; Smith, 2012).

This study is also in its first phase of treatment development and testing as it is a novel intervention (Gallo, Comer, & Barlow, 2013). In order to provide preliminary evidence that the intervention provides meaningful change, this study was undertaken as a pilot study. The pilot study was implemented using a sub section of the police who were a representative of the population for whom the treatment was designed for, this may not be represented of the wider public (Gallo et al., 2013). Using a pilot study also allowed the researcher time to refine the treatment and make necessary changes to procedure before attempting a larger-scale intervention (Clark & Watson, 2016).
ii. **Hypothesis**

By identifying people who are not coping with or are unable to cope with stress, but are already moderately active the effect of high intensity intermittent training (HIIT) on decreasing stress and aiding in the recovering of the immune system was examined. We hypothesise

1. HIIT will have a positive effect and alleviate symptoms of stress by decreasing Perceived Stress Scale Scores.
2. HIIT will help normalise the immune proteins in persons who have abnormal immune system levels/function due to stress and.

3. Cortisol levels of the participants will change to a more normalised level during and after the prescribed 10-week HIIT intervention.

iii. **Ethics**

Ethics was granted by the institutional ethics (Application 16/22) committee. All potential participants signed a consent form after a verbal explanation of the study.

iv. **Participants**

   a. Recruitment

Participants of this study were recruited from one unit of the NZ Police department who were considered to be facing high stress. An invitation to participate and information sheets were distributed to any member of the police who were interested in the study. Members of the New Zealand Police who wished to participate contacted the investigator individually due to confidentiality.

b. Confidentiality

It was originally intended that the three participants would remain anonymous to each other and to those in their work place. However, what was not foreseen was that the chosen participants soon confided in each other and co-workers as to their selection for the study and the study processes. The Police unit targeted in this research is small and close unit, and after discussions with the participants it was decided that the lack of anonymity could not be maintained but would not be
detrimental to the study or participants. Although the participants had been told before volunteering for the study that they could use work time for their trainings their managers still needed to be communicated with as to their involvement in the study. The managers of participants were not provided with any specific information about the study or any details regarding the participants personal stress levels.

c. Inclusion

Inclusion to participate in this study was based on three factors; 1) the participant had to be a part of the unit, 2) older than 18 years, and 3) a score 35 or above on the PSS. This lower cut-off limit was calculated based on adding 2s.d. to the mean score recorded in the Cohen and Williamson (1988) study of the perceived stress scale in a probability sample in the United States of America. Although no cut-offs are suggested by Cohen and Williamson, higher scores indicate more perceived stress with the maximum score being 45. Our lower cut-off score was adjusted down as it was found that participants below the cut-off were reporting feelings and symptoms of high stress levels.

When calculating the lower limit for the study gender bias had not been considered as a result the score for receiving admission into the study was too high. This is substantiated by Cohen and Williamson’s (1988) where there is a clear difference between the male and female scores, with females reporting on average higher stress levels. When Cohen and Williamson (1998) calculated the mean scores in their study males scored an average of 18.8. If 2s.d., were added to this as was done in the study protocol here the cut-off recalculated score for Cohen and Williamson's (1998) study was 32.5. However, the females mean PSS score was 20.2 which was 36 with the addition of 2.s.d. The results of this study linked well with the scores that had been collected from the three potential participants, with the two males scoring lower than the female participant. It has been suggested that this occurs as females report more distress, fear producing and stressful experiences than men, demonstrating a females tendency to experiences negative emotions at a greater frequency and intensity (Kelly, Tyrka, Anderson, Price, & Caroenter, 2008). It can then be concluded that the set limit needed for entry into this study suited females who report high stress and distress, but not males who
routinely respond less to interpersonal stressors (Kelly et al., 2008). It was for this reason that the lower limit PSS score for entry into the study was re-calculated. The new score needed for males and females to gain admission into the study on the PSS questionnaire then became 32. As the purpose of this study was to help those who volunteered and saw themselves as chronically stressed, turning down those who still had high levels of stress but did not necessarily score 35 seemed to contradict the purpose of the study. The supporting of past literature that indicated that males routinely record lower stress than females reinforced this discussion.

d. Exclusion

Exclusion to the study occurred if volunteers were not a part of the chosen unit of the New Zealand Police, or did not score over 32 on the PSS scale. It was important that potential participants understood the large time commitment this study would require. This was clearly outlined to any potential participants in the information sheet, as well as at the interview with the primary researcher. This interview was conducted in order to get the potential participant to complete a PSS as part of the inclusion criteria. Participants had score a PSS score of above 32 to be admitted into the study. We could only interview and test one participant at a time for ethical considerations. If the potential participant did not believe they could fully commit to the training within the study they were also excluded. Participants were excluded if they were injured or lacked a suitable base level fitness as obtained via interview and the International Physical Activity Questionnaire (IPAQ) that would be necessary to complete the HIIT intervention.

e. The participant group

Each of the participants worked for a subset of the New Zealand police. It is for this reason that specific details regarding participant age, job description and type of work cannot be disclosed in order to preserve anonymity. However, it must be noted that these participants all worked a nine to five job. Their roles were predominantly desk jobs with the occasional day of active duty.

i. Participant One
Participant one was female placed in the age range 40-65 which is considered a middle adult as specified by Erikson (1994). Participant one scored 42 on her eligibility PSS questionnaire. She weighed 73kg and had a height of 172cm. The baseline measure of fitness for participant one was a level of 6.0 for the beep test indicating a VO₂ Max 32.50 ml/kg/min, which is considered average for her age (Thompson, Gordon, & Pescatello, 2010). Responses to the International Physical Activity Questionnaire indicated that she had normal physical activity habits (i.e.), she was not sedentary but also not over training. The Mini Nutritional Assessment showed that participant one had normal nutritional and hydration habits. All pretesting indicated that participant one was a relatively healthy individual who could manage a 10-week exercise intervention.

**ii. Participant Two**

Participant two was male considered a middle adult in the age range of 40-65 as specified by Erikson (1994). Participant two scored 33 on his eligibility PSS questionnaire. He weighed 113kg and had a height of 182cm at baseline testing. Participant two reached level 6.2 on his first beep testing indicating a VO₂ max of 35.66 ml/kg/min and considered to be fair for his age group (Thompson et al., 2010). Participant two responses in the IPAQ indicated moderate physical activity habits as he was training for a cycle race at the time of interview. Participant two also had normal nutritional consumption based on his MNA.

**iii. Participant Three**

Participant three was a male considered to be a young adult in the age range of 19-40 as specified by Erikson (1994). Participant three was accepted into the study after scoring 32 on his eligibility PSS questionnaire. At baseline participant three weighed 86kg and was 182cm tall. He attained level 8.0 on his baseline beep test, indicating a VO₂ max of 41.98 ml/kg/min which is average for his age (Thompson et al., 2010). His physical activity habits before the study were considered as moderate based on his IPAQ responses and his nutritional intake was considered normal after analysis of his MNA.

v. *Measures*

a) *Perceived Stress Scale Information*
Stress can be measured through the use of the perceived stress scale (PSS). The PSS measures the degree to which situations in one's life are appraised as stressful (Cohen, Kamarck, & Mermelstein, 1983). There are 14 items on the PSS questionnaire, the questions are designed to tap the degree to which respondents find their lives unpredictable, uncontrollable and overloading (Cohen et al., 1983). The issues are central components of the experience of stress (Wheaton, 1997; Wheaton & Montazer, 2010). This scale also includes a number of questions related to current levels of experienced stress. The PSS is user friendly and can be completed in a few minutes. The scoring process is easy to negotiate and requires only small calculations. Because stress levels change rapidly due to daily influences, major event and changes in the ability to cope the validity of the PSS drops off after four to eight weeks (Cohen et al., 1983). Higher scores indicate more perceived stress with the 14-point scale having a score range of 0-45. Evidence of the PSS concurrent validity is found in studies of college students and studies of those in a community smoking cessation programme (Cohen et al., 1983). Cohen and colleagues (1983) indicated that the PSS has adequate internal and test-retest reliability. The PSS correlates with life-event scores, depressive and physical symptomology, utilization of health services, social anxiety and smoking reduction maintenance (Cohen et al., 1983; Cohen & Williamson, 1988). In all comparisons between the PSS and the life-events scores, PSS was a better predictor than life-event scores when evaluating how stressful one perceived life. The PSS is a brief and easy to use questionnaire which shows the degree to which one's life is stressful. It is proven to have both reliability and validity; thus it is a good tool for examining issues about the role of stress in disease and behavioural disorder (Cohen et al., 1983; Cohen & Williamson, 1988). The PSS questionnaire used can be viewed at appendix D.

b) Revised Mini Nutritional Assessment (Revised MNA)

The MNA is a validated nutritional screening tool that asks fairly detailed nutritional questions to identify older adults who are at risk of malnourishment or malnutrition (Bauer, Kaiser, Anthony, Guigoz, & Sieber, 2008). This assessment has been validated by 3 studies on more than 600 elderly subjects (Guigoz, 2006). The tool consists of 18 items on general, nutritional and subjective assessment.
The MNA represents a valuable tool for rapid and reliable nutritional screening (Bauer et al., 2008; Guigoz, 2006; Hagström, Oja, & Sjöström, 2017). For this study the assessment was adapted given that all three participants were <60 years and not all questions were appropriate (e.g. question 1 “pressure sores or skin ulcers” was deleted and replaced with “do you snack during the day”). An example of the MNA used in this study can be found at appendix E. This screening tool was only used to ensure that each of the participants had an adequate nutritional diet prior to the study. The scoring system was not used instead the participant’s questionnaires were instead examined to analyse whether or not participants showed any areas that indicated that the HIIT programme would not be suitable for them.

c) *International Physical Activity Questionnaire (IPAQ)*

The IPAQ is an instrument designed for adult population (15-69 years). The purpose of the questionnaire is to provide estimates on physical activity. This study used the long version of the IPAQ which provides more detailed information for research or evaluation purposes. The IPAQ has reasonable measurement properties for monitoring adults level of physical activity (Craig et al., 2003; Hagström et al., 2017). The IPAQ has acceptable validity when assessing levels of physical. No scoring was completed of the IPAQ; however, each completed IPAQ was thoroughly examined to ensure that they were not sedentary and had a base level of fitness that enabled them to manage the HIIT intervention. Participants had to be adhering to minimal physical activity guideline as supplied by American College of Sport Medicine that adults should get 150 minutes of moderate intensity exercise (Haskell et al., 2007) These recommendations could have been met in a variety of ways; 30-60 minutes of moderate-intensity exercise 5 days a week, 20-60 minutes of vigorous-intensity exercise three days per week or one continuous session and multiple shorter sessions of at least 10 minutes (Haskell et al., 2007). Physical activity is seen as a range of activities from running, cycling and gardening to cleaning, played games and planned exercise. This questionnaire can be found at appendix F. Participants one and two spent 30 minutes 3-4 times a week exercising at a moderate intensity while also mentioning vigorous and moderate physical activity around the house two times a week for up to one hour. With
participant three indicating that he partook in vigorous physical activity twice a week for 1 hour and 30 minutes. These questionnaires indicated that all three participants had the necessary base fitness as part of the inclusion criteria to partake in the HIIT intervention.

\textit{d) Fitness Test}

As part of baseline measures participants partook in a fitness test to estimate their maximum oxygen uptake as well as the HRmax following the immediate completion of the beep test. The beep test was used as it has been validated to accurately predict VO$_2$max and in the filed provides useful information regarding the aerobic fitness of adults especially in determining the optimal intensity of training (Paradisis, Zacharogiannis, Mandila, Smirtiotou, Argeitaki & Cooke, 2014). Results of the 20-m shuttle run beep test such as HRmax and VO$_2$max would be used to gauge intensity of the HIIT intervention. Leger and Lamberts (1982) 20m Multi Stage Shuttle Run test and Mahar, Guerieri, Hanna and Kemble (2011) provided the protocol for the administration of the beep test. A standard beep test has 21 levels, with each level consisting of a different number of sub-level shuttles. Participants run continuously between two sets points that are 20 meters apart at set time intervals for each shuttle and level. The initial speed for the test is 8.5 (km/h) and consists of 7 shuttles. The speed and number of shuttles increases from there with participants who reach level 21 and complete the test having a final speed of 18.5 (km/h). The participants were expected to synchronise their run to a pre-recorded CD which plays the beeps at the required set intervals. As the test proceeds between levels the time interval between each beep reduces forcing the participant to increase their speed in order to keep up with the following beep. The participants needed to ensure that they arrived at the marker before the beep sounded, they were then to remain at the marker until the next beep. The process was repeated until the participants were unable to maintain pace with two consecutive beeps. The participants final score was the last level of the shuttle completed before missing the beep.

