THE INFLUENCE OF PSYCHOSOCIAL AND LIFESTYLE FACTORS ON ILLNESS AND INJURY IN BRITISH ARMY RECRUITS

By

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A thesis submitted in partial fulfilment of the

requirements of Liverpool John Moores University

for the degree of **Doctor of Philosophy**

May 2026

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LIST OF ABBREVIATIONS

ACEs – Adverse Childhood Experiences

BMI – Body Mass Index

CRP – C-reactive Protein

CTQ – Childhood Trauma Questionnaire

LOT-R – Life Orientation Test–Revised

MSKI – Musculoskeletal Injury

OR – Odds Ratio

PLANS – Parents lived apart and never spoke

PSS – Perceived Stress Scale

PSQ – Perceived Sleep Quality

PSQI – Pittsburgh Sleep Quality Index

RTI – Respiratory Tract Infection

SES – Socioeconomic Status

URTI – Upper Respiratory Tract Infection

ABSTRACT

Starting military training represents for many a major life transition. Both the high physical and high psychological demands of military training are thought to increase the risk of illness and injury in military recruits, however, the role of these demands in military recruits undertaking Army training is not well understood. In young adults embarking on basic military training, this thesis aimed to examine how childhood adversity and sleep behaviour influence infection and injury risk and whether changes in psychosocial and lifestyle factors during training impact the influence. In **Chapter 4**, childhood adversity, particularly exposure to multiple or abuse-related events, was associated with a significantly higher risk of respiratory tract infection (RTI) during adulthood. This relationship persisted after adjusting for potential confounders, demonstrating that early-life adversity exerts a lasting impact on adult immune function. Perceived stress and poor sleep quality during training partially account for this relationship. **Chapter 5** examined the protective role of optimism on the relationship between childhood adversity and RTI. Recruits with a history of childhood adversity who showed increases in optimism across the first month of training exhibited no increased RTI risk, whereas those with decreases in optimism were over five times more likely to suffer RTI. **Chapters 6 – 8** focused on the role of sleep on illness and injury. In **Chapter 6**, sleep restriction significantly increased upper respiratory tract infection (URTI) risk; however, this association was moderated by perceived sleep quality (PSQ). Recruits experiencing sleep restriction who reported good PSQ were at no greater risk of URTI compared to non-sleep-restricted individuals, whereas those with sleep restriction and poor PSQ were twice as likely to suffer infection. **Chapter 7** extended these findings, demonstrating that poor sleep quality, independent of sleep duration, was consistently associated with increased RTI risk throughout training. Recruits who improved their sleep quality from civilian life to training were protected against infection, while those with enduring poor sleep were most at risk. Finally, **Chapter 8** demonstrated that poor sleep quality and short sleep duration were associated with higher incidence of musculoskeletal injury (MSKI). Recruits with poor or short sleep were three to four times more likely to sustain an injury during training, suggesting that sleep disturbances compromise MSKI integrity. Collectively, this thesis presents novel evidence that both early-life psychosocial experiences and current behavioural factors shape adult health outcomes during military training.

DECLARATION

I hereby declare that this thesis is the results of my own investigations, except where otherwise stated. All other sources are acknowledged by bibliographic references. This work has not previously been accepted in substance for any degree and is not being concurrently submitted in candidature for any degree unless, as agreed by the University, for approved dual awards.

ACKNOWLEDGEMENTS

First and foremost, I would like to express my deepest gratitude to my supervisor, Prof. Neil Walsh. I am forever grateful for the countless insightful discussions, feedback on my work, and the opportunities you provided for me to grow and learn throughout my PhD journey.

I would also like to give great thanks to my postdoc, Dr Jason Edwards, for his constant support and guidance throughout the years together. Further appreciation goes to my colleague Catherine Gage; I consider myself lucky to have worked alongside and developed friendships with you and Jason. I will always hold fond memories from our countless research trips up the M1 to Catterick. I would also like to thank Dr. Martin Eubank for taking on co-supervising duties and being open and willing to help me when needed.

I would further like to thank all the postgraduate and undergraduate students who formed the research teams to assist with data collection in each of the studies, as well as the training staff, medical staff, and military recruits at The Infantry Training Centre, Catterick for their cooperation and participation in the series of projects.

This PhD was sponsored by the Army Recruiting and Initial Training Command (ARITC), Ministry of Defence, who helped shape the study design and supported data collection. Again, I am thankful to Neil and Jason as well as the team at ARITC for affording me the opportunity to work on an impactful, exciting, and challenging project.

I am eternally thankful to all my friends and family for their continued care and encouragement throughout the ups and downs of this journey. In particular, my wife Sarah, for her confidence in me and her warmth during stressful times, providing a constant source of strength and steadiness for me. Finally, to my parents for their unwavering love and support throughout my many years of education.

PUBLICATIONS

Publications arising from work presented within this thesis

Walsh, N. P., Kashi, D. S., Edwards, J. P., **Richmond, C.**, Oliver, S. J., Roberts, R., ... & Greeves, J. P. (2023). Good perceived sleep quality protects against the raised risk of respiratory infection during sleep restriction in young adults. Sleep, 46(1), zsac222.

Conference proceedings

Richmond, C., Edwards, J. P., Gage, C. R., Vedhara, K., Cole, S. W., Fairclough, L. C., … & Walsh, N. P. (2024). Childhood adversity and respiratory infection risk in adulthood: a prospective cohort study in military recruits. Verbal presentation at the International Society of Exercise Immunology, University of Vienna, September 2024. Presented by **C. Richmond**.

CHAPTER 1 – General Introduction

The British Army trains approximately 9,000 new military recruits each year. New recruits are required to complete a 28 week (Phase 1 & Phase 2) syllabus of military training consisting of basic military skills including physical training, weapon handling, map reading and field craft. The early weeks of Phase 1 are widely recognised as the most physically and psychologically demanding period of the training pipeline, with recruits facing a combination of novel stressors including geographic relocation, separation from family and social networks, and a training tempo that escalates rapidly in intensity. Phase 2 follows successful completion of Phase 1 and delivers the specialist role-specific skills required for operational deployment. While generally less physically intense than Phase 1, Phase 2 introduces distinct pressures associated with more advanced performance assessment, greater individual accountability and the anticipation of operational deployment. Both the high physical and high psychological demands of military training are thought to increase the risk of illness and injury in military recruits (O'Leary et al., 2023; Walsh, 2018), resulting in lost training days and medical attrition that incur a significant financial burden to military organisations and reduced operational effectiveness (e.g., increased training wastage and reductions in gains to trained strength). Evidence indicates that psychological stress and anxiety raise the risk of illness and injury in athletes competing at major sporting events (Drew et al., 2018; Timpka et al., 2017). However, the role of psychosocial stress and lifestyle stress in military recruits undertaking Army training is not well understood, underpinning the requirement for this thesis.

Recently, there has been increased interest among researchers working in the field of psycho-neuro-immunology in understanding how childhood adversity influences immunity and life-long health. Childhood adversity, including physical, emotional and sexual abuse, and physical and emotional neglect, is a major public health concern with estimated incidence rates between 30-50% in developed countries (Ho et al., 2020; Rod et al., 2020) and an estimated lifetime economic burden of £1.6 trillion (Peterson, Florence, & Klevens, 2018). However, the incidence of childhood adversity in British Army infantry recruits remains to be determined. Recruits who experience childhood adversity are more likely to report higher levels of negative psychosocial factors (e.g., psychological stress, depressive symptoms) and poorer levels of coping at the start and during military training, with potential deleterious impacts on immune health and injury (Tiesman et al., 2006). Evidence suggests that unfavourable childhood

environments are associated with an array of physical and mental health problems in adulthood including infection, cardiovascular disease, accelerated biological aging, suicide risk and premature mortality (Brown et al., 2009; Hughes et al., 2017; Murphy, Cohen, Janicki-Deverts, & Doyle, 2017; Norman et al., 2012; Sumner, Colich, Uddin, Armstrong, & McLaughlin, 2019). The effects of childhood adversity likely persist into adulthood because childhood is a developmentally sensitive period that shapes stress-sensitive biological pathways, with numerous studies showing associations between childhood adversity and dysregulated inflammation and hypothalamic-pituitary-adrenal (HPA) axis activity in adulthood (Appelmann, Manigault, Shorey, & Zoccola, 2021; Chen & Lacey, 2018; Iob, Lacey, & Steptoe, 2020; Kuras et al., 2017; Renna et al., 2021). Moreover, studies have shown that exposure to multiple childhood adversities or exposure to abuse related adversity are related with poorer biological outcomes including shorter telomere length, dysregulated diurnal cortisol rhythm, altered cortisol reactivity to acute stress and impaired cellular immunity (Busso, McLaughlin, & Sheridan, 2017; Fagundes, Glaser, Malarkey, & Kiecolt-Glaser, 2013; Hughes et al., 2017; Kiecolt-Glaser et al., 2011; LoPilato et al., 2020; Slopen, McLaughlin, Dunn, & Koenen, 2013; Whittaker, 2018). As such, dysregulation of biological pathways may result in increased respiratory infection risk, but the influence of childhood adversity on immune health in British Army recruits remains unknown. Identifying the influence of childhood adversity on immune health will pave the way for practical mitigation strategies to reduce the adverse effects of childhood adversity by reducing the levels of negative psychosocial factors (e.g., high psychological stress/poor sleep) and increasing coping resources (e.g., resilience/optimism) during military training.

The incidence of RTI is high during basic training, with previous findings showing that approximately 1 in 10 male and female Army recruits who complete Phase One training suffer a RTI requiring a visit to the Doctor, resulting in an average of more than three days downgraded from training (Harrison et al., 2021; Wentz et al., 2018). The true burden of RTI on training effectiveness is likely to be much greater as sick recruits are often reluctant to visit the Doctor when they should. Research is now looking beyond the effects of physical training and considering the role of other lifestyle factors e.g., psychological, sleep disruption etc. in the well-known decrease in host defence and increase in infections during military training (Walsh, 2018). It is likely that psychosocial stress and other lifestyle factors in Army recruits (e.g., poor sleep) contribute to increased susceptibility to infection during training. It is important to identify factors that reduce host-defence and increase RTI, paving the way for

simple, cost-effective strategies to reduce illness leading to fewer training days downgraded or lost, better sickness and absence management and improved operational effectiveness.

Musculoskeletal injuries (MSKIs) are common in Army recruits as they face a new living environment combined with high-intensity physical training, and present a considerable threat to completion of training, occupational fitness, and overall military readiness. Reports from Basic Combat Training in the U.S Army highlight that MSKI incidence is as high as 42% in men, and recruits who suffer an injury during training are more likely to be discharged from the military than those who do not incur an injury (Jones, Bovee, Harris, & Cowan, 1993; Nye, Pawlak, Webber, Tchandja, & Milner, 2016). MSKI is also the leading cause of training days lost among military personnel (Bullock, Jones, Gilchrist, & Marshall, 2010). Poor sleep is a proposed risk factor for MSKI with elite athletes and soldiers particularly susceptible to poor sleep pertaining to training and travel (Sargent, Lastella, Halson, & Roach, 2021; Walsh et al., 2020). Yet prospective studies investigating the association between poor sleep and MSKI in athletic populations are lacking. It is important to better understand the relationship between sleep and MSKI in order to identify effective mitigation strategies to help reduce injury burden in recruits, in turn optimising health and military readiness.

CHAPTER 2 – Literature Review

This chapter reviews the existing literature on psychosocial and lifestyle factors that may influence health outcomes in young adults, with particular focus on populations experiencing physical and psychological stress. The review is organised into four main sections. First, the relationship between childhood adversity and adult health is examined, including potential mechanisms involving stress-sensitive biological pathways and immune function (Section 2.1). Second, the bidirectional relationship between sleep and health is explored, encompassing sleep duration, quality, and restriction (Section 2.2). Third, psychological resilience factors, particularly optimism, are reviewed as potential moderators of stress-related health outcomes (Section 2.3). Fourth, military training is discussed as a unique research context for examining these relationships (Section 2.4). The chapter concludes by summarising key gaps in knowledge that this thesis addresses (Section 2.5).

2.1. Childhood adversity and health

Childhood adversity encompasses a range of negative experiences occurring during developmentally sensitive periods, including physical, emotional, and sexual abuse, and physical and emotional neglect. Understanding the long-term health consequences of childhood adversity is essential for identifying vulnerable populations and developing interventions that may mitigate these effects. This section examines the prevalence and health impact of childhood adversity, with particular attention to mechanisms involving stress physiology and immune function.

2.1.1 Prevalence and scope of childhood adversity

Childhood adversity is a major public health concern with estimated incidence rates between 30-50% in developed countries (Ho et al., 2020; Rod et al., 2020) and an estimated lifetime economic burden of £1.6 trillion (Peterson et al., 2018). These figures likely underestimate the true prevalence, as childhood adversity is often underreported due to shame, memory gaps, or reluctance to disclose traumatic experiences (Danese, 2020; Felitti et al., 1998). The prevalence of childhood adversity may be particularly elevated in certain populations. Compared with the general UK population, the incidence of childhood adversity is likely greater in military recruits, who typically come from lower socioeconomic backgrounds where childhood adversity is more prevalent (Walsh, McCartney, Smith, & Armour, 2019). Military servicemen and women may join the armed forces to escape traumatic home environments characterised

by abuse, violence, or other adverse experiences, potentially contributing to heightened prevalence in this population (Blosnich, Dichter, Cerulli, Batten, & Bossarte, 2014; Ginexi, 1994). However, the specific incidence of childhood adversity in UK Army infantry recruits remains to be determined.

2.1.2 Childhood adversity and adult health outcomes

A substantial body of evidence links childhood adversity to adverse health outcomes across the lifespan. Large cohort studies have demonstrated associations between childhood adversity and increased risk of cardiovascular disease, type 2 diabetes, psychiatric disorders, and premature mortality in adulthood (Anda, Butchart, Felitti, & Brown, 2010; Eriksson, Raikkonen, & Eriksson, 2014; Tomasdottir et al., 2015). These associations persist after adjustment for demographic and lifestyle factors, suggesting that childhood adversity exerts effects on health that are not entirely explained by socioeconomic circumstances or health behaviours in adulthood.

Recent research has moved beyond examining childhood adversity as a binary construct (present vs. absent) to consider its multidimensional nature. Studies examining the number and type of adverse experiences have revealed dose-response relationships and differential effects depending on adversity characteristics. For example, older adults who reported multiple adverse childhood experiences (ACEs) (three or more) exhibited chronically elevated CRP levels during adulthood compared to those who did not experience any adversity (Iob et al., 2020). This finding suggests that cumulative exposure to adversity may produce greater biological dysregulation than single adverse events. Furthermore, research distinguishing between different types of adversity has shown that threat-related childhood adversities (e.g., physical, emotional, or sexual abuse), but not deprivation-related adversities (e.g., physical or emotional neglect), were associated with accelerated biological aging in children and adolescents, as demonstrated by shorter telomere length (Sumner et al., 2019). These findings suggest that the biological consequences of childhood adversity are not uniform but depend on the specific nature and severity of the adverse experience.

Despite extensive evidence linking childhood adversity to chronic diseases, a critical gap exists in the literature regarding the influence of childhood adversity on acute infectious illness, particularly RTI. The closest evidence to date comes from a study showing that adults whose parents lived apart and never spoke to each other during childhood (PLANS) were more than

three times as likely to develop a common cold following experimental viral exposure compared to adults from intact families (Murphy et al., 2017). However, interpreting this finding as evidence of childhood adversity effects is challenging. The use of parental relationship status as a proxy for childhood adversity does not capture the breadth, severity, or specific types of adverse experiences (e.g., abuse, neglect) that may differentially impact health. Moreover, the PLANS measure does not distinguish between families in which parents maintained civil relationships despite separation and families characterised by ongoing conflict or trauma. Consequently, whether childhood adversity, assessed using validated measures that distinguish between adversity types and severity, increases RTI risk in adulthood remains an important unanswered question.

2.1.3 Stress, immune health, and mechanisms of childhood adversity effects

To understand how childhood adversity might influence adult health, it is necessary to consider the interplay between psychological stress and immune function. The immune system is a network of organs, cells, and tissues working together as a defence to protect the body against pathogenic microorganisms (bacteria, viruses, fungi, and protozoa). It has two main barriers of defence, firstly; the innate system, and secondly; the acquired system (Walsh, 2018). The innate system is fast to react and consists of physical, chemical, and cellular lines of protection. The acquired system is slower yet more specialised in its attack against microorganisms. It comprises of many different lymphocytes, specifically T and B lymphocytes, each with a specific role to carry out to destroy and protect against invading pathogens. For example, T and B lymphocytes can carry out a multitude of roles from B cell antibody production to the development of T memory cells which provide a heightened response following re-exposure of the pathogen (Walsh, 2018).

Psychological stress profoundly influences immune function. When an individual perceives a situation as threatening, the brain activates a coordinated stress response involving the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic-adrenal-medullary (SAM) axis (Dhabhar & McEwen, 1997). These systems release the “big three” stress hormones, norepinephrine, epinephrine, and cortisol, that modulate immune cell distribution, function, and gene expression. Acute stress can enhance certain aspects of immunity (e.g., redistributing immune cells to sites of potential infection), whereas chronic stress typically suppresses immune function, increasing susceptibility to infectious diseases (Dhabhar, 2018; Segerstrom & Miller, 2004). The relationship between stress and infection susceptibility has been

demonstrated in both naturalistic and experimental studies. For example, in a seminal study, 394 healthy adults were administered nasal drops containing respiratory viruses or saline and then quarantined for observation. Participants who reported higher levels of psychological stress prior to viral exposure exhibited higher rates of respiratory infection in a dose-response manner (Cohen, Tyrrell, & Smith, 1991).

2.1.4 Mechanisms linking childhood adversity to immune dysregulation

The effects of childhood adversity likely persist into adulthood because childhood represents a developmentally sensitive period during which environmental experiences shape the maturation of stress-sensitive biological systems, including the HPA axis, autonomic nervous system, and immune system (Danese & McEwen, 2012; McLaughlin, Sheridan, & Lambert, 2014; Nelson et al., 2020). Two hypotheses have been proposed to explain how childhood adversity leads to lasting immune alterations:

Hypothesis 1: Direct immune programming.

The first hypothesis, articulated by Miller, Chen, and Parker (2011) proposes that childhood adversity directly modifies the developmental trajectory of the immune system, leading to persistent changes in immune cell function and gene expression. This concept of "biological embedding" suggests that early-life stress creates a phenotype characterised by heightened inflammatory activity and diminished antiviral defences. Consistent with this hypothesis, research examining transcriptional profiles in individuals with childhood trauma has identified a conserved transcriptional response to adversity (CTRA), characterised by upregulated expression of pro-inflammatory genes (e.g., IL-1β, IL-6, TNF-α) and downregulated expression of genes involved in antiviral defence, including type I interferon response genes (Cole, 2014). CTRA has been observed in diverse populations exposed to early-life stress, including former child soldiers and adults reporting childhood maltreatment (Bower et al., 2020; Kohrt et al., 2016), suggesting that this molecular signature represents a common pathway through which adversity becomes biologically embedded. The childhood adversity-associated immune phenotype comprises three interrelated features: accelerated immunosenescence, elevated circulating inflammatory markers, and impaired cellular immunity (Elwenspoek, Kuehn, Muller, & Turner, 2017) **(Figure 1)**. These alterations form a self-perpetuating cycle in which chronic inflammation accelerates immune aging, which in turn further impairs cellular immunity and promotes inflammation.

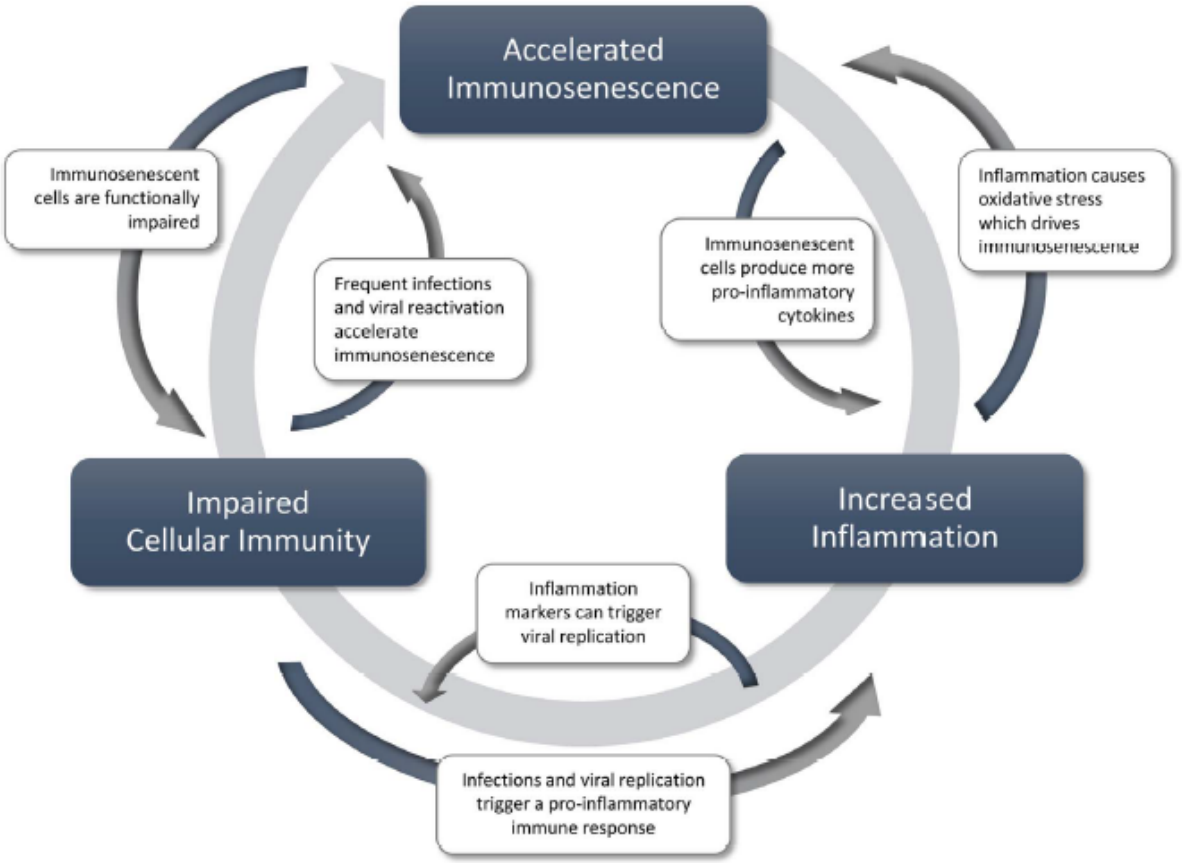

Figure 1. *Childhood adversity phenotype. Accelerated immunosenescence, increased circulatory inflammation markers, and impaired cellular immunity are the three principle immune effects associated with childhood adversity and form a self-perpetuating cycle. Figure taken from Elwenspoek et al., (2017).*

Hypothesis 2: Altered stress physiology.

The second hypothesis proposes that childhood adversity alters HPA axis function and stress reactivity, which indirectly affects immune function throughout the lifespan (Carpenter et al., 2007; Carpenter, Shattuck, Tyrka, Geracioti, & Price, 2011). Adults with childhood adversity histories exhibit greater immune dysregulation and display enhanced emotional and physiological stress sensitivity to subsequent stress. They also present with fewer coping resources to manage such stress (Fagundes, Glaser, & Kiecolt-Glaser, 2013). Research supporting this notion demonstrated that women with a history of childhood adversity exhibited increased HPA and autonomic responses to stress in adulthood compared with controls (Heim et al., 2000). Furthermore, increased psychological stress sensitivity has been demonstrated in women with a history of childhood adversity whereby those who experienced sexual abuse were more likely to suffer depression following a severe stressful life event in adulthood (Dougherty, Klein,

& Davila, 2004). This pattern of heightened stress perception and reactivity may amplify physiological stress responses during challenging life periods, leading to chronic elevations in stress hormones that suppress cellular immunity and increase infection susceptibility.

These two hypotheses are not mutually exclusive. Childhood adversity may directly programme immune cell function while simultaneously altering stress physiology, creating a collaborative vulnerability to health problems. Moreover, both pathways likely interact with ongoing life stressors. Adults with childhood adversity histories not only carry biological vulnerability but also tend to have fewer psychosocial resources (e.g., lower optimism, poorer coping skills) to manage new stressors (Fagundes, Glaser, & Kiecolt-Glaser, 2013), potentially exacerbating stress-related immune dysregulation during challenging transitions such as military training.

The present thesis focuses on clinical health outcomes rather than biomarkers. However, understanding these mechanistic pathways provides a theoretical foundation for interpreting associations between childhood adversity and health outcomes during military training.

2.2 Sleep and Health

Sleep is essential for maintaining physical health, cognitive function, and emotional well-being. Recommended sleep duration varies across the lifespan, with young adults and adults advised to obtain 7-9 hours of sleep per night (Hirshkowitz, Whiton, Albert, Alessi, Bruni, DonCarlos, Hazen, Herman, Katz, et al., 2015). However, modern society has witnessed widespread sleep restriction as individuals curtail sleep to accommodate work, social, and leisure demands (Grandner, Patel, Gehrman, Perlis, & Pack, 2010). Habitual short sleep and poor sleep quality are associated with increased morbidity and mortality, including elevated risk of cardiovascular disease, metabolic disorders, and infectious illness (Cappuccio, D'Elia, Strazzullo, & Miller, 2010a, 2010b; Liu et al., 2017). This section examines the bidirectional relationship between sleep and stress, the effects of sleep duration, restriction, and quality on immune health, and the association between sleep and injury risk.

2.2.1 Sleep stages and architecture

There are 4 main stages of sleep that humans cycle through every night and can be broken down into non-rapid eye movement sleep (NREM) and rapid eye movement sleep (REM) (see **Figure 2**). NREM sleep is further divided down into three stages:

1. NREM 1 – the lightest stage of sleep where breathing and heart rate slow.
2. NREM 2 – a deeper stage of sleep where brain activity slows further and muscle activity decreases.
3. NREM 3 – the deepest stage of sleep, also known as slow-wave sleep (SWS) and is the most restorative stage of sleep.

Following NREM sleep comes the stage of REM where muscle paralysis occurs and brain activity increases, typically resulting in dreaming.

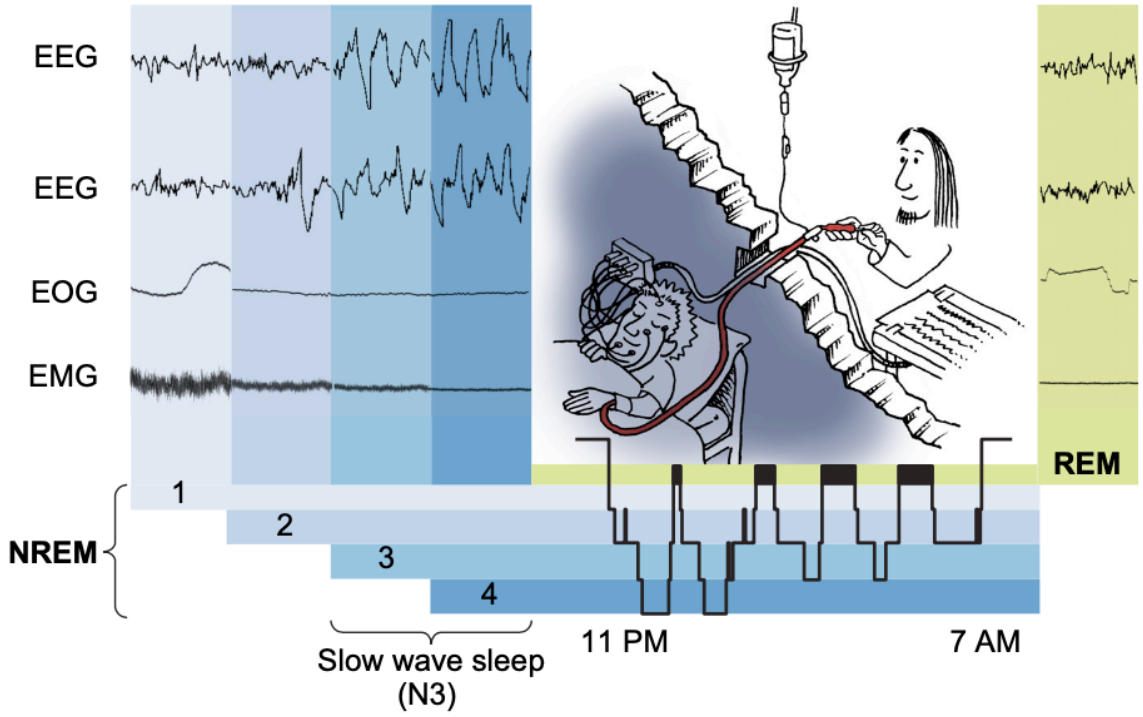

Figure 2. *Prototypical hypnogram with EEG characteristics and sleep laboratory setting. Sleep is divided into stages N1, N2, N3 and rapid-eye-movement (REM) sleep based on specific patterns of brain activity [measured with electroencephalography (EEG)], eye movements [measured with electrooculography (EOG)], and muscle activity [measured with electromyography (EMG)]. The course of the different sleep stages across a sleep period is typically visualized in a hypnogram (bottom right). Figure taken from Besedovsky, Lange, and Haack (2019).*

A typical night of sleep involves cycling through these stages approximately every 90 minutes, with the proportion of time spent in each stage varying across the night. SWS predominates in the first half of the night, whereas REM sleep becomes more prominent in the second half. Disruptions to sleep architecture, such as reduced SWS or fragmented sleep, may have

particularly adverse effects on health, as SWS is thought to be the most restorative sleep stage and plays a critical role in immune function (Besedovsky, Lange, & Haack, 2019).