As a result of the initial fitness test (beep test) each of the participants VO$_2$ max and HR at test completion was recorded. This data was then used to calculate the intensity of the exercise sessions. HR was taken at the beginning of the beep test, at
each level of the beep test and then immediately following the cessation of the beep test. Heart Rate was recorded via HR monitors. The HR of each participant at the end of the beep test was considered to be their HRmax. The HRmax for each participant was then correlated to the age predicted HR max calculation that allowed the researcher to see if the participant worked to what is assumed to be there predicted HRmax.

The predicted HR max calculation is below as stated by:

\[ 220 - \text{age} \]

As based on HIIT protocol 80-90% of HR max was then needed to find the HR range each participant need to work at. In order to do the below calculation was used:

\[
HRmax \times .80 = 80\% \text{ HRmax} \\
HRmax \times .90 = 90\% \text{ HRmax}
\]

Vo2 max was also used in the determination of each individuals exercise intensities.

According to ACSM 55% HRmax corresponds to 40% of VO2 max, 70% HRmax to 60% VO2 max, 85% HRmax to 80% VO2 max and 90% HRmax to 85% VO2 max. These targets values are based on a number of studies that determine regression equations for %HRmax vs % VO2 max (Swain, Abernathy, Smith, Lee, & Bunn, 1994). VO2 max was determined using the below equation.

\[
VO2 \text{ max} = (vel \times 6.65 - 35.8) \times 0.95 + 0.182
\]

This equation was obtained via Flouris, Metsios and Koutedakis (2005). This particular algorithm increases the efficacy of the 20m shuttle run test to accurately evaluate VO2 max and for this reason it was used (Flouris, Metsios, Koutedakis, & Koutedakis, 2005). VO2 max ranges were then determined via

vi. Study Protocol

The study consisted of three repeated baseline measures, one mid intervention measure and three post measures based around an exercise intervention. The exercise component of the study was a 10-week High Intensity
Interval Training intervention (HIIT), which participants performed three times a week. For a visual interpretation of the protocol please refer to appendix G.

A. Baseline

In order to minimise errant values, baseline was repeated weekly for three weeks. A baseline period is necessary for single case study designs. Baseline is a period of time in which target behaviours are observed repeatedly (Gallo et al., 2013). Baseline demonstrates the stability of the target behaviour prior to the intervention, so that the effects of the intervention can be evaluated against the natural occurrence of the behaviour pre intervention (Gallo et al., 2013). Baseline periods allow for the researcher to then demonstrate decreasing or increasing of symptoms after the initiation of the intervention (Smith, 2012). It is important that a stable baseline pattern occurs as demonstrating that treatment decreases symptoms after initiation of the intervention may be less compelling if the symptoms were already showing a decrease during the baseline period.

In this study baseline line testing occurred for three weeks prior to intervention onset. On the same day of each week at 08.30am a venous blood sample was drawn to be analysed for cortisol, a full blood count and lymphocyte subsets. A further blood sample was collected at 15.30pm on the same day for cortisol alone due to its diurnal variations. Standard operating phlebotomy procedures were used for the gathering of all vena puncture samples. Cortisol samples were collected in an 8ml yellow Vacutainer SST Gel Separator Tubes (BD Medical Technologies, New Zealand). Full blood count and lymphocyte samples were collected in individual 4ml purple EDTA tubes (BD Medical Technologies, 2010). Each tube was inverted eight times to ensure for adequate anticoagulation of the specimen as specified by BD medical technologies. Each tube was labelled with the participant’s unique code to ensure anonymity, the study code (HIIT Study), the date and the time. All blood samples were delivered for analysis within half an hour of the blood being drawn, and were analysed by Wellington Southern Community Labourites at Wellington Hospital.

The participants also completed a PSS questionnaire at each of the three baseline sessions. In addition to this, participants were given a revised Mini Nutritional
Assessment (MNA) and an International Physical Activity Questionnaire (IPAQ) to be completed as part of their base-line measures at their first pre-test. The IPAQ was used to ensure that all participants were engaged in at least a frequent moderate level of activity and were not over-training so they would each have the necessary base level of fitness to complete a 10 week HIIT intervention. Participants completed a beep test so that the researcher could then calculate the participants VO$_2$ max. The participants VO$_2$ max was then used as a basis of what intensity the exercise intervention would be administered at. The beep test was conducted at week three of base line testing. The beep test was administered at this point of baseline testing to ensure that the intensity of the exercise intervention was set to their fitness level just prior to starting the intervention.

B. Mid-intervention Testing

At week five of the HIIT intervention blood sample collection and PSS testing was conducted. Blood samples were again collected at 08.30am and 15.30pm for the same blood analyses as baseline testing (i.e., cortisol, a full blood count and lymphocyte subsets analysis). A PSS questionnaire was completed to assess how their stress levels where tracking in regards to other measures.

C. Post -testing

At the completion of the 10 weeks HIIT intervention each participant completed three weeks of post-testing to assess the effectiveness of the program. During this period of time the participants continued exercise, but not at the same intensity as the HIIT programme. Post-testing was conducted over three weeks in the same manner as base-line tests (i.e., blood samples were collected at 08.30 and 15.30 on three consecutive Fridays). The participants completed a PSS questionnaire at each of the three mornings of blood collection sessions. They were also required to complete a MNA to ensure nutrition had been maintained, and an IPAQ to re-evaluate exercise habits after the conclusion of the 10 week HIIT intervention. A beep test was also administered during the first week of the post-test stage. The purpose of using the beep test at this stage was to see if the PSS scores had improved as a direct relation to the participant’s beep test scores.

vii. High Intensity Intermittent Training Intervention
A. Intervention Structure

This study consisted of a ten-week exercise intervention. Each week consisted of three High Intensity Intermittent Training sessions lasting a maximum on 30 minutes. To ensure continuity there were three different training session formats these being: session one: an individual session with the trainer, session two: a team training session with the other participants and a trainer and session three: a home based individual training session with no trainer. Each exercise session consisted of a 5-minute warm up, 20-minutes of a high intensity intermittent training protocol and a 5-minute cool down. Research indicates that for high intensity intermittent exercise to be successful the intervention needed to last between eight to twelve weeks and have three exercise sessions a week (Boutcher, 2011; Laursen & Jenkins, 2002). Participants were required to work at 80-90% of their HRmax in the working periods to ensure that a high intensity was being reached. Each participant wore a heart rate monitor to ensure they were working at the appropriate intensity. Participants were notified by the HR monitor by a repetitive beeping sound when they neared their target HR range.

The exercise sessions gradually increased in difficulty and intensity as the weeks and progressed refer to appendix H to see these progressions. At week one session one participants completed 20-seconds of near max followed by a 30-second rest, a work to rest ratio of 1:1.5. The session was broken into three circuits with a 1-minute rest between each circuit. The circuits were each repeated three times. At week three and four, participants would work for a max of 40-seconds at a time with varying rest periods. Participants would complete a 40-second strength exercise such as box jumps then move stations and perform 20-seconds of a cardio exercise such a high-knees. The sessions remained in this pattern for 2-weeks with a work to rest ratio of 1:0.5. Weeks five and six consisted of work periods of 2-minutes followed by a 1-minute rest (i.e., 2:1 ratio. In week seven the programme increased significantly in difficulty as participants had begun to plateau with each finding it difficult to achieve their target heart rate. It was at this point that the session structures changed and the participants focused more on completing a certain number of repetitions in the given time. For example, in week seven there were four, 4-minute circuits. Within each circuit there was four
exercises for the participants to complete. They needed to complete 20 repetitions of each exercise, and if the timer was not finished at the end of this they then completed 10 repetitions. This gave the participants a goal number to work towards which increased their motivation. These sessions were worked at the ratio of 1:0.25. Weeks 8 and 9 continued to challenge the participants with varying time ratio and repetitions being used. Week 8 followed closely to the exercise regime of week 7 but with the work period being 5-minutes work to 1-minute rest 1:0.2 ratio. Week 10 reverted back to shorter work periods but with more difficult exercises within the sessions. Week 10 focused more on explosive movements and body weight movements ensuring the heart rates remained in the target zone.

B. Exercise

Before starting each HIIT sessions, the trainer ran through each exercise with the participants to ensure for correct form and technique. At the last session of each week with the trainer, participants were demonstrated the exercises that were planned for their home based sessions. Participants also received a handout with a written description and diagram of each exercise (see appendix I). Each week comprised of a variety of exercises to ensure all large muscle groups were worked. Initially attention was paid to using body weight and small hand weights (2-3kg range), but as the weeks progressed explosive exercise such as box jumps, bounds and tuck jumps were introduced. Heavier weights (5-10kg range) were incorporated to increase the difficulty as exercises became easier, such as squats and lunges as well as punches and multi joint movement such as squat with shoulder presses.

At the beginning of at least one session a week it was ensured that participants completed sprints. These sprints were also completed in the form of intervals with participants often sprinting for 30 seconds followed by 20 seconds of active rest (walking) for a max of three repetitions. For examples and additional details of each week of the HIIT intervention refer to the appendices (appendix H)

viii. Data Analysis

Data was gathered and graphed using Microsoft Excel. The data was initially assessed using visual analysis. In addition to visual analysis, the Percentage of Non
Overlapping data (PND) statistic was used to show effect size (Scruggs & Mastropieri, 1998; Scruggs, Mastropieri, & Casto, 1987). The PND statistic is the percentage of post testing data that is more extreme than the single most extreme point of data at baseline (Scruggs et al., 1987). In this study the single most extreme data point at baseline testing was each participant’s lowest baseline PSS score. The PND statistic represents the proportion of observations that exceed any behaviours observed during baseline testing (Scruggs et al., 1987). The PND statistic was calculated by identifying the lowest PSS score at baseline and drawing a horizontal line that extended from this point across treatment and post testing. The number of points that fell below the extended line were counted and divided by the total number of points plotted after baseline measures. This was then multiplied by 100 and expressed as a percentage. If performance during the intervention phase did not overlap with the performance in baseline testing, then the effect was regarded as reliable. If the PND score was below 50% the treatment was considered ineffective. When the PND was between 50-70% it suggested questionable treatment effectiveness whereas scores of 70-90% were considered to be effective. Any scores over 90% suggest that the treatment is very effective (Scruggs & Mastropieri, 1998).

Individual’s data was graphed using Brinley plots, to show any change in pre to post measures. The Brinley plot is a type of scatter plot that can show data from conditions. This study closely follows Stunkard and Penick (1979) use of Brinley Plots to show individuals weight loss data at pre- and post-therapy. In this study Baseline PSS scores were plotted on the x-axis and their follow up post intervention PSS scores on the y-axis. If PSS scores were maintained post intervention the point would lie close to the 45° line. PSS increase or decrease shown by data points lying above or below the 45° line. Each data point represents the average PSS score of each participant at baseline testing and post testing.

The Standardised Mean Difference (SMD) is a measure of effect and was also used in the analysis of the results. The SMD expresses the size of the intervention effect (Faraone, 2008). Generally, the comparator is a placebo but a similar calculation can be used if the comparator is an alternative to active treatment. The SMD is computed with the following formula (Higgins & Green, 2011).
\[
SMD = \frac{\text{New treatment improvement} - \text{comparator}}{\text{Standard deviation of outcome}}
\]

For this study the formulae used was adjusted slightly as the comparison was between to conditions these being; baseline before intervention and post intervention. The formula was adjusted to;

\[
SMD = \frac{\text{New treatment improvement mean (post test)} - \text{baseline mean}}{\text{SD of baseline}}
\]

When an SMD = 0 it means that the treatment and the placebo or non-treatment conditions have equivalent effects. If improvements are associated with lower scores as seen with the PSS scale, SMD’s <0 indicate the degree to which treatment is more efficacious. A negative number in this case would represent superiority of the intervention over base line measures as the PSS scale is negatively orientated. Cohen offered a guideline for interpreting the magnitude of the SMD; small=0.2, medium=0.5 and large=0.8 (Faraone, 2017; Faraone, 2008).
Chapter Four Results

Key information emerged in regards to the effect of HIIT to decrease stress.
Baseline and Post-intervention results are presented alongside the changes in VO2.
The key areas reported on are (1) perceived stress scale, (2) cortisol slope
differences, and (3) full blood count and lymphocyte subset analysis. In the section
below each of these key areas is discussed alongside graphs and tables displaying
the appropriate data.

i. Anthropometric Data

Outlined in Table 1 below is each participant’s anthropometric data for baseline
and post intervention measures.

Table 1. Anthropometric data for participants one to three from baseline to post-
intervention testing.