2.2.2 Bi-directional link between stress and sleep

Sleep and stress exhibit a bidirectional relationship: 1) stress disrupts sleep, and 2) poor sleep amplifies stress reactivity (Prather, 2020; Vgontzas & Chrousos, 2002). Understanding this relationship is crucial because it suggests that sleep disturbances may exacerbate the health consequences of psychosocial stress, including stress stemming from childhood adversity.

1) Stress disrupts sleep.

Psychological stress is a well-established cause of sleep disturbances. One of the earliest studies demonstrating this link found that slow-wave sleep exerts an inhibitory effect on the HPA axis and cortisol secretion (Weitzman, Zimmerman, Czeisler, & Ronda, 1983). Conversely, stress-induced HPA axis activation and elevated cortisol can suppress SWS and fragment sleep, creating a vicious cycle. In occupational settings, individuals reporting high work strain exhibit substantially greater prevalence of disturbed sleep compared to those reporting low work strain (30% vs. 5%; Kalimo R. (2000)). Similarly, poor perceived social support has been associated with disrupted sleep in Vietnam War veterans, suggesting that psychosocial stressors broadly undermine sleep quality (Fabsitz, Sholinsky, & Goldberg, 1997). Importantly, individuals with childhood adversity histories report poorer sleep quality in adulthood, even decades after the adverse experiences occurred, indicating that early-life stress may have lasting effects on sleep regulation (Greenfield, Lee, Friedman, & Springer, 2011; Liu et al., 2023).

2) Poor sleep amplifies stress reactivity.

The reverse pathway, poor sleep exacerbating stress, is equally important. Sleep restriction elevates circulating cortisol levels and amplifies HPA axis and sympathetic nervous system responses to subsequent stressors (Guyon et al., 2014; Vgontzas et al., 1998). Chronic short sleep has been conceptualised as a state of chronic stress, characterised by sustained activation of stress-responsive systems and associated health consequences (Vgontzas & Chrousos, 2002). This bidirectional relationship underscores the potential for sleep disturbances to compound the effects of other stressors, such as childhood adversity or the demands of military training, on health outcomes.

2.2.3 Sleep duration and immune health

Substantial evidence links sleep duration to immune function and infection susceptibility. In a landmark study, 125 healthy midlife adults received the standard three-dose hepatitis B vaccination series. Sleep duration was assessed via sleep diaries and wrist actigraphy in the days surrounding vaccination. Results showed that objectively measured short sleep duration (<6 hours per night) was associated with reduced antibody response to vaccination (Prather et al., 2012). These findings indicate that insufficient sleep impairs the acquired immune response, potentially compromising protection against infectious diseases. The association between short sleep and infectious illness has also been demonstrated in experimental challenge studies. In one influential study, 164 healthy adults wore wrist actigraphy and completed sleep diaries for seven consecutive nights. They were then administered nasal drops containing rhinovirus and quarantined for five days. Participants sleeping less than six hours per night were more than four times as likely to develop a clinical cold compared to those sleeping more than seven hours (Prather, Janicki-Deverts, Hall, & Cohen, 2015). This study provides strong evidence that habitual short sleep increases infection susceptibility under controlled conditions of pathogen exposure.

Findings from military populations corroborate these experimental results. Military recruits commonly experience sleep restriction during training, with a large proportion sleeping less than the recommended 7-9 hours per night (Wentz et al., 2018). In a large cohort of British Army recruits, those sleeping less than six hours per night during training were four times more likely to be diagnosed with a URTI compared to those sleeping 7-9 hours (Wentz et al., 2018). Moreover, short sleepers lost more training days due to URTI, highlighting the operational consequences of sleep restriction. The transition from civilian life to military training is typically accompanied by significant sleep restriction; Wentz et al. (2018) reported a mean decrease of 1.5 hours per night in self-reported sleep duration from civilian life to training, underscoring the challenge of maintaining adequate sleep during this transition.

2.2.4 Sleep restriction and immune health

While the adverse effects of habitual short sleep are well-established, there is growing interest in understanding how changes in sleep, particularly acute or sustained sleep restriction, affect immune function. Sleep restriction studies in laboratory settings have predominantly examined severe restriction (e.g., total sleep deprivation or restriction to 4 hours per night), which may not reflect the more modest sleep curtailment commonly experienced in real-world settings.

Laboratory studies of severe sleep restriction have demonstrated clear effects on inflammatory markers. One study examined the effect of total sleep deprivation (88 consecutive hours of wakefulness) and partial sleep deprivation (sleep restricted to 4.2 hours per night for 10 days) on high-sensitivity CRP levels in healthy adults. Both conditions resulted in increased CRP levels, whereas a control group maintaining 8.2 hours of sleep per night showed no increase (Meier-Ewert et al., 2004). Longitudinal analyses in population-based studies have similarly shown that reductions in sleep duration across time points are associated with increased circulating CRP and IL-6 levels (Ferrie et al., 2013). These findings suggest that sleep curtailment elevates systemic inflammation, a risk factor for chronic diseases and potentially for acute infections. However, the magnitude of sleep restriction in laboratory studies (e.g., 4 hours per night) is extreme and not representative of typical sleep habits. More modest sleep restriction, such as the difference between weekday and weekend sleep observed in population studies, has also been associated with adverse health outcomes. For example, individuals reporting "sleep debt" (defined as ≥2 hours difference between weekday and weekend sleep) exhibited poorer ideal cardiovascular health compared to those without sleep debt (Cabeza de Baca et al., 2019). Whether such modest sleep restriction increases respiratory infection risk, and whether individual differences in habitual sleep patterns moderate this risk, remains an important question warranting investigation.

2.2.5 Sleep quality and immune health

While much research has focused on sleep duration, emerging evidence suggests that sleep quality may be equally or more important for health. Sleep quality encompasses multiple dimensions, including the subjective perception of sleep adequacy, sleep onset latency, sleep continuity (number and duration of awakenings), and sleep architecture (time spent in restorative sleep stages). Importantly, sleep quality and duration are not perfectly correlated; some individuals sleeping short durations report good sleep quality, whereas others sleeping adequate durations report poor quality.

A striking demonstration of the importance of sleep quality comes from a large epidemiological study examining pneumonia risk. Short sleep duration (≤5 hours per night) was associated with increased pneumonia risk; however, this association was only observed in individuals who perceived their sleep as inadequate (Patel et al., 2012). Short sleepers who perceived their sleep as adequate showed no elevated pneumonia risk, suggesting that subjective sleep quality

modifies the health consequences of short sleep duration. Similarly, other studies have shown that good PSQ is associated with reduced cardiovascular disease risk and better overall health, independent of sleep duration (Cribbet et al., 2014; Hoevenaar-Blom, Spijkerman, Kromhout, van den Berg, & Verschuren, 2011).

The mechanisms by which PSQ influences health independently of duration remain incompletely understood but likely involve multiple pathways. First, good PSQ may reflect better sleep architecture, particularly greater amounts of SWS, which is associated with reduced cortisol secretion, increased growth hormone and prolactin release, and enhanced immune cell redistribution and activation (Akerstedt, Hume, Minors, & Waterhouse, 1997; Besedovsky et al., 2019). During SWS, immune cells exhibit increased migration to lymph nodes where they encounter antigens and undergo activation, supporting the formation of immunological memory (Lange, Dimitrov, Bollinger, Diekelmann, & Born, 2011). Experimental studies have demonstrated that SWS activity the night following vaccination predicts subsequent antibody response, with reductions in cortisol and increases in growth hormone during SWS correlating with antigen-specific T-cell expansion (Lange et al., 2011). Individuals who report good sleep quality despite reduced duration may maintain adequate SWS, thereby preserving immune function. Second, good PSQ may indicate better circadian alignment, even when total sleep time is reduced. Circadian misalignment, such as sleeping at biologically inappropriate times or experiencing irregular sleep-wake schedules, disrupts HPA axis regulation, increases inflammatory markers, and impairs immune function independent of sleep duration (Wright et al., 2015). Individuals who perceive their sleep as good may be better aligned with their endogenous circadian rhythms, minimising inflammatory consequences. Third, PSQ is strongly associated with psychological well-being. Poor sleep quality is linked to higher levels of negative affect, anxiety, and perceived stress (Bower, Bylsma, Morris, & Rottenberg, 2010; Triantafillou, Saeb, Lattie, Mohr, & Kording, 2019), all of which independently increase infection risk (Cohen, Doyle, & Skoner, 1999; Falagas, Karamanidou, Kastoris, Karlis, & Rafailidis, 2010). Conversely, individuals who perceive their sleep as good may experience less psychological distress, which could protect against stress-related immune suppression.

2.2.6 Sleep and injury

In addition to affecting immune function and infection susceptibility, poor sleep has emerged as a potential risk factor for MSKI, particularly in athletic and military populations

(Dobrosielski, Sweeney, & Lisman, 2021; Grier, Dinkeloo, Reynolds, & Jones, 2020; Milewski et al., 2014). Athletes and soldiers are especially vulnerable to sleep disturbances due to high training demands, travel across time zones, irregular schedules, and psychological stress (Sargent et al., 2021; Walsh et al., 2020). Despite growing recognition of sleep as a potential injury risk factor, prospective evidence remains limited, and findings are inconsistent. The Centers for Disease Control and Prevention (CDC) estimated that approximately 8.6 million sports and recreation-related injuries occur annually in the United States (Sheu, Chen, & Hedegaard, 2016). In military settings, MSKI are the leading cause of training days lost and a major contributor to medical attrition, presenting significant financial and operational burdens (Bullock et al., 2010). Identifying modifiable risk factors for MSKI, including sleep, is therefore a high priority for injury prevention.

Cross-sectional and retrospective studies have suggested associations between short sleep duration and increased injury risk in adolescent athletes and military personnel (Grier et al., 2020; Milewski et al., 2014; von Rosen, Frohm, Kottorp, Friden, & Heijne, 2017). However, these studies are limited by reliance on self-reported injuries, recall bias, and inability to establish exact timings of injury occurrence. A systematic review highlighted the need for well-controlled prospective studies examining the association between sleep and MSKI in adult athletic populations (Dobrosielski et al., 2021). One prospective study in Chinese military recruits found that poor sleep quality before commencing military training was associated with increased risk of injury during training (Ruan et al., 2020); however, this study only used subjective measures of sleep and did not examine whether changes in sleep during training predict subsequent injury risk, nor did it distinguish between traumatic and overuse injuries.

The mechanisms linking poor sleep to MSKI likely include multiple pathways. First, sleep loss impairs neurocognitive function, including attention, reaction time, and motor control (Avedesian, Forbes, Covassin, & Dufek, 2022; Wilke & Groneberg, 2022). Slowed reaction times and impaired motor sequence preparation may reduce protective neuromuscular responses during high-impact activities, increasing the likelihood of acute traumatic injuries such as sprains, strains, and fractures. Second, sleep restriction disrupts hormonal balance, including reductions in testosterone and growth hormone, which are important for muscle repair and adaptation (Leproult & Van Cauter, 2011). Third, poor sleep may compromise tissue repair processes, reducing musculoskeletal integrity and increasing vulnerability to both acute and overuse injuries. Given these mechanisms, the association between poor sleep and injury

may be particularly strong for traumatic injuries (caused by single acute events) rather than overuse injuries (resulting from cumulative microtrauma).

Despite the theoretical rationale and emerging evidence, prospective studies investigating sleep as a predictor of MSKI in military training are lacking. Furthermore, no studies have examined whether changes in sleep quality during training predict subsequent injury risk, or whether improvements in sleep quality afford protection against injury. Addressing these gaps is important for developing evidence-based injury prevention strategies in military and athletic populations.

2.2.7 Measuring sleep

Sleep can be assessed using multiple methods, each with distinct advantages and limitations. Polysomnography (PSG), conducted in laboratory settings, is the gold standard for measuring sleep, providing detailed information about sleep stages, respiratory function, and physiological parameters (heart rate, muscle activity, brain activity). However, PSG is expensive, requires technical expertise, and is impractical for large-scale studies or assessment of habitual sleep in naturalistic settings. Actigraphy, which uses wrist-worn accelerometers to estimate sleep-wake patterns based on movement, offers a portable and objective alternative. Actigraphy can record sleep over extended periods (days to weeks), providing data on sleep duration, sleep onset latency, sleep efficiency, and sleep fragmentation. However, actigraphy algorithms vary, and there is inconsistency in definitions of sleep onset and offset, which can affect comparability across studies (Fekedulegn et al., 2020). Subjective measures, including sleep diaries and validated questionnaires such as the Pittsburgh Sleep Quality Index (PSQI), capture individuals' perceptions and experiences of sleep. While subjective measures do not provide physiological detail, they are practical for large studies, capture dimensions of sleep quality not easily measured objectively (e.g., satisfaction with sleep), and importantly, relate to objectively measured sleep parameters and health outcomes (Akerstedt, Hume, Minors, & Waterhouse, 1994; Akerstedt et al., 1997; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Discrepancies between objective and subjective sleep measures have been documented (Baker, Maloney, & Driver, 1999); however, this does not invalidate subjective assessments. Rather, it suggests that objective and subjective measures capture different but complementary aspects of sleep. For example, individuals may perceive their sleep as poor due to psychological factors (worry, stress) even when objective sleep duration and efficiency are adequate. Importantly, recent evidence suggests that PSQ may moderate the relationship between sleep duration and

health outcomes, indicating that subjective assessments capture health-relevant information independent of objective measures (Patel et al., 2012).

2.3 Psychological resilience and optimism

While childhood adversity and poor sleep represent risk factors for poor health, psychological resilience factors may buffer against these risks. Resilience is broadly defined as the capacity to maintain or regain mental health despite experiencing adversity (Dantzer, Cohen, Russo, & Dinan, 2018). Among psychological resilience factors, optimism; defined as the generalised expectancy that good rather than bad outcomes will occur, has received research attention due to its associations with better physical and mental health outcomes (Scheier, Carver, & Bridges, 1994).

2.3.1 Optimism and health outcomes

Optimism has been prospectively associated with numerous health benefits, including reduced risk of cardiovascular events and all-cause mortality (Giltay, Geleijnse, Zitman, Hoekstra, & Schouten, 2004; Rozanski, Bavishi, Kubzansky, & Cohen, 2019). Optimism has also been linked to enhanced antibody responses following vaccination (Dantzer et al., 2018) and reduced susceptibility to respiratory infections (Segerstrom, Taylor, Kemeny, & Fahey, 1998). For example, in one study of law students undergoing the stress of examinations, those with higher optimism exhibited better immune responses (e.g., higher natural killer cell activity) and reported fewer physical symptoms compared to less optimistic students (Segerstrom et al., 1998).

2.3.2 The stress-buffering hypothesis

The mechanisms by which optimism protects health are likely multifaceted. One influential framework is Cohen's stress-buffering model, which proposes that psychosocial resources, including optimism, social support, and coping skills, protect health primarily during times of stress by altering stress appraisals and enhancing coping capacity (Cohen & Wills, 1985). According to this model, optimists appraise potentially stressful situations as less threatening and more manageable, reducing the intensity and duration of physiological stress responses. Optimists also engage in more adaptive coping strategies, such as problem-focused coping and cognitive reframing, which effectively resolve or mitigate stressors (Carver et al., 1993; Scheier et al., 1994). By reducing stress perception and improving coping, optimism may minimise activation of the HPA axis and sympathetic nervous system, thereby protecting immune

function. In contrast, Cohen also proposed a main effects model, suggesting that psychosocial resources promote health regardless of stress levels by fostering positive emotions, healthy behaviours (e.g., better sleep, exercise, nutrition), and social connections that directly support physiological well-being (Cohen & Wills, 1985).

2.3.3 Optimism and childhood adversity

Childhood adversity can be conceptualised as a source of chronic stress that may persist in its effects long after the adverse experiences have ended. According to the stress-buffering hypothesis, individuals with high levels of psychological resilience may be less susceptible to the long-term health consequences of early adverse experiences. Consistent with this framework, research has shown that psychological resilience factors can moderate the biological impact of childhood adversity. For example, in a study of Nepali former child soldiers, those who exhibited greater psychological resilience despite experiencing severe trauma showed less upregulation of pro-inflammatory gene expression compared to those with post-traumatic stress disorder (Kohrt et al., 2016). This finding suggests that resilience can buffer against the inflammatory consequences of severe adversity, potentially reducing subsequent health risks. However, individuals with childhood adversity histories often report lower levels of optimism and other resilience factors in adulthood (Beutel et al., 2017), creating a double vulnerability: biological sensitivity to stress combined with fewer psychological resources to manage stress. This pattern raises an important question: can improvements in optimism during stressful life transitions (e.g., military training) mitigate the health risks associated with childhood adversity? If so, optimism-enhancing interventions could represent a promising strategy for reducing health burden in vulnerable populations.

2.3.4 Modifiability of optimism

An important consideration is whether optimism is a stable trait or can be modified through intervention. While optimism shows moderate stability over time, evidence indicates that it can be enhanced through psychological interventions (Malouff & Schutte, 2016). Techniques that have been shown to increase optimism include best possible self exercises (visualising and writing about positive future outcomes), cognitive restructuring (identifying and challenging pessimistic thinking patterns), gratitude practices, and positive psychology interventions emphasising strengths and positive emotions (Meevissen, Peters, & Alberts, 2011; Peters, Flink, Boersma, & Linton, 2010).

In military contexts, resilience training programmes have been implemented with the goal of enhancing psychological resilience, including optimism, coping skills, and stress management (Precious & Lindsay, 2019). For example, a brief resilience intervention in U.S. Army soldiers produced increases in optimism over a one-month period (Rozek et al., 2019), suggesting that even time-limited interventions may yield meaningful psychological changes. Whether such changes in optimism translate to reduced health problems, particularly in individuals with childhood adversity, is an important question that merits investigation.

2.4 Military training as a research context

Military training provides a unique natural laboratory for examining the influence of psychosocial and lifestyle factors on health. Several features of military training make it particularly well-suited for addressing the research questions posed in this thesis.

2.4.1 Standardised conditions and controlled exposure

Military recruits undergo training in highly standardised environments. Recruits live in shared accommodation, eat meals at centralised dining facilities with limited menu options, follow prescribed daily schedules, and participate in uniform physical training activities under close supervision. This standardisation minimises variability in environmental factors that typically confound observational health research, including diet quality, physical activity levels, pathogen exposure, and socioeconomic circumstances during the study period. By controlling these factors, military training allows for clearer examination of how individual differences in psychosocial factors (e.g., childhood adversity, optimism) and lifestyle behaviours (e.g., sleep) influence health outcomes.

2.4.2 High-stress context and life transition

Military training represents a major life transition involving relocation to a new environment, separation from family and friends, adoption of military culture and discipline, and exposure to novel physical and psychological demands. The training environment is intentionally challenging, designed to develop physical fitness, military skills, and mental resilience under pressure. This high-stress context is valuable for research because stress is known to reveal underlying vulnerabilities and resilience factors that may not be apparent under normal circumstances. For example, individuals with childhood adversity histories may exhibit heightened stress reactivity and poorer health outcomes specifically during stressful periods, consistent with the stress-sensitisation model (LoPilato et al., 2020). Similarly, the stress-

buffering effects of psychological resilience factors like optimism may be most evident during high-stress contexts such as military training (Cohen & Wills, 1985).

2.4.3 Sleep restriction

Sleep restriction is common practice during military training. The combination of early morning physical training, evening duties and tasks, and the need for time to complete personal administration (cleaning, ironing, paperwork) often results in recruits sleeping substantially less than they did in civilian life. Previous research has documented mean reductions of 1.5-2 hours per night from civilian life to military training (Wentz et al., 2018). This naturally occurring sleep restriction provides an opportunity to examine the health consequences of sleep loss in a real-world setting, complementing laboratory studies of experimental sleep deprivation. Moreover, because sleep opportunity is relatively uniform across recruits (all must rise early for morning training), differences in sleep duration and quality primarily reflect individual differences in bedtime choices, sleep efficiency, and subjective sleep experiences.

2.4.4 Relevance to military health and broader populations

Understanding the determinants of illness and injury during military training has direct practical relevance for military organisations. Respiratory infections and musculoskeletal injuries are leading causes of training days lost and medical attrition, imposing substantial costs and reducing operational readiness (Bullock et al., 2010; Wentz et al., 2018). Identifying modifiable risk factors could inform targeted interventions to reduce health burden and improve training effectiveness. Beyond military populations, the insights gained from studying recruits may generalise to other populations experiencing high stress, physical demands, sleep restriction, and life transitions, including university students, shift workers, healthcare workers, elite athletes, and new parents. Thus, research in military training contexts has the potential to inform health promotion strategies across diverse settings.

2.5 Summary and thesis objectives

This literature review has identified several important findings and knowledge gaps that provide the foundation for the present thesis.

Childhood adversity is highly prevalent (30-50% in general populations, potentially higher in military recruits) and is associated with increased risk of chronic diseases and premature mortality in adulthood. The mechanisms linking childhood adversity to poor health likely

involve biological embedding of stress reactivity and immune dysregulation, characterised by elevated inflammation, impaired cellular immunity and dysregulated HPA axis function. However, a critical gap exists in understanding whether childhood adversity increases susceptibility to acute infectious illnesses, particularly respiratory infections, remains largely unknown.

Psychological resilience factors, particularly optimism, are associated with better health outcomes and may buffer against the negative effects of stress according to Cohen's stress-buffering hypothesis (Cohen & Wills, 1985). However, few studies have tested whether optimism moderates the association between childhood adversity and health outcomes. Furthermore, most research has treated optimism as a stable trait rather than examining the health consequences of changes in optimism during stressful life transitions. Given evidence that optimism can be enhanced through psychological interventions, understanding whether improvements in optimism protect against illness in individuals with childhood adversity would have important practical implications.

Sleep is essential for immune function and physical health. Short sleep duration is associated with increased susceptibility to respiratory infections and impaired vaccine responses (Cohen, Doyle, Alper, Janicki-Deverts, & Turner, 2009; Prather et al., 2012; Prather et al., 2015). Sleep restriction, defined as reductions in sleep duration from habitual levels, has been studied primarily in laboratory settings using extreme restriction (e.g., 4 hours per night), with less attention to more modest, real-world sleep restriction. Importantly, emerging evidence suggests that sleep quality may be as important as sleep duration for health outcomes. Good PSQ has been associated with better cardiovascular health and, in one key study, perceived adequate sleep eliminated the association between short sleep duration and pneumonia risk (Patel et al., 2012). However, whether good sleep quality protects against respiratory infections during sleep restriction, and whether sleep quality predicts infection risk independently of sleep duration, remains unknown. Furthermore, the relationship between sleep and MSKI has been inadequately studied, with most research being retrospective and focused on adolescent athletes. Prospective studies examining whether sleep quality and duration predict injury risk in adults undergoing physical training are lacking.

Military training provides a unique opportunity to address these gaps. The standardised living conditions, high stress, naturally occurring sleep restriction, and access to objective health

outcomes make military training an ideal context for examining how childhood adversity, psychological resilience, and sleep influence illness and injury risk. Moreover, the life transition into military training allows for examination of whether changes in psychological and behavioural factors (e.g., improvements in optimism or sleep quality) predict health outcomes.

With this information in mind, this thesis aims to examine:

1. The influence of psychosocial factors (e.g., childhood adversity) and lifestyle factors (e.g., sleep) at the start of military training on illness and injury incidence in British Army infantry recruits.

2. Whether changes in psychosocial and lifestyle factors during training impact illness and injury incidence.

CHAPTER 3 – General Methods

3.1 Ethical approval

Approval was obtained for **Chapters 4, 5, 7 & 8** by the UK Ministry of Defence Research Ethics Committee (1070/MODREC/20) and was conducted in accordance with the Declaration of Helsinki (study registration at www.clinicaltrials.gov [NCT04780867]).

3.2 Study design and participants

A prospective, longitudinal cohort design was used for **Chapters 4, 5, 7 & 8** to examine the influence of psychosocial and lifestyle factors at the start and during basic military training on immune health and injury incidence. A total of N = 1,500 new recruits starting basic military training at the Infantry Training Centre, Catterick were verbally briefed, in the absence of training staff, and assessed for eligibility between October 2020 and April 2022 across 35 platoons. A total of N = 1,188 recruits provided written informed consent to participate in the study 24 h after a verbal briefing.

3.3 Procedures

A total of 1,188 healthy recruits (98% male) provided written informed consent to participate in the study for **Chapters 4, 5, 7 & 8**. Within 5 days of arrival at the Training Centre, recruits underwent a medical assessment with a physician to ensure they were in good health and fit to commence training. Medical reasons for exclusion from training included psychiatric disorders (e.g., clinical anxiety, depression, post-traumatic stress disorder, history of self-harm), chronic lung diseases and symptoms or treatment for asthma in the past year. To provide further confidence that recruits were healthy at the start of training those who had self-reported RTI symptoms (Jackson, Dowling, Spiesman, & Boand, 1958), serum C-reactive protein (CRP) values > 10 mg/L (Pearson et al., 2003) at the medical assessment or were diagnosed with a RTI **(Chapters 4, 5 & 7)** or MSKI **(Chapter 8)** by medical personnel within one week of arrival were excluded from analyses. At the medical assessment, demographic, lifestyle, and psychosocial data were collected, and height and body mass were measured.

3.4 Childhood adversity

Childhood adversity was assessed using the Childhood Trauma Questionnaire (CTQ) (Bernstein et al., 2003). Participants were afforded the opportunity to either opt-in or -out of

completing the CTQ due to the sensitivity of the questions presented. Respondents who opted-in to completing the CTQ were asked to report childhood adverse events that occurred before 11 years of age (Bernstein et al., 2003); this time-period provides separation between childhood and adulthood and is a notable time for children due to the transition from primary to secondary school in the UK. The CTQ consists of 25 scored items with each item ranging from 1 'never true' to 5 'very often true', yielding a total severity score from 25–125; higher scores indicate more severe and frequent adverse experiences. The CTQ assesses two types of adversity: abuse and neglect. Each type is also broken down into subtypes, with established cut-off scores for each subtype, as previously described (Walker et al., 1999): physical abuse (> 7), emotional abuse (> 9), sexual abuse (> 7), physical neglect (> 7) and emotional neglect (> 14). Childhood adversity was determined when the score for any subtype exceeded the threshold. We recorded the number of subtypes (one or multiple) and type of adversity (abuse or neglect). We also created quartiles (Q) of the CTQ to conduct analyses on CTQ severity (Q1 – Q4) with Q4 representing the most severe experience of childhood adversity.

3.5 Perceived psychological stress

Perceived psychological stress was assessed using the 10-item Perceived Stress Scale (PSS) (Cohen, Kamarck, & Mermelstein, 1983): the PSS is a widely used psychological instrument for measuring the perception of stress and measures the degree to which life situations are considered stressful by the individual during the previous month (Cohen et al., 1983). Responses were measured on a five-point Likert scale (from 0 'never' to 4 'very often') with a range of scores from 0—40, and greater scores indicating higher levels of psychological stress.

3.6 Optimism

Optimism is the expectancy for good rather than bad things to happen and is an important component of resilience (Dantzer et al., 2018); the process that allows individuals to adapt to adverse conditions and recover from them. Optimism was assessed using the 10-item Life Orientation Test–Revised (LOT-R). Each item was scored on a 5-point Likert scale (0 = strongly disagree to 4 = strongly agree), with 6 items scored; 3 negatively worded items that were reverse scored, 3 positively worded items and four filler items. Scores ranged from 0─24, with higher summed scores reflecting higher levels of optimism (Scheier et al., 1994). Psychological interventions are acknowledged to increase optimism (Malouff & Schutte, 2016; Meevissen et al., 2011), with one study showing increases in optimism following a one-month intervention in US Army soldiers (Rozek et al., 2019).

3.7 Subjective sleep

Sleep in the previous month was assessed using Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989). The PSQI is a commonly used measure of subjective sleep quality and quantity. It consists of 19-items assessing PSQ, sleep duration, latency, efficiency disturbances, daytime dysfunction and sleep medication use. Scores on the PSQI range from 0—21, with higher scores indicating poorer sleep quality. A sleep diary was used to assess PSQ and quantity the night before the first medical assessment.