<table>
<thead>
<tr>
<th>General Information</th>
<th>Baseline</th>
<th>Post Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gender</td>
<td>Height (cm)</td>
</tr>
<tr>
<td>Participant 1</td>
<td>F</td>
<td>172</td>
</tr>
<tr>
<td>Participant 2</td>
<td>M</td>
<td>182</td>
</tr>
<tr>
<td>Participant 3</td>
<td>M</td>
<td>188</td>
</tr>
</tbody>
</table>

As can be seen in Table 1 each of the participants VO2 max readings increased from
the baseline testing to post intervention testing. The percentage increase for each
participant was as follows P1: 35.25%, P2: 12.9% and P3: 20.05%. Participant
beep test scores also increased from baseline to post intervention testing citing an
increased in fitness level. Table 1 also displays anthropometrical data such as
weight over the course of the intervention, as can be seen weight loss was not
significant. With participant one being the only participant to reduce weight by
10kgs.
Table 2. Calculations and observations made at the completion of the Beep Test, in order to determine the HR and VO_{2max} ranges for each participant.

<table>
<thead>
<tr>
<th></th>
<th>Age Predicted HRmax (bpm)</th>
<th>HRmax Beep Test (bpm)</th>
<th>VO_{2max} (ml/kg/min)</th>
<th>HR (bpm) Range for Intervention</th>
<th>VO_{2max} (ml/kg/min) Range for Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participant 1</td>
<td>173</td>
<td>179</td>
<td>32.50</td>
<td>143-161</td>
<td>26.00-29.25</td>
</tr>
<tr>
<td>Participant 2</td>
<td>177</td>
<td>176</td>
<td>35.66</td>
<td>141-158</td>
<td>28.53-32.09</td>
</tr>
<tr>
<td>Participant 3</td>
<td>181</td>
<td>186</td>
<td>41.98</td>
<td>149-167</td>
<td>33.58-37.78</td>
</tr>
</tbody>
</table>

Table 2 shows the data in regards to participants HRmax and VO_{2max} as determined via the beep test. This data was then used to calculate participants exercise intensities using the calculations outlined in chapter three part two data analysis. The HR and VO_{2max} ranges for the intervention are 80-90% of each participants HRmax and VO_{2max}. These ranges were supplied to the participants and this was the intensity that their HIIT intervention was completed at.

ii. Perceived Stress Scale

The changes in PSS scores and VO_{2max} scores following the three stages of the intervention are displayed in Figure 2. Based on the visual interpretation of the data, it appears that there was a decrease in PSS as the beep test scores increased. The calculated percentage decrease from baseline to post-intervention in each participant’s PSS scores were P1, 38%; P2, 36%; and P3, 40%. The changes in each participant’s PSS scores in relation to their fitness levels is displayed in Figure 2 below. The data is presented in the way that 1, 2 and 3 on the graph corresponds to baseline testing week 1, baseline testing week two, and baseline testing week three. Stage four corresponds to mid intervention testing at week five and stages six, seven and eight correspond to post intervention testing weeks one, two and three.
Figure 2. Perceived Stress scale scores and VO2max changes over the course of baseline, mid- and post-intervention testing.
The data in Figure 2 show all three of the participant’s VO₂max scores and PSS scores were maintained in the three weeks of post intervention with all participants keeping a PSS score well below that of their baseline measures. What can be observed is the decrease in PSS scores in relation with the participants increasing fitness level.

Perceived Stress scores showed a decrease from baseline to post intervention testing as can be seen below in Table 3. All three participants showed reductions in their initial PSS score.

**Table 3. Mean scores for each of the three participants PSS questionnaires at baseline and post-intervention.**

<table>
<thead>
<tr>
<th>Participants</th>
<th>Baseline Test</th>
<th>Post Intervention Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>44</td>
<td>27.3</td>
</tr>
<tr>
<td>P2</td>
<td>40.3</td>
<td>25.7</td>
</tr>
<tr>
<td>P3</td>
<td>29.3</td>
<td>17.3</td>
</tr>
</tbody>
</table>

The data in Table 3 indicates a decrease in each participants PSS from baseline averages to post-intervention averages. The Percentage of Non Overlapping Data statistics for each individual’s PSS scores as a result of the 10-week HIIT intervention shows a decrease across all three participants. In addition to this, the standard mean of difference (SMD) was also calculated. All participants indicated an effective treatment as denoted by the large SMD values i.e., SMD for P1 (-16.7); P2 (-4.76); and P3 (-3.97). With -0.2 indicating small effectiveness, -0.5 indicating medium effectiveness and -0.8 indicating large effectiveness.

Displayed below in Figure 3 are Brinley plots comparing each participant’s average PSS score at pre- and post-intervention. The plots indicate decreased stress for participants 1, 2 and 3 post intervention.
Figure 3. Brinley plots comparing participant averages of PSS scores at baseline and post-intervention.

Each of the three participants PSS average scores from baseline and post intervention testing, are sitting below the regression line. This indicates a sustained decreased in PSS scores throughout the study.

iii. Cortisol

Table 4 shows the cortisol concentrations for each participant. Cortisol was measured at both 08.30am and 15.30pm for each of the three measurement periods i.e., baseline, mid- and post-intervention testing. Note the diurnal variation of a decreased cortisol level in the afternoons. All morning samples fell within the reference range standard for normal values except the first and third sample for Participant 3.

Table 4. Cortisol levels (nmol/L) of all three participants at both morning (08.30am) and afternoon (15.30pm) at baseline, mid- and post-intervention.

<table>
<thead>
<tr>
<th></th>
<th>Baseline 1</th>
<th>Baseline 2</th>
<th>Baseline 3</th>
<th>Mid Intervention</th>
<th>Post Intervention 1</th>
<th>Post Intervention 2</th>
<th>Post Intervention 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AM</td>
<td>PM</td>
<td>AM</td>
<td>PM</td>
<td>AM</td>
<td>PM</td>
<td>AM</td>
</tr>
<tr>
<td>P1</td>
<td>321</td>
<td>128</td>
<td>237</td>
<td>165</td>
<td>296</td>
<td>55</td>
<td>425</td>
</tr>
<tr>
<td>P2</td>
<td>208</td>
<td>186</td>
<td>238</td>
<td>175</td>
<td>220</td>
<td>105</td>
<td>260</td>
</tr>
<tr>
<td>P3</td>
<td>162</td>
<td>316</td>
<td>475</td>
<td>195</td>
<td>561</td>
<td>434</td>
<td>435</td>
</tr>
</tbody>
</table>
Note. Normal reference range for a cortisol reading is 170-500nmol/L

**Table 5.** Baseline, mid intervention and post testing cortisol level (nmol/L) averages at both morning (8.30am) and afternoon (15.30pm).

|       | Baseline Averages |          |          |          |          |          |
|-------|-------------------|----------|----------|----------|----------|
|       | AM    | PM    | AM    | PM    | AM    | PM    |
| **P1** | 285   | 116   | 425   | 311   | 437   | 244   |
| **P2** | 222   | 155   | 260   | 179   | 278   | 185   |
| **P3** | 399   | 315   | 435   | 183   | 440   | 310   |

Wellington Southern Community Laboratories stated their reference range for cortisol to be between 170-500nmol/L for any blood samples taken in the hours between 6am and 10am (Wellington Southern Community Laboratories, 2016). Table 4 displays each participant’s cortisol levels at morning and afternoon over the course of the study. Table 5, displays participants baseline, mid and post intervention cortisol averages for both morning (8.30am) and afternoon (15.30pm) tests. These averages show that as the study progressed participants show higher morning cortisol levels (34.7% for P1, P2 at 20.14% and 9.3% for P3). P1 displayed the greatest percentage increase between baseline testing and post intervention, these results are also consistent with the PSS score of P1 which also decreased significantly throughout the 10-week intervention showing a percentage decrease of 37.95%.

In order to show the changes from morning to evening cortisol slopes graphs were calculated. The slopes of graphs were calculated from the averages for baseline, mid- and post-intervention. Results indicate a steeper slope for all three Participants at post-intervention as can be seen in Figure 4. The mid-intervention slopes were higher than those at baseline for Participants 2 and 3, but not Participant 1 where it was lower than both the baseline and post-intervention slopes.
Figure 4. Cortisol slope measures for all three participants calculated from averages at baseline, mid- and post-intervention.
These results indicate that there was and increased difference between morning and evening cortisol levels which follows more normalised cortisol patterns.

**iv. Full Blood Count and Lymphocyte Subset analysis**

The full blood count and lymphocyte subsets analyses for each participant at baseline, mid intervention and post intervention are shown in Table 6 below.

There was no significant baseline to post intervention differences seen in any of the immune response blood markers except for Eosinophils. However, some values were flagged by the Wellington Southern Community Laboratory as being either above or below what they consider to be their specified normal ranges. These are indicated in red on the table. There did not appear to be any pattern as to whether the out-of-normal range values fall at baseline or post-intervention.

**Table 6. Immune Protein Blood markers at baseline, mid- and post-intervention for each of the three participants.**

<table>
<thead>
<tr>
<th>Participant One</th>
<th>Baseline 1</th>
<th>Baseline 2</th>
<th>Baseline 3</th>
<th>Mid Intervention</th>
<th>Post 1</th>
<th>Post 2</th>
<th>Post 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lymphocyte Subsets</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Lymphocytes 10^9/L (1.0-4.0)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD1 10^9/L (0.1-0.4)</td>
<td>0.18</td>
<td>0.13</td>
<td>0.45</td>
<td>0.17</td>
<td>0.11</td>
<td>0.15</td>
<td>0.12</td>
</tr>
<tr>
<td>CD2 10^9/L (0.7-2.9)</td>
<td>1.13</td>
<td>0.91</td>
<td>2.57</td>
<td>1.01</td>
<td>0.582</td>
<td>1.33</td>
<td>0.91</td>
</tr>
<tr>
<td>CD3 10^9/L (0.1-0.4)</td>
<td>48%</td>
<td>43%</td>
<td>53%</td>
<td>44%</td>
<td>44%</td>
<td>48%</td>
<td>48%</td>
</tr>
<tr>
<td>CD4 10^9/L (0.6-1.4)</td>
<td>0.75</td>
<td>0.58</td>
<td>1.79</td>
<td>0.64</td>
<td>0.53</td>
<td>0.9</td>
<td>0.6</td>
</tr>
<tr>
<td>CD8 10^9/L (0.2-0.6)</td>
<td>0.34</td>
<td>0.29</td>
<td>0.72</td>
<td>0.33</td>
<td>0.26</td>
<td>0.4</td>
<td>0.28</td>
</tr>
<tr>
<td>CD4/CD8 RATIO (1.0-1.5)</td>
<td>2.21</td>
<td>2</td>
<td>2.49</td>
<td>1.94</td>
<td>2.04</td>
<td>2.25</td>
<td>2.14</td>
</tr>
<tr>
<td>CD56 10^9/L (0.2-0.4)</td>
<td>0.25</td>
<td>0.28</td>
<td>0.32</td>
<td>0.25</td>
<td>0.25</td>
<td>0.35</td>
<td>0.22</td>
</tr>
</tbody>
</table>