3.8 Objective sleep (Actigraphy)

Objective sleep was assessed using Actigraphy, with recruits wearing a small triaxial accelerometer (Actigraph GT3X, Pensacola, USA) on the wrist of the non-dominant hand. Data were collected at a sampling rate of 30Hz using 1-min epochs and downloaded using Actilife software (V6). Sleep onset and wake times were set using a sleep diary, with sleep parameters calculated using the Cole-Kripke algorithm.

3.9 C-Reactive Protein

CRP levels in week 1 were assessed using from a fingerprick capillary blood sample using a point-of-care device (Quikread Go, Aidan, Finland) The Quikread Go has been shown to perform well compared with a standard immunoturbidimetric method performed on a routine analyser (mean difference 0.2; 95% CI -1.2─1.5 in samples < 20 mg/L, 93% of samples in the current study were < 20 mg/L) (Minnaard et al., 2013). In addition, internal pilot data in 93 samples ranging from 0-120 mg/L showed a strong correlation between CRP concentrations assessed using a particle-enhanced immunoturbidimetric assay (Roche Cobas, UK) and the point-of-care testing device ($r = 0.88$; $P < 0.01$).

3.10 Training outcome

Data on training outcome were extracted from the British Army's Training, Administration and Financial Management Information System (TAFMIS). These data outlined whether recruits completed the training course on time, had a delayed completion or failed to complete the training course.

3.11 Illness and injury

Recruits presenting with symptoms of illness or injury who reported to the medical facility at ITC(C) and underwent a consultation by medical personnel. As this study was conducted

during the COVID-19 pandemic, in line with MOD policy, recruits who presented to the medical facility with RTI symptoms indicative of COVID-19 (e.g., fever, continuous cough, anosmia) had a nasopharyngeal swab collected using standard procedures before being placed in viral transport medium. Samples were transported to James Cook University Hospital for qPCR analysis for determination of COVID-19 pathology (Dutta et al., 2022).

Injuries were identified as either traumatic or overuse injuries. Traumatic musculoskeletal injuries were defined as those caused by a single abrupt overload of the tissue or joint with sudden onset and a known cause (Ekstrand, Hagglund, & Walden, 2011; Robinson et al., 2016). Overuse injuries were defined as those that occurred in the absence of a single, identifiable traumatic cause and considered the result of a cumulative process of tissue damage rather than instantaneous energy transfer (Knapik et al., 2001; Robinson et al., 2016).

Diagnosed respiratory infections, injuries and lost training days were obtained via participant medical records on the Defence Medical Information Capability Programme (DMICP) three months after the scheduled completion of the course; this is because some recruits repeat elements of the course and thus take longer to finish than scheduled. For **Chapters 4 and 5**, RTI incidence was reported across the full 28-week training period. For **Chapters 6 – 8**, RTI and MSKI incidence was reported across the first 12-week Phase 1 training period.

CHAPTER 4 – Childhood adversity and respiratory infection risk in adulthood

4.1 Abstract

To assess potential effects of childhood adversity on adult host defence, we quantified the association between childhood adversity and the incidence of RTI in a cohort of young adults, considering the type (abuse and neglect) and number of adversities. In a prospective cohort study, we consented 1,188 healthy young adults commencing a 28-week military training course in the UK. Logistic regression quantified associations between childhood adversity (CTQ) and physician-diagnosed RTI, as well as distinct effects of adversity type. Regressions were adjusted for SES, lifestyle, current perceived stress and sleep. Almost half of participants reported childhood adversity (47%), with 54% of these experiencing multiple adversities. A total of 100 respiratory infections were recorded over the 28-week course. After adjustment for confounders, childhood adversity was associated with increased respiratory infection, particularly in participants reporting multiple adversities (OR = 2.26, 1.35–3.79, P = 0.002), abuse alone (OR = 2.56, 1.30–5.05, P = 0.007) or those in the highest CTQ severity quartile (OR = 2.65, 1.33–5.27, P = 0.005; vs Q1). Neglect alone was not associated with increased respiratory infection. Associations were independent of demographic confounders but attenuated after adjusting for potential mediators during training (e.g., perceived stress and sleep quality). In conclusion, childhood adversity is associated with respiratory infection risk in adulthood, particularly in individuals who experienced abuse.

4.2 Introduction

Children exposed to adversity are more vulnerable to mental and physical illness across the lifespan (Felitti et al., 1998; Nelson et al., 2020). National surveys show that more than 30% of all mental disorders in the global population are preceded by childhood adversity, with adversity incidence rates of 30–50% reported in developed countries and an estimated economic burden equivalent to 3–4% of gross domestic product in Europe and North America (Hughes, Ford, Kadel, Sharp, & Bellis, 2020; Kessler et al., 2010; McLaughlin et al., 2012; Rod et al., 2020).

Childhood is a developmentally sensitive period when exposure to adverse environments can disrupt cognitive and social development and the functioning of stress-sensitive biological pathways (McLaughlin & Sheridan, 2016). Childhood adversity encompasses a broad range of experiences including physical, sexual and emotional abuse and physical and emotional neglect (McLaughlin et al., 2014). Research examining the number and type of adverse childhood experiences highlights that adults exposed to multiple childhood adversities (Wang et al., 2024) or the more severe abuse-related adversities experience more pronounced negative health outcomes (Aas et al., 2017; Kiecolt-Glaser et al., 2011; Sumner et al., 2019). Although the different dimensions of childhood experience relating to abuse and neglect often co-occur, they are considered to have unique effects on neural development, behaviour and health. For example, exposure to neglect-related adversities in childhood leads to developmental delays, in turn affecting cognitive, social and emotional abilities, whereas exposure to abuse-related adversities activates the hypothalamic-pituitary-adrenal (HPA) axis, with chronic activation leading to neuroendocrine and immune dysregulation (Evans, Li, & Whipple, 2013; McLaughlin et al., 2014). Adults who experienced abuse-related adversities in childhood are likely to perceive challenging situations as more threatening and be particularly vulnerable to psychological stress (LoPilato et al., 2020) and sleep pathology (Greenfield et al., 2011), both independent risk factors for RTI (Cohen et al., 1991; Vedhara et al., 2022; Walsh et al., 2023). Impaired innate antiviral response, cellular immunity, and poorer regulation of inflammation in adults who experienced childhood abuse may increase susceptibility to respiratory tract infection (RTI), particularly as innate antiviral response plays a key role in preventing symptomatic RTIs (McNab, Mayer-Barber, Sher, Wack, & O'Garra, 2015) and subsequent inflammation drives illness symptoms during an RTI episode (Cohen et al., 1999; Fagundes, Glaser, Malarkey, et al., 2013; Pinto Pereira, Stein Merkin, Seeman, & Power, 2019). However, it remains unknown whether abuse-related adversities in childhood might distinctively increase

adult RTI risk relative to neglect-related adversities. The small number of studies investigating associations between childhood adversity and adulthood RTI risk have not taken account of the different dimensions of childhood adversity.

Using parental relationship quality as a measure of childhood adversity, one study showed that adults whose parents separated and did not speak to each other during childhood were more than three times as likely to develop an RTI following viral exposure than adults from intact childhood families (Murphy et al., 2017). However, interrogating the UK Biobank database revealed only a weak association between childhood maltreatment and the incidence of COVID-19 that was lost after adjustment for potential mediators including socio-economic status and lifestyle-related factors (Wang et al., 2024). It is important to acknowledge the difficulty separating differential pathogen exposure from differential host resistance in most health survey or national cohort research. One strategy to distinguish those effects is to assess the relationship between childhood adversity and adulthood RTI within a relatively homogenous cohort occupying the same general infection exposure environment, so that differential exposure is a less likely explanation for RTI episodes than persisting immune sequelae. Basic military training offers a unique opportunity to investigate the relationship between childhood adversity and RTI, under standardised living and working conditions, in adults facing the challenges associated with relocating to a new living and working environment alongside the physical and psychological demands of the course. Respiratory infections are important not only as a direct morbidity burden, but also as a sentinel read-out of broader host resistance to disease. Moreover, RTI incidence is a substantial risk factor for other types of morbidity and the third most commonly reported cause of death in developed and developing countries (Akinyemi & Morakinyo, 2018; Hak, Rovers, Kuyvenhoven, Schellevis, & Verheij, 2006; Petrie et al., 2016; Smeeth et al., 2004).

With this information in mind, in a cohort of 1,188 initially healthy adults embarking on basic military training, we examined the association between childhood adversity and RTI risk. We also tested the extent to which childhood adversity might be associated with RTI risk during training via potential mediators e.g., psychological stress and poor sleep. We hypothesised that childhood adversity would be associated with increased RTI susceptibility, particularly in those reporting abuse-related adversities.

4.3 Methods

4.3.1 Study design and participants

Study design and participants are outlined in **Chapter 3, section 3.1 – 3.3.**

4.3.2 Procedures

A total of 1,188 healthy recruits (98% male) provided written informed consent to participate in the study (**Figure 3**). Procedures are detailed in **Chapter 3, section 3.3**.

Psychosocial and lifestyle measures were assessed in private using online surveys (LimeSurvey, Hamburg, Germany) via an electronic tablet. Childhood adversity was assessed using the CTQ-SF and perceived psychological stress (PSS) and sleep quality (PSQI) were assessed at week 1 and one-month later to examine changes during training (see **Chapter 3, sections 3.4 – 3.7**).

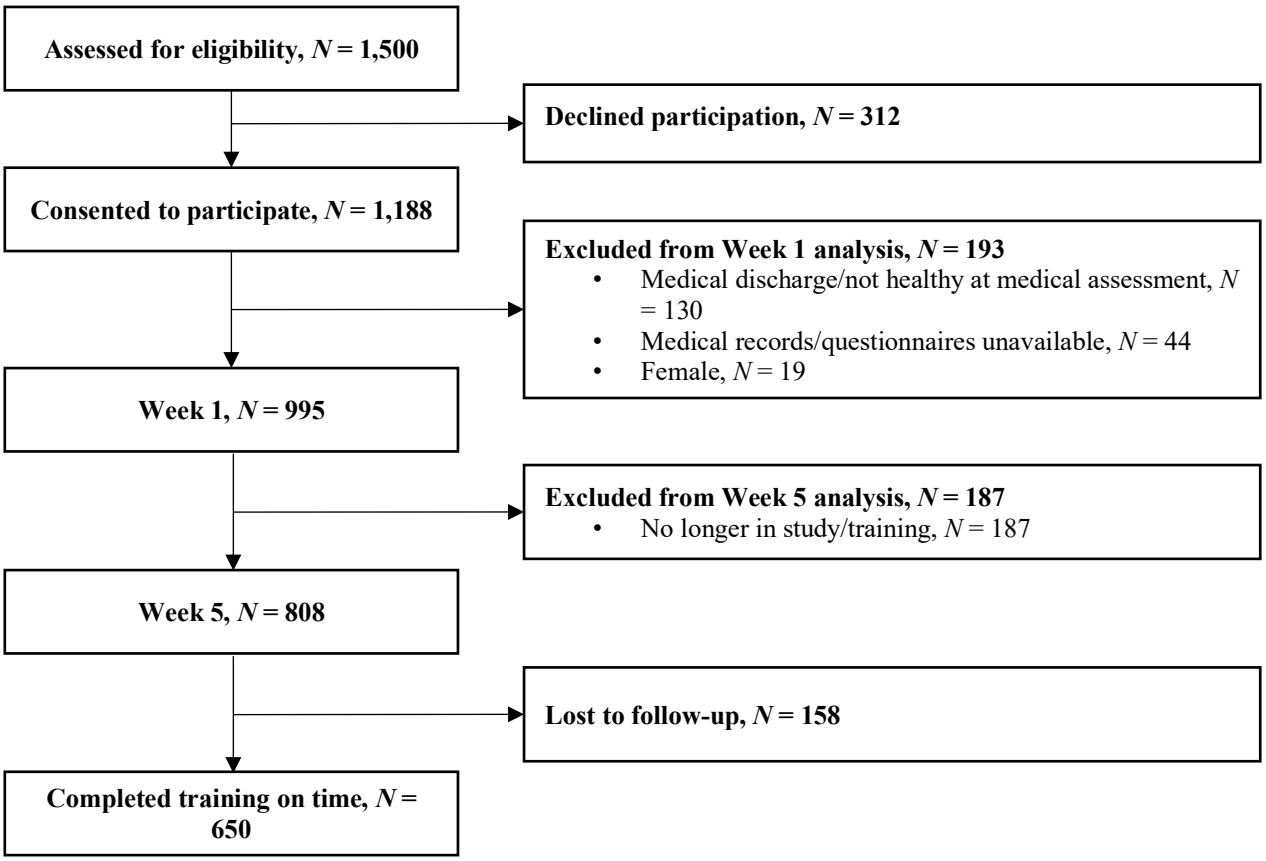

Figure 3. *Flowchart outlining recruits assessed for eligibility, consented and available at the start and end of the training course. Recruits presenting with serum CRP levels ≥ 10mg/L or common cold symptoms at the medical assessment were classed as 'not healthy' and therefore excluded from analyses. Incomplete data refers*

to missing demographic, anthropometric or psychological questionnaire data. The Ministry of Defence Research Ethics Committee required female inclusion in study recruitment; however, due to the limited number of females entering training they were excluded from data analysis due to the lack of statistical power.

4.3.3 Outcomes

The primary outcome of this study was RTI during military training. RTI diagnosis is described in **Chapter 3, section 3.11**.

4.3.4 Covariates

Season of enrolment (autumn/winter/spring) and region of recruitment (Indices of Multiple Deprivation; 1–5 (Abel, Barclay, & Payne, 2016)) were recorded at study entry. Demographic, lifestyle, psychosocial and serum CRP data were collected at the medical assessment at week 1, including age (years), ethnicity (white/non-white), smoking status (smoker/non-smoker), alcohol use (yes/no), socioeconomic status (SES; low/high, determined as presence or absence of formal qualifications (Case, Fertig, & Paxson, 2005)), body mass index (BMI; kg/m^2), perceived stress (PSS; moderate-to-high >13 and low ≤13 (Cohen et al., 1983)), sleep quality (PSQI; poor ≥5 and good <5 (Buysse et al., 1989)) and serum CRP (≥1/<1 mg/L). BMI, perceived stress, sleep quality, and serum CRP were re-assessed at week 5. To control for differential infection exposure, the number of days each participant spent in training was obtained from military records following completion or withdrawal from the training course.

4.3.5 Statistical analysis

Participant demographic, lifestyle, psychosocial and childhood adversity data are presented as mean ± SD for continuous variables or absolute numbers and percentages for categorical variables. Comparisons were made using independent t-tests, paired samples t-tests, ANOVA, one-way repeated measures ANOVA and Chi-square, where appropriate (**Table 1 – 6**).

A small proportion of PSQI data at week 5 were missing, assumed missing at random, with 17% (N = 166) of recruits missing PSQI data e.g. due to administrative issues with questionnaires. To avoid a potential bias, multiple imputation was carried out using predictive mean matching (50 iterations and 40 imputed datasets), in line with recommendations (Graham, 2009). After accounting for participants who withdrew from Army training and the

study by week 5, PSQI collected at week 1 and all variables collected at week 5 were included in multiple imputation, including the outcome variable RTI incidence.

Hierarchical logistic regression was used to examine the association between childhood adversity and adulthood RTI risk. After conducting diagnostic checks, we first examined the influence of childhood adversity on RTI risk (**Figure 4**A), followed by the effects of the number of adversity subtypes and the type of adversity on RTI risk (**Figure 4**B-C); recruits with no childhood adversity were considered the reference group. We also investigated the effects of childhood adversity severity on RTI risk, with severity assessed using both the continuous CTQ score, and quartiles of the CTQ, with Q1 considered as the reference (**Figure 4**D). Regression analysis was performed using a 4-step model approach; whereby, Model 1 investigated the unadjusted association between childhood adversity and RTI risk; Model 2 adjusted for age, ethnicity, season of recruitment, region of recruitment and days spent in training to control for RTI exposure; Model 3 adjusted for all covariates included in Model 2 plus a set of mediator variables that could potentially serve as a mechanistic pathway between childhood adversity and RTI, including smoking status, alcohol status, socioeconomic status, BMI, perceived stress, and sleep quality; Model 4 included Model 3 plus adjustment for serum CRP (fully adjusted model). This multiple regression modelling approach allowed us to determine the total effect of childhood adversity on adulthood RTI risk and compare to direct and indirect effects from models accounting for any potential mediators.

As a supplementary analysis, the same 4-model approach was repeated in the subset of participants present at week 5 (**Table 7**, N = 741), to examine whether changes in potential mediator variables during training accounted for the association between childhood adversity and RTI risk. In this supplementary analysis, BMI, perceived stress and sleep quality in Model 3 were reassessed at week 5, and serum CRP in Model 4 was additionally assessed at week 5. Age, ethnicity, smoking, alcohol, SES, season and days spent in training were retained from Week 1 assessments. Participants diagnosed with an RTI in close proximity to the week 5 assessment (weeks 4–6) were excluded from this supplementary analysis (N = 16) to reduce the risk of reverse causation.

Further analyses were conducted to evaluate the independent effects of these potential mediator variables on RTI risk. These analyses were performed in the full sample at week 1 and in the subsample available at week 5, using a 2-step model approach; whereby, Model 1 examined

the unadjusted association between each mediator variable and RTI risk and Model 2 was adjusted for age, ethnicity, season, region of recruitment and days spent in training. Analyses were conducted using SPSS 28.0 (IBM, Armonk, NY, USA) with statistical significance set at $P < 0.05$.

4.4 Results

4.4.1 Descriptives, lifestyle information and childhood adversity

Demographic, lifestyle and psychosocial descriptive data are presented in **Table 1**. Participants were young adult males, with a mean age of 21 ± 4 years (range 17–35 years). The majority of participants were white (84%), with 16% identifying as non-white. Most participants were enrolled during autumn (36%) or winter (44%), with the remainder enrolled during spring (20%); no participants commenced training during summer. Mean BMI was 24.2 ± 3.2 kg/m², 27% were smokers and 70% reported alcohol use at the start of training.

The proportion of recruits reporting childhood adversity was 47%, with 54% of those individuals reporting multiple adversity subtypes (**Table 2**). There was a tendency for recruits who experienced childhood adversity to report both abuse and neglect (45%) compared with abuse alone (20%) or neglect alone (35%; **Table 2**). In recruits reporting multiple adversity subtypes, 84% experienced both abuse and neglect and only 4% abuse alone and 12% neglect alone.

Table 1. *Descriptive information in the population that commenced training.*

	Total sample
Demographic and lifestyle	***N* = 995**
Age (y) [mean ± SD]	21 ± 4
Ethnicity, white [*N* (%)]	837 (84)
Ethnicity, non-white [*N* (%)]	158 (16)
BMI (kg/m^2) [mean ± SD]	24.2 ± 3.2
Smoker [*N* (%)]	266 (27)
Alcohol use [*N* (%)]	695 (70)
Region of recruitment, 1 – least deprived [*N* (%)]	150 (15)
Region of recruitment, 2 – 4 [*N* (%)]	535 (54)
Region of recruitment, 5 – most deprived [*N* (%)]	310 (31)
Low SES [*N* (%)]	168 (17)
Season of enrolment	
Autumn [*N* (%)]	355 (36)
Winter [*N* (%)]	441 (44)
Spring [*N* (%)]	199 (20)
Summer [*N* (%)]	0 (0)

BMI = body mass index. Region of recruitment categories determined using Indices of Multiple Deprivation (Abel, Barclay, & Payne, 2016). SES = socioeconomic status; low SES determined as percentage of participants with no formal qualifications (Case, Fertig, & Paxson, 2005). Non-white ethnicity is made up of; 'Mixed / multiple ethnic groups', 'Asian / Asian British', 'Black / African / Caribbean / Black British' and 'Other'.

Table 2. *The composition of childhood adversity (number and type) assessed using the Childhood Trauma Questionnaire (CTQ).*

	Adversity Type			
	Abuse	**Neglect**	**Abuse & Neglect**	**Total**
Adversity Number				
One	75 (39) [88]	119 (61) [81]	–	**194 [46]**
Multiple	10 (4) [12]	28 (12) [19]	191 (84) [100]	**229 [54]**
Total	**85 (20)**	**147 (35)**	**191 (45)**	**423**

Data are shown as *N* (% of row) and *N* [% of column]. Adversity number refers to the incidence of one or more of the five subtypes: physical, sexual and emotional abuse and physical and emotional neglect. *N* = 908 of 995 opted-in to completing the CTQ and *N* = 423 (47%) experienced childhood adversity and *N* = 485 (53%) experienced no childhood adversity.

Compared to no childhood adversity, participants reporting childhood adversity were less likely to be White, more likely to be a smoker, have no formal qualifications and report higher perceived stress and poorer sleep quality at the start of training ($P < 0.05$; **Table 3**). Participants with the most severe childhood adversity (CTQ severity quartiles 3 and 4) reported higher perceived stress and poorer sleep quality at both week 1 and week 5 (CTQ severity quartiles 3 and 4 > quartile 1, $P < 0.05$; **Table 4**). Notwithstanding, participants with the most severe childhood adversity were no less likely to complete training than those without childhood adversity (completion: CTQ severity quartile 1 = 66%, quartile 4 = 61%, $P = 0.209$). Participants who opted out of completing the CTQ (9%) were similar to those with childhood adversity for demographic information, perceived stress and sleep quality at week 1 (**Table 3** and **Table 5**). For example, compared to those who reported no childhood adversity, participants who opted out of completing the CTQ were less likely to be white, more likely to be a smoker, have no formal qualifications and report higher perceived stress and poorer sleep quality at week 1 ($P < 0.05$; **Table 3**).

Table 3. *Descriptive information in the populations that reported no childhood adversity, childhood adversity, and those who opted-out of answering the Childhood Trauma Questionnaire.*

	No childhood adversity	**Childhood adversity**	**Opted out of answering CTQ**
	***N* = 485**	***N* = 423**	***N* = 87**
Demographic and lifestyle			
Age (y) [mean ± SD]	21 ± 3	21 ± 4	20 ± 3
Ethnicity, white [*N* (%)]	426 (88)	344 (81)[aa]	68 (79)[a]
Ethnicity, non-white [*N* (%)]	59 (12)	79 (19)[aa]	18 (21)[a]
BMI (kg/m^2) [mean ± SD]	24.3 ± 3.2	24.3 ± 3.1	23.6 ± 3.2
Smoker [*N* (%)]	108 (22)	126 (30)[a]	32 (37)[aa]
Alcohol use [*N* (%)]	365 (75)	277 (66)[aa]	55 (64)[aa]
Region of recruitment, 1 – least deprived [*N* (%)]	73 (15)	62 (15)	15 (17)
Region of recruitment, 2 – 4 [*N* (%)]	264 (54)	229 (54)	42 (49)
Region of recruitment, 5 – most deprived [*N* (%)]	148 (31)	132 (31)	30 (34)
Low SES [*N* (%)]	54 (11)	91 (22)[aa]	24 (28)[aa]
Season of enrolment			
Autumn [*N* (%)]	184 (38)	143 (34)	28 (32)
Winter [*N* (%)]	220 (45)	182 (43)	39 (45)
Spring [*N* (%)]	81 (17)	98 (23)	20 (23)
Summer [*N* (%)]	0 (0)	0 (0)	0 (0)
Perceived stress and sleep quality at week 1			
Perceived stress (PSS) [mean ± SD]	10.6 ± 5.4	12.9 ± 5.9[aa]	13.0 ± 6.5[aa]
Cut-off score >13 [N (%)]	138 (29)	178 (42)[aa]	41 (47)[aa]
Sleep quality (PSQI) [mean ± SD]	4.8 ± 2.5	5.6 ± 2.9[aa]	5.5 ± 3.0[aa]
Cut-off score ≥5 [N (%)]	243 (50)	266 (63)[aa]	51 (59)[aa]

BMI = body mass index. Region of recruitment categories determined using Indices of Multiple Deprivation (Abel, Barclay, & Payne, 2016). SES = socioeconomic status; low SES determined as percentage of participants with no formal qualifications (Case, Fertig, & Paxson, 2005). PSS = Perceived Stress Scale (10-item). PSQI = Pittsburgh Sleep Quality Index. [a] = significantly different to no childhood adversity. Single letter denotes $P < 0.05$ (e.g., [a]); double letter denotes $P < 0.01$ (e.g., [aa]).

Table 4. *Health variables by Childhood Trauma Questionnaire severity score quartiles.*

	All	CTQ Severity Score Quartile			
		Q1	**Q2**	**Q3**	**Q4**
	N = 724	*N* = 194	*N* = 188	*N* = 166	*N* = 176
Variables at week 1					
BMI (kg/m^2) [mean ± SD]	24.4 ± 3.1	24.4 ± 3.1	24.5 ± 3.2	24.5 ± 3.3	24.3 ± 2.9
Perceived stress (PSS) [mean ± SD]	11.1 ± 5.5	9.6 ± 5.5	10.3 ± 5.3	11.9 ± 4.8^{aa}	12.9 ± 5.6^{aa}
Cut-off score >13 [N (%)]	230 (31)	45 (23)	51 (27)	62 (37)^{aa}	72 (39)^{aa}
Sleep quality (PSQI) [mean ± SD]	5.0 ± 2.7	4.4 ± 2.5	4.7 ± 2.7	4.8 ± 2.2	6.2 ± 3.0^{aa}
Cut-off score ≥5 [N (%)]	400 (54)	89 (45)	89 (47)	97 (58)^{aa}	125 (68)^{aa}
CRP (mg/L) <1	461 (64)	118 (61)	122 (65)	110 (66)	111 (63)
CRP (mg/L) 1–3	202 (28)	57 (29)	51 (27)	38 (23)	56 (32)
CRP (mg/L) >3	61 (8)	19 (10)	15 (8)	18 (11)	9 (5)
Variables at week 5					
BMI (kg/m^2) [mean ± SD]	24.3 ± 2.7	24.3 ± 2.7	24.5 ± 2.9	24.3 ± 2.8	24.1 ± 2.5
Perceived stress (PSS) [mean ± SD]	13.4 ± 6.5**	12.0 ± 6.6**	12.2 ± 6.0**	14.2 ± 6.4^{**aa}	15.8 ± 6.0^{**aa}
Cut-off score >13 [N (%)]	373 (50)**	76 (38)**	81 (42)**	90 (54)^{**aa}	126 (67)^{**aa}
Sleep quality (PSQI) [mean ± SD]	5.3 ± 2.4**	4.8 ± 2.5*	5.0 ± 2.3	5.3 ± 2.2**	6.0 ± 2.5^{aa}
Cut-off score ≥5 [N (%)]	439 (60)*	97 (49)	108 (57)*	104 (62)^{a}	130 (71)^{aa}
CRP (mg/L) <1	353 (49)**	96 (50)	92 (49)*	77 (46)*	88 (50)
CRP (mg/L) 1–3	262 (36)**	72 (37)	70 (37)*	62 (37)**	58 (33)
CRP (mg/L) >3	109 (15)**	26 (13)	26 (14)**	27 (16)**	30 (17)**

CTQ = childhood trauma questionnaire. Q = quartile. PSS = Perceived Stress Scale (10-item). PSQI = Pittsburgh Sleep Quality Index. CRP = c-reactive protein where clinically recognised thresholds were adopted (Pearson et al., 2003). *N* = 741 of 808 opted-in to completing the CTQ and were present at week 5. To reduce the risk of reverse causation, participants diagnosed with an RTI in close proximity to the week 5 assessment (weeks 4–6) were excluded (*N* = 16), leaving *N* = 724 available. * = significantly different to week 1 of corresponding group. ^{a} = significantly different to Q1. Single letter denotes $P < 0.05$ (e.g., ^{a}); double letter denotes $P < 0.01$ (e.g., ^{aa}).

4.4.2 Childhood adversity is associated with increased risk of adulthood RTI

A total of 100 physician diagnosed RTI incidences were recorded during the 28-week training course (Phase 1 $N = 61$, Phase 2 $N = 39$). Childhood adversity was associated with increased RTI risk (**Figure 4**; **Table 6**). Participants reporting childhood adversity were nearly twice as likely as those without childhood adversity to develop an RTI (Model 1: OR = 1.86, 1.18–2.93, $P = 0.007$). This association persisted after adjustment for age, ethnicity, season of enrolment, region of recruitment and days spent in training (Model 2: OR = 1.78, 1.12–2.83, $P = 0.016$, **Figure 4**A). This association also remained after further adjustment for potential mediator variables assessed at week 1, including smoking, alcohol use, SES, BMI, perceived stress and sleep quality (Model 3: OR = 1.71, 1.06–2.76, $P = 0.029$) and serum CRP (Model 4: OR = 1.74, 1.08–2.81, $P = 0.024$).