<p>| <strong>Full Blood Count</strong> |            |            |            |                  |        |        |        |
| <strong>Hemoglobin g/L</strong> | 150        | 147        | 142        | 145              | 144    | 140    | 146    |
| <strong>Hematocrit (Ratio)</strong> | 0.442      | 0.425      | 0.434      | 0.439            | 0.43   | 0.427  | 0.435  |
| <strong>Red Cell Count 10^12/L</strong> | 4.78       | 4.62       | 4.58       | 4.61             | 4.55   | 4.47   | 4.6    |
| <strong>Mean Cell Volume fL (80-99)</strong> | 92.5       | 92         | 94.8       | 95.2             | 94.5   | 95.5   | 94.6   |
| <strong>Mean Cell Hemoglobin pg (27-33)</strong> | 31.4       | 31.8       | 31         | 31.5             | 31.6   | 31.3   | 31.7   |
| <strong>Platelets 10^9/L (150-400)</strong> | 301        | 273        | 320        | 286              | 282    | 252    | 283    |
| <strong>WBC 10^9/L (4.0-11.0)</strong> | 5.2        | 4.8        | 7.8        | 5.1              | 5.2    | 5      | 4.8    |
| <strong>Neutrophils 10^9/L (1.9-7.5)</strong> | 2.9        | 2.6        | 3.8        | 2.9              | 3.3    | 2.8    | 3.1    |
| <strong>Lymphocyte 10^9/L (1.0-4.0)</strong> | 1.5        | 1.4        | 3.1        | 1.5              | 1.2    | 1.6    | 1.1    |
| <strong>Monocytes 10^9/L (0.2-1.0)</strong> | 0.5        | 0.4        | 0.6        | 0.4              | 0.4    | 0.4    | 0.3    |</p>
<table>
<thead>
<tr>
<th>Eosinophils 10^4/L (0.0-0.5)</th>
<th>0.3</th>
<th>0.3</th>
<th>0.3</th>
<th>0.3</th>
<th>0.2</th>
<th>0.2</th>
<th>0.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basophils 10^4/L (0.0-0.2)</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Nucleated Red Cell Count 10^4/L</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Participant Two</th>
<th>Baseline 1</th>
<th>Baseline 2</th>
<th>Baseline 3</th>
<th>Mid Intervention</th>
<th>Post 1</th>
<th>Post 2</th>
<th>Post 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lymphocyte Subsets</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphocytes 10^4/L (1.0-4.0)</td>
<td>1.9</td>
<td>2</td>
<td>2.2</td>
<td>1.8</td>
<td>2.3</td>
<td>1.8</td>
<td>2.2</td>
</tr>
<tr>
<td>CD1 10^4/L (0.1-0.4)</td>
<td>0.19</td>
<td>0.23</td>
<td>0.27</td>
<td>0.17</td>
<td>0.28</td>
<td>0.20</td>
<td>0.25</td>
</tr>
<tr>
<td>CD3 10^4/L (0.7-2.9)</td>
<td>1.48</td>
<td>1.55</td>
<td>1.75</td>
<td>1.47</td>
<td>1.79</td>
<td>1.49</td>
<td>1.73</td>
</tr>
<tr>
<td>CD4% (38-46)</td>
<td>48%</td>
<td>46%</td>
<td>51%</td>
<td>51%</td>
<td>48%</td>
<td>51%</td>
<td>48%</td>
</tr>
<tr>
<td>CD4 10^4/L (0.6-1.4)</td>
<td>0.89</td>
<td>0.97</td>
<td>1.12</td>
<td>0.92</td>
<td>1.10</td>
<td>0.93</td>
<td>1.07</td>
</tr>
<tr>
<td>CD8 10^4/L (0.2-0.6)</td>
<td>0.58</td>
<td>0.58</td>
<td>0.62</td>
<td>0.54</td>
<td>0.68</td>
<td>0.56</td>
<td>0.65</td>
</tr>
<tr>
<td>CD4/CD8 RATIO (1.0-1.5)</td>
<td>1.54</td>
<td>1.67</td>
<td>1.81</td>
<td>1.70</td>
<td>1.62</td>
<td>1.66</td>
<td>1.65</td>
</tr>
<tr>
<td>CD56 10^4/L (0.2-0.4)</td>
<td>0.17</td>
<td>0.15</td>
<td>0.15</td>
<td>0.14</td>
<td>0.23</td>
<td>0.13</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>Full Blood Count</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemoglobin g/L</td>
<td>156</td>
<td>155</td>
<td>149</td>
<td>147</td>
<td>152</td>
<td>152</td>
<td>149</td>
</tr>
<tr>
<td>Hematocrit (Ratio)</td>
<td>0.475</td>
<td>0.469</td>
<td>0.460</td>
<td>0.453</td>
<td>0.46</td>
<td>0.46</td>
<td>0.45</td>
</tr>
<tr>
<td>Red Cell Count 10^12/L</td>
<td>5.50</td>
<td>5.39</td>
<td>5.24</td>
<td>5.18</td>
<td>5.34</td>
<td>5.36</td>
<td>5.22</td>
</tr>
<tr>
<td>Mean Cell Volume fL (80-99)</td>
<td>86.4</td>
<td>87</td>
<td>87.8</td>
<td>87.5</td>
<td>86.7</td>
<td>86.6</td>
<td>87</td>
</tr>
<tr>
<td>Mean Cell Hemoglobin pg (27-33)</td>
<td>28.4</td>
<td>28.8</td>
<td>28.4</td>
<td>28.4</td>
<td>28.5</td>
<td>28.4</td>
<td>28.5</td>
</tr>
<tr>
<td>Platelets 10^4/L (150-400)</td>
<td>193</td>
<td>196</td>
<td>196</td>
<td>103</td>
<td>196</td>
<td>175</td>
<td>200</td>
</tr>
<tr>
<td>WBC 10^4/L (4.0-11.0)</td>
<td>5.5</td>
<td>5.9</td>
<td>5.4</td>
<td>4.8</td>
<td>5.8</td>
<td>4.8</td>
<td>6.3</td>
</tr>
<tr>
<td>Neutrophils 10^4/L (1.9-7.5)</td>
<td>3</td>
<td>3.4</td>
<td>2.7</td>
<td>2.6</td>
<td>3.2</td>
<td>2.5</td>
<td>3.4</td>
</tr>
<tr>
<td>Lymphocyte 10^4/L (1.0-4.0)</td>
<td>1.8</td>
<td>1.9</td>
<td>2.1</td>
<td>1.7</td>
<td>1.9</td>
<td>1.7</td>
<td>2.2</td>
</tr>
<tr>
<td>Monocytes 10^4/L (0.2-1.0)</td>
<td>0.5</td>
<td>0.4</td>
<td>0.4</td>
<td>0.3</td>
<td>0.5</td>
<td>0.4</td>
<td>0.6</td>
</tr>
<tr>
<td>Eosinophils 10^4/L (0.0-0.5)</td>
<td>0.2</td>
<td>0.2</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Basophils 10^4/L (0.0-0.2)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Nucleated Red Cell Count 10^4/L</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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</table>

<table>
<thead>
<tr>
<th>Participant Three</th>
<th>Baseline 1</th>
<th>Baseline 2</th>
<th>Baseline 3</th>
<th>Mid Intervention</th>
<th>Post 1</th>
<th>Post 2</th>
<th>Post 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lymphocyte Subsets</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphocytes 10^4/L (1.0-4.0)</td>
<td>1.2</td>
<td>1.6</td>
<td>1.7</td>
<td>1.4</td>
<td>1.7</td>
<td>1.6</td>
<td>1.5</td>
</tr>
<tr>
<td>CD1 10^4/L (0.1-0.4)</td>
<td>0.13</td>
<td>0.2</td>
<td>0.24</td>
<td>0.17</td>
<td>0.18</td>
<td>0.17</td>
<td>0.17</td>
</tr>
<tr>
<td>CD3 10^4/L (0.7-2.9)</td>
<td>0.78</td>
<td>1.1</td>
<td>1.19</td>
<td>0.99</td>
<td>1.1</td>
<td>1.02</td>
<td>0.97</td>
</tr>
<tr>
<td>CD4% (38-46)</td>
<td>40%</td>
<td>44%</td>
<td>47%</td>
<td>45%</td>
<td>42%</td>
<td>38%</td>
<td>41%</td>
</tr>
<tr>
<td>CD4 10^4/L (0.6-1.4)</td>
<td>0.5</td>
<td>0.72</td>
<td>0.79</td>
<td>0.65</td>
<td>0.7</td>
<td>0.61</td>
<td>0.61</td>
</tr>
<tr>
<td>CD8 10^4/L (0.2-0.6)</td>
<td>0.25</td>
<td>0.33</td>
<td>0.34</td>
<td>0.3</td>
<td>0.35</td>
<td>0.36</td>
<td>0.31</td>
</tr>
<tr>
<td>CD4/CD8 RATIO (1.0-1.5)</td>
<td>2</td>
<td>2.18</td>
<td>2.32</td>
<td>2.17</td>
<td>2</td>
<td>1.69</td>
<td>1.97</td>
</tr>
<tr>
<td>CD56 10^4/L (0.2-0.4)</td>
<td>0.31</td>
<td>0.32</td>
<td>0.24</td>
<td>0.27</td>
<td>0.38</td>
<td>0.41</td>
<td>0.32</td>
</tr>
<tr>
<td><strong>Full Blood Count</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
Hemoglobin \( g/L \) & 153 & 155 & 151 & 153 & 156 & 155 & 152 \\
Hematocrit (Ratio) & 0.456 & 0.456 & 0.448 & 0.455 & 0.46 & 0.45 & 0.46 \\
Red Cell Count \( 10^{12}/L \) & 5.07 & 5.16 & 5.03 & 5.1 & 5.21 & 5.11 & 5.19 \\
Mean Cell Volume \( \mu l(80-99) \) & 89.9 & 88.4 & 89.1 & 89.2 & 88.7 & 89.4 & 88.8 \\
Mean Cell Hemoglobin pg (27-33) & 30.2 & 30 & 30 & 30 & 29.9 & 30.3 & 29.3 \\
Platelets \( 10^{4} /L \) (150-400) & 209 & 251 & 236 & 216 & 212 & 200 & 212 \\
WBC \( 10^{9} /L \) (4.0-11.0) & 8.6 & 6.3 & 6 & 5.5 & 3.7 & 5.8 & 4.5 \\
Neutrophils \( 10^{9} \) (1.9-7.5) & 6.4 & 3.9 & 3.5 & 3.4 & 1.4 & 3.6 & 2.6 \\
Lymphocytes \( 10^{9} /L \) (1.0-4.0) & 1.2 & 1.6 & 1.7 & 1.5 & 0.6 & 1.6 & 1.4 \\
Monocytes \( 10^{9} /L \) (0.2-1.0) & 0.7 & 0.6 & 0.5 & 0.4 & 0.1 & 0.5 & 0.4 \\
Eosinophils \( 10^{9} /L \) (0.0-0.5) & 0.3 & 0.2 & 0.2 & 0.2 & 0 & 0.1 & 0.1 \\
Basophils \( 10^{9} /L \) (0.0-0.2) & 0.1 & 0.1 & 0.1 & 0.1 & 0 & 0.1 & 0 \\
Nucleated Red Cell Count \( 10^{9} /L \) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\

It must be noted that the CD4/CD8 ratio was flagged (in red) in each of the Participant’s tests throughout baseline, mid intervention and post intervention as can be seen in Table 6. These all presented higher than the Southern Community Laboratories range of 1.0-1.5. However, the ratios did not seem to follow in a particular pattern and appear to fluctuate throughout the study by only small degrees.

**Table 7. Eosinophil count \( \times10^{9} /L \ ) of all three participants from baseline, mid- and post-intervention**

<table>
<thead>
<tr>
<th></th>
<th>Baseline 1</th>
<th>Baseline 2</th>
<th>Baseline 3</th>
<th>Mid</th>
<th>Post 1</th>
<th>Post 2</th>
<th>Post 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>P2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>P3</td>
<td>0.3</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0</td>
<td>0.1</td>
<td>0.1</td>
</tr>
</tbody>
</table>

All the Eosinophil values in Table 7 fell within the normal range as stated by the Wellington Southern Community Laboratory i.e., 0.0-0.5 \( \times10^9 /L \). However, note that all the post-intervention measures are lower than those at baseline for all three participants.

v. **Summary of results**

Baseline to post intervention changes
- Fitness increased for all three participants
- PSS scores decreased in all participants
- Steeper slopes for cortisol diurnal changes am too pm for all participants
- Decreased eosinophil levels in all participants
- Heightened CD4/CD8 ratio in all participants.
Chapter Five Discussion

i. General Discussion

A 10-week High Intensity Intermittent Training (HIIT) intervention appears to successfully combat chronic levels of high stress in moderately fit individuals through decreased PSS scores. The observations of each participant’s results also indicated a more normal cortisol profile and decreased inflammation (eosinophil count). This further supports the aim of the study which was to show through single case studies that HIIT can in fact decrease a person’s perceived stress.

The participants demonstrated a visible reduction in perceived stress scale scores at the conclusion of the 10-week HIIT intervention. The mid intervention testing as seen in Figure 2 demonstrates that from mid intervention at week five to post testing at weeks 11-13 each of the three participants showed a clear decrease and maintenance of lowered stress in comparison to their baseline testing scores. These findings are consistent with past research which has demonstrated that exercise diminishes psychological stress and its associated symptoms (Norris, Carroll, & Cochrane, 1992).

Literature suggests that exercise creates an increased ability to cope with one’s life stressors while developing a resistance to the stress effect (Assis et al., 2008; Norris et al., 1992). Theories and hypothesis as to why exercise works to aid psychological recovery suggest that exercise provides distraction, increases self-efficacy and offers social interaction (Bandura, 1977; Faulkner and Biddle, 2004; North, McCullagh & Tran, 1990). These theories and hypothesis provide reasons as to why exercise may aid in the reduction of psychological issues (Greenwood & Fleshner, 2011; Kobasa, Maddi, & Puccettp, 1982). However, this study provides physiological evidence of the effects exercise has on reducing the physiological aspects of stress such as decreased cortisol and increased inflammation (Puterman et al., 2010; Scully et al., 1998).

In broad terms exercise contributes to general health and well-being. Exercise can be beneficial to the cardiovascular system in decreasing a person’s susceptibility to cardiovascular disease through reducing blood pressure, heart rate (HR),
cholesterol, and obesity (Agarwal, 2012; Kohl, 2001; Thompson et al., 2003). Regular exercise also helps strengthen muscle and bone, and can benefit the mental well-being of a person though its anti-depressive and anxiety reducing effects (Craft & Perna, 2004; Haskell et al., 2007b; Thompson et al., 2003). Physical activity is positively correlated with improved wellbeing and fitness in addition to its ability to help participant cope more successfully with stressors is reflected in the results of our study (MacFarlane, 2010). However, when interpreting the results, it must be remembered that each of the participants in this study were already fit and active, with each exercising at a moderate intensity at least three times a week. Therefore, it is suggested that it is not the amount of training a person partakes that causes the decrease in stress, but may in fact be the type and intensity of exercise.