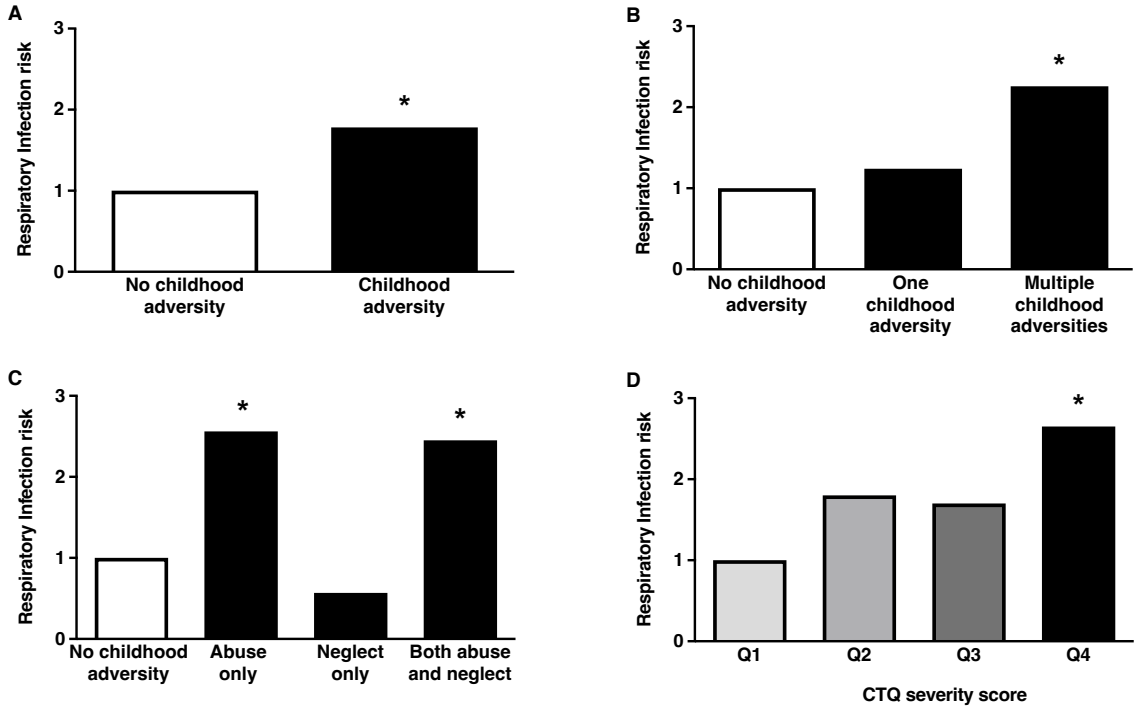

***Figure 4**. The influence of childhood adversity on respiratory tract infection (RTI) risk during a 28-week military training course. Panels show any adversity (A), the number of adversities (B), the type of adversity (C), and the severity of adversity by quartile (D). Odds ratios presented are from Model 2 accounting for; age, ethnicity, season, region of recruitment and days in training. CTQ = Childhood Trauma Questionnaire. N = 908 of 995 opted-in to completing the CTQ. For panels A, B and C no childhood adversity is considered as reference; for panel D, Q1 is considered as reference. * P < 0.05 vs no childhood adversity/Q.*

Further analysis examined the influence of the number and type of childhood adversities on adulthood RTI risk during military training (**Figure 4**B–C; **Table 6**). Fully adjusted regression showed that participants reporting multiple childhood adversity subtypes were at a significantly greater risk of RTI during training (*vs* no childhood adversity; Model 4: OR = 2.19, 1.28–3.76, $P = 0.005$; **Figure 4**B). However, experiencing one childhood adversity subtype did not significantly raise the risk of RTI (*vs* no childhood adversity; Model 4: OR = 1.26, 0.67–2.37, $P = 0.472$; **Figure 4**B). When examining the association between childhood adversity type (i.e., only abuse or only neglect) and RTI, individuals reporting only abuse were at increased risk of RTI (*vs* no childhood adversity; Model 4: OR = 2.67, 1.35–5.29, $P = 0.005$, **Figure 4**C) whereas those reporting only neglect showed no differential RTI risk, despite both groups showing a similar overall CTQ severity score (only neglect 38 ± 6 *vs* only abuse 38 ± 5*,* $P = 0.206$). Participants experiencing the most severe childhood adversity (CTQ severity score quartile 4) were nearly two and a half times more likely to suffer RTI than those reporting the lowest level of adversity (*vs* quartile 1, Model 4: OR = 2.55, 1.26–5.19, $P = 0.010$; **Figure 4**D; **Table 6**). This association between CTQ severity score and adulthood RTI risk was also observed in regressions using the continuous CTQ score (Model 4: OR = 1.02 per CTQ score unit, 1.01–1.04, $P = 0.010$).

To further examine whether potential mediator variables (e.g., perceived stress and sleep quality) during training might contribute to the association between childhood adversity and RTI risk, analyses were repeated in the subset of participants present at week 5, with BMI, perceived stress, sleep quality and serum CRP reassessed at week 5 (**Table 7**). To reduce the risk of reverse causation, participants diagnosed with an RTI in close proximity to the week 5 assessment (weeks 4–6) were excluded from this analysis ($N = 16$), leaving $N = 724$ available. In this analysis, childhood adversity remained associated with increased RTI risk after adjustment for potential confounders (*vs* no childhood adversity; Model 2: OR = 2.08, 1.16–3.73, $P = 0.014$). However, after further adjustment for potential mediator variables assessed during training, the level of overall association between childhood adversity and RTI risk (*vs* no childhood adversity; Model 3: OR = 1.81, 0.98–3.33, $P = 0.058$) was attenuated, suggesting partial mediation. Similar attenuation was observed in analyses relating RTI risk to the highest CTQ severity quartile (Q4) (*vs* quartile 1, Model 3: OR = 2.41, 0.99–5.86, $P = 0.052$). Participants reporting multiple childhood adversities remained at greater RTI risk across all models (*vs* no childhood adversity; Model 4: OR = 2.27, 1.16–4.48, $P = 0.018$), as did those

reporting abuse only (*vs* no childhood adversity; Model 4: OR = 2.75, 1.10–6.89, $P = 0.030$). The continuous CTQ severity score also remained associated with RTI risk across all models (Model 4: OR = 1.03 per CTQ score unit, 1.01–1.05, $P = 0.015$).

4.4.3 Potential mediator variables assessed during training independently predict RTI risk

To examine whether the potential mediator variables included in the regression models were themselves independently associated with RTI risk, further analyses were conducted separately from models examining childhood adversity (**Table 8**). None of the variables assessed at week 1, including BMI, smoking status, alcohol use, low SES, perceived stress or sleep quality were independently associated with RTI risk. However, when assessed during training at week 5, moderate-to-high perceived stress (*vs* low perceived stress; OR = 2.49, 1.46–4.24, $P < 0.001$) and poor sleep quality (*vs* good sleep quality; OR = 1.76, 1.02–3.04, $P = 0.041$) were both independently associated with increased RTI risk after adjustment for age, ethnicity, season, region of recruitment and days spent in training.

***Table 5**. Descriptive information in the population who opted-out of answering the Childhood Trauma Questionnaire compared to those who opted-in.*

	Opted in to	**Opted out of**
	***N* = 908**	***N* = 87**
Demographic and lifestyle		
Age (y) [mean ± SD]	21 ± 4	20 ± 3
Ethnicity, white [*N* (%)]	770 (85)	68 (79)
Ethnicity, non-white [*N* (%)]	138 (15)	18 (21)
BMI (kg/m^2) [mean ± SD]	24.3 ± 3.2	23.6 ± 3.2
Smoker [*N* (%)]	234 (26)	32 (37)[a]
Alcohol use[*N* (%)]	640 (71)	55 (64)
Region of recruitment, 1 – least deprived [*N*	135 (15)	15 (17)
Region of recruitment, 2 – 4 [*N* (%)]	493 (54)	42 (49)
Region of recruitment, 5 – most deprived [*N*	280 (31)	30 (34)
Low SES [*N* (%)]	145 (16)	24 (28)[aa]
Season of enrolment		
Autumn [*N* (%)]	327 (36)	28 (32)
Winter [*N* (%)]	402 (44)	39 (45)
Spring [*N* (%)]	179 (20)	20 (23)
Summer [*N* (%)]	0 (0)	0 (0)

BMI = body mass index. Region of recruitment categories determined using Indices of Multiple Deprivation (Abel, Barclay, & Payne, 2016). SES = socioeconomic status; low SES determined as percentage of participants with no formal qualifications (Case, Fertig, & Paxson, 2005). CTQ = childhood trauma questionnaire. [a] = significantly different to opted-in to answering CTQ. Single letter denotes $P < 0.05$ (e.g., [a]); double letter denotes $P < 0.01$ (e.g., [aa]).

Table 6. *Association between childhood adversity and RTI during military training, accounting for potential mediator variables assessed at week 1.*

	Model 1: Unadjusted	**Model 2:** Adjusted for age, ethnicity, season, region of recruitment & days in training	**Model 3:** Model 2 + smoking, alcohol, SES, BMI, perceived stress & sleep quality	**Model 4:** Model 3 + CRP
Childhood adversity (reference: no childhood adversity)	**1.86 (1.18–2.93) *P* = 0.007**	**1.78 (1.12–2.83) *P* = 0.016**	**1.71 (1.06–2.76) *P* = 0.029**	**1.74 (1.08–2.81) *P* = 0.024**
Childhood adversity severity score (continuous)	**1.03 (1.01–1.04) *P* < 0.001**	**1.02 (1.01–1.04) *P* = 0.003**	**1.02 (1.01–1.04) *P* = 0.011**	**1.02 (1.01–1.04) *P* = 0.010**
One childhood adversity (reference: no childhood adversity)	1.27 (0.69–2.34) *P* = 0.435	1.24 (0.66–2.30) *P* = 0.507	1.27 (0.68–2.38) *P* = 0.458	1.26 (0.67–2.37) *P* = 0.472
Multiple childhood adversities (reference: no childhood adversity)	**2.39 (1.45–3.95) *P* < 0.001**	**2.26 (1.35–3.79) *P* = 0.002**	**2.12 (1.23–3.65) *P* = 0.007**	**2.19 (1.28–3.76) *P* = 0.005**
Neglect only adversities (reference: no childhood adversity)	0.57 (0.23–1.38) *P* = 0.435	0.57 (0.23–1.40) *P* = 0.217	0.57 (0.23–1.41) *P* = 0.226	0.57 (0.23–1.40) *P* = 0.225
Abuse only adversities (reference: no childhood adversity)	**2.80 (1.45–5.41) *P* = 0.002**	**2.56 (1.30–5.05) *P* = 0.007**	**2.68 (1.35–5.30) *P* = 0.005**	**2.67 (1.35–5.29) *P* = 0.005**
Both abuse and neglect adversities (reference: no childhood adversity)	**2.57 (1.53–4.32) *P* < 0.001**	**2.45 (1.43–4.18) *P* = 0.001**	**2.38 (1.35–4.18) *P* = 0.003**	**2.35 (1.33–4.15) *P* = 0.003**
Q2 CTQ severity score (reference: Q1 CTQ severity score)	1.88 (0.92–3.83) *P* = 0.082	1.80 (0.87–3.71) *P* = 0.111	1.83 (0.89–3.78) *P* = 0.102	1.84 (0.89–3.81) *P* = 0.099
Q3 CTQ severity score (reference: Q1 CTQ severity score)	1.75 (0.84–3.64) *P* = 0.133	1.70 (0.81–3.59) *P* = 0.164	1.67 (0.78–3.55) *P* = 0.185	1.66 (0.78–3.53) *P* = 0.191
Q4 CTQ severity score (reference: Q1 CTQ severity score)	**2.88 (1.47–5.63) *P* = 0.002**	**2.65 (1.33–5.27) *P* = 0.005**	**2.49 (1.22–5.09) *P* = 0.012**	**2.55 (1.26–5.19) *P* = 0.010**

RTI = respiratory tract infection. Region of recruitment categories determined using Indices of Multiple Deprivation (Abel, Barclay, & Payne, 2016). SES = socioeconomic status; low SES determined as percentage of participants with no formal qualifications (Case, Fertig, & Paxson, 2005). BMI = body mass index. CRP = C-reactive protein. CTQ = childhood trauma questionnaire. All variables included in the models were assessed at week 1.

Table 7. *Association between childhood adversity and RTI during military training, accounting for potential mediator variables assessed at Week 5.*

	Model 1: Unadjusted	**Model 2:** Adjusted for age, ethnicity, season, region of recruitment & days in training	**Model 3:** Model 2 + smoking, alcohol, SES, BMI, perceived stress & sleep quality	**Model 4:** Model 3 + CRP
Childhood adversity (reference: no childhood adversity)	**2.22 (1.26–3.91) *P* = 0.006**	**2.08 (1.16–3.73) *P* = 0.014**	1.81 (0.98–3.33) *P* = 0.058	1.81 (0.98–3.33) *P* = 0.058
Childhood adversity severity score (continuous)	**1.03 (1.02–1.05) *P* < 0.001**	**1.03 (1.01–1.05) *P* < 0.001**	**1.03 (1.01–1.05) *P* = 0.013**	**1.03 (1.01–1.05) *P* = 0.015**
One childhood adversity (reference: no childhood adversity)	1.41 (0.66–3.01) *P* = 0.380	1.32 (0.61–2.89) *P* = 0.482	1.28 (0.58–2.85) *P* = 0.540	1.28 (0.58–2.86) *P* = 0.540
Multiple childhood adversities (reference: no childhood adversity)	**2.97 (1.60–5.50) *P* < 0.001**	**2.75 (1.46–5.19) *P* = 0.002**	**2.28 (1.16–4.48) *P* = 0.017**	**2.27 (1.16–4.48) *P* = 0.018**
Neglect only adversities (reference: no childhood adversity)	1.15 (0.44–3.00) *P* = 0.781	1.20 (0.45–3.20) *P* = 0.723	1.13 (0.41–3.07) *P* = 0.818	1.13 (0.41–3.09) *P* = 0.814
Abuse only adversities (reference: no childhood adversity)	**3.57 (1.50–8.52) *P* = 0.004**	**2.86 (1.16–7.07) *P* = 0.023**	**2.71 (1.09–6.76) *P* = 0.032**	**2.75 (1.10–6.89) *P* = 0.030**
Both abuse and neglect adversities (reference: no childhood adversity)	**3.20 (1.58–6.48) *P* = 0.001**	**2.87 (1.38–5.96) *P* = 0.005**	2.17 (0.99–4.78) *P* = 0.054	2.17 (0.98–4.77) *P* = 0.055
Q2 CTQ severity score (reference: Q1 CTQ severity score)	1.87 (0.77–4.57) *P* = 0.169	1.88 (0.76–4.66) *P* = 0.173	1.87 (0.75–4.69) *P* = 0.179	1.81 (0.72–4.53) *P* = 0.209
Q3 CTQ severity score (reference: Q1 CTQ severity score)	1.81 (0.72–4.55) *P* = 0.205	1.67 (0.66–4.27) *P* = 0.282	1.47 (0.56–3.84) *P* = 0.429	1.35 (0.51–3.55) *P* = 0.544
Q4 CTQ severity score (reference: Q1 CTQ severity score)	**3.34 (1.45–7.72) *P* = 0.005**	**3.06 (1.30–7.19) *P* = 0.012**	2.41 (0.99–5.86) *P* = 0.052	2.29 (0.94–5.58) *P* = 0.069

RTI = respiratory tract infection. SES = socioeconomic status; low SES determined as percentage of participants with no formal qualifications (Case, Fertig, & Paxson, 2005). BMI = body mass index. CRP = C-reactive protein. CTQ = childhood trauma questionnaire. Smoking, alcohol, and SES were assessed at Week 1. BMI, perceived stress, sleep quality and CRP were assessed at week 5. N = 741 of 808 opted-in to completing the CTQ and were present at week 5. To reduce the risk of reverse causation, N = 16 participants were removed as they were diagnosed with an RTI in close proximity to the week 5 CRP measurement (weeks 4-6), leaving *N* = 724 available.

Table 8. *Association between potential mediator variables and RTI risk.*

	Model 1: Unadjusted	**Model 2:** Adjusted for age, ethnicity, season, region of recruitment & days in training
Variables at week 1		*N* = 908
BMI	1.11 (0.89–1.38) *P* = 0.348	1.14 (0.90–1.44) *P* = 0.270
Smoker [reference: non-smoker]	1.30 (0.83–2.03) *P* = 0.248	1.26 (0.80–1.99) *P* = 0.314
Alcohol use [reference: no alcohol use]	1.24 (0.75–2.06) *P* = 0.401	1.20 (0.71–2.04) *P* = 0.492
Low SES [reference: high SES]	1.35 (0.77–2.37) *P* = 0.299	1.26 (0.70–2.26) *P* = 0.435
Perceived stress (PSS) *Cut-off score >13* [reference: ≤13]	1.00 (0.63–1.60) *P* = 0.987	1.12 (0.69–1.81) *P* = 0.648
Sleep quality (PSQI) *Cut-off score ≥5* [reference: <5]	1.52 (0.96–2.42) *P* = 0.077	1.56 (0.97–2.51) *P* = 0.068
Variables at week 5		*N* = 724
BMI	1.00 (0.76–1.32) *P* = 0.975	1.07 (0.83–1.38) *P* = 0.612
Perceived stress (PSS) *Cut-off score >13* [reference: ≤13]	**2.27 (1.35–3.81) *P* = 0.002**	**2.49 (1.46–4.24) *P* < 0.001**
Sleep quality (PSQI) *Cut-off score ≥5* [reference: <5]	1.64 (0.97–2.76) *P* = 0.066	**1.76 (1.02–3.04) *P* = 0.041**

RTI = respiratory tract infection. BMI = body mass index. SES = socioeconomic status; low SES determined as percentage of participants with no formal qualifications (Case, Fertig, & Paxson, 2005). PSS = Perceived Stress Scale (10-item). PSQI = Pittsburgh Sleep Quality Index. At week 1, *N* = 908 of 995 opted-in to completing the CTQ. N = 741 of 808 opted-in to completing the CTQ and were present at week 5. To reduce the risk of reverse causation, participants diagnosed with an RTI in close proximity to the week 5 assessment (weeks 4–6) were excluded (*N* = 16), leaving *N* = 724 available.

4.5 Discussion

These new findings are consistent with the hypothesis that childhood adversity has a lasting influence on host resistance to infectious disease. Among recruits undertaking a 28-week military training course, childhood adversity was associated with an approximately 2-fold increased risk of physician-diagnosed respiratory infection in adulthood. This association was stronger in those reporting multiple and the most severe childhood adversities. For example, fully adjusted regression analyses showed that those who experienced abuse related adversities during childhood were nearly three times more likely to suffer RTI in adulthood; however, neglect related adversities were not associated with increased RTI risk. Those reporting childhood adversity also reported higher perceived stress and poorer sleep quality during the training period (*vs* no childhood adversity), and these variables were in turn associated with elevated RTI risk. Statistical control for perceived stress and sleep quality attenuated the strength of some associations, suggesting that differential adult experience may contribute to the differential risk of RTI during adulthood. However, RTI associations with multiple adversities and abuse-related adversities remained robust despite control for perceived stress and sleep quality, suggesting the existence of additional mediating pathways that remain to be identified in future research.

The mechanism(s) responsible for the observed association between childhood adversity and adulthood RTI risk remains to be elucidated. Childhood adversity is multi-dimensional and can be divided into experiences of abuse (reflecting potential harm) and neglect (reflecting an absence of expected environmental inputs), that can disrupt neural development and the functioning of stress-sensitive pathways, impairing host defence and immunity, as well as cognitive and social development (McLaughlin et al., 2014; Nelson et al., 2020). Individuals exposed to childhood adversity often develop heightened stress reactivity, impaired emotional regulation, and reduced coping capacity (Fagundes, Glaser, & Kiecolt-Glaser, 2013); maladaptations likely to amplify emotional and physiological stress sensitivity to the challenging scenarios faced during military training. In the present study, a greater proportion of individuals with the most severe childhood adversity (CTQ severity score quartile 4) reported moderate-high levels of perceived stress at week 1 (39% *vs* 23% quartile 1) and strikingly so at week 5 (68% *vs* 38% quartile 1); this highlights that those with the most severe childhood adversity struggled to cope with the multifaceted demands of military training. Consistent with this, perceived stress and poor sleep quality assessed during training were independently associated with increased RTI risk (**Table 8**), consistent with previous research

in the general population (Cohen et al., 2009; Cohen et al., 1991). Adjusting for these variables during training partially attenuated the level of association between childhood adversity and adult RTI risk in some analyses, suggesting that perceived stress and poor sleep quality constitute important, though not exclusive, pathways through which childhood adversity increases RTI susceptibility. However, associations with multiple adversities and abuse-related adversities remained robust across all models, indicating that additional mediating pathways beyond stress and sleep remain to be identified in future research. These may include stress-biological pathways that impair host resistance to infection, as well as differences in disease-mediating behavioural processes stemming from effects of early adversity on social or cognitive development.

Although experiences of childhood abuse and neglect tended to co-occur (**Table 2**), as is widely reported (McLaughlin et al., 2014), analysis of adversity type (i.e., only abuse or only neglect) showed the strongest association between childhood adversity and RTI for those who experienced abuse, whereas neglect was not associated with a raised risk of RTI (**Figure 4**C). Assuming similar infection exposure in the shared living and working conditions, the biological mechanism(s) by which the experience of childhood abuse but not neglect increased adulthood RTI risk during military training requires investigation. Evidence points to a 'biological embedding' of the most severe forms of childhood adversity (e.g., abuse and trauma) in immune cell gene expression that manifests low-grade inflammation and downregulated anti-viral responses in adulthood (Cole, 2019; Kohrt et al., 2016; Miller et al., 2011). In addition to this 'molecular liability', adults with early life experiences of abuse have reduced coping, exaggerated stress sensitivity and increased vulnerability to the purported effects of chronic stress on neuroendocrine-immune dysregulation and pathology (as conceptualised in the allostatic load model) (LoPilato et al., 2020; McEwen, 1998). Together with potential differences in health behaviour (e.g., patterns of social interaction, preventive healthcare, etc.), these biological and psychosocial mechanisms may account for the robust association between abuse-related adversities and RTI risk observed in the present study, even after adjustment for perceived stress, sleep quality and serum CRP. Notably, adjustment for serum CRP did not attenuate the associations between childhood adversity and RTI risk, and CRP did not differ across CTQ severity quartiles at either week 1 or week 5 (**Table 4**). This suggests that low-grade systemic inflammation is unlikely to be the primary biological pathway linking childhood adversity to RTI susceptibility in this young adult cohort, and instead, the

mechanism points more towards impaired cellular immunity and downregulated innate antiviral responses (Cole, 2019).

In the context of the existing research linking early adversity to lifespan health and well-being, this study has several strengths. We used the validated CTQ to assess reports of childhood adversity, which is widely used in research and clinical settings (de Koning, Kuzminskaite, Vinkers, Giltay, & Penninx, 2022; Heany et al., 2018; Hinkelmann et al., 2013; Liebschutz et al., 2018). Furthermore, military training offered a unique opportunity to investigate the relationship between childhood adversity and adulthood RTI risk under standardised living and working conditions (e.g., housing, diet and physical activity) in young adults facing the challenges associated with relocating to a new living and working environment alongside the physical and psychological demands of the course. Standardisation of housing (infection exposure), diet, physical activity and sleep opportunity are important because these factors may influence RTI risk (Ahmad et al., 2020; Bermon et al., 2017; Simpson et al., 2020; Walsh et al., 2023).

Despite the strengths listed above, the present study also has several limitations. Our measurement of childhood adversity was retrospective which can be prone to memory bias and reporting errors; albeit moderate agreement has been shown between prospective and retrospective reports of childhood adversity (Hardt & Rutter, 2004). Prospective studies following children through the life course are ethically challenging, take years to conduct, and the findings of which regarding the influence of childhood adversity on adulthood RTI risk would remain associational, like the present study (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Liebschutz et al., 2018). To provide separation between childhood and adulthood we recorded adverse events occurring before 11 years of age. We acknowledge the limitation that childhood adversity is typically recorded up to 18 years of age (Felitti et al., 1998). As such, we cannot exclude the possibility that some participants may have been experiencing forms of abuse or neglect immediately prior to commencing training. In this regard, the military training environment provides separation from everyday home life, offering a unique opportunity to observe the effects of childhood adversity on adulthood RTI under controlled conditions that limit the influence of ongoing adversity related to home life. Perceived stress and sleep quality were assessed at week 1 and week 5 only. Given that the training course lasted 28 weeks, these variables may have changed meaningfully beyond week 5 as the physical and psychological demands of training continues. As such, our regression analyses may underestimate the extent

to which stress and sleep quality during training potentially mediate the association between childhood adversity and RTI risk in adulthood, and future studies should incorporate repeated psychosocial assessments across the full duration of training. We also acknowledge that the sample population comprised of young adult males, primarily white (84%) and of lower SES (Kiernan, Arthur, Repper, Mukhuty, & Fear, 2016), limiting the generalisability of these findings. Whether these findings are generalisable to females and ethnically minoritised individuals warrants investigation, particularly as these individuals are disproportionately affected by the most severe childhood adversity experiences (e.g., abuse) which given the present findings may conceivably increase their risk of adulthood RTI (Chen et al., 2021; Nelson et al., 2002). We anticipated a greater incidence of childhood adversity compared with wider population studies as military recruits typically come from lower SES backgrounds where childhood adversity is more prevalent (Campbell-Sills et al., 2025; Walsh et al., 2019). The proportion of participants with childhood adversity (47%) was greater than a retrospective cohort study using UK Biobank data (33%) (Ahmad et al., 2020) but comparable to that reported in the Danish nationwide register (45%) (Miller et al., 2011). By barring entry to military training in those with psychiatric disorders (e.g., clinical anxiety and depression) and chronic lung diseases (e.g., asthma), conditions associated with childhood adversity and RTI susceptibility (Corne et al., 2002; Helby, Nordestgaard, Benfield, & Bojesen, 2017; Vedhara et al., 2022), the study sample may not fully represent the broader population of individuals with severe childhood adversity. Furthermore, although their childhood adversity experience is unknown, participants who opted out of answering the CTQ (9%) were similar to those with childhood adversity for demographic information (e.g., smoking and SES) and perceived stress and sleep quality at the start of training (**Table 3**). This points to another potential source of bias and underreporting of childhood adversity in this study and the wider literature, as discussed elsewhere (Danese, 2020; Felitti et al., 1998). Finally, future studies should perform pathological analysis on throat swabs and assess inflammation to confirm the infectious origin of physician diagnosed RTI.

In conclusion, childhood adversity was associated with increased risk of respiratory infection in adults undergoing military training, particularly in those reporting experiences of multiple adversities and childhood abuse. Associations were independent of demographic confounders, and some analyses suggest that higher perceived stress and poorer sleep quality in adulthood may partially mediate these effects. These findings highlight the long-term impact of childhood adversity on infection risk, offering novel insights into how the number, type and severity of

adverse childhood experiences shape vulnerability to respiratory infection in adulthood. Future research must explore targeted interventions to reduce the health burden of childhood adversity and inform evidence-based public health policies to improve lifelong health outcomes.

Recommendations for the MOD deriving from this chapter's findings:

1. **Adversity screening at recruitment.** Approximately half of infantry recruits reported a history of childhood adversity, yet this is not currently assessed at entry. Incorporating a brief validated screening tool, such as the short-form Childhood Trauma Questionnaire, into recruitment or early training assessments could identify recruits at elevated infection risk, enabling targeted monitoring and support without requiring disclosure of specific experiences.
2. **Enhanced welfare support during Phase 1 of training.** Recruits with childhood adversity reported significantly higher perceived stress and poorer sleep quality at the start and during training, both of which partially mediate elevated infection risk. Consideration should be given to whether enhanced welfare contact or access to psychological services during the early period of Phase 1 would be beneficial for these individuals.
3. **Awareness training for training and medical staff.** Training staff and medical personnel should be made aware that early life psychosocial experiences can elevate infection risk independently of psychosocial and lifestyle factors. Brief awareness training regarding the health consequences of childhood adversity could support earlier identification of at-risk recruits at minimal cost.

CHAPTER 5 – Childhood adversity and respiratory infection risk in adulthood: The protective role of optimism

5.1 Abstract

To assess whether optimism moderates the association between childhood adversity RTI in adulthood, we examined 1,188 healthy young adults commencing a 28-week military training course in the UK. Logistic regression quantified associations between childhood adversity (CTQ), changes in optimism (LOT-R), and physician-diagnosed RTI, whereby recruits with and without childhood adversity were grouped into increased or decreased optimism. Regressions were adjusted for SES, lifestyle, perceived stress, depressive symptoms, and sleep quality. Almost half of recruits reported childhood adversity (47%). Changes in optimism during the first month of training significantly moderated the association between childhood adversity and subsequent RTI incidence after week 5 (unadjusted: B = -0.22, SE 0.11, P = 0.041). Recruits with childhood adversity who showed increases in optimism were at no greater risk of subsequent RTI compared to those without childhood adversity and increased optimism (OR = 2.24, 0.62–8.05, P = 0.219). In contrast, recruits with childhood adversity who experienced decreases in optimism were over five times more likely to suffer subsequent RTI (OR = 5.40, 1.70–17.11, P = 0.004 *vs* childhood adversity and increased optimism). This pattern was consistent for multiple adversities, abuse-related adversities, and the most severe adversity (CTQ quartile 4). Findings remained after adjustment for covariates. In conclusion, increases in optimism during military training protected against the negative effects of childhood adversity on adulthood RTI.

5.2 Introduction

Childhood adversity encompasses a wide range of negative experiences that can occur during childhood, including physical, emotional, and sexual abuse, and physical, and emotional neglect. A growing body of evidence links childhood adversity with adverse psychological and physical health outcomes across the life course (Brown et al., 2009; Hughes et al., 2017; Norman et al., 2012; Sumner et al., 2019). Extending this literature, findings from **Chapter 4** demonstrated that childhood adversity was associated with increased respiratory infection risk in adulthood, particularly in individuals reporting multiple, abuse, or the most severe experiences of adversity. However, it is important to note that not all individuals with childhood adversity develop poor health in later life, suggesting a role of potential protective factors moderating this risk.

One framework for understanding such moderation is Cohen's Stress-buffering Model, which proposes that psychosocial resources (e.g., optimism and social support) protect health by reducing the appraisal of stressors as threatening and by enhancing coping resources (Cohen & Wills, 1985). Stress-buffering factors are consistently associated with better health and immune outcomes, including a reduced likelihood of cardiovascular events (Giltay et al., 2004), mortality (Rozanski et al., 2019), improved antibody response to vaccination (Dantzer et al., 2018; Kohrt et al., 2016) and RTI (Dantzer et al., 2018; Kohrt et al., 2016; Segerstrom et al., 1998). However, it remains unknown whether such stress-buffering factors provide a protective effect on the association between childhood adversity and RTI.

Mechanistic pathways help to explain why adults with a history of childhood adversity are at elevated risk to negative health outcomes and how optimism may provide a protective effect. Individuals with childhood adversity have an increased stress perception in adulthood whereby they are more likely to report higher psychological stress (LoPilato et al., 2020) and depressive symptoms in daily life (Dougherty et al., 2004), poorer sleep quality (Liu et al., 2023), and have less coping resources (e.g. resilience) (Beutel et al., 2017) to protect against the deleterious effects of childhood adversity on health (Hughes et al., 2017). Chronic stress and adversity can lead to dysregulation of the HPA axis; disrupting the normal functioning of the immune system and causing chronic inflammation (Miller, Chen, & Zhou, 2007). Psychological resilience (e.g., optimism) is thought to provide a protective effect on the negative influence of stress on HPA dysregulation by reducing the perceived threat of stress and providing effective coping mechanisms (Dantzer et al., 2018). Evidence to support this has

demonstrated resilience to buffer against the effects of childhood adversity on immune health (upregulated inflammatory and downregulated anti-viral gene expression) (Kohrt et al., 2016). Whether optimism protects against the purported negative effects of childhood adversity on RTI remains unknown. This is particularly appealing because studies have shown that practical, short-term interventions can improve levels of resilience in both civilian and military populations (Joyce et al., 2018; Malouff & Schutte, 2016; Meevissen et al., 2011; Peters et al., 2010).

Military training focuses on the development of physical, technical, and psychological abilities of a soldier, with a key focus placed on improving mental resilience (Precious & Lindsay, 2019). This scenario provides an ideal opportunity to investigate changes in resilience on health outcomes. With this information in mind, in a cohort of 1,188 healthy young adults embarking on a military career, we examined the protective role of optimism on the association between childhood adversity and RTI. We hypothesised that increases in optimism would provide a protective effect on the association between childhood adversity and subsequent RTI.

5.3 Methods

5.3.1 Study design and participants

Study design and participants are outlined in **Chapter 3, section 3.1 – 3.3**.

5.3.2 Procedures

A total of 1,188 healthy recruits (98% male) provided written informed consent to participate in the study (**Chapter 4, Figure 3**). Procedures are detailed in **Chapter 3, section 3.3**.

Psychosocial and lifestyle measures were assessed in private using online surveys (LimeSurvey, Hamburg, Germany) via an electronic tablet. Childhood adversity was assessed using the CTQ-SF and optimism (LOT-R), perceived psychological stress (PSS) and sleep quality (PSQI) were assessed at week 1 and one-month later to examine changes during training (see **Chapter 3, sections 3.4 – 3.7**).

5.3.3 Outcomes

The primary outcome of this study was RTI during military training. RTI diagnosis is described in **Chapter 3**.

5.3.4 Statistical analysis

Participant demographic, lifestyle, psychosocial and childhood adversity data are presented as mean ± SD for continuous variables or absolute numbers and percentages for categorical variables. Comparisons were made using independent t-tests, paired samples t-tests, ANOVA, one-way repeated measures ANOVA and Chi-square, where appropriate (**Table 9** & **Table 10**).

A small proportion of PSQI data at week 5 were missing, assumed missing at random, with 17% (N = 166) of recruits missing PSQI data e.g. due to administrative issues with questionnaires. To avoid a potential bias, multiple imputation was carried out using predictive mean matching (50 iterations and 40 imputed datasets), in line with recommendations (Graham, 2009). After accounting for participants who withdrew from Army training and the study by week 5, PSQI collected at week 1 and all variables collected at week 5 were included in multiple imputation, including the outcome variable RTI incidence.

Hierarchical logistic regression was used to examine the association between childhood adversity, optimism and RTI risk. After checking assumptions, we first examined the moderating role of optimism and other psychosocial variables on childhood adversity and RTI risk. Moderation analyses examining adversity type and severity are presented unadjusted, as small subgroup sizes were insufficient to support reliable covariate-adjusted estimation. Secondly, logistic regression was also used to examine the combined influence of optimism and childhood adversity (including the number and type) on RTI susceptibility, whereby recruits with and without childhood adversity were grouped into increased or decreased optimism by reporting at least a one unit increase or decrease in optimism levels between week 1 and week 5 (Kronstrom et al., 2011); recruits without childhood adversity who reported increased optimism were considered the reference group (**Figure 5**). We also investigated the effects of childhood adversity severity and changes in optimism on RTI risk, with severity assessed using quartiles of the CTQ; recruits with Q1 who reported increased optimism were considered the reference group (**Figure 5**). Regression analysis was performed using a 3-step model approach: Model 1 investigated the unadjusted association with RTI; Model 2 was adjusted for likely RTI risk factors including, BMI (Murugan & Sharma, 2008), smoking (Arcavi & Benowitz, 2004), SES (Cohen, Chiang, Janicki-Deverts, & Miller, 2020), season (Heikkinen & Jarvinen, 2003) and days spent in training to control for RTI exposure; Model 3 was adjusted for all covariates included in model 2, plus adjustment for perceived stress, and

sleep quality (fully adjusted model) (Cohen et al., 1991; Falagas, Mourtzoukou, & Vardakas, 2007; Walsh et al., 2023). Analyses were conducted using SPSS 28.0 (IBM, Armonk, NY, USA) with statistical significance set at $P < 0.05$.

5.4 Results

5.4.1 Sample characteristics, childhood adversity and RTI incidence

Demographic, lifestyle and childhood adversity descriptive data in the population that commenced training are presented in **Chapter 4 Table 1** and **Table 2**.

A greater proportion of recruits with childhood adversity had no formal qualifications and reported greater levels of perceived stress, and poorer optimism and sleep quality at the start of training (vs no childhood adversity; **Chapter 4 Table 3**). During the first month of training, perceived stress, sleep quality, and optimism levels decreased; a trend similar to first-year university students experiencing a major life transition (Duffy et al., 2020). These decreases in psychosocial factors were exacerbated in those with childhood adversity (**Table 9**). Despite this, 40% of recruits reported increased optimism in the first month of training, with comparable proportions who increased their optimism between those with and without childhood adversity (**Table 10**). Furthermore, in those who reported decreased optimism in the first month of training, the magnitude of decreased optimism was similar between those with and without childhood adversity (**Table 10**). Recruits who experienced childhood adversity and reported decreases in optimism reported the highest levels of perceived stress, and poor sleep at week 5 (**Table 10**).

A total of 100 physician diagnosed RTI incidences were recorded during training, of which 70 occurred after week 5.

Table 9. *Perceived stress, sleep quality and optimism by childhood adversity classifications.*

	All	No Childhood Adversity	Childhood Adversity	CTQ Severity Score Quartile			
				Q1	Q2	Q3	Q4
	N = 741	*N* = 396	*N* = 345	*N* = 199	*N* = 191	*N* = 167	*N* = 184
Variables at week 1							
Perceived stress (PSS)	11.1 ± 5.5	10.0 ± 5.2	12.3 ± 5.5^{aa}	9.6 ± 5.5	10.3 ± 5.3	11.9 ± 4.8^{bb}	12.9 ± 5.6^{bb}
Cut-off score ≥13[§] [N (%)]	230 (31)	96 (24)	134 (39)^{aa}	45 (23)	51 (27)	62 (37)^{bb}	72 (39)^{bb}
Sleep quality (PSQI)	5.0 ± 2.7	4.6 ± 2.5	5.5 ± 2.8^{aa}	4.4 ± 2.5	4.7 ± 2.7	4.8 ± 2.2	6.2 ± 3.0^{bb}
Cut-off score ≥5[#] [N (%)]	400 (54)	186 (47)	214 (62)^{aa}	89 (45)	89 (47)	97 (58)^{bb}	125 (68)^{bb}
Optimism (LOT-R)	14.0 ± 3.9	14.3 ± 3.9	13.5 ± 4.0^{aa}	14.9 ± 3.9	14.3 ± 3.7	13.4 ± 3.9^{bb}	13.0 ± 4.0^{bb}
Cut-off score ≥14[◇] [N (%)]	408 (51)	236 (60)	172 (50)^{aa}	128 (64)	116 (61)	79 (47)^{bb}	85 (46)^{bb}
Variables at week 5							
Perceived stress (PSS)	13.5 ± 6.5**	12.3 ± 6.4**	14.8 ± 6.3^{**aa}	12.0 ± 6.6**	12.2 ± 6.0**	14.2 ± 6.4^{**bb}	15.8 ± 6.0^{**bb}
Cut-off score ≥13 [N (%)]	373 (50)**	163 (41)	210 (60)^{aa}	76 (38)**	81 (42)**	90 (54)^{**bb}	126 (67)^{**bb}
Sleep quality (PSQI)	5.3 ± 2.4**	5.0 ± 2.3**	5.6 ± 2.4^{aa}	4.8 ± 2.5*	5.0 ± 2.3	5.3 ± 2.2**	6.0 ± 2.5^{bb}
Cut-off score ≥5 [N (%)]	439 (60)*	217 (55)	222 (64)^{aa}	97 (49)	108 (57)*	104 (62)^{b}	130 (71)^{bb}
Optimism (LOT-R)	13.8 ± 4.4	14.3 ± 4.2	13.2 ± 4.4^{aa}	14.6 ± 4.2	14.3 ± 4.1	13.6 ± 4.5^{bb}	12.5 ± 4.4^{**bb}
Cut-off score ≥14 [N (%)]	381 (51)	225 (57)	156 (45)^{aa}	120 (60)	110 (58)	78 (47)^{bb}	73 (40)^{bb}

Values presented as mean ± SD unless otherwise stated. CTQ = childhood trauma questionnaire. Q = quartile. *N* = 741 of 808 opted-in to completing the CTQ and were present at week 5. * = significantly different to week 1 of corresponding group. ^{a} = significantly different to No Childhood Adversity. ^{b} = significantly different to Q1. Single letter denotes $P < 0.05$ (e.g., ^{a}); double letter denotes $P < 0.01$ (e.g., ^{aa}). [§] Cohen et al., 1983; [#] Buysse et al., 1989; [◇] Scheier et al., 1994.

Table 10. *Psychosocial variables by Childhood Trauma Questionnaire severity score quartiles and changes in optimism.*

	No Childhood Adversity		Childhood Adversity	
	↑ optimism	**↓ optimism**	**↑ optimism**	**↓ optimism**
	***N* = 147 (42%)**	***N* = 198 (44%)**	***N* = 118 (38%)**	***N* = 175 (46%)**
Variables at week 1				
Perceived stress (PSS)	11.0 ± 5.2	9.0 ± 5.1[aa]	12.4 ± 5.6	12.4 ± 5.2[b]
Sleep quality (PSQI)	4.6 ± 2.2	4.7 ± 2.8	5.3 ± 2.6	5.7 ± 3.0[aabb]
Optimism (LOT-R)	13.7 ± 3.5	14.9 ± 4.0[a]	12.2 ± 3.9[aa]	14.0 ± 3.8[cc]
Variables at week 5				
Perceived stress (PSS)	10.9 ± 5.8	13.4 ± 6.6[aa]	14.0 ± 6.3[aa]	15.9 ± 6.2[aabbc]
Sleep quality (PSQI)	4.8 ± 2.0	5.1 ± 2.5	5.1 ± 2.4	6.0 ± 2.5[aabbc]
Optimism (LOT-R)	16.4 ± 3.6	12.8 ± 4.0[aa]	14.9 ± 4.1[a]	11.5 ± 4.1[aabbcc]
Changes in psychosocial variables				
Perceived stress (PSS)	-0.1 ± 4.6	4.4 ± 5.4[aa]	1.6 ± 5.2[a]	3.5 ± 6.0[aacc]
Sleep quality (PSQI)	0.2 ± 2.6	0.4 ± 3.2	-0.2 ± 2.5	0.3 ± 2.8
Optimism (LOT-R)	2.7 ± 2.1	-2.1 ± 2.9[aa]	2.7 ± 1.8	-2.5 ± 2.2[aacc]
Childhood adversity severity score	28.9 ± 3.5	29.0 ± 3.5	44.9 ± 11.8[aa]	46.7 ± 12.5[aabb]
Completed training on time [N (%)]	88 (60)	129 (45)[aa]	73 (62)	114 (45)[aacc]

Values presented as mean ± SD unless otherwise stated. Increases and decreases in optimism were determined by reporting a change of either a at least a one unit increase or decrease in optimism levels between week-1 and week-5 (Kronstrom, 2011). *N* = 103 recruits who experienced no change in optimism are not presented. [a] = significantly different to no childhood adversity ↑ optimism; [b] = significantly different to equivalent no childhood adversity group. [c] = significantly different to childhood adversity ↑ optimism. Single letter denotes $P < 0.05$ (e.g., [a]); double letter denotes $P < 0.01$ (e.g., [aa]).

5.4.2 Improved optimism buffers the association between childhood adversity and RTI

Changes in optimism, but not perceived stress, in the first month of the training course significantly moderated the association between childhood adversity and subsequent RTI incidence, whereby the association between childhood adversity and the raised risk of RTI was driven by those who experienced a decrease in optimism in the first month of training (unadjusted: B = -0.22, SE 0.11, P = 0.041). Findings remained for multiple adversities (unadjusted: B = -0.10, SE 0.05, albeit P = 0.059), abuse-related adversities (unadjusted: B = -0.08, SE 0.04, P = 0.028), and the most severe adversity (Q4) (unadjusted: B = -0.43, SE 0.19, P = 0.021). Fully adjusted hierarchical logistic regression showed that at the start of training, perceived stress and optimism were not independently associated with RTI susceptibility (P values all > 0.700).

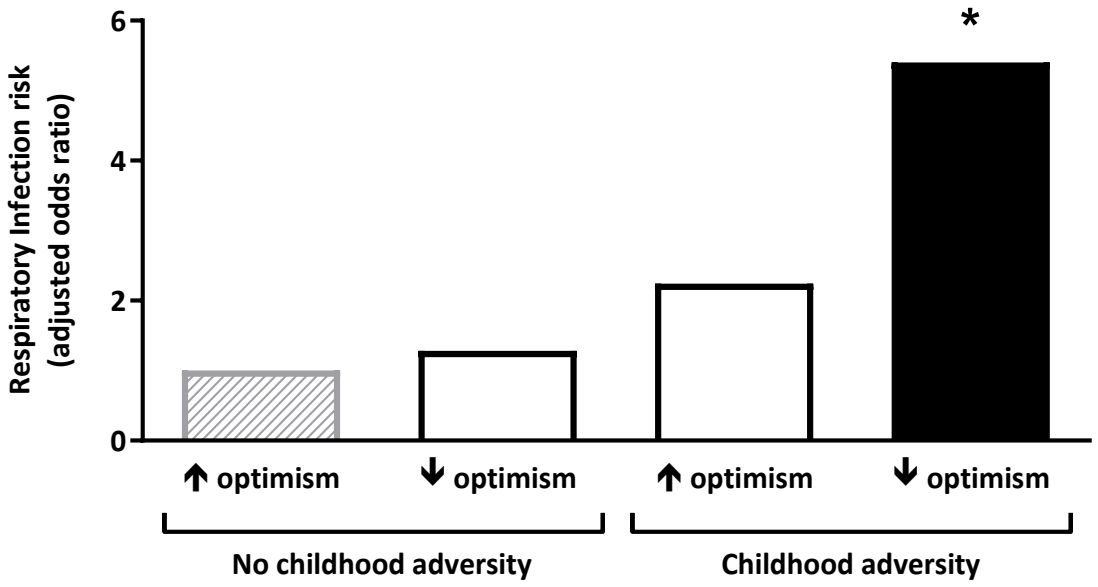

Figure 5. *The interaction between childhood adversity and changes in optimism at the start of training, on respiratory tract infection (RTI) occurring after the first 5 weeks of military training. Odds ratios presented are from adjusted analyses accounting for; BMI, smoking status, education level, season of enrolment, perceived stress, depressive symptoms, civilian sleep quality (PSQI), and days spent in training. No childhood adversity and ↑ optimism is considered as reference. * P < 0.05 vs no childhood adversity and ↑ optimism.*

Further analysis examining the combined influence of childhood adversity and changes in optimism on RTI risk showed that recruits without childhood adversity who reported decreases in optimism in the first month of training were at no greater risk of RTI (fully adjusted: OR = 1.28, 0.33–5.06, P = 0.723). Similarly, recruits with childhood adversity who reported increases in optimism were at no greater risk of RTI (fully adjusted: OR = 2.24, 0.62–8.05, P

= 0.219), compared to individuals with no childhood adversity and increases in optimism. However, recruits who experienced childhood adversity and reported decreases in optimism in the first month were over five times more likely to suffer a subsequent RTI (fully adjusted: OR = 5.40, 1.70–17.11, P = 0.004; **Figure 5**), despite comparable childhood adversity scores in recruits with childhood adversity who reported increases and decreases in optimism (increases 44.9 ± 11.8, decreases 47.1 ± 14.5; P = 0.192).

These associations remained when examining multiple adversities, abuse-related adversities those with the most severe adversity (Q4) **Figure 6**. In summary, the association between childhood adversity, multiple adversities, abuse-related adversities and the most severe adversity and RTI was driven by recruits reporting decreases in optimism in the first month of training, highlighting the protective role of increases in optimism against respiratory infection in recruits with childhood adversity.

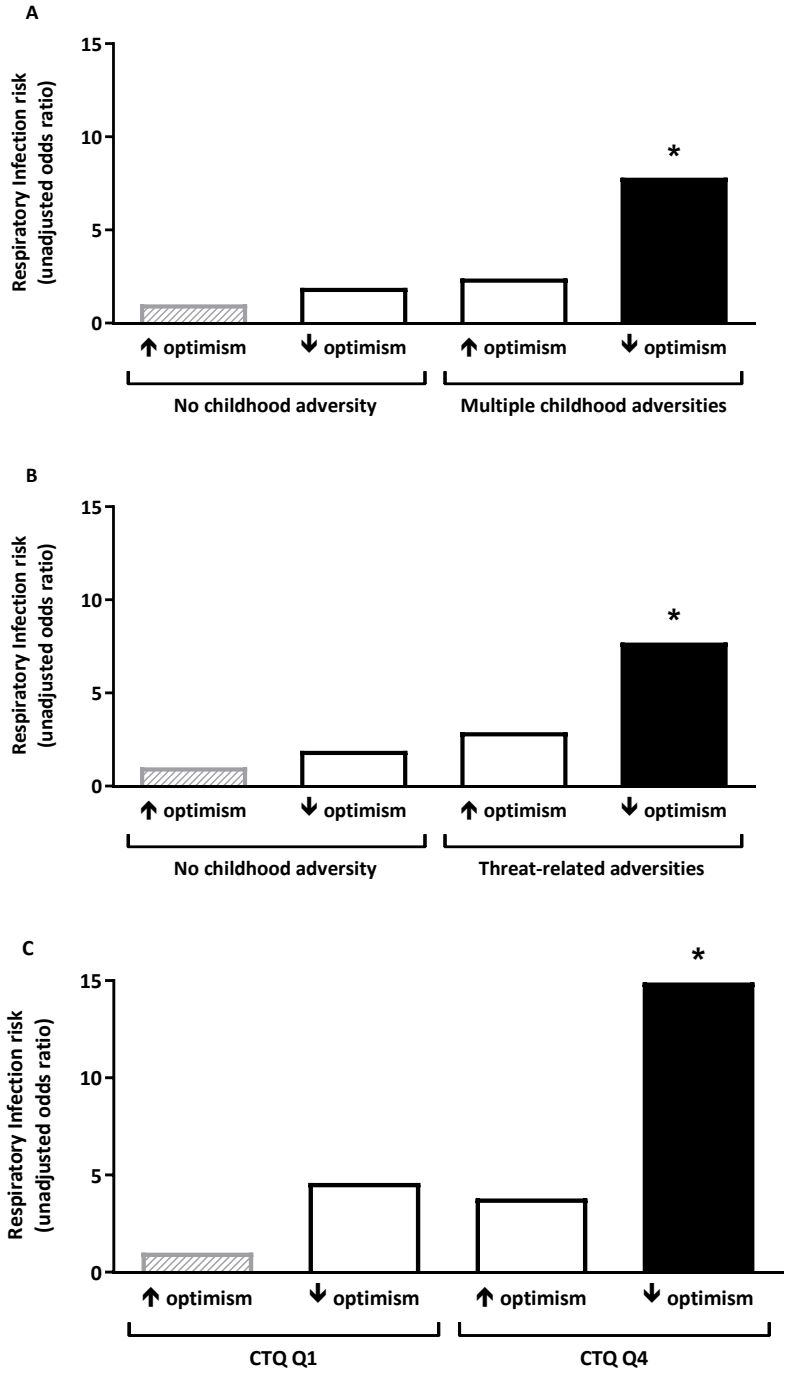

Figure 6. *The interaction between the number of childhood adversities (A), the type of adversity (B), and the severity of adversity (C) and changes in optimism during the start of training, on respiratory tract infections (RTI) occurring after the first 5 weeks of military training. CTQ = Childhood Trauma Questionnaire. Q = quartile. Odds ratios presented are from unadjusted analyses. No childhood adversity/CTQ Q1 and ↑ optimism is considered as reference. * P < 0.05 vs no childhood adversity/CTQ Q1 and ↑ optimism.*

5.5 Discussion

These findings provide further insights into the protective role of optimism on immune health in those with a history of childhood adversity. Changes in optimism during the first month of military training moderated the relationship between childhood adversity and subsequent RTI after week 5, whereby the association between childhood adversity and a raised risk of RTI was driven by those who experienced a decrease in optimism (**Figure 5**). Recruits with childhood adversity and an increase in optimism were protected against RTI.

The current findings presented include several strengths to further provide confidence in the results. Recruits presenting with subjective common cold symptoms or CRP values > 10 mg/L (indicative of acute infection) (Pearson et al., 2003) at the start of training or were diagnosed with a RTI within one week of arrival were deemed not healthy and subsequently excluded from analyses to mitigate reverse causation. Regression models were adjusted for variables known to be associated with increased RTI risk, including BMI (Harpsoe et al., 2016), smoking (Arcavi & Benowitz, 2004), education, season (Heikkinen & Jarvinen, 2003), perceived stress (Cohen et al., 1999; Cohen et al., 1991), sleep quality (Robinson et al., 2021), and further adjustment to account for the number of days spent in training.

The potential biological rationale by which optimism acts as a protective buffer on the effects of stressors and adversities on health is not yet fully understood. Chronic stress is associated with dysregulated immune function and increased inflammation however, optimism has been shown to moderate the negative impact of such stress on immunity (Brydon, Walker, Wawrzyniak, Chart, & Steptoe, 2009; Segerstrom, 2005; Segerstrom et al., 1998). Optimists hold more coping resources to manage stressors, experience less negative mood, and display better health behaviours (Brissette, Scheier, & Carver, 2002; Carver et al., 1993; Scheier et al., 1994). Dysregulation of the immune system becomes increased during times of more recent stress due to increased stress sensitivity in individuals with a history of childhood adversity, coupled with having fewer coping resources to manage stress (Dougherty et al., 2004; Fagundes, Glaser, Malarkey, et al., 2013). Evidence of this can be seen in the present study whereby we followed recruits throughout military training and demonstrated that in those reporting childhood adversity and increases in optimism, lower perceived stress, and better sleep quality were demonstrated compared to individuals reporting childhood adversity and decreases in optimism. Furthermore, increases in optimism provided a protective role on the negative influence of childhood adversity on RTI risk.

We acknowledge that the present study isn't without limitation. The associative design cannot determine causality, and there may be environmental factors influencing the relationship between childhood adversity and RTI. Nevertheless, associations remained after accounting for demographic and lifestyle behaviours. Also, we provide confidence about reverse causality excluding those with RTI symptoms, elevated CRP and RTI cases occurring in the first week. Secondly, our measurement of childhood adversity was retrospective which can be prone to memory bias, recall bias and reporting errors, however, moderate agreement has been shown between prospective and retrospective reports of childhood adversity (Hardt & Rutter, 2004). In addition, we used the CTQ to assess reports of childhood adversity, a questionnaire that was been widely used in research and clinical settings (de Koning et al., 2022; Heany et al., 2018; Hinkelmann et al., 2013; Liebschutz et al., 2018). Experimental studies have prospectively followed children and performed assessments at different time points through the life course over many decades (Danese et al., 2007; Liebschutz et al., 2018). These studies are incredibly difficult to undertake and take years. Lastly, the sample population was homogeneous, whereby the population was limited to young males from a low SES background and therefore not representative of the general population. Despite this, and due to the nature of military training, all recruits followed a rigid training schedule with similar diet, sleep opportunity and physical training demand, adding strength to the study as these are all factors known to influence immune health. Further investigation should be carried out to understand the buffering role of optimism on the association between childhood adversity and subsequent RTI risk in females. This is especially important as research has indicated differences in childhood adversity exposure between the genders, with females more likely to experience sexual abuse (Nelson et al., 2002).

In conclusion, increases in optimism during military training protected against the negative effects of childhood adversity on subsequent RTI. These findings contribute further evidence of the buffering role of optimism on adversity and health outcomes. Future research should prioritise potential interventions to improve resilience and optimism in those with childhood adversity, to subsequently improve health.

Recommendations for the MOD deriving from this chapter's findings:

1. **Embed optimism-building activities into early Phase 1 training.** Brief psychological techniques shown to increase optimism, including cognitive-behavioural approaches, positive goal-setting exercises, and best-possible-self interventions, could feasibly be incorporated into the educational framework during the first month of training. Given that optimism changes over just four weeks meaningfully predicted health outcomes, even short-duration, low-cost psychological inputs during this window may yield meaningful reductions in infection risk.

CHAPTER 6 – Good perceived sleep quality protects against the raised risk of respiratory infection during sleep restriction in young adults

The analyses presented in this chapter were conducted in a cohort of recruits from a separate research programme than those studied in Chapters 4, 5, 7 and 8. This population includes males and females from the Infantry Training Centre Catterick, UK, and the Army Training Centre, Pirbright, UK, with data collected between January 2014 and November 2015.

6.1 Abstract

To examine whether sleep restriction increases URTI susceptibility and whether PSQ moderates this association, we prospectively studied 1,318 healthy young adults (68% male) commencing Phase 1 12-week military training. Sleep restriction was defined as an individualised reduction in sleep duration of ≥2 hours/night compared with civilian life and URTIs were retrieved from medical records. PSQ was assessed using a four-point Likert scale with a dichotomous grouping created to represent 'good' and 'poor' PSQ. Logistic regression examined associations between sleep restriction, PSQ, and physician-diagnosed URTI, adjusted for sex, BMI, smoking, season, mood disturbance, and civilian sleep duration. Upon training commencement, approximately half of recruits were sleep restricted (52%; 2.1 ± 1.7 hours); despite the sleep debt, 58% of recruits with sleep restriction reported good PSQ. A total of 93 URTIs were recorded. Sleep restriction was independently associated with increased URTI susceptibility during the first four weeks of training and across the full 12-week course (4 weeks: unadjusted OR = 2.26, 1.08–4.73, P = 0.031). After adjustment for covariates, regression showed that these findings were driven by poor PSQ (4 weeks: OR = 3.18, 1.20–8.43, P = 0.020) as no significant association between sleep restriction and URTI was observed in those reporting good PSQ, despite a similar magnitude of sleep restriction. In recruits with sleep restriction, fully adjusted models showed that good PSQ was significantly associated with half the likelihood of URTI (12 weeks: OR = 0.51, 0.27–0.96, P = 0.027). Associations between sleep restriction, PSQ and URTI remained in recruits who endured sleep restriction throughout training. In conclusion, good PSQ is associated with protection against the raised risk of respiratory infection during sleep restriction.