The results of this study show a HIIT intervention of 10-weeks reduced stress in the already moderately active participants. As indicated by the IPAQ both participants one and three partook in either sports or running multiple times a week which indicated that would be considered moderately active. Participant 3 was also considered moderately active as he biked at various stages throughout the week, comfortably training for a long distance cycle race. Nonetheless, he continually scored high on the PSS at baseline testing as did participant one and two. As the participants were considered moderately active it came to a surprise when each participant completed the beep test and all VO$_2$max scores resulted in average or fair for their specified age range (Thompson et al., 2010). This reinforced the conclusion that although each participant was active they were not necessarily deemed “fit”. Furthermore, when participant one and three felt the need to include HIIT exercise into their lifestyles at the conclusion of the study in order to continue to manage their stress levels, it demonstrated that their level of fitness baseline had not been enough to curtail the symptoms of chronic stress. As can be seen from the results the three participant’s fitness increased in terms of VO$_2$max which is denoted by the fact that exercise intensity had to be increased weekly in order for each of the participants to reach their target heart rates.

Although weight loss was not a main focus of this it additionally must be noted here the substantial weight loss of 10kgs participant one experienced as a result of
the HIIT intervention. As each participant completed a MNA at pre and post testing we can confidently say that the participants diet did not change and that they were not on a diet of any kind. The weight loss of participant one was real and it would not be unreasonable to say that it was largely due to the HIIT intervention and decreased stress levels.

These results further develop the link between HIIT and its ability to decrease a person’s stress levels. Previous evidence suggests that moderate exercise three times a week for twelve weeks increases physical condition, and facilitates the individuals capability for dealing with stress (Scully et al., 1998). Had participants been facing a short bout of acute stress, it is possible that their current fitness ability and amount of exercise being undertaken would have been enough to manage the symptoms associated with stress. Research indicates that moderate aerobic exercise appears to influence the stress response, in this study however, this was not seen (Klaperski, von Dawans, Heinrichs, & Fuchs, 2013; Scully et al., 1998). Contrary to evidence participants showed that with moderate fitness they were unable to handle the stresses presented in their daily life, and that it did not play a protective role in the stress system dysregulation, lowering of the stress response or reduce the sensitivity to stress as cited in literature (Tsatsoulias & Fountoulakis, 2006). This solidifies the idea that in those who are chronically stressed, moderate intensity exercise does not have the ability to decrease chronic stress symptoms, as all three participants presented at baseline with high PSS scores, cortisol levels that were at the lower end of the normal range and heightened eosinophil count. At the conclusion of the study all three participants had experienced an increase in their VO2max and a decrease in their PSS scores. This shows a link between increased fitness and the ability of the body to handle stress.

When confronting acute stress, the stress response increases cortisol output in order for the individual to manage the situation. However, contrary to this when an individual is chronically stressed evidence suggests that their daily cortisol output is decreased (Miller et al., 2007). As mentioned in chapter 1 cortisol acts on a diurnal rhythm (Chung, Son, & Kim, 2011). On waking cortisol is at its highest, it ebbs and flows during the day and is followed by its lowest point in the evening (Chan & Debono, 2010; Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009).
However, the cortisol results as seen in Table 4 and Figure 4 indicate that the participants had blunted cortisol responses as their cortisol profiles from morning to evening were flattened, where there was no high morning peak followed by the evening drop at baseline (Table 4). Participant three demonstrated baseline cortisol levels averaging 399 (nmol/L) for the morning sample. This value is at the higher end of the normal sample it is suspected that this higher baseline morning cortisol sample could be linked too participant threes lower PSS score on entering the study.

Participant one may have shown lower cortisol levels than participant three due to the fact that females routinely report and exhibit higher stress symptoms and stress reactivity than males (Nolen-Hoeksema, 2001). It has been hypothesised that this gender difference could be due to women being more likely to have a dysregulated HPA response that either presents elevated or lowered hormone responses to stress (Nolen-Hoeksema, 2001). Kirschbaum et al (1999) however, argued that gender difference is masked in plasma cortisol stress response, so it is impossible to conclude as to whether there is a definite gender difference in this study, as salivary cortisol was not taken (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). Kirschbaum et al., (1999) did however state that men seemed to have a stronger hypothalamic drive in response to stressful stimulation than women, creating a difference in salivary free cortisol levels. Research also shows that men show double the elevation in cortisol when facing stressful situations, and that women show negative affect (Foley & Kirschbaum, 2010; Kelly et al., 2008). So although it cannot be confidently concluded that participant one’s cortisol levels presented as lower due to a gender difference, it is a possible hypothesis that could account for the significant difference in cortisol levels between participant one and three.

Participant two’s cortisol levels however, display as relatively low in comparison to participant one and three. It is suggested that this could be a product of the additional responsibility and therefore higher stress role participant two has. In comparison to participant one and three, participant two has a lower baseline cortisol level at wakening with an average of 222 (nmol/L). The stress level experienced is reinforced by the PSS scores participant two received at baseline.
testing indicating that participant two had at baseline, a PSS average of 40.3. This score is considered relatively high considering Cohen and Williamsons, (1998) average score for males completing the PSS questionnaire to be 32.5.

Regardless of gender and perceived stress scores all participants showed particularly low morning cortisol levels, with only a small decrease at evening measures as seen in Tables 4 and 5. Figure 4 additionally shows the slope changes from morning measures to evening measures for each participant. As can be seen in each of the participant baseline testing participant slopes are smaller in comparison to post intervention testing. The reduced slope suggests a smaller cortisol change from morning to evening with no clear morning peak and evening trough. This blunting/flattening of the cortisol response or a decrease in cortisol from already low baseline measures in individuals exposed too severe acute, or severe chronic stress has been termed hypocortisolism (Heim, Ehlet, & Hellhammer, 2000).

Hypocortisolism has been described as a disorder that occurs when an individual has a hypoactive adrenal gland usually caused by traumatic events eventually leading to post traumatic stress disorder (Heim et al., 2000). However, recent evidence has shown that hypocortisolism also occurs in those who live under conditions of constant chronic stress (Heim et al., 2000; Raison & Miller, 2003). Low cortisol has been observed in patients with a range of different stress-related disorders where the person is often accompanied by symptoms such as increased reactivity to stress, pain and/or fatigue (Fries et al., 2005). Hypocortisolism is determined by alterations to the HPA axis, and can be seen by reductions in cortisol levels, due to reduced release of cortisol from the adrenal glands. The mechanisms behind hypocortisolism are complex and may differ between and within patients and unfortunately beyond the scope of this research, but for further and more detailed information on these mechanisms refer to Hiem et al., (2000).

The reduced availability of cortisol is key to this research as cortisol provides a protective effect to an individual. The immnosuppressive action of cortisol as a primary immune defensive mechanism that is activated in response to stress becomes an issues in those who are chronically stressed (Raison & Miller, 2003).
With a decrease in cortisol there is a permanent lack of the protective effects cortisol provides, and so chronically stressed individuals may become vulnerable to autoimmune disease, inflammation, chronic pain, allergies and asthma (Heim et al., 2000). Decreased cortisol levels can be due to a depletion in cortisol, insufficient free unbound cortisol, impaired cortisol secretion of corticotropin-releasing hormone (CRH) function, glucocorticoid receptors (GR) resistance or down regulation and/or hypersensitivity to of the negative feedback system (Hannibal & Bishop, 2014). Under normal circumstances cortisol binds to GR and acts as an anti-inflammatory agent (Raison & Miller, 2003). However, prolonged cortisol secretion can result in a down regulation or resistance to GR that blocks the cortisol from binding (Hannibal & Bishop, 2014). With the reduction or resistance to GR the duration and or intensity of the inflammatory response increases, heightening the risk for asthma, autoimmune diseases, as well as the onset and progression of inflammatory diseases (Sheldon Cohen et al., 2012). Albeit identifying the specific low cortisol mechanism in chronic stress is beyond the scope of this research, the long term effect of chronic stress remains the same: cortisol fails to correctly function and as a result increased systematic inflammation occurs (Hannibal & Bishop, 2014).

As discussed glucocorticoids such as cortisol exert immunosuppressive actions on certain immune functions such as lymphocyte function, macrophage activity and T Cell proliferation (Glaser & Kiecolt-Glaser, 2005; Miller et al., 2007; Sapolsky, Romero, & Munck, 2000). Moreover, the release of cortisol reduces natural killer cell activity and inhibits the secretion of cytokines such as interleukins (Sergerstrom & Miller, 2004). These types of cytokines are important for immune and inflammatory mediators which in turn help to regulate cellular immunity, however the immunosuppressive action of cortisol prevents the toxic effects of the body’s primary defensive mechanism that are activated within the body as a response to stress and disease (Glaser & Kiecolt-Glaser, 2005; Miller et al., 2007; Sergerstrom & Miller, 2004). As this study shows each of the participants displayed low cortisol levels and high eosinophil counts at baseline testing. Thus suggesting that in those that are chronically stressed and experiencing a decrease in cortisol levels lack the protective effect that cortisol might promote disinhibition of immune functions leaving them increasingly venerable to immune disorder and
disease (Chrousos, 2009). Meagher, Cousin, Seckl and Haslett, 1996 also demonstrated that glucocorticoids have a direct effect on eosinophils by resulting in a decreased eosinophils concentration, clearly implicating glucocorticoids as key to the body’s an anti-inflammatory and anti-allergic systems (Meagher et al., 1996). This study demonstrates that HIIT increases cortisol production to more normalised diurnal variation levels and as a result each participant’s eosinophil levels halved. This evidence shows the direct effect, cortisol has on the body’s immune functions, specifically focusing on inflammation in the body. This evidence displays the link between chronic bad stress, low cortisol and increased inflammation levels as demonstrated in Figure 5.

These results are consistent with past research which indicates that high (80%) intensity exercise provokes an increase in circuiting cortisol levels due to HPA axis stimulation which is effected by those who experience chronic stress (Hill, Battaglini, Viru & Hackney, 2008). Corral, Mahon, Duncan, Howe & Craig, (1994) additionally showed that 30 minutes of exercise at 70% VO₂max significantly elevated serum cortisol concentrations in male children during and after the exercise session. It is interesting to note that although some studies protocols indicate that serum cortisol is to be taken during or just after exercise sessions there is evidence to indicate that cortisol levels remain significantly higher than baseline 30-minutes after the high intensity intermittent training session (VanBruggen, Hackney, McMurray, & Ondrak, 2011). This lends further support to the results above that show an overall elevation in cortisol from baseline to post testing suggests that the changes in cortisol levels do not immediately decline following the completion of the HIIT exercise session.

In contrast past literature suggests that those facing psychological stressors, trauma, significant life events or have documented chronic stress have an higher secretion of cortisol (Ko Pitman & Orr, 1990; Kotozaki et al., 2012; Schulz, Kirschbaum, Prüßner, & Hellhammer, 1998; van Eck, Berkhof, Nicolson, & Sulon, 1996). In our study, participants showed lowered cortisol levels, and a higher eosinophil count at baseline. As demonstrated above lowered cortisol is called hypocortolism however this effect can be explained via the Glucocorticoid Resistance Model. This model explains that stress diminishes the immune
response by decreasing its sensitivity to glucocorticoid hormones that would usually terminate the inflammation (Sheldon Cohen et al., 2012; Miller et al., 2007; Sapolsky et al., 2000). The resistance model suggests that the receptors responsible for binding with glucocorticoid hormones like cortisol are down regulating subsequently weakening the immune systems and its ability to respond to the anti-inflammatory needs that cortisol initiates, thus leading to an increase in the body’s inflammatory processes (Chrousos, 2009; Sheldon Cohen et al., 2012; Miller et al., 2007).

The Glucocorticoid Resistance Model could help explain some of the results reported for participant one. During baseline testing prior to the 10-week HIIT intervention participant one exhibited viral cold symptoms such as coughing, sneezing and headaches in addition to a significant increase in asthmatic symptoms. The participant was unable to fight the infection and as a result took sick leave for two weeks in an attempt to manage the infection. The escalation of participant’s asthmatic symptoms, along with their increased susceptibility to catch a viral infection indicates a compromised immune system prior to beginning the HIIT intervention. It was at baseline testing that participant one’s eosinophil count was at its highest of 0.3. However, as a result of the 10-week HIIT intervention participant one’s eosinophil count dropped to 0.2 indicating that the inflammation the participants body was facing was decreasing. In accordance with the Glucocorticoid Resistance Model, it can be suggested that participant one’s stress response was beginning to resume its natural function where an increase in cortisol (glucocorticoid hormone) allowed the body to suppress pro-inflammatory cytokines therefore increasing the participant’s cellular immunity due to increased glucocorticoid function.