6.2 Introduction

Sleep is considered an important factor in the promotion and maintenance of physical and cognitive health. However, modern day society has seen adults restrict their sleep to cope with the demands of work and other leisure activities, resulting in a high proportion of habitual short sleepers in the general population (Grandner et al., 2010). Evidence suggests that short sleep is associated with increased morbidity and mortality (Cappuccio et al., 2010a; Liu et al., 2017). For example, habitual short sleep is associated with a raised risk of respiratory infection (Cohen et al., 2009; Patel et al., 2012; Prather et al., 2015) and with diseases associated with inflammation (Ferrie et al., 2013), including cardiovascular disease (Altman et al., 2012) and diabetes (Lee, Ng, & Chin, 2017). An example of such is seen in a large population of UK military recruits, whereby those who reported sleeping <6h per night during training were four times more likely to be diagnosed with an URTI compared to those sleeping 7-9 hours (Wentz et al., 2018). As such, The National Sleep Foundation recommend that young adults sleep 7-9 hours per night for health, well-being, and optimal neurocognitive function (Hirshkowitz, Whiton, Albert, Alessi, Bruni, DonCarlos, Hazen, Herman, Adams Hillard, et al., 2015), however there is a long debate about the minimum amount of sleep required for health (Grandner et al., 2010), and that intra-individual differences are an important consideration (Besedovsky et al., 2019; Bin, 2016). A striking demonstration of this can be found in the results of one large epidemiological investigation, the principal finding of which was that short sleep was associated with pneumonia risk; however, pneumonia risk was only increased in short sleepers who perceived they had inadequate sleep, not in those who perceived their sleep to be adequate (Patel et al., 2012).

There is a growing interest in the influence of changes in sleep (e.g., sleep restriction) on the modulation of the immune system and the potential mechanisms involved. Similar to other forms of stress, sleep restriction influences immunity via activation of the HPA axis and the sympathetic nervous system (Irwin, 2015). Laboratory studies of sleep restriction, performed in those who routinely achieve sleep recommendations (7–9 h sleep/night) (Hirshkowitz, Whiton, Albert, Alessi, Bruni, DonCarlos, Hazen, Herman, Katz, et al.), in which sleep was restricted to 4 hours/night, demonstrate circadian misalignment e.g., disrupted HPA axis regulation of the diurnal cortisol rhythm (Guyon et al., 2014; Simpson et al.), increased inflammation e.g., raised circulating interleukin-6 and CRP (Simpson et al., 2016; van Leeuwen et al., 2009) and an impaired immune response to vaccination (Spiegel, Sheridan, & Van Cauter). Whilst the impact of a more modest sleep restriction of ~2 hours on cardiovascular

(Cabeza de Baca et al., 2019) and inflammatory (Vgontzas et al., 2004) outcomes have been examined, it remains unknown whether these findings translate to increased URTI, and the impact that accounting for individual sleep habits among a wider spread of sleeper warrants enquiry e.g., ~40% of the US adult population report sleeping <7 or >9 (Liu et al., 2016).

Thinking beyond the direct effects of sleep duration and sleep restriction on immune health, it is yet known whether good PSQ provides any protection against the negative effects of sleep restriction on URTI incidence. Subjective assessment of sleep quality, e.g., PSQ using a four point Likert scale, scored from one very poor to four very good (Prather et al., 2012), relates to objectively assessed sleep parameters, including sleep onset latency and sleep continuity (Akerstedt et al., 1994, 1997). The idea of sleep quality being just as important, if not more important than sleep duration in optimising immune health is commonly overlooked (Bin, 2016), however, evidence supports the notion of health benefits in those reporting good rather than poor PSQ e.g., to cardiovascular and immune health (Cribbet et al., 2014; Hoevenaar-Blom et al., 2011). Whilst the mechanism(s) for a purported beneficial effect of good PSQ on immune health requires elucidation, good PSQ has been associated with more restorative slow wave sleep (SWS) (Akerstedt et al.), considered the most relevant sleep stage in mediating the effect of sleep on the immune system (Besedovsky et al., 2019). Accordingly, SWS activity the night after hepatitis A vaccination was predictive of the antibody response; and reductions in circulating cortisol and increases in growth hormone and prolactin during SWS correlated strongly with the expression of antigen-specific CD4 T cells measured up to one year after vaccination (Lange et al., 2011). Analogous are study findings showing that poor PSQ is associated with disrupted circadian cortisol rhythm (Backhaus, Junghanns, & Hohagen, 2004; Lasikiewicz, Hendrickx, Talbot, & Dye) with likely consequences including dysregulated inflammation and immunity and associated poor health outcomes (Adam et al., 2017; Sephton et al., 2013). From a practical standpoint, should good PSQ provide protection against URTI during sleep restriction, it is encouraging to note that non-pharmacological interventions can improve PSQ (Irwin, Olmstead, & Motivala, 2008) and promote SWS (Beck, Loretz, & Rasch).

With this information in mind, in a cohort of 1,318 healthy young males and females entering basic military training, we prospectively examined the association between sleep restriction (an individualised reduction in sleep duration of ≥2 hours/night compared with civilian life (Vgontzas et al.), PSQ and URTI susceptibility. We hypothesised that sleep restriction would

be associated with increased URTI susceptibility, and that good PSQ would afford some protection against URTI in those with sleep restriction.

6.3 Methods

6.3.1 Study design and participants

The data presented herein were collected as part of a prospective, observational program of research investigating immune health and physical performance in British Army recruits undergoing phase one training; the findings from which have been presented elsewhere (Carswell et al., 2018; Harrison et al., 2021; Kashi et al., 2021; Wentz et al., 2018). Here we present previously unpublished findings showing the protective effect of good PSQ on URTI susceptibility during sleep restriction.

A total of $N = 1{,}546$ civilian males and females, ≥18 years of age, entering British Army Phase One Training were assessed for eligibility between January 2014 and November 2015; males were located at the Infantry Training Centre Catterick, UK, and females were located at the Army Training Centre, Pirbright, UK (**Figure 7** & **Table 11**). British Army Phase One Training follows a 12-week syllabus of fundamental military skills including physical training, weapon handling, map reading and fieldcraft. Phase One Training takes place under controlled living and working conditions: recruits reside on the military base in shared living accommodation (up to 12 per room), they all perform largely the same daily activities in groups and eat their meals at a military catering facility, all whilst under the close supervision of their superiors. With the exception of discharge for medical or welfare reasons in Week-1, recruits remain enrolled on the training course, residing on the military base, for at least the first six weeks **Figure 7**. After this time weekend leave is permitted and recruits are afforded the opportunity to voluntarily discharge from training. During the busy daily schedule there is scant opportunity for napping and during the evenings there is little opportunity for down time; recruits are required to prioritise this time to complete tasks such as cleaning, ironing, and paperwork over relaxing and using mobile phones. Notwithstanding the rigorous control over day-to-day living and working conditions, including the requirement to arise relatively early in the morning (0500–0630 h), recruits are afforded considerable flexibility over the time they typically retire to bed at night (2100–0100 h). Sleep during civilian life and military training is characterised in **Table 12**.

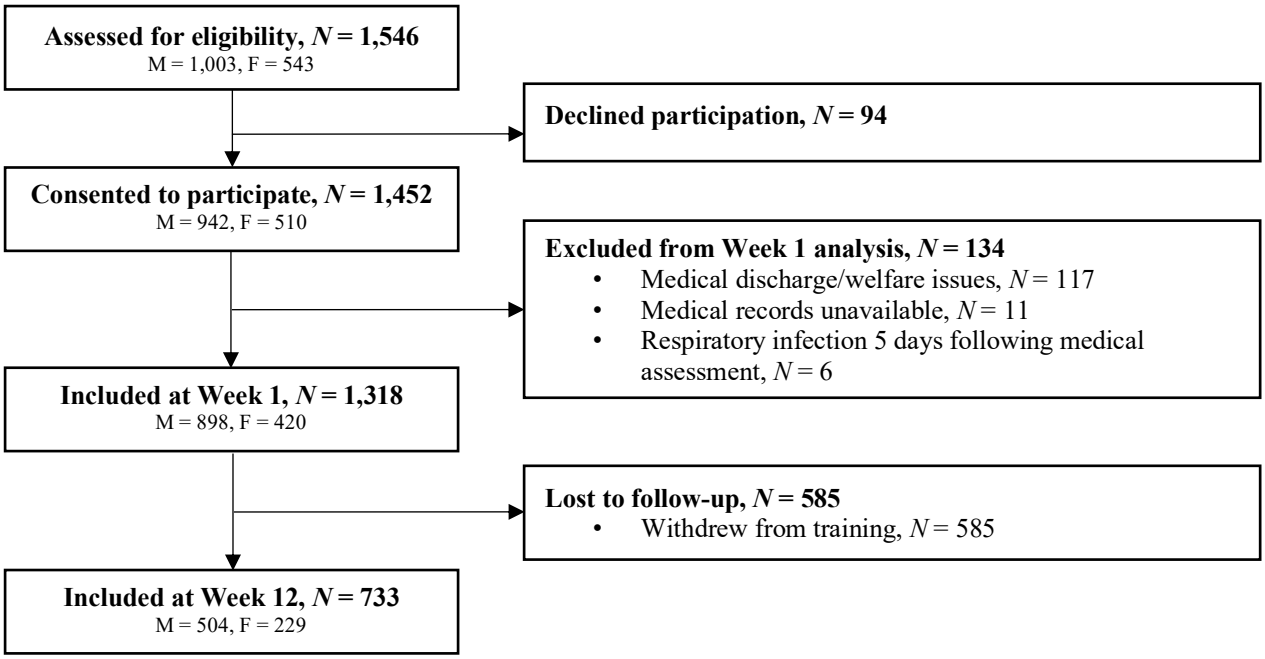

Figure 7. *Flowchart outlining recruits assessed for eligibility, consented and available at the start and end of the training course.*

6.3.2 Procedures

A total of *N* = 1,452 healthy males and females provided written informed consent to participate in the study before completing a medical assessment with a physician either the day after arrival at the military base or the following day. Medical reasons for exclusion from training included sleep and psychiatric disorders, chronic lung diseases and symptoms or treatment for asthma in the past year. Demographic and lifestyle data were collected at the medical at the start of training, and sleep assessments were made at the start and end of training; participant flow is summarised in Figure 1. Ethical approval was obtained from the UK, Ministry of Defence Research Ethics Committee (reference: 165/Gen/10), with all protocols conducted in accordance with the 2013 Declaration of Helsinki.

At the medical assessment, recruits participating in the study were asked to report the time they went to sleep and woke up on a normal night during civilian life and the night prior to the medical assessment. Recruits also provided a PSQ rating for the night before the medical assessment using a four point Likert scale, where 1 = very poor, 2 = poor, 3 = good and 4 = very good, as previously described (Prather et al.). To account for an association between disturbed mood and URTI (Falagas et al., 2010), mood in the past week was assessed at the start of training using the Brunel Mood Scale, which measures five negative mood states

(anger, tension, confusion, depression and fatigue) and one positive mood state (vigour), as previously described (Lewis, Annett, Davenport, Hall, & Lovatt, 2016). In Week-12, recruits were asked to report the time they went to sleep and woke up on a normal night during training and the time they went to sleep and woke up the previous night with a corresponding PSQ rating.

6.3.3 Outcomes

Recruits presenting with URTI symptoms during training reported to the medical facility for a physician consultation. At the end of the training course, participant medical records were accessed by a single independent physician to obtain diagnosed URTIs and associated lost training days due to URTI. The physician's assessment at the Week-1 medical that the recruit was in good health, ready to commence training, mitigates the risk of reverse causation (i.e., ongoing respiratory illness influencing the Week-1 sleep assessment). Additionally, at the statistical analysis stage, we excluded physician-diagnosed URTIs that occurred in the five days following the Week-1 sleep assessment; a step taken because subjective sleep is typically affected in the first few days of a URTI (Lasselin et al., 2019). There were no cases of physician diagnosed URTI in the five days before the Week-12 sleep assessment.

6.3.4 Statistical analysis

All analyses were conducted using SPSS 28.0 (IBM, Armonk, NY, USA) with statistical significance set at $P < 0.05$. Sleep restriction was defined as an individualised reduction in sleep duration of ≥2 hours/night compared with civilian life; studies have chosen two hours as a modest threshold for sleep restriction (Cabeza de Baca et al., 2019; Vgontzas et al., 2004). As a large proportion of participants reported PSQ ratings of 2 or 3 (81%), a dichotomous grouping was created whereby scores of 1 and 2 were combined to represent 'poor' PSQ and scores of 3 and 4 were combined to represent 'good' PSQ. Participant demographic, lifestyle and sleep data are presented as mean ± SD for continuous variables or absolute numbers and percentages for categorical variables; comparisons were made using independent t-tests, one-way analysis of variance and Chi-square, where appropriate (**Tables 9, 10, 11** and **13**).

A small proportion of data were missing, assumed missing at random, with 16% ($N = 211$) of recruits missing one or more data points e.g., due to administrative issues with questionnaires. To avoid a potential bias due to excluding incomplete cases, and to maximise statistical power, multiple imputation was carried out using predictive mean matching (50 iterations and 40

imputed datasets), in line with recommendations (Galli, Jones, Larson, Basner, & Dinges, 2022; Graham, 2009). All variables were included in multiple imputation, including the outcome variable URTI (Graham, 2009). The small number of recruits missing medical records (N = 11 with no URTI data, **Figure 7**) were excluded from subsequent statistical analyses, as recommended (von Hippel, 2007). Similar demographic and sleep characteristics are shown for complete cases (N = 1,107) and the dataset following multiple imputation (N = 1,318; **Table 12**).

Hierarchical logistic regression was used to examine the moderating effect of PSQ on the association between sleep restriction and URTI susceptibility. After checking assumptions, step 1 examined whether URTI susceptibility was predicted from the independent main effects of sleep restriction and PSQ, followed by step 2 examining the interaction effect of sleep restriction and PSQ. Logistic regression was also used to examine the combined influence of sleep restriction and PSQ on URTI susceptibility; whereby, recruits classified as non-sleep restricted who reported good PSQ were considered the reference group (**Figure 9A–B** and **Table 16**). Separate analyses were used to predict URTI occurring during the first four weeks of training and across the full 12-Week training course. The first four weeks of training was chosen as an additional period of interest because almost half of all URTI's occurred during this period after recruits, traveling from various locations in the United Kingdom, Republic of Ireland and Commonwealth Nations, moved into shared living accommodation (i.e., at a time of heightened pathogen exposure); in addition, recruits were still enrolled in training and remained on the military base during the first four weeks. To account for a selection bias due to loss to follow up, logistic regression was also used to predict URTI cases occurring across the full 12-Week training course in the N = 733 who completed training. This population also provided the opportunity to predict URTI susceptibility using logistic regression in a comparative analysis of groups reporting enduring sleep measures e.g., those reporting poor PSQ at both the start and end of training *vs* good PSQ at the start and end of training. For each logistic regression analysis, after checking assumptions, model 1 investigated the unadjusted association with URTI. Adjustment for covariates (likely URTI risk factors) was made as follows: model 2 was additionally adjusted for sex (Falagas et al., 2007) and BMI (Harpsoe et al., 2016); model 3 included model 2, plus adjustment for smoking (Arcavi & Benowitz, 2004); model 4 included model 3, plus adjustment for season (Heikkinen & Jarvinen, 2003); model 5 included model 4, plus adjustment for total mood disturbance (Falagas et al., 2010); model 6 included model 5, plus adjustment for long civilian sleep (> 10 h per night) (Hirshkowitz,

Whiton, Albert, Alessi, Bruni, DonCarlos, Hazen, Herman, Katz, et al., 2015); model 7 included model 5, plus adjustment for short civilian sleep (< 6 h per night) (Hirshkowitz, Whiton, Albert, Alessi, Bruni, DonCarlos, Hazen, Herman, Katz, et al., 2015); and model 8 included all previous covariates (fully adjusted model).

6.4 Results

6.4.1 Sample characteristics, sleep and URTI incidence

Demographic and lifestyle data for the *N* = 1,318 recruits who commenced training and the *N* = 733 who completed training are presented in **Table 11** and **Table 15**, and sleep characteristics are presented in **Table 12**. During civilian life, average sleep duration was 8.5 ± 1.5 h (**Table 12**) and the proportion of short sleepers (3%, < 6 h) and long sleepers (11%, > 10 h) was comparable to a representative sample (Lo, Leong, Loh, Dijk, & Chee, 2014). Accounting for selection bias due to loss to follow-up, demographic, lifestyle, and sleep measures were comparable in those who completed the course and those lost to follow-up (**Table 13**). Moreover, recruits with sleep restriction were no less likely to complete training and the proportion of recruits completing training did not differ across the sleep restriction and PSQ classifications (**Table 11**).

During civilian life, average sleep duration was 8.5 ± 1.5 h, lowering to 6.4 ± 0.8 h at the start of training. Sleep duration, when measured during civilian life or at the start of training, was not associated with URTI incidence (civilian life: OR = 1.13, 95% CI 0.98–1.31, *P* = 0.081; start of training: OR = 0.93, 95% CI 0.71–1.22, *P* = 0.601). At the start of training, approximately half of all recruits were sleep restricted compared with civilian life (52%; sleep restriction: 2.1 ± 1.6 h; **Table 12**) and 62% reported good PSQ. Recruits with sleep restriction (*vs* no sleep restriction) slept longer during civilian life, owing to later morning awakening, and slept less at the start of training (both *P* < 0.001; **Table 12**). Despite the sleep debt, at the start of training over half of recruits with sleep restriction reported good PSQ (58% *vs* 67% for no sleep restriction). Compared with the start, recruits experienced less sleep restriction during training (1.4 ± 1.3 h, *P* < 0.001; **Table 12**); nevertheless, sleep restriction classification tended to be consistent as 93% of recruits with no sleep restriction at the start of training experienced no sleep restriction during training and 71% with sleep restriction at the start endured sleep restriction. The proportion of recruits with a consistent PSQ classification at both the start and end of training was 71% for good and 43% for poor. A total of 93 physician diagnosed URTI

episodes were recorded during training, of which 47% occurred during the first four weeks. Each URTI episode resulted in 3.3 ± 3.7 lost training days.

Table 11. *Descriptive information in the population that commenced training.*

	Total Sample	**No Sleep Restriction**			**Sleep Restriction**		
		All	Good PSQ	Poor PSQ	All	Good PSQ	Poor PSQ
Population that commenced training	***N* = 1,318**	***N* = 628**	***N* = 420**	***N* = 208**	***N* = 690**	***N* = 400**	***N* = 290**
Demographic and lifestyle							
Age	22 ± 3	22 ± 3	22 ± 3	22 ± 3	21 ± 3^{bb}	21 ± 3^{aa}	22 ± 3^{bcc}
Sex, Male [*N* (%)]	898 (68)	500 (80)	342 (81)	158 (76)	398 (58)^{bb}	262 (66)^{aa}	136 (47)^{aabbcc}
Sex, Female [*N* (%)]	420 (32)	128 (20)	78 (19)	50 (24)	292 (42)^{bb}	138 (35)^{aa}	154 (53)^{aabbcc}
Ethnicity, White [*N* (%)]	1253 (95)	594 (95)	398 (95)	196 (94)	659 (96)	380 (95)	279 (96)
BMI (kg/m^2)	23.9 ± 2.7	24.1 ± 2.7	24.1 ± 2.8	24.2 ± 2.7	23.7± 2.6^{bb}	23.8 ± 2.8	23.6 ± 2.4
Smoker [*N* (%)]	777 (59)	397 (63)	279 (66)	118 (57)^{a}	380 (55)^{bb}	235 (59)^{a}	145 (50)^{aac}
Season of enrolment							
Winter [*N* (%)]	280 (21)	144 (23)	77 (18)	67 (32)	136 (20)	82 (21)	54 (19)
Spring [*N* (%)]	214 (16)	87 (14)	55 (13)	32 (15)	127 (18)	65 (16)	62 (21)
Summer [*N* (%)]	416 (32)	214 (34)	152 (36)	62 (30)	202 (29)	123 (31)	79 (27)
Fall [*N* (%)]	408 (31)	183 (29)	136 (33)	47 (23)	225 (33)	130 (32)	95 (33)
Total mood disturbance	5 ± 9	3 ± 8	2 ± 8	6 ± 9^{aa}	6 ± 9^{bb}	4 ± 9^{aa}	8 ± 10^{aacc}
Completed training [N (%)]	733 (56)	365 (58)	248 (59)	117 (56)	368 (53)	218 (55)	150 (52)

Values presented as mean ± SD unless otherwise stated. PSQ = perceived sleep quality; BMI = body mass index. Sleep restriction is defined as an individualised reduction in sleep duration of ≥ 2 hours from civilian life. ^{a} = significantly different to no sleep restriction Good PSQ; ^{b} = significantly different to equivalent no sleep restriction group. ^{c} = significantly different to sleep restriction Good PSQ. Single letter denotes $P < 0.05$ (e.g., ^{a}); double letter denotes $P < 0.01$ (e.g., ^{aa}).

Table 12. *Sleep characteristics in the population that commenced and completed training.*

	Total Sample	No Sleep Restriction			Sleep Restriction		
		All	Good PSQ	Poor PSQ	All	Good PSQ	Poor PSQ
Population that commenced training	***N* = 1,318**	***N* = 628**	***N* = 420**	***N* = 208**	***N* = 690**	***N* = 400**	***N* = 290**
Civilian life							
Duration (h)	8.5 ± 1.5	7.3 ± 1.0	7.3 ± 1.0	7.4 ± 1.1	9.5 ± 1.1^{bb}	9.6 ± 1.1^{aa}	9.4 ± 1.2^{aabb}
Bed/waketime	23:24–07:56	23:22–06:36	23:21–06:36	23:26–06:35	23:26–09:04	23:27–09:11	23:25–08:53
Start of training							
Duration (h)	6.4 ± 0.8	6.6 ± 0.7	6.7 ± 0.6	6.4 ± 0.7^{aa}	6.2 ± 0.8^{bb}	6.4 ± 0.6^{aa}	6.0 ± 0.9^{aabbcc}
Bed/waketime	22:56–05:23	22:46–05:26	22:43–05:27	22:55–05:24	23:05–05:21	22:54–05:21	23:15–05:15
Sleep restriction (h)	2.1 ± 1.6	0.7 ± 1.0	0.6 ± 1.0	1.0 ± 0.9^{aa}	3.3 ± 1.1^{bb}	3.2 ± 1.0^{aa}	3.4 ± 1.2^{aabb}
Population that completed training	***N* = 733**	***N* = 365**	***N* = 248**	***N* = 117**	***N* = 368**	***N* = 218**	***N* = 150**
Civilian life							
Duration (h)	8.4 ± 1.6	7.3 ± 1.0	7.3 ± 0.9	7.2 ± 1.2	9.6 ± 1.1^{bb}	9.7 ± 1.0^{aa}	9.4 ± 1.2^{aabb}
Bed/waketime	23:21–07:50	23:21–06:34	23:16–06:29	23:32–06:45	23:21–08:56	23:27–09:10	23:11–08:36
Start of training							
Duration (h)	6.4 ± 0.8	6.6 ± 0.7	6.7 ± 0.6	6.4 ± 0.7^{a}	6.2 ± 0.9^{bb}	6.4 ± 0.7^{aa}	6.0 ± 1.0^{aabbcc}
Bed/waketime	22:58–05:24	22:47–05:25	22:45–05:25	22:53–05:25	23:07–05:22	22:55–05:20	23:25–05:25
Sleep restriction (h)	2.0 ± 1.7	0.7 ± 0.9	0.6 ± 0.9	0.8 ± 1.0	3.4 ± 1.1^{bb}	3.3 ± 1.0^{aa}	3.4 ± 1.2^{aabb}
During training							
Duration (h)	7.0 ± 0.8	7.0 ± 0.7	7.0 ± 0.7	7.1 ± 0.9	7.0 ± 0.9	7.0 ± 0.8	7.0 ± 1.0
Bed/waketime	22:31–05:33	22:31–05:33	22:33–05:32	22:28–05:35	22:31–05:34	22:32–05:34	22:30–05:33
Sleep restriction (h)	1.4 ± 1.3	0.3 ± 1.1	0.3 ± 1.1	0.1 ± 1.3	2.6 ± 1.3^{bb}	2.7 ± 1.3^{aa}	2.4 ± 1.3^{aabb}
End of training							
Duration (h)	7.0 ± 1.2	6.9 ± 1.1	6.9 ± 1.2	7.0 ± 1.1	7.1 ± 1.3^{b}	7.0 ± 1.2	7.3 ± 1.3^{a}
Bed/waketime	22:51–05:58	22:53–05:51	22:52–05:47	22:57–06:01	22:54–06:04	22:54–05:55	22:55–06:16
Sleep restriction (h)	1.4 ± 1.9	0.4 ± 1.4	0.4 ± 1.3	0.2 ± 1.4	2.5 ± 1.7^{bb}	2.7 ± 1.6^{aa}	2.1 ± 1.8^{aabbcc}

Values presented as mean ± SD unless otherwise stated. PSQ = perceived sleep quality. Sleep restriction is defined as an individualised reduction in sleep duration of ≥ 2 hours from civilian life. ^{a} = significantly different to no sleep restriction Good PSQ; ^{b} = significantly different to equivalent no sleep restriction group. ^{c} = significantly different to sleep restriction Good PSQ. Single letter denotes $P < 0.05$ (e.g., ^{a}); double letter denotes $P < 0.01$ (e.g., ^{aa}).

6.4.2 Sleep restriction is independently associated with URTI

Unadjusted regression analysis showed that sleep restriction was independently associated with increased URTI susceptibility during the first four weeks of training (**Figure 8A**) and across the full 12-Weeks (model 1: OR = 2.99, 95% CI 1.35–6.65, P = 0.007). This association remained in the fully adjusted model accounting for sex, BMI, smoking, season, mood disturbance and long and short civilian sleep duration (model 8: OR = 2.93, 1.29–6.69, P = 0.011). Associations between sleep restriction and URTI across the full 12-Week course also remained in both unadjusted (**Figure 8B**) and adjusted models in recruits who completed training, accounting for a selection bias due to loss to follow-up (model 8: OR = 3.41, 1.09–10.67, P = 0.032). Moreover, those with sleep restriction at both the start and during training were more likely to suffer URTI (model 1: OR = 1.80, 95% CI 0.97–3.35, albeit P = 0.062). Further analysis using sleep restriction on a continuous scale provided associations with increased URTI susceptibility (first 4 weeks: OR = 1.21, 95% CI 1.00–1.47, P = 0.048; full 12-Weeks: OR = 1.16, 95% CI 1.00–1.33, P = 0.045). Poor PSQ at the start of training was not significantly associated with URTI; however, recruits with poor PSQ at both the start and end of training were more likely to suffer URTI across the full 12-Week course (model 8: OR = 3.16, 95% CI 1.31–7.61, P = 0.010).

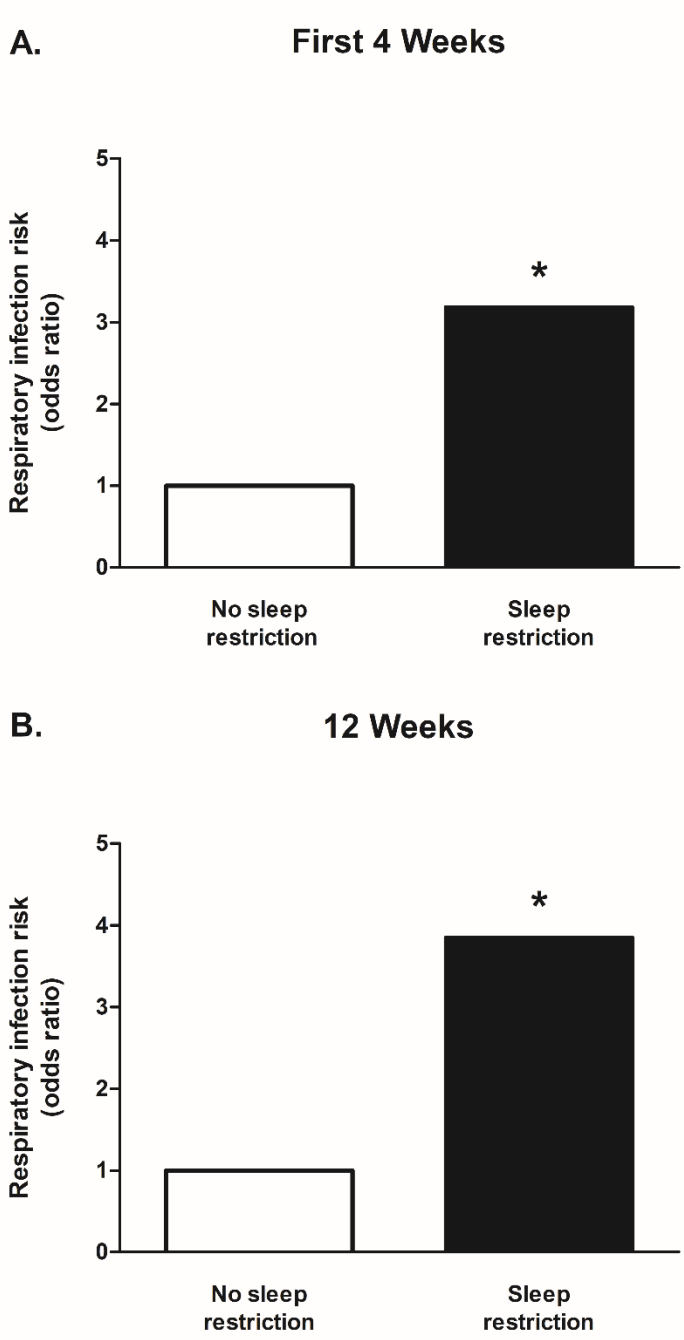

Figure 8. *The influence of sleep restriction on URTI susceptibility during the first four weeks of military training (A), and across the 12-week training course in recruits who completed training (B). Sleep restriction is defined as an individualised reduction in sleep duration of ≥ 2 hours from civilian life. Odds ratios presented are from unadjusted analyses. No sleep restriction is considered as reference. URTI = upper respiratory tract infection. * P < 0.05 vs no sleep restriction. These associations persisted in the fully adjusted model accounting for sex, BMI, smoking, season, mood disturbance and long and short civilian sleep duration.*

6.4.3 Good PSQ protects against the raised risk of URTI during sleep restriction

Hierarchical logistic regression showed that PSQ significantly moderated the association between sleep restriction and URTI susceptibility across the full 12 weeks (model 1: B = -1.12, SE 0.50, P = 0.023), whereby the association between sleep restriction and a raised risk of URTI was only observed in those with poor PSQ but not good PSQ. This moderating influence remained in the fully adjusted model accounting for sex, BMI, smoking, season, mood disturbance and long and short civilian sleep (model 8: B = -1.13, SE 0.50, P = 0.025). A moderating effect of PSQ on the association between sleep restriction and URTI susceptibility was also observed in the population who completed the training course, accounting for loss to follow-up (model 1: B = -1.28, SE 0.69, albeit P = 0.061).

Further analysis examined the combined influence of sleep restriction and PSQ on URTI susceptibility; with recruits who did not experience sleep restriction and reported good PSQ as the reference group (**Figure 9A-B** and **Table 16**). Fully adjusted regression showed that recruits with no sleep restriction who reported poor PSQ at the start of training were at no greater risk of URTI in either the first four weeks (**Figure 9A**) or the full 12-Week course in those who completed training (model 8: OR = 0.65, 95% CI 0.20–2.04, P = 0.455; **Table 16**). In contrast, recruits with sleep restriction who reported poor PSQ at the start of training were twice as likely to suffer URTI during the first four weeks of training (**Figure 9A**) and across the full 12-Week course in those who completed training (model 1: OR = 2.12, 95% CI 1.03–4.34, P = 0.040; **Figure 9B** and **Table 16**). Despite a similar magnitude of sleep restriction in recruits reporting poor and good PSQ (poor 2.4 ± 1.3 h and good 2.7 ± 1.3 h during training; P = 0.152; **Table 12**), recruits with sleep restriction who reported good PSQ were at no greater risk of URTI in either the first four weeks or across the full 12-Week course in those who completed training (model 8: OR = 1.00, 95% CI 0.45–2.20, P = 0.999; **Figure 9B** and **Table 16**). In summary, the independent association between sleep restriction and URTI was driven by recruits reporting poor PSQ; recruits with sleep restriction reporting good PSQ were at no greater risk of URTI compared with the reference group.

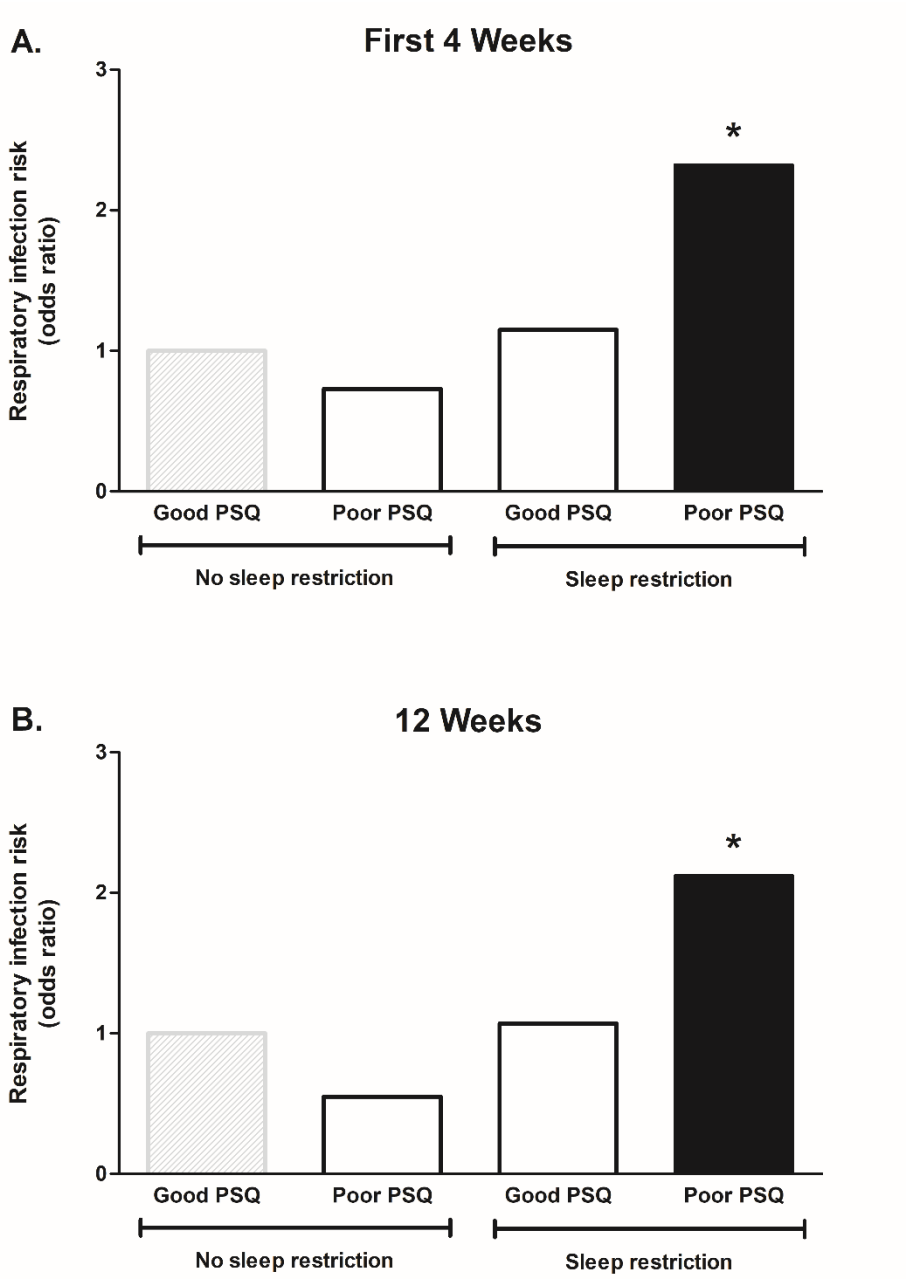

Figure 9. *The combined influence of sleep restriction and PSQ on URTI susceptibility during the first four weeks of military training (A), and across the 12-week training course in recruits who completed training (B). Sleep restriction is defined as an individualised reduction in sleep duration of ≥ 2 hours from civilian life. Odds ratios presented are from unadjusted analyses. No sleep restriction and good PSQ considered as reference (grey shaded bar). URTI = upper respiratory tract infection; PSQ = perceived sleep quality. * P < 0.05 vs reference group. These associations persisted in the fully adjusted model accounting for sex, BMI, smoking, season, mood disturbance and long and short civilian sleep duration.*

Table 13. *Descriptive information in the populations that completed training and were lost to follow-up.*

	Completed training	**Lost to follow-up**
	***N* = 733**	***N* = 585**
Demographic and lifestyle		
Age	22 ± 3	21 ± 3
Sex, Male [*N* (%)]	504 (69)	394 (67)
Sex, Female [*N* (%)]	229 (31)	191 (33)
Ethnicity, White [*N* (%)]	699 (95)	554 (95)
BMI (kg/m^2)	24.0 ± 2.7	23.9 ± 2.7
Smoker [*N* (%)]	433 (59)	344 (59)
Season of enrolment		
Winter [*N* (%)]	127 (17)	153 (26)†
Spring [*N* (%)]	137 (19)	77 (13)†
Summer [*N* (%)]	189 (26)	227 (39)†
Fall [*N* (%)]	280 (38)	128 (22)†
Total mood disturbance	4 ± 8	5 ± 10†
Sleep measures		
Sleep duration during civilian life (h)	8.4 ± 1.6	8.5 ± 1.4
Bed/waketime	23:21–07:50	23:29–08:04
Sleep duration at the start of training (h)	6.4 ± 0.8	6.4 ± 0.7
Bed/waketime	22:58–05:24	22:54–05:23
Sleep restriction at the start of training (h)	2.0 ± 1.7	2.1 ± 1.5
Sleep quality, Good [*N* (%)]	466 (64)	354 (61)
Sleep quality, Poor [*N* (%)]	267 (36)	231 (39)

Values presented as mean ± SD unless otherwise stated. BMI = body mass index. Sleep restriction is defined as an individualised reduction in sleep duration from civilian life. $^{\dagger}P < 0.05$ *vs* the population that completed training.

Table 14. *Descriptive information in complete cases and in the total sample following multiple imputation.*

	Complete Cases	Total Sample
	***N* = 1,107**	***N* = 1,318**
Demographic and lifestyle		
Age	22 ± 3	22 ± 3
Sex, Male [*N* (%)]	760 (69)	898 (68)
Sex, Female [*N* (%)]	347 (31)	420 (32)
Ethnicity, White [*N* (%)]	1051 (95)	1253 (95)
BMI (kg/m^2)	23.9 ± 2.7	23.9 ± 2.7
Smoker [*N* (%)]	642 (58)	777 (59)
Season of enrolment		
Winter [*N* (%)]	129 (12)	280 (21)
Spring [*N* (%)]	191 (17)	214 (16)
Summer [*N* (%)]	392 (35)	416 (32)
Fall [*N* (%)]	395 (36)	408 (31)
Total mood disturbance	4 ± 10	5 ± 9
Sleep measures		
Sleep duration during civilian life (h)	8.5 ± 1.6	8.5 ± 1.5
Sleep duration at the start of training (h)	6.4 ± 0.8	6.4 ± 0.8
Sleep restriction at the start of training (h)	2.1 ± 1.7	2.1 ± 1.6
Sleep quality, Good [*N* (%)]	718 (65)	820 (62)
Sleep quality, Poor [*N* (%)]	389 (35)	498 (38)

Values presented as mean ± SD unless otherwise stated. BMI = body mass index. Sleep restriction is defined as an individualised reduction in sleep duration from civilian life.

Table 15. *Descriptive information in the population that completed training.*

	Total Sample	**No Sleep Restriction**			**Sleep Restriction**		
		All	Good PSQ	Poor PSQ	All	Good PSQ	Poor PSQ
Population that completed training	***N* = 733**	***N* = 365**	***N* = 248**	***N* = 117**	***N* = 368**	***N* = 218**	***N* = 150**
Demographic and lifestyle							
Age	22 ± 3	22 ± 3	22 ± 3	22 ± 3	21 ± 3[bb]	21 ± 3[aa]	22 ± 3
Sex, Male [*N* (%)]	504 (69)	284 (78)	201 (81)	83 (71)[a]	220 (60)[bb]	152 (70)[aa]	68 (45)[aabbcc]
Sex, Female [*N* (%)]	229 (31)	81 (22)	47 (19)	34 (29)[a]	148 (40)[bb]	66 (30)[aa]	82 (55)[aabbcc]
Ethnicity, White [*N* (%)]	699 (95)	343 (94)	233 (94)	110 (94)	356 (97)	209 (96)	147 (98)
BMI (kg/m^2)	24.0 ± 2.7	24.1 ± 2.8	24.1 ± 2.9	24.3 ± 2.7	23.8 ± 2.6	23.9 ± 2.6	23.7 ± 2.5
Smoker [*N* (%)]	433 (59)	219 (60)	159 (64)	60 (51)[a]	214 (58)	135 (62)	79 (53)[a]
Season of enrolment							
Winter [*N* (%)]	127 (17)	72 (20)	43 (17)	29 (25)	55 (15)	35 (16)	20 (13)[b]
Spring [*N* (%)]	137 (19)	64 (18)	39 (16)	25 (21)	73 (20)	42 (19)	31 (21)
Summer [*N* (%)]	189 (26)	100 (27)	71 (29)	29 (25)	89 (24)	53 (24)	36 (24)
Fall [*N* (%)]	280 (38)	129 (35)	95 (38)	34 (29)	151 (41)	88 (41)	63 (42)[b]
Total mood disturbance	4 ± 8	3 ± 8	2 ± 8	5 ± 8[aa]	5 ± 9[bb]	4 ± 9	7 ± 9[aacc]

Values presented as mean ± SD unless otherwise stated. PSQ = perceived sleep quality; BMI = body mass index. Sleep restriction is defined as an individualised reduction in sleep duration of ≥ 2 hours from civilian life. [a] = significantly different to no sleep restriction Good PSQ; [b] = significantly different to equivalent no sleep restriction group. [c] = significantly different to sleep restriction Good PSQ. Single letter denotes $P < 0.05$ (e.g., [a]); double letter denotes $P < 0.01$ (e.g., [aa]).

Table 16. *Association between sleep restriction, PSQ and URTI during training.*

	No Sleep Restriction		Sleep Restriction	
	Good PSQ	Poor PSQ	Good PSQ	Poor PSQ
Population that commenced training	OR	OR	OR	OR
First 4 weeks				
Model 1: unadjusted (sleep restriction and PSQ)	*Reference*	0.73 (0.23–2.32)	1.15 (0.50–2.64)	2.32 (1.07–5.02)[a]
Model 2: sex + BMI	*Reference*	0.70 (0.22–2.23)	1.06 (0.46–2.46)	1.95 (0.86–4.39)
Model 3: model 2 + smoking	*Reference*	0.73 (0.23–2.34)	1.09 (0.47–2.52)	2.05 (0.91–4.64)
Model 4: model 3 + season	*Reference*	0.74 (0.23–2.39)	1.09 (0.47–2.54)	2.02 (0.89–4.57)
Model 5: model 4 + total mood disturbance	*Reference*	0.74 (0.23–2.38)	1.09 (0.47–2.53)	1.99 (0.87–4.59)
Model 6: model 5 + long sleep	*Reference*	0.74 (0.23–2.38)	1.06 (0.44–2.57)	1.97 (0.84–4.60)
Model 7: model 5 + short sleep	*Reference*	0.77 (0.24–2.51)	1.03 (0.44–2.40)	1.89 (0.82–4.35)
Model 8: model 5 + long + short sleep duration	*Reference*	0.74 (0.23–2.39)	1.11 (0.47–2.63)	2.02 (0.87–4.68)
12 weeks				
Model 1: unadjusted (sleep restriction and PSQ)	*Reference*	0.56 (0.25–1.25)	0.97 (0.56–1.69)	1.68 (0.98–2.86)
Model 2: sex + BMI	*Reference*	0.55 (0.24–1.22)	0.95 (0.55–1.67)	1.59 (0.91–2.80)
Model 3: model 2 + smoking	*Reference*	0.56 (0.25–1.26)	0.97 (0.55–1.70)	1.65 (0.94–2.90)
Model 4: model 3 + season	*Reference*	0.56 (0.25–1.26)	0.97 (0.55–1.70)	1.66 (0.94–2.93)
Model 5: model 4 + total mood disturbance	*Reference*	0.57 (0.25–1.29)	0.98 (0.56–1.73)	1.71 (0.96–3.06)
Model 6: model 5 + long sleep	*Reference*	0.57 (0.25–1.30)	0.91 (0.50–1.65)	1.63 (0.90–2.95)
Model 7: model 5 + short sleep	*Reference*	0.57 (0.25–1.30)	0.98 (0.55–1.73)	1.71 (0.95–3.07)
Model 8: model 5 + long + short sleep duration	*Reference*	0.57 (0.25–1.29)	0.94 (0.53–1.67)	1.67 (0.93–3.00)
Population that completed training				
12 weeks				
Model 1: unadjusted (sleep restriction and PSQ)	*Reference*	0.55 (0.18–1.70)	1.07 (0.50–2.26)	2.12 (1.03–4.34)[a]
Model 2: sex + BMI	*Reference*	0.54 (0.17–1.66)	1.07 (0.50–2.28)	2.11 (0.99–4.48)
Model 3: model 2 + smoking	*Reference*	0.58 (0.19–1.80)	1.07 (0.50–2.29)	2.19 (1.02–4.67)[a]
Model 4: model 3 + season	*Reference*	0.64 (0.20–2.00)	1.04 (0.48–2.23)	2.19 (1.02–4.72)[a]
Model 5: model 4 + total mood disturbance	*Reference*	0.65 (0.21–2.05)	1.05 (0.49–2.26)	2.26 (1.03–4.96)[a]
Model 6: model 5 + long sleep	*Reference*	0.65 (0.21–2.06)	0.97 (0.43–2.18)	2.15 (0.96–4.79)
Model 7: model 5 + short sleep	*Reference*	0.65 (0.21–2.07)	1.04 (0.48–2.25)	2.24 (1.02–4.95)[a]
Model 8: model 5 + long + short sleep duration	*Reference*	0.65 (0.20–2.04)	1.00 (0.45–2.20)	2.20 (0.99–4.86)

PSQ = perceived sleep quality; URTI = upper respiratory tract infection; BMI = body mass index. Sleep restriction is defined as an individualised reduction in sleep duration of ≥ 2 hours from civilian life. Long sleep was defined as >10 hours civilian sleep duration. Short sleep was defined as <6 hours civilian sleep duration. [a] = $P < 0.05$.

6.5 Discussion

Meeting a one-size-fits-all recommendation for sleep duration (e.g., 7–9 hours' sleep each night (Hirshkowitz, Whiton, Albert, Alessi, Bruni, DonCarlos, Hazen, Herman, Katz, et al., 2015) is unlikely necessary for all adults: individual sleep needs are likely an important consideration for optimal health (Besedovsky et al., 2019; Bin, 2016). Empirical research shows that adults who habitually fall short of the recommended 7–9 hours' sleep each night are more susceptible to respiratory infections (Cohen et al., 2009; Patel et al., 2012; Prather et al., 2015), but the influence of restricting habitual sleep, now commonplace in modern society (e.g., rising early for work), and PSQ on respiratory infection incidence remains unknown. To this end, here we prospectively examined the association between sleep restriction, an individualised reduction in sleep duration ≥2 hours (Cabeza de Baca et al., 2019; Vgontzas et al., 2004), PSQ and physician diagnosed URTI in young adult male and female civilians embarking on a 12-Week military training course. Two new and noteworthy findings align with our hypotheses: first, sleep restriction was associated with increased URTI susceptibility during training (**Figure 8A–B**); second, and most notably, the observed association between sleep restriction and URTI was driven by recruits reporting poor PSQ. Fully adjusted regression analyses showed that, compared with recruits with no sleep restriction reporting good PSQ, recruits with sleep restriction at the start of training who reported poor PSQ were twice as likely to suffer URTI during the first four weeks of training, a time of heightened pathogen exposure, and during the full 12-Week course (**Figure 9A–B** and **Table 16**). The likelihood of URTI was not significantly increased in sleep restricted recruits who reported good PSQ, despite a similar magnitude of sleep restriction during training in recruits reporting good and poor PSQ. Notwithstanding the inevitable impact of loss to follow-up reducing the number of respiratory infections available for comparisons, recruits enduring sleep restriction during training were more likely to suffer URTI. Moreover, recruits reporting poor PSQ when healthy at both the start and end of training were more than three times as likely to suffer URTI than recruits consistently reporting good PSQ. These findings show that sleep restriction is associated with increased URTI susceptibility, but only in recruits reporting poor not good PSQ.

The present findings are restricted to the level of association and thus require cautious interpretation. Nonetheless, confidence in their significance is increased by rigorous steps taken including: excluding from analysis recruits with URTI symptoms at the time of or in close proximity to sleep assessments (i.e., mitigating reverse causation); showing consistent effects in recruits who not only completed training (i.e., accounting for selection bias due to loss to

follow up) but also in recruits who endured sleep restriction during training (*vs* no sleep restriction) and in recruits who reported poor (*vs* good) PSQ at both the start and end of training. Regression models were adjusted for URTI risk factors including sex (Falagas et al., 2007), BMI (Harpsoe et al., 2016), smoking (Arcavi & Benowitz, 2004) and season (Heikkinen & Jarvinen, 2003), and further adjustments were made to account for long and short civilian sleep and mood disturbance, not only because these are likely URTI risk factors (Cohen et al., 2009; Falagas et al., 2010; Patel et al., 2012; Robinson et al., 2021) but also because of their known association with poor PSQ (Bin, 2016; Bower et al., 2010; Park et al., 2010; Triantafillou et al., 2019); for example, concordant are the present findings that recruits reporting poor PSQ also reported greater mood disturbance (**Table 11**). Confidence is increased that the observed associations with URTI susceptibility are not driven by mood disturbance because the data show comparable mood disturbance in recruits reporting poor PSQ with and without sleep restriction, yet URTI susceptibility was significantly greater than the reference group only in recruits with sleep restriction reporting poor PSQ (**Figure 9A–B** and **Table 16**).

Candidate mechanisms to explain the observed associations between sleep restriction, PSQ and URTI require elucidation but likely include neuro-endocrine immune modulation (Irwin, 2015) associated with circadian misalignment (Wright et al., 2015), subjective stress (Williams, Magid, & Steptoe, 2005) and/or alterations in sleep architecture (e.g., SWS) (Akerstedt et al., 1997; Besedovsky et al., 2019; Irwin, 2015). In the present study, sleep restriction was largely due to consistently earlier morning awakening during military training than civilian life (**Table 12**), likely resulting in circadian misalignment, particularly for sleep restricted recruits. Our findings tentatively point to another explanation rather than circadian misalignment because bed and wake times during civilian life and military training were comparable in recruits with sleep restriction reporting good and poor PSQ (**Table 12**), yet URTI susceptibility was significantly greater (*vs* the reference group) only in sleep restricted recruits reporting poor PSQ. It is conceivable that sleep restricted recruits reporting poor PSQ experienced increased subjective stress and associated immune perturbations via disrupted HPA axis regulation of diurnal cortisol. Indeed, underground railway staff experienced a greater cortisol awakening response when rising early for a morning shift but only when the early shift coincided with poor PSQ and increased subjective stress (Williams et al., 2005). Further research is warranted to explore these possibilities by adopting recommended measurements of circadian rhythm (e.g., melatonin) (Nguyen & Wright, 2010), subjective stress (e.g., PSS) (Cohen et al., 1983) and HPA activity (e.g., diurnal cortisol) (Adam et al., 2017; Stalder et al., 2016).

We acknowledge that the present study has several limitations. The sample population comprised of young male and female infantry recruits, primarily White and of lower SES (Kiernan et al., 2016), limiting the generalisability of these findings. Whether these findings are generalisable to other populations where sleep restriction is commonplace such as workers rising early for long commutes (Basner, Spaeth, & Dinges, 2014) and new parents (Richter, Kramer, Tang, Montgomery-Downs, & Lemola, 2019) warrants investigation, particularly as low SES has been associated with increased vulnerability to URTI amongst short sleepers (Prather, Janicki-Deverts, Adler, Hall, & Cohen, 2017). Nevertheless, civilians embarking on a military training course affords a unique opportunity to examine the association between sleep restriction, PSQ and URTI in a healthy, pre-screened population under standardised conditions. For example, although we did not assess physical training load and diet, both of which are considered to influence immune health (Bermon et al., 2017; Simpson et al., 2020), these factors are relatively well controlled during military training because recruits perform largely the same daily physical activities and eat their meals from a limited menu at a military catering facility. We acknowledge that sleep was assessed in this study by self-report, e.g., PSQ was assessed using a single self-report item assessing the previous night's sleep quality, and that objective sleep measures using actigraphy or polysomnography would have provided a more comprehensive sleep characterisation. Having said this, self-reported sleep measures capture behaviours or perceptions that individuals can self-monitor, are practical for use in large population studies and relate to objectively assessed sleep (Akerstedt et al., 1994, 1997) and health outcomes (Cabeza de Baca et al., 2019; Cribbet et al., 2014; Hoevenaar-Blom et al., 2011). The present finding that sleep restriction (defined as an individualised $\geq$ 2 h reduction in sleep duration) is associated with increased URTI susceptibility extends beyond those of previous studies, using the same sleep restriction threshold, showing that sleep restriction increased inflammation and cardiovascular disease risk (Cabeza de Baca et al., 2019; Vgontzas et al., 2004); however, given the limited empirical data, research should further investigate the suitability of this sleep restriction threshold for neuro-endocrine immune and other health outcomes. As sleep was assessed only at the start and end of the training course, future research would also benefit from more regular sleep assessments to better characterise variations in sleep restriction and PSQ classifications across the measurement period. For example, although sleep restriction classification tended to be consistent at both time points in the present study, PSQ improved in over half of recruits who reported poor PSQ at the start of training. Despite this, confidence in the observed association between PSQ and URTI is bolstered by analysis

showing that recruits reporting poor PSQ at both the start and end of training were more likely to suffer URTI (*vs* consistently good PSQ). Finally, future studies should confirm the infectious origin of physician diagnosed URTI by performing pathological analysis on throat swabs.

In conclusion, sleep restriction was associated with increased respiratory infection susceptibility during military training. However, the observed association between sleep restriction and respiratory infection was driven by recruits reporting poor PSQ; good PSQ was associated with protection against respiratory infection during sleep restriction. These findings advance our knowledge of how sleep restriction influences immune health, highlighting the need for future studies to account for individual sleep habits and sleep quality. A critical remaining knowledge gap, ripe for further enquiry, is whether improvements in sleep quality arising from behavioural sleep interventions translate to reduced respiratory infection during sleep restriction.

Recommendations for the MOD deriving from this chapter's findings:

1. **Prioritise sleep quality over sleep duration alone in recruit health monitoring.** Approximately half of recruits entering Phase 1 training were sleep restricted, yet those who perceived their sleep as good were at no greater risk of respiratory infection despite losing a comparable amount of sleep. This finding suggests that sleep quality, rather than duration alone, is the more operationally relevant target for intervention, and that monitoring and reporting systems should capture how recruits experience their sleep, not simply how long they sleep.
2. **Develop and deliver targeted sleep hygiene education during Phase 1.** Evidence-based sleep hygiene guidance, covering sleep environment, pre-sleep routines and strategies to improve sleep quality within a constrained sleep window, should be delivered to all recruits during the first week of Phase 1 training. This is particularly important given that recruits have limited control over wake time but retain flexibility over sleep time and pre-sleep behaviours, providing a realistic opportunity for quality improvement even where duration cannot be increased.

CHAPTER 7 – Poor sleep quality is associated with increased respiratory infection, irrespective of sleep duration.

7.1 Abstract

To examine whether sleep quality predicts RTI risk independently of sleep duration, we prospectively studied 995 healthy young adults (male) commencing Phase 1 12-week military training. Sleep quality and duration were assessed using the PSQI in civilian life and during training. Objective sleep was measured using actigraphy. Logistic regression examined associations between sleep measures and physician-diagnosed RTI, adjusted for BMI, smoking, season, training exposure, and perceived stress. In civilian life, mean sleep duration was 8.0 ± 1.5 h, with 24% reporting very poor sleep quality (PSQI ≥ 7). A total of 61 RTIs were recorded during the 12-week training course. Recruits reporting very poor sleep quality in civilian life were twice as likely to suffer RTI during training compared to those with good sleep quality (PSQI < 5) (Model 3: OR = 1.96, 1.05–3.64, $P = 0.034$). Short sleep duration (< 7 h) during civilian life was associated with increased RTI during the first month of training, and this association persisted after full adjustment (Model 3: OR = 2.66, 1.08–6.59, $P = 0.033$); however, good sleep quality provided protection against infection in short sleepers. Very poor sleep quality during the first month of training was associated with increased risk of subsequent RTI in unadjusted analyses (Model 1: OR = 3.28, 1.23–8.76, $P = 0.018$); however, this association was attenuated following adjustment for perceived stress and did not reach significance in the fully adjusted model (Model 3: OR = 2.74, 0.90–8.28, $P = 0.057$). Similarly, recruits with enduring very poor sleep quality across both civilian life and training had a sixfold greater risk of RTI after week 5 in unadjusted analyses (Model 1: OR = 6.72, 1.42–31.87, $P = 0.016$). Recruits who improved their sleep quality were protected against RTI ($P > 0.05$). In conclusion, poor sleep quality, rather than short sleep duration alone, was consistently associated with increased respiratory infection risk.