In addition to this at the completion of the 10-week HIIT intervention participant two and three’s eosinophil count had halved also indicating a decreased inflammation in the immune system. Participant two’s average eosinophil count at baseline was 0.17 and by post testing this had decreased to 0.10(2dp). Participant three displayed an eosinophil count of 0.23(2dp) and by baseline this had also reduced to 0.07(2dp). As can be seen each of the participant’s inflammation levels as displayed by the eosinophil counts in Table 7 were reduced due to the effect of
increased and normalised cortisol as a result of the HIIT exercise intervention. The participants’ immune response to infection was curtailed by suppressing the secretion of cytokine inflammatory agents such as eosinophils which resulted in a lowered eosinophil count (Dedovic & Duchesne, 2012). This evidence shows HIIT decreased stress via reduced systematic inflammation achieved via normalisation of cortisol levels.

Eosinophils usually play a role in promoting inflammation that is necessary and beneficial to the body (Fulkerson & Rothenberg, 2013). However, at times there is an increase in eosinophils that leads to troublesome symptoms. Sometimes such an increase in eosinophils play apart in the development of asthma, hay fever and autoimmune diseases (Fulkerson & Rothenberg, 2013). As can be seen in Table 6 and Table 7 each of the participants had higher eosinophil counts during their baseline testing. However, all participants displayed a decrease by the end of the HIIT intervention. This data indicates that the participant’s inflammation in the body was decreasing. It is thought that this could be linked to the more normalised cortisol rhythms which were displaying a morning high with a more obvious drop off towards the evening. The less flattened cortisol pattern indicates that participant’s cortisol levels were returning to a more sufficient level, therefore increasing glucocorticoid signalling and therefore immunosuppressive control.

In addition to eosinophil counts, data was also recorded in regards to CD4, CD8 and CD56 as they are all part of the immune spectrum and play significant roles in the body’s immune response. During the baseline and post intervention testing a number of participant blood results regarding CD4, CD8, CD4/CD8 Ratio and CD56 were flagged by the laboratory as being abnormally high or low. However, there did not appear to be a discernible pattern that emerged for all participants in regards to CD4, CD8 or CD56. Small increases and decreases were observed across all participants but these did not appear to be significant or directly related to the increases in cortisol response or lowering of eosinophil count.

In regards to the CD4/CD8 ratio however, all three participants experienced high CD4/CD8 ratios across majority of the testing undertaken. Usually CD4, CD8 and CD4/CD8 ratios are used to determine the progression and severity of the disease HIV/Aids (Sainz et al., 2013; Taylor, Fahey, Detels, & Giorgi, 1989). Normal
CD4/CD8 ratios in humans are difficult to judge and ratios sitting within 1.5-2.5 are usually considered normal (Amadori et al., 1995). However, in this case Southern Community Laboratories reference range is 1.0-1.5. It has been suggested by Taylor et al., (1989) that the CD4/CD8 ratio is not the best indicator of immune function because of its reciprocal fluctuating nature between CD4 and CD8. I thought that the repeated measures (three weeks’ baseline and three weeks’ post interventions) would show more stable averages as was seen for PSS scores and cortisol levels. However, this proved not to be the case. The fluctuations could be caused by a variety of causes and it has been reported that diet and even sun exposure can affect the CD4/CD8 ratio (Myrick et al., 2002). However, if the ratio had been flagged as below the set reference ranges or had decreased throughout the intervention this is would have been a clear determinant of immunodeficiency and a decline in immune function (Scanlan, Vitaliano, Ochs, Savage, & Borson, 1998). We can only assume that HIIT either maintained the CD4/CD8 ratio or had little to no effect.

Decreased stress could be due not only to the ability to cope with stress as a whole but an increased capability to handle the symptoms that may adjourn stress. All participants cited at the beginning of the intervention that they felt they lacked energy, had fluctuating moods, sleep trouble, irritability and increased tension as a result of the stressors they faced in their lives. Exercise offers less to alter the stressful event or stressors itself but provides a way where a person can come more resilient to the effects and symptomology it produces (Kobasa et al., 1982). The increased ability to manage emotions and symptoms that often accompany stress is extremely important in a person’s capability to cope.

Ellis, Griffith, Allen, Thorpe and Bruce (2015), showed that exercise has been proved to help decrease stress symptoms. Similar results are reflected in participant three with a 40% change in PSS scores from baseline to post testing. Participant three stated that although work was still stressful he found himself less irritable at home and in a more stable mood having used exercise as a stress release to stabilise his emotions. Participant two stated that exercise dramatically influenced energy levels. While usually feeling tired and lacking focus and energy around 10am, after a morning exercise session participant two cited experiencing
an energy boost which increased mood and ability to handle the stressful tasks presented during the day. Participant one cited feeling anxious and stressed at the beginning of the study, but with the increase in fitness and the regular sessions eventually felt a change in mood and decreased. This difference is observed for participant one with PSS scores decreasing by 38% at the end of the study. The HIIT intervention provided a buffering mechanism that comes into play when life presents stressful problems and situations by allowing participants a respite to alleviate some of the symptoms associated with stress. Additionally, building up a resistance to the mechanisms of stress so that when faced with stressful situations the physiological process that occurs is not as severe.

Figure 5 shows chronic stress and its effects as a negative spiralling cycle which when not broken can lead to constant stress and illness. It is for these reasons that psychological and physiological interventions need to be implemented to develop resilience and decrease vulnerability to the effects of chronic stress. deTerte et al., (2014), propose a three-part model of psychological resilience that solidifies the need for adaptive health behaviours such as exercise to aid in the reduction and alleviation of mental health difficulties. In addition to exercise behaviours, deTerte et al., (2014) suggested that greater social support from colleagues, adaptive health practices and coping strategies plus greater personal understanding of emotions and optimism were related to decreased symptoms of PTSD, better physical health and less psychological distress. Involving those who are chronically stressed in a HIIT programme, with other members of their work environment provided an individual with social support and an alternative coping mechanism needed to help participants handle their chronic stress levels and symptomology. Thus, breaking the negative stress spiral and developing behaviours that may make them more resilient to stress in the future.
Figure 5. Model of stress and inflammation.

However, although this study shows that a HIIT intervention can decrease perceived stress, normalise cortisol and reduce inflammation I am unsure as to the potential mechanisms as to how chronic stress directly effects a person’s cortisol and inflammation. As displayed in Figure 5, it can be seen that stress affects an individual’s physiological and psychological ability to cope. Although in these three case studies there is a link between cortisol, eosinophil and psychological stress, I cannot state which one is cause and effect, whether inflammation causes the psychological stress or whether psychological stress effects the immune system and then effects cortisol. Although I do believe there is an interplay/relationship between all three with each affecting the other. This supports emerging evidence
that systemic inflammation affects mental health disease. We are however, unsure by which pathway this occurs at the present time.

**ii. Limitations**

Although this study provided important information in regards to the effect of high intensity intermittent training has on decreasing perceived stress scores and normalising immune function. There are limiting factors that must be addressed as they were crucial to the development and generalisability of this study.

At the outset of the study it was stated that all participants would come from the same Police team to ensure that they faced similar stresses, what was not accounted for was the different roles within each team that the participants may have been involved in. Each participant took part in various cases throughout the study. Some of these were said to be more or less demanding than others. In addition to this each participant held a different role within the team which meant their stress was not caused by the same cases or issues. As a result, it was impossible to control for the stress each individual was facing. However, each participant perceived the stressors they were faced with to be at the same level throughout the intervention. During the intervention, when asked how they were feeling in regards to their stress load all participants reported that they felt they were experiencing similar stress to that of their baseline. At any which time, each participant reported that the stress of their job and their life was the same regardless of which case they were on and what was happening in their home lives.

Supplementary to this it was ensured that participants completed three PSS baseline questionnaires, one mid intervention questionnaire and three post-test questionnaire to gain an average, so that the PSS score was relative to weekly changes.

Participants were also required to complete one homebased HIIT session a week. This was to ensure that if the programme was to become replicable with the NZ police that it was cost effective, and manageable for those with busy lives in and outside of work. However, as the researcher was not present to observe intensities it is possible that the participants did not work out at the intensity specified for them based on their VO₂max. This limitation was overcome in two ways. Firstly, each participant received a personal HR monitor that was set to beep when they
reached their specified intensity. They were taught during their personally training sessions as to how to use the monitors and were advised on ensuring they reached the necessary HR in order for the training to be effective. Secondly participants had to record their HR at rest intervals throughout their home based session, on printed recording sheets see appendix J. These were used to ensure that the researcher could see if the participants were working at their specified heart rates.

Each of the participants believed themselves to be fit and active, and based on the IPAQ results each participant recorded moderate amounts of activity. However, each participants VO₂max was average or below average percentile. To account for the varying levels of fitness, VO₂max and HRmax was used to establish what 80-90% of each participants VO₂max and HRmax was. This then regulated the intensity each participant trained at, based on their fitness ability. To ensure that fitness continued to increase and that participants maintained the correct intensity the HIIT programme improved in difficulty each week see appendix H.

The researcher demonstrated the exercise, explained key learning points and asked for demonstrations from each participant to avoid unnecessary injury. Exercises were only made more complex and increased in difficulty when the researcher was sure the participant could manage. Each exercise was shown in progressive steps from easy, medium and hard so that each participant could train at their individual ability while also maintaining intensity. They were also provided with an instruction sheet on how to complete home-based exercise session to ensure for safety and wellbeing (appendix J)

Additionally, to this was the issue of participant confidentiality. This was made difficult due to the small nature of the task force the participants worked within and of the police itself. Any details in regards to age, job description and time in the force had to be removed from the participant’s section of the study. Age in the participant section has instead been changed to age range. The age ranges were determined through Erikson’s eight stages of psychosocial development (Erikson, , 1994; Marcia, 2002). Information regarding the participant’s length of time in the police and small descriptions about their role was also removed. This kind of information could have made the participants easily identifiable and would have breached their anonymity and therefore could not be left in the thesis. This
information also did not affect any of the results displayed or conclusions made. To ensure that each participant anonymity was maintained in the publication of this study, the participants were sent the participants section of the thesis. The participants were required to sign a consent as seen in appendix K. This consent form asked the participants to ensure they had read the participants section to check that there was no personal information or information of any nature that made them identifiable. Once this had been checked the participants were required to sign the consent form and agree to publication of the participant section of the thesis.

It may have been prudent to repeat the beep test at mid intervention testing (weeks five) in order to ensure that the intensity the participants were working at in each HIIT sessions was correct. However, HRmax was established based on the participant’s beep tests results prior to the study and from that the participant’s intensity of exercise was increased every week to ensure that participants continually worked at 80-90% of their HRmax.

At the conclusion of the study it was noted that due to the high level of stress the participants were under and the type of work completed by the police that a validated sleep questionnaire could have been introduced to baseline, mid intervention and post intervention testing. However, in this study PSS questionnaire scores decreased indicating that there was a reduction in the overall stress experienced by each participant. It can be expected that this would have had a positive effect on participants sleep as sleep disruption is a common symptom of stress (Han, Kim & Shim, 2012; Kerstedt, Knutsson, Westerholm, Theorell, Alfredsson & Kecklund, 2002).

It is also unfortunate that we are unable to give the inter-intra coefficient validations for the blood work that was analysed. However, the analysis was carried out at a government pathology laboratory so it was expected that the bloods had a high level of precision and repeatability.

As discussed earlier on it is understood that gender can have an effect on cortisol and that this would result in a knock on effect in the eosinophil counts as discussed. In hindsight it would have been better to back this up with female/male
hormone levels. Nevertheless, all the participant’s subjective and objective measures all changed in the same direction, it is however the level of change that could be attributed to gender.

iii. Future Directions

Following the clear results reported here, this study needs to be attempted on a larger population. This would allow increased generalisability and demonstrate whether it is possible to replicate the above results. In order to show long term resilience of the effects of the HIIT intervention a study involving a follow up of one year should be included. This would allow for researcher to assess the longevity of the effects caused by the HIIT intervention. Increased assessment of the immune system also needs to looked into as this would allow researchers to further analyse the other important immune markers that are effected by stress and HIIT.
Chapter Six Conclusion

The results of this study show that trained people who thought themselves to be physically fit displayed high stress and compromised immune functioning due to blunted cortisol secretion and increased eosinophil count. The level of stress these individuals faced, essentially meant that the moderate exercise they were currently partaking in was not enough to curtail the physiological and psychological stress related symptoms they were facing. Changing participants training to high intensity intermittent training had a positive effect on each participant’s cortisol, immune and inflammation makers. Additional to these results all participants displayed lowered perceived stress scale even though the stress they were facing from their occupation and family lives was maintained throughout the study. This evidence all shows that in moderately active New Zealand Police, high intensity intermittent training appears to be an effective mechanism for lowering overall perceived stress scores, increasing cortisol function and decreasing inflammation in the body. In addition to decreasing effects of high occupational stress in New Zealand Police this research has provided further evidence for the relationship between systemic inflammation and mental health disease.
Chapter Seven References


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Chapter Eight Appendices

**Appendix A**: Participant information sheet
**Appendix B**: Participant Consent form
**Appendix C**: Personal Letter of Invite
**Appendix D**: Perceived Stress Scale
**Appendix E**: Revised MNA
**Appendix F**: IPAQ
**Appendix G**: Flow diagram of methodology
**Appendix H**: Snapshot of Exercise programme
**Appendix I**: Exercise demonstration sheet
**Appendix J**: HR recording sheet
**Appendix K**: Participant Information Consent Form for Participants Section
Appendix A: Participant Information Sheet

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

High Intensity Intermittent Training in Clinically Stressed Persons to Relieve Symptoms

INFORMATION FOR PARTICIPANTS

Thank you for showing an interest in this study. Please read everything below before deciding if you want to take part. This information sheet will tell you a little more about the study and what we would like you to do.

The research is being conducted by Ms Rebecca Kurtovich, Masters Student Massey University, School of Sport and Exercise. During the study Rebecca will be supervised by the following academic staff: Dr. Sally Lark and Dr Ian de Terte.

Why is this research important?

Stress is becoming an ever present issue in society. People are constantly faced with stressful situations and environments which more often than not can have an effect for prolonged periods of time. There is now an increased awareness in regards to how stress can affect a person both physically and mentally. The symptoms of stress can cumulate so gradually that often an individual is coping with the resulting symptoms as well as the source of stress. Certain groups of the work force face stressful situations more frequently and are therefore predisposed to developing chronic stress. Police officers can suffer anticipatory stress at the start of each shift, while also dealing with the stress of critical incidents which happen during their working day. A result of constant stress is that people often find their immunity decreases and that illness occurs more frequently. Stress can often disrupt the immune system and leave it unbalanced, therefore not working as effectively as it could.

There is evidence to suggest that regular exercise helps reduce depression, anxiety and stress in individuals. It is thought that for those in occupations where alternative treatment methods are being sought, that high intensity exercise could be an adjunctive therapy to treat chronic stress. This research is important as it will endeavour to show
an alternative treatment to deal with stress that is not pharmacological. For this reason the researchers of this study believe that exercise in the form of high intensity intermittent training (HIIT) could relieve symptoms of chronic stress and improve immune function in a population who are already well exercise trained and yet still subject to chronic stress.

**Who can take part in this study?**

Healthy 18 to 35-year-old females and males, who are sworn members of the New Zealand Police, and work as part of the Wellington Police District Child Protection Team at Koru House and live in the Wellington region.

**What will I be asked to do if I take part?**

Members of the NZ Police who wish to partake will arrange a meeting with the primary researcher to sign a consent form and then complete a perceived stress scale questionnaire. From the results of the stress scale questionnaire, three persons will be asked to participate further in the study with the exercise intervention. Completing the questionnaire should only take approximately 10 minutes. Candidates that do not meet criteria for inclusion for the exercise intervention will be sent a letter/email stating that they are not eligible for further participation in the study.

Those who are invited and consent to participate further in this study will have a series of pre-, mid- and post- exercise intervention measures as follows:

*Baseline*

For the first 3 weeks, once a week at the same time of day you will complete the same stress-scale self-report measure, and a blood sample will be taken from you. The blood sample is to measure your immune function and this is repeatedly measured once a week over the first 3 weeks for a more accurate baseline. Finally, you will complete an exercise and health questionnaire and a nutritional assessment questionnaire just before the HIIT program. On the third the third week we will conduct a treadmill submaximal fitness test with you. This will take place at Massey University, Sport and Exercise Science rooms. Each pretest should take approximately 40 minutes.

*Half-way Point*

After 5 weeks, the same stress self-report measure and a further blood test from you will be taken. This test should take approximately 40 minutes.

*End of the Study*

At the end of the 10 weeks of supervised training you will be asked to complete the same stress self-report measure along with a blood sample once a week for 3 weeks
post the HIIT programme. Each post test should take approximately 40 minutes.

The Training

The HIIT training will take place three times per week for 10 weeks. This will be supervised either at Massey University or with permission at the NZ Police Gymnasium in Wellington city. The training will comprise of a 5-minute warm up followed by ~20 minutes of short bursts of maximal effort upper and lower body exercises. The cool down of 5 minutes will be light exercise such as walking and stretching. The exercise intervention should take approximately 40 minutes, three times a week a total time of 20 hours.

The estimated time for the completion of the entire study is 24.55 hours.

What are the benefits and risks of taking part in this study?

The personal benefits of taking part in both parts of this study are you should see an increase in your fitness level. If the hypothesis of the study is correct your immune function will be improved and stress levels decrease. You will also have information on your immune response pre and post-exercise, which you can choose to share with your doctor.

The risk of taking part is an initial discomfort from the high intensity exercise. This is common and is known as delayed onset soreness. It is at its worse ~48 hours after your first bout of high intensity exercise, but then will ease. The pain is minor and should not interfere with normal daily functioning. To account for this risk at each training there will be a compulsory five-minute warm up and down.

What will happen to this information?

The data will be used only for the purposes of this project, the data will be coded so that no individual can be identified. No individual information will be disclosed to any other members of the police including management. Individuals Perceived Stress Scale scores are personal and will not be shared with anyone other than the researcher. Only the investigators of the study will have access to personal information and this will be kept secure and strictly confidential. Results of this project may be published or presented at conferences or seminars, and disseminated in scientific research journals, but no individual will be able to be identified. At the end of the study you will receive a brief report summarizing the main findings of the research project via post or e-mail if you wish to receive the summary of the study.

Participant’s Rights

You are under no obligation to accept this invitation. If you decide to participate, you
have the right to:

- decline to answer any particular question;
- withdraw from the study at any time
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used,
- be given access to a summary of the project findings when it is concluded.

Who may I contact for further information?

For any questions, please feel free to contact Rebecca Kurtovich or one of the study supervisors Dr. Sally Lark or Dr Ian de Terte.

Rebecca Kurtovich

Dr. Ian de Terte
i.deterte@massey.ac.nz 04-979-3603

Dr. Sally Lark
S.Lark@Massey.ac.nz; 04-979-3497

Thank you for considering participating in this study!

Compensation for Injury

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Accident Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements, and must help you access those entitlements. Entitlements may include, but not be limited to, treatment costs, travel costs for rehabilitation, loss of earnings, and/or lump sum for permanent impairment. Compensation for mental trauma may also be included, but only if this is incurred as a result of physical injury.

If your ACC claim is not accepted, you should immediately contact the researcher. The researcher will initiate processes to ensure you receive compensation equivalent to that to which you would have been entitled had ACC accepted your claim.

Mental Health

All participants that are identified as having high perceived stress from the stress scale questionnaire will be offered the option of seeing a clinical psychologist via the college of clinical psychologists or the New Zealand Psychological Society (NZPsS). The primary researcher will refer you to the Massey Psychology Clinic via a letter. In addition to this
it is noted that the New Zealand Police also offer psychological support services that can be accessed by any of the participants if additional support is required.

Committee Approval Statement
This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 16/22. If you have any concerns about the conduct of this research, please contact Mr Jeremy Hubbard, Chair, Massey University Human Ethics Committee: Southern A, telephone 04 801 5799 x 63487, email humanethicssoutha@massey.ac.nz
Appendix B: Consent Form

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

High Intensity Intermittent Training in Clinically Stressed Persons to Relieve Symptoms

PARTICIPANT CONSENT FORM - INDIVIDUAL

I have read the Information Sheet concerning this project and have had the details of the study explained to me. My questions have been answered to my satisfaction. I understand that I may ask further questions and request further information at any time.

I know that:

1. My participation in the project is entirely voluntary
2. I am free to withdraw at any time without any disadvantage to myself.
3. Any personal information such as my name will not be used in the publication of this study. Any information that is used in the publication of the study will be such information that I will not be identifiable in order to preserve my anonymity.

Please indicate yes or no for the following:

- Would you like to receive back the remaining bloods after analysis for appropriate cultural disposable? Y / N

I agree to participate in this study under the conditions set out in the Information Sheet.

Signature: ........................................................................................................... Date: ........................................

Full Name - printed ........................................................................................................
Appendix C: Personal Letter of Invite

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAOORA TANGATA

18/03/2016
NZ Police

Dear member of the NZ Police,

I am currently a 5th year Sport and Exercise Masters student at Massey University. As part of my research, I will be examining the effects of High Intensity Intermittent Training (HIIT) on people’s stress symptoms and immune function, and invite you to participate. If you agree to voluntarily participate in this study there will be no physical or psychological harm to you from participating in this study, but there may be some strenuous exercise.

The result of constant chronic stress is that people often find their immunity decreases and that illness occurs more frequently. Stress can often disrupt the immune system and leave it unbalanced, therefore not working as effectively as it could. There is evidence to suggest that regular exercise helps reduce depression, anxiety and stress in individuals. It is thought that a high intensity intermittent exercise programme could be an alternative therapy to treat chronic stress.

An information sheet outlining all procedures and that participation is voluntary and confidential is enclosed.

We ask that you read the information sheet and if you have any questions about the study or require further information please do not hesitate to contact me or my research supervisor’s Dr Sally Lark or Dr Ian de Terte. My direct line is [hidden], or alternatively my email is [hidden]. Dr Sally Lark’s direct line is 04 801 5799 ext 6461, or alternatively her e-mail address is: s.lark@massey.ac.nz. Dr Ian de Terte’s direct line is 4-979 3603, or alternatively his email address is: i.deterte@massey.ac.nz.

Yours sincerely

Rebecca Kurtovich
Primary Investigator

Sport and Exercise Science, Massey University
Appendix D: Perceived Stress Scale

*Perceived Stress Scale Questionnaire (PSS)*

Name: ........................................................................................................................................................................

Date: ........................................................................................................................................................................

**Instructions**

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate *how often* you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly and quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate.

For each question choose from the following alternatives:

1. Never
2. Almost never
3. Sometimes
4. Fairly often
5. Very Often

1. In the last month, how often have you been upset because of something that happened unexpectedly?

2. In the last month, how often have you felt that you were unable to control the important things in your life?

3. In the last month, how often have you felt nervous and “stressed”?  

4. In the last month, how often have you dealt successfully with irritating life hassles?

5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?

6. In the last month, how often have you felt confident about your ability to handle your personal problems?

7. In the last month, how often have you felt that thing were going your way?

8. In the last month, how often have you found that you could not cope with all the things you had to do?
9. In the last month, how often have you been able to control irritations in your life? □
10. In the last month, how often have you felt that you were on top of things? □
11. In the last month, how often have you been angered because of things that happened that were outside of your control? □
12. In the last month, how often have you found yourself thinking about things that you have to accomplish? □
13. In the last month, how often have you been able to control the way you spend your time? □
14. In the last month, how often have you felt difficulties were piling up so high that you could not overcome? □
Appendix E: Revised Mini Nutritional Assessment.

**Amended Mini Nutritional Assessment**

<table>
<thead>
<tr>
<th>LAST NAME:</th>
<th>FIRST NAME:</th>
<th>CODE:</th>
</tr>
</thead>
<tbody>
<tr>
<td>SEX:</td>
<td>AGE:</td>
<td>WEIGHT (kg):</td>
</tr>
<tr>
<td>DATE</td>
<td>WAIST CIRCUMFERENCE:</td>
<td>HIP CIRCUMFERENCE:</td>
</tr>
</tbody>
</table>

**Please circle the correct answer to the questions below.**

<table>
<thead>
<tr>
<th>SCREENING:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Has your food intake declined over the past 3 months due to loss of appetite or digestive problems?</td>
<td></td>
</tr>
<tr>
<td>a) Severe decrease in food intake</td>
<td>b) Moderate decrease in food intake</td>
</tr>
<tr>
<td>B. Have you experienced weight loss during the last 3 months?</td>
<td></td>
</tr>
<tr>
<td>a) Weight loss greater than 3kgs</td>
<td>b) Don’t know</td>
</tr>
<tr>
<td>C. Have you suffered psychological stress or acute disease in the past 3 months</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>D. Have you experienced psychological distress or acute disease in the past 3 months</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>E. Do you consider you have?</td>
<td></td>
</tr>
<tr>
<td>a) Normal nutritional status</td>
<td>b) At risk malnutrition</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ASSESSMENT</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>F. Do you live alone</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>G. Take prescription drugs each day</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>H. How many full meals do you have each day</td>
<td></td>
</tr>
<tr>
<td>a) 1 meals</td>
<td>b) 2 meals</td>
</tr>
<tr>
<td>I. Do you snack during the day?</td>
<td></td>
</tr>
<tr>
<td>a) Once</td>
<td>b) Twice</td>
</tr>
<tr>
<td>J. Selected consumption markers for protein intake</td>
<td></td>
</tr>
<tr>
<td>- At least one serving of dairy products (milk, cheese or yoghurt) per day.</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>- Two or more servings of legumes or eggs per week</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>- Meat, fish or poultry every day</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>K. Do you consume 3 or more servings of fruit and vegetables per day</td>
<td></td>
</tr>
<tr>
<td>a) Yes</td>
<td>b) No</td>
</tr>
<tr>
<td>L. How much fluid (water, juice, coffee, tea, milk) do you consume per day</td>
<td></td>
</tr>
<tr>
<td>a) less than 3 cups</td>
<td>b) 3 to 5 cups</td>
</tr>
<tr>
<td>M. In comparison with other people the same age, how do you consider your health status</td>
<td></td>
</tr>
<tr>
<td>a) not as good</td>
<td>b) don’t know</td>
</tr>
</tbody>
</table>
Appendix F: International Physical Activity Questionnaire (IPAQ)
17. How much time did you usually spend on one of those days doing moderate physical activities in the garden or yard?
   ___ hours per day
   ___ minutes per day