7.2 Introduction

Sleep plays a crucial role in maintaining overall health and well-being. Indeed, poor or short sleep (<6 or 7 hours/night) is associated with the onset and development of a number of chronic illnesses (Cappuccio et al., 2010a, 2010b; King et al., 2008) and premature mortality (Hammond, 1964; Kripke, Simons, Garfinkel, & Hammond, 1979). As such, The National Sleep Foundation, American Academy of Sleep Medicine, and Sleep Research Society recommend that young adults sleep 7-9 hours per night for health, well-being and optimal neurocognitive function. Despite these recommendations, a significant proportion of the population fails to achieve the recommended sleep duration (Scott et al., 2024), leading to various negative health outcomes. As discussed previously in **Chapter 6**, studies have demonstrated that shorter sleep duration and poor sleep efficiency, assessed both objectively using wrist worn actigraphy and subjectively using sleep diaries, are associated with increased susceptibility to biological verified cold following experimental rhinovirus challenge (Cohen et al., 2009; Prather et al., 2015). Similarly, in a population of UK military recruits, those who reported sleeping <6h per night during training were 4x more likely to be diagnosed with a URTI compared to those sleeping the recommended 7-9h (Wentz et al., 2018).

Building on from **Chapter 6**, the negative consequences of short sleep or restricted sleep are not observed in everyone. This is in part attributable to the vast majority of literature focusing on the absolute influence of sleep duration per se on immune and other health parameters. Recent developments in sleep research have gone further than examining main effects, with our recent publication **(Chapter 6)** highlighting the protective effect of good PSQ during sleep restriction on respiratory infection risk (Walsh et al., 2023). However, it remains unknown whether good PSQ protects against RTI in short sleepers, and whether changes in sleep are associated with RTI risk. This chapter also incorporates objectively measured sleep using actigraphy, to enhance the accuracy of sleep assessment and explore the agreement between objective and subjective sleep measurements.

The potential mechanism(s) for a purported beneficial effect of good PSQ on immune health are discussed in **Chapter 6**.

With this information in mind, in a cohort of 1,188 young athletic adults embarking upon Phase 1 of military training (12-week course), we prospectively investigated whether sleep quality and sleep duration predict RTI risk. We also examined whether changes in sleep are associated

with RTI. We hypothesised short sleepers, and poor sleepers will have a greater risk of RTI. We also hypothesised that improved sleep quality will afford protection against RTI.

7.3 Methods

7.3.1 Study design and participants

Study design and participants are outlined in **Chapter 3, section 3.1 – 3.3.**

7.3.2 Procedures

A total of 1,188 healthy recruits (98% male) provided written informed consent to participate in the study (**Figure 10**). Procedures are detailed in **Chapter 3, section 3.3**.

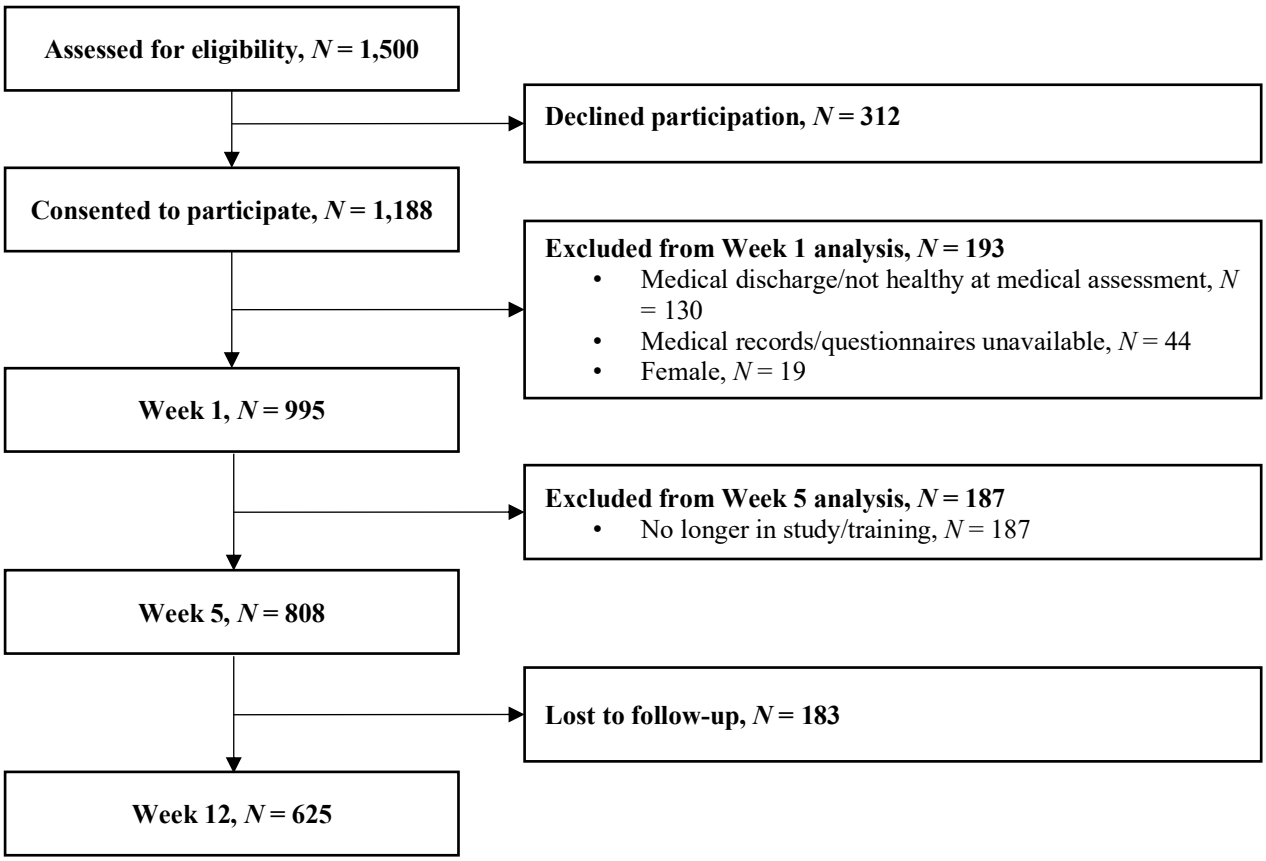

Figure 10. *Flowchart outlining recruits assessed for eligibility, consented and available at the start and end of Phase 1 training. Recruits presenting with serum CRP levels ≥ 10mg/L or common cold symptoms at the medical assessment were classed as 'not healthy' and therefore excluded from analyses. Incomplete data refers to missing demographic, anthropometric or psychological questionnaire data. The Ministry of Defence Research Ethics Committee required female inclusion in study recruitment; however, due to the limited number of females entering training they were excluded from data analysis due to the lack of statistical power.*

Psychosocial and lifestyle measures were assessed in private using online surveys (LimeSurvey, Hamburg, Germany) via an electronic tablet. Sleep quality and duration in the previous month was assessed at week 1 and one-month later with the widely used PSQI (see **Chapter 3, section 3.7** for details) (Buysse et al., 1989). Actigraphy was used to obtain objective measures of sleep at the start of training (see **Chapter 3, section 3.7** for details).

7.3.3 Outcomes

The primary outcome of this study was RTI during military training. RTI diagnosis is described in **Chapter 3, section 3.11**. At the statistical analysis stage, we excluded diagnosed RTIs that occurred in the five days following the week 1 sleep assessment; a step taken because subjective sleep is typically affected in the first few days of a RTI (Lasselin et al., 2019).

7.3.4 Statistical analysis

All analyses were conducted using SPSS 28 (IBM, Armonk, NY, USA) with statistical significance set at $P < 0.05$. Participant demographic, lifestyle and sleep data are presented as mean ± SD for continuous variables or absolute numbers and percentages for categorical variables; comparisons were made using one-way analysis of variance and Chi-square, where appropriate (**Table 17**).

A small proportion of PSQI data at week 5 were missing, assumed missing at random, with 17% (N = 166) of recruits missing PSQI data e.g. due to administrative issues with questionnaires. To avoid a potential bias, multiple imputation was carried out using predictive mean matching (50 iterations and 40 imputed datasets), in line with recommendations (Graham, 2009). After accounting for participants who withdrew from Army training and the study by week 5, PSQI collected at week 1 and all variables collected at week 5 were included in multiple imputation, including the outcome variable RTI incidence.

To examine the association between sleep and RTI, sleep quality and sleep duration were assessed separately as continuous and categorical data. Sleep quality was categorised using global PSQI scores < 5 (good sleep quality), 5 - < 7 and ≥ 7 (poor sleep quality); ≥ 7 is a threshold recognised to identify clinically significant sleep complaints (Buysse et al., 1989). Sleep duration was categorised into < 7 h, 7 – 9 h and > 9 h. Sleep restriction was defined as an individualised reduction in sleep duration of ≥2 hours/night compared with civilian life.

Hierarchical logistic regression analysis was used to examine the independent main effects of sleep duration, sleep quality and sleep restriction on RTI susceptibility. After checking assumptions, we first examined whether RTI susceptibility was predicted from the independent main effects of sleep quality (**Figure 12**), sleep duration (**Figure 13A**), and sleep restriction. We next examined the combined influence of sleep duration and sleep quality on RTI risk; whereby recruits classified as sleeping 7 – 9 h who reported good sleep quality (PSQI ≥ 7) were considered the reference group (**Figure 13B**). Separate analyses were carried out to predict RTI incidence from sleep at Week-1 and sleep at Week-5. For sleep assessed at Week-5, prospective analyses were conducted by isolating RTI incidences occurring after 5 weeks.

Potential confounding factors on the association between poor sleep and RTI include BMI (Murugan & Sharma, 2008), smoking (Arcavi & Benowitz, 2004), season (Heikkinen & Jarvinen, 2003), days spent in training (to account for exposure bias), and perceived stress (Cohen et al., 1991). For each logistic regression analysis, after checking assumptions, Model 1 investigated the unadjusted association with RTI. Model 2 was adjusted for BMI, smoking, season and days spent in training; Model 3 included model 2 plus adjustment for perceived stress.

7.4 Results

7.4.1 Sample characteristics sleep, and RTI incidence

Demographic and lifestyle data for the $N = 995$ recruits who commenced training are presented in **Chapter 4 Table 1**, and sleep characteristics across the training period are presented in **Table 17**. In those who commenced training, civilian life, sleep duration was 8.0 ± 1.6 h, similar to the general young adult population (Oginska & Pokorski, 2006; Steptoe, Peacey, & Wardle, 2006), with the greatest proportion sleeping 7-9 h (62%). Despite this, 49% reported a PSQI global score ≥ 5 and 1 in 4 recruits reported very poor sleep quality determined using a global PSQI score ≥ 7; a clinically relevant threshold previously shown to be associated with sleep disorder patients (Buysse et al., 2007).

During the first month of training, self-reported sleep duration was 6.2 h, a reduction of almost 2 h compared with civilian life (**Table 17**), in line with **Chapter 6**. Despite this sleep restriction, somewhat surprisingly, global sleep quality did not decrease from civilian life to training (**Table 17**). Potential explanations for this observation include increased sleepiness

(e.g., shortened sleep latency and improved sleep efficiency). Accordingly, the proportion of recruits reporting subjective poor sleep latency decreased from 51% in civilian life to 15% at week 5 and the proportion of recruits reporting subjective sleep efficiency > 85% significantly increased from 71% to 87% at week 5 ($P < 0.001$).

Comparisons between self-reported and objective sleep quality (Actigraphy) were made the night before the week 1 medical, whereby similar proportions of recruits who reported "good" or "very good" subjective PSQ also demonstrated good sleep efficiency scores as measured from Actigraphy (77 *vs* 79% respectively). In addition, the greatest proportion of recruits reporting 'very poor' sleep quality also had the greatest proportion of objective poor sleep efficiency (**Figure 11**). These findings indicate that subjective sleep quality is reflective of objectively assessed sleep quality.

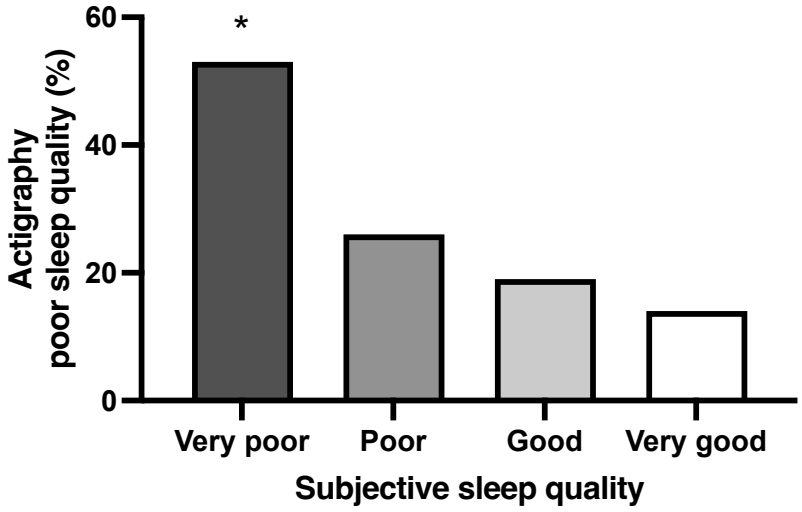

Figure 11. *The proportion of recruits reporting perceived sleep quality and Actigraphy assessed sleep efficiency. Poor Actigraphy assessed sleep efficiency < 85%. * P < 0.01 vs all other groups.*

A total of 61 physician diagnosed RTI episodes were recorded during the 12 weeks of Phase 1 training, of which 49% occurred during the first month of training. Each RTI episode resulted in 4.4 ± 2.7 lost training days.

Table 17. *Global sleep quality and sleep quality components in civilian life, during the first month of training and in the last month of military training in those that completed training.*

	Civilian life	First month of training	Last month of training
Week of assessment	1	5	12
PSQI			
Global score (0-21) [mean ± SD]	5.0 ± 2.7	5.1 ± 2.3	5.0 ± 3.0
Score ≥ 5 [*N* (%)]	332 (53)	351 (56)	327 (52)
Score ≥ 7 [*N* (%)]	152 (24)	152 (24)	160 (26)
PSQ components			
Average sleep duration (h) [mean ± SD]	8.0 ± 1.5[ab]	6.2 ± 0.9[c]	6.6 ± 1.1[c]
7–9-hour sleep duration [*N* (%)]	399 (64)[ab]	135 (22)[bc]	292 (46)[ac]
Poor sleep quality [*N* (%)]	95 (15)	104 (17)	101 (16)
Poor sleep latency [*N* (%)]	320 (51)[ab]	96 (15)[bc]	145 (23)[ac]
Good sleep efficiency [*N* (%)]	441 (71)[ab]	546 (87)[bc]	508 (81)[ac]
Sleep disturbance [*N* (%)]	38 (6)[b]	27 (4)[c]	60 (10)[ac]
Daytime dysfunction [*N* (%)]	58 (9)[ab]	99 (16)[c]	94 (15)[c]
Use of sleep medication ≥ 1 a week [*N* (%)]	11 (2)	6 (1)	22 (4)

PSQI = Pittsburgh Sleep Quality Index. Data for perceived sleep quality, sleep latency, sleep disturbance, daytime dysfunction and sleep medication show the proportion scoring '2 or 3' on these components. Data for sleep efficiency shows the proportion scoring '0'. [a] $P < 0.05$ *vs.* first month of training; [b] $P < 0.05$ *vs.* last month of training; [c] $P < 0.05$ *vs.* civilian life. Data is presented for the sample of participants who were present at 'Week 12', $N = 625$.

7.4.2 The association between civilian life sleep and RTI risk during training

Regression analyses showed that recruits who reported very poor sleep quality (PSQI ≥7) during civilian life were twice as likely to suffer RTI during training compared to good quality sleepers (PSQI < 5) (Model 1: OR = 2.13, 1.19–3.80, *P* = 0.011, **Figure 12**). This association remained in the covariate adjusted Model 2 accounting for BMI, smoking status, season of enrolment, days spent in training, as well as in the fully adjusted Model 3 that additionally controlled for perceived stress (Model 3: OR = 1.96, 1.05–3.64, *P* = 0.034, **Table 18**). There was a trend with very poor sleep quality (PSQI ≥7) during civilian life and RTI during the first month of training, however this did not reach significance (Model 3: OR = 1.87, 0.74–4.67, *P* = 0.091 *vs* PSQI < 5, **Table 18**). Furthermore, short (< 7 h) and long (> 9 h) sleep duration during civilian life was associated with increased susceptibility to RTI during the first month of training compared to those sleeping the recommended 7 – 9h, and these associations persisted after full adjustment (Model 3: < 7 h; OR = 2.66, 1.08–6.59, P = 0.033, > 9 h; OR = 3.07, 1.27–7.42, *P* = 0.028, **Figure 13A**). These associations did not remain in all RTIs during training (**Table 19**).

There was no association between sleep restriction and increased RTI susceptibility during the first month of training or across the full 12 weeks in any model (*P* > 0.05).

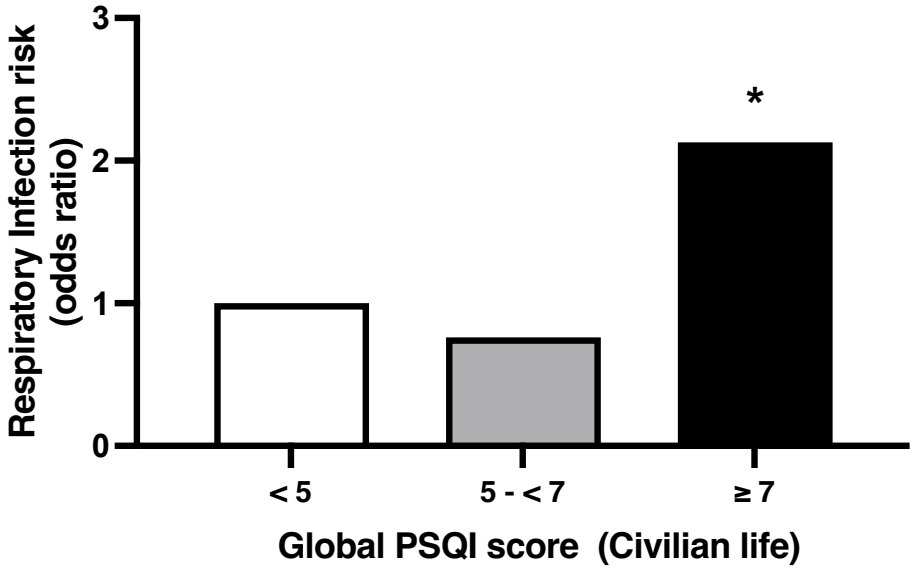

Figure 12. *Association between sleep quality during civilian life and respiratory infection risk during training. PSQI = Pittsburgh Sleep Quality Index. PSQI global score < 5 was reference. Odds ratios presented are from unadjusted analyses. * P < 0.05 vs reference. These associations persisted in the fully adjusted model accounting for BMI, smoking status, season of enrolment, days spent in training and perceived stress.*

Further regression analyses showed that very poor sleep quality (PSQI ≥7) in civilian life was associated with increased RTI during training irrespective of sleep duration (**Figure 13B**). Good sleep quality (PSQI < 5) was associated with protection against RTI. Recruits sleeping the recommended 7 – 9h but reporting very poor sleep quality were over 2x increased risk of RTI compared to 7 – 9h sleepers reporting good sleep quality (Model 1: OR = 2.26, 1.01–5.02, *P* = 0.046, **Figure 13B**). These associations did not remain in the fully adjusted analyses, likely due to limited statistical power in sub analyses. Short (< 7 h) and long (> 9 h) sleepers with good sleep quality were at no greater risk of RTI compared to recruits who slept the recommended 7 – 9h and also reported good sleep quality (Model 1: < 7 h; OR = 0.97, 0.12–7.75, *P* = 0.980, > 9 h; OR = 1.65, 0.65–4.21, *P* = 0.296, **Figure 13B**).

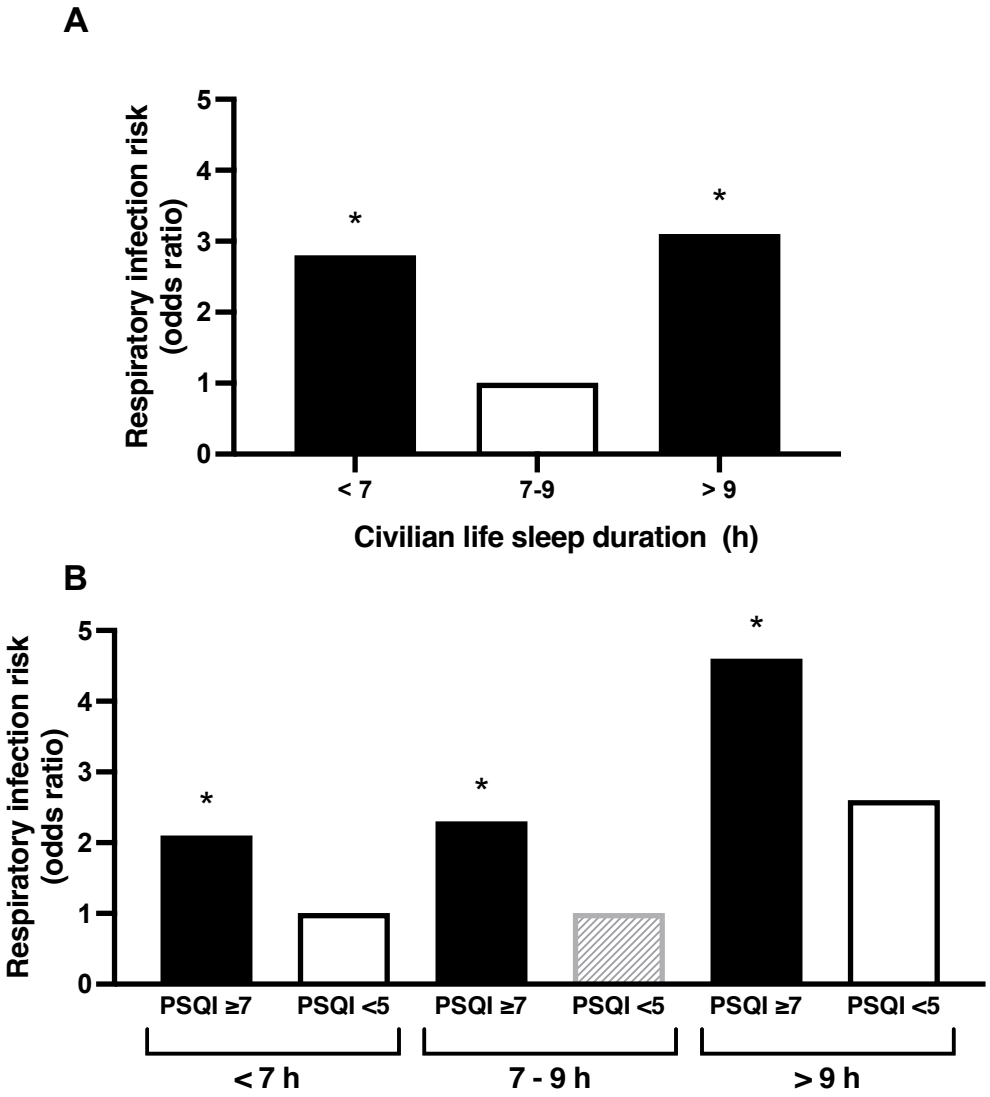

Figure 13. *Association between civilian life sleep duration, quality and respiratory infection during training. A. Sleep duration. B. Sleep duration stratified by PSQI. PSQI = Pittsburgh Sleep Quality Index. Reference group was A) 7-9 h, B) 7-9 h PSQI <5. Odds ratios presented are from unadjusted analyses. * P < 0.05 vs reference.*

7.4.3 The association between sleep in the first month of training and subsequent RTI risk

During the first month of military training when sleep opportunity was reduced and sleep pressure increased (**Table 17**), very poor sleep quality (PSQI score ≥ 7) was associated with over a 3x increased risk of suffering subsequent RTI compared to good sleep quality (PSQI < 5) (Model 1: OR = 3.28, 1.23–8.76, *P* = 0.018, **Table 18**). Furthermore, recruits with enduring very poor sleep quality in both civilian life and the first month had a 6x greater risk of suffering a RTI after week 5 (Model 1: OR = 6.72, 1.42─31.87, *P* = 0.016 vs enduring good quality sleepers; **Figure 14**). Recruits who improved their sleep quality during the first month of training had protection against the raised risk of RTI (*P* > 0.05, **Figure 14**). There was no association with short sleep (< 7 h) during the first month of training and subsequent RTI incidence (Model 1: OR = 1.27, 0.52─3.12, *P* = 0.606). These associations did not remain in the fully adjusted analyses, likely due to limited statistical power in sub analyses.

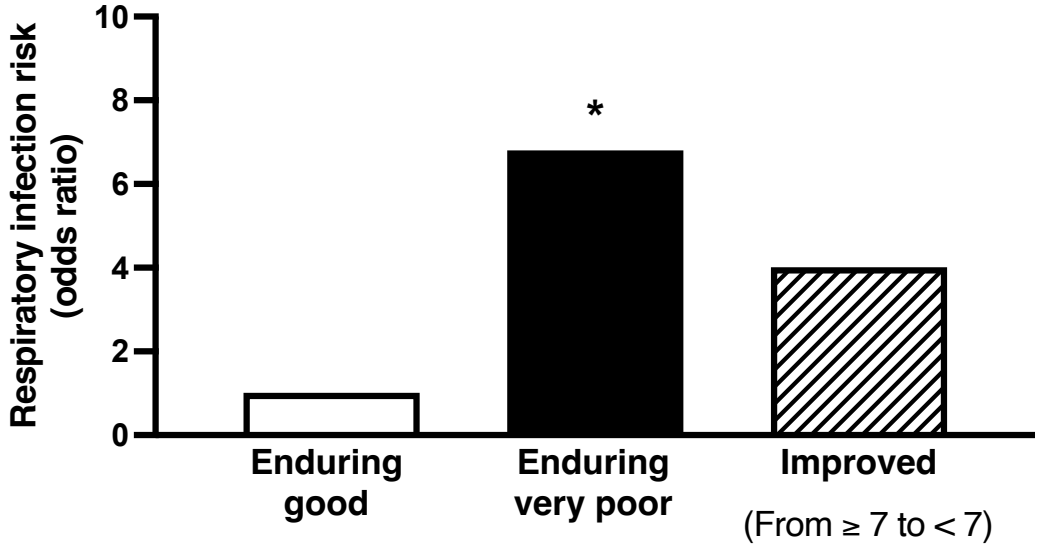

Figure 14. *Association between enduring sleep quality (civilian life and first month of training) and respiratory infection risk after week 5. PSQI = Pittsburgh Sleep Quality Index. Enduring good was reference group. Odds ratios presented are from unadjusted analyses. * P < 0.05 vs reference.*

Table 18. *Association between global PSQI assessed during civilian life and risk of RTI during training.*

	Global PSQI score		
	Good (< 5)	Poor (5 - < 7)	Very poor (≥ 7)
	OR	OR	OR
Population that commenced training			
First 4 weeks			
Model 1: unadjusted (PSQI sleep quality)	*Reference*	1.55 (0.61–3.96)	2.16 (0.90–5.20)
Model 2: BMI + smoking + season + days in training	*Reference*	1.56 (0.61–4.00)	1.98 (0.82–4.82)
Model 3: model 2 + perceived stress	*Reference*	1.51 (0.58–3.91)	1.87 (0.74–4.67)
12 weeks			
Model 1: unadjusted (PSQI sleep quality)	*Reference*	0.76 (0.36–1.59)	2.13 (1.19–3.80)[a]
Model 2: BMI + smoking + season + days in training	*Reference*	0.74 (0.35–1.56)	2.19 (1.22–3.94)[aa]
Model 3: model 2 + perceived stress	*Reference*	0.70 (0.33–1.49)	1.96 (1.05 –3.64)[a]
Population present at week 5 follow up visit			
5 – 12 weeks			
Model 1: unadjusted (PSQI sleep quality)	*Reference*	2.97 (1.13–7.85)[a]	3.28 (1.23–8.76)[a]
Model 2: BMI + smoking + season + days in training	*Reference*	2.74 (1.02–7.39)[a]	3.55 (1.31–9.63)[a]
Model 3: model 2 + perceived stress	*Reference*	2.40 (0.86–6.67)	2.74 (0.90–8.28)

PSQI = Pittsburgh Sleep Quality Index; BMI = body mass index. Sleep quality was categorised using global PSQI scores < 5 (good sleep quality), 5 - <7 (poor sleep quality, and ≥ 7 (very poor sleep quality); ≥ 7 is a threshold recognised to identify clinically significant sleep complaints [a] = $P < 0.05$; [aa] = $P < 0.01$.

Table 19. *Association between civilian life sleep duration and risk of RTI during training.*

	Sleep duration in civilian life (hours)		
	< 7	7 – 9	> 9
	OR	OR	OR
Population that commenced training			
First 4 weeks			
Model 1: unadjusted (Sleep duration)	2.82 (1.15–6.91)[a]	*Reference*	3.13 (1.31–