18. Once again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate activities like carrying light loads, washing windows, scrubbing floors and sweeping inside your home?
   ___ days per week
   ❑ No moderate activity inside home ➔ Skip to PART 4: RECREATION, SPORT AND LEISURE-TIME PHYSICAL ACTIVITY

19. How much time did you usually spend on one of those days doing moderate physical activities inside your home?
   ___ hours per day
   ___ minutes per day

PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY

This section is about all the physical activities that you did in the last 7 days solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during the last 7 days, on how many days did you walk for at least 10 minutes at a time in your leisure time?
   ___ days per week
   ❑ No walking in leisure time ➔ Skip to question 22

21. How much time did you usually spend on one of those days walking in your leisure time?
   ___ hours per day
   ___ minutes per day

22. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities like aerobics, running, fast bicycling, or fast swimming in your leisure time?
   ___ days per week
   ❑ No vigorous activity in leisure time ➔ Skip to question 24

11. How much time did you usually spend on one of those days to bicycle from place to place?
   ___ hours per day
   ___ minutes per day

12. During the last 7 days, on how many days did you walk for at least 10 minutes at a time to go from place to place?
   ___ days per week
   ❑ No walking from place to place ➔ Skip to PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

13. How much time did you usually spend on one of those days walking from place to place?
   ___ hours per day
   ___ minutes per day

PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

This section is about some of the physical activities you might have done in the last 7 days in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family.

14. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, chopping wood, shoveling snow, or digging in the garden or yard?
   ___ days per week
   ❑ No vigorous activity in garden or yard ➔ Skip to question 16

15. How much time did you usually spend on one of those days doing vigorous physical activities in the garden or yard?
   ___ hours per day
   ___ minutes per day

16. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate activities like carrying light loads, sweeping, washing windows, and raking in the garden or yard?
   ___ days per week
   ❑ No moderate activity in garden or yard ➔ Skip to question 18
23. How much time did you usually spend on one of those days doing vigorous physical activities in your leisure time?
   _____ hours per day
   _____ minutes per day

24. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities like bicycling at a regular pace, swimming at a regular pace, and doubles tennis in your leisure time?
   _____ days per week
   □ No moderate activity in leisure time → Skip to PART 5: TIME SPENT SITTING

25. How much time did you usually spend on one of those days doing moderate physical activities in your leisure time?
   _____ hours per day
   _____ minutes per day

PART 5: TIME SPENT SITTING

The last questions are about the time you spend sitting while at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television. Do not include any time spent sitting in a motor vehicle that you have already told me about.

26. During the last 7 days, how much time did you usually spend sitting on a weekday?
   _____ hours per day
   _____ minutes per day

27. During the last 7 days, how much time did you usually spend sitting on a weekend day?
   _____ hours per day
   _____ minutes per day

This is the end of the questionnaire, thank you for participating.
Appendix G: Methodology Flow Dig.

1) Information sheets distributed to Police Force.

2) Volunteers for study fill out consent form and perceived stress scale questionnaire.

Case Study One

Case Study Two

Case Study Three

3) IPAQ and MNA questionnaires

4) Pre-test measures A, B and C, once per week over 3 weeks (weekly intervals)

Pre A: complete PSS.

Pre B: Base line BP and HR assessed

Pre C: Bloods. Cortisol, leukocytes, cytokines CD-4

A sub max test

5) start 10 week HIIT training
   3x per week

5) start 10 week HIIT training
   3x per week

6) Post-measures A, B and C, once per week over 3 weeks (weekly intervals)

at week 5 of training repeat measures A, B and C
Appendix H: Exercise Intervention sample of day one of weeks one, five and ten.

<table>
<thead>
<tr>
<th>Week One: Monday morning 7.30am individual session</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Warm Up: Jogging 3 minutes back and forward, plus plyometric stretching</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exercises</th>
<th>Time/Reps/sets</th>
<th>Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardio Starter: Sprints</td>
<td>30 seconds, 3x1</td>
<td>30 seconds between rep.</td>
</tr>
<tr>
<td>Circuits</td>
<td>Time/Reps/Sets</td>
<td>Rest</td>
</tr>
<tr>
<td>1) Circuit Lower Body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Squats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Low box jump</td>
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<td></td>
</tr>
<tr>
<td>- Lunge</td>
<td></td>
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<tr>
<td>Complete exercise for 20 seconds, followed by 30 seconds of active rest. Repeat circuit 3xs. At completion of the 3rd circuit participants get 1minute recovery.</td>
<td></td>
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<tr>
<td>2) Circuit Upper Body</td>
<td></td>
<td></td>
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<tr>
<td>- Bent over row</td>
<td></td>
<td></td>
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<tr>
<td>- Curl to press</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Press up</td>
<td></td>
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</tr>
<tr>
<td>Complete exercise for 20 seconds, followed by 30 seconds of active rest. Repeat circuit 3xs. At completion of the 3rd circuit participants get 1minute recovery.</td>
<td></td>
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<tr>
<td>3) Circuit Core</td>
<td></td>
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<tr>
<td>- Spider Planks</td>
<td></td>
<td></td>
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<tr>
<td>- Walk out</td>
<td></td>
<td></td>
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<tr>
<td>- Russian Twist</td>
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<td></td>
</tr>
<tr>
<td>Complete exercise for 20 seconds, followed by 30 seconds of active rest. Repeat circuit 3xs. At completion of the 3rd circuit participants get 1minute recovery.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warm Down- Jogging 2 minutes, walking 2 minutes, followed by static stretching.</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Week Five: Individual Session</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Warm Up: Jogging 3 minutes back and forward, plus plyometric stretching</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exercises</th>
<th>Time/Reps/sets</th>
<th>Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardio Starter: Sprints</td>
<td>30 seconds, 3x1</td>
<td>15 seconds between rep.</td>
</tr>
<tr>
<td>Circuits</td>
<td>Time/Reps/Sets</td>
<td>Rest</td>
</tr>
<tr>
<td>1) Circuit One</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- 5 mountain climbers + 1 push up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Kettlebell pull up</td>
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<td></td>
</tr>
<tr>
<td>- Burpee Row</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complete each exercise for 40 seconds, rest in between each exercise for 10 seconds. Repeat the circuit 3xs. Rest for one minute at the end of the this circuit then move station.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
2) Circuit Two  
- Round the world lunge  
- Snap Jumps  
- Triple pulse squat  
Complete each exercise for 40 seconds, rest in between each exercise for 10 seconds. Repeat the circuit 3xs. Rest for one minute at the end of the circuit then move station.

3) Circuit Three  
- Sit up and twist  
- In outs  
- Flutter Kicks  
Complete each exercise for 40 seconds, rest in between each exercise for 10 seconds. Repeat the circuit 3xs. Rest for one minute at the end of the circuit then move station.

Challenge- QUICK AS YOU CAN!!  
- 10 sit ups  
- 10 kettle bell swings  
- 10 box jumps  
Complete the challenge at the start of the session after sprints. Then at end of session before warm down. Not timed, participants to complete as quick as possible with correct form and technique. Compete against your group.

Warm Down- Jogging 2 minutes, walking 2 minutes, followed by static stretching.

---

**Week 10: Group Session**

**Warm Up:** Jogging 3 minutes back and forward, plus plyometric stretching

**Cardio Starter**

- Suicide Run: 4 minutes of work followed by a 2-minute rest and final stretch before circuit.
  - Row One- Basketballers x10
  - Row Two- Plank Shoulder Tap x10
  - Row Three- Squat x10
  - Row Four- Star Jump Toe Taps x10

Participant runs out to line completes exercise runs back to start, participant runs out to second line completes exercise runs back to start, and so on for 4 minutes.

<table>
<thead>
<tr>
<th>Circuits</th>
<th>Time</th>
<th>Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Circuit One</td>
<td>Each circuit will be run for 2:30. Each participant will complete exercise at correlating number of reps then move to the next exercise. They will continue</td>
<td>30 seconds rest at the end of the 2:30 circuit. Each participant must complete each circuit x2</td>
</tr>
<tr>
<td>Squat Jump x20</td>
<td></td>
<td></td>
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<tr>
<td>Sprint x2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jump Lunge x20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sprint x2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2) Circuit 2</td>
<td>to complete each exercise until the 2.30 is complete</td>
<td></td>
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<tr>
<td>----------------------------------</td>
<td>------------------------------------------------------</td>
<td></td>
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<tr>
<td>Punches x50</td>
<td></td>
<td></td>
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<tr>
<td>Bum Flicks x50</td>
<td></td>
<td></td>
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<tr>
<td>X Jumps x20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Broad Jumps x20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3) Circuit Three</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fast feet x110, drop burpee</td>
<td></td>
<td></td>
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<tr>
<td>x1 repeat x5</td>
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<td></td>
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<tr>
<td>High knees x10, mountain climber</td>
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<tr>
<td>x1 repeat 5xs</td>
<td></td>
<td></td>
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<tr>
<td>Crab walk, jump x5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snap jump squat x5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circuit Four</td>
<td></td>
<td></td>
</tr>
<tr>
<td>180 squat and lunge x10</td>
<td></td>
<td></td>
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<tr>
<td>Side to sides x19</td>
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<td></td>
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<tr>
<td>Medicine ball wall throws x10</td>
<td></td>
<td></td>
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<tr>
<td>Push Ups x10</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Warm Down- Jogging 2 minutes, walking 2 minutes, followed by static stretching.
Appendix I: Exercise Demonstration Sheet

WEEK THREE HOME SESSION: this handout runs you through any new exercise, that we have not used before but have gone over in our group sessions.

CIRCUIT ONE:

Step up Lunge Back

- Place right foot on the bench in front of you
- Step up and drive left knee towards chest
- Then step backwards off box placing left foot on the floor
- Right foot then steps backwards into a lunge
- Remember to keep toes facing forward, and do not allow knee to come over the front foot’s toes.
- Keep torso upright and chest out at all times,

Tricep Dips

- Using either floor, chair or box
- Place palms on floor, chair or box so that finger tips face towards you with hands shoulder width apart and sitting just under shoulders
- Keep feet flat on floor shoulder width apart
- Bend at elbows so that bum is just above the floor.
- Extend elbows pushing body up and off the floor. Your thighs shoulder move upwards towards the roof, and your bottom should be off the floor.
CIRCUIT TWO

Scissors

- Lie on floor
- Black flat legs extended in front of you raised off the ground
- Engage core, while in this position, move legs in a horizontal direction with each foot crossing over the other.

CIRCUIT THREE

Push up with shoulder touches

- Complete a standard push up
- When in extended position remove one hand from ground and tap opposite shoulder
- Repeat with opposite hand
- Lower pack into push up and repeat
- Remember to keep core tight and do not leave tummy behind when you push up.
- When in plank position keep bum tucked in and back straight

Tuck Jumps

- Stand feet shoulder width apart.
- Engage core
- Push off group with both feet using heels
- Tuck legs as close to chest as possible
- Land softly with knees bent
Appendix J: Heart Rate recording sheet

<table>
<thead>
<tr>
<th>HR Recording During Sessions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date:</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>P1 Target HR</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Circuit 1 HR</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>Circuit 2</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>Circuit 3</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>Sprints</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
</tbody>
</table>
Appendix K: Participant Information Consent Form for Participants Section

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

**High Intensity Intermittent Training in Clinically Stressed Persons to Relieve Symptoms**

**PARTICIPANT INFORMATION CONSENT FORM – INDIVIDUAL**

I have read the Participant information section of the thesis and I am satisfied that my anonymity has been maintained. My questions have been answered to my satisfaction and any changes I have asked for have been completed. I understand that as of the 21st of February no further changes can be made to this section of the thesis.

**Please check that**

1. Any personal information such as my name will not be used in the publication of this study.
2. Any information that is used in the publication of the study is not identifiable in order to preserve my anonymity.
3. I am happy with what has been written and feel it is preserves my anonymity.

I agree to publication of the Participant Information section of the thesis regarding my involvement in a HIIT programme through Massey University.

Signature:  

Date:  

Full Name - printed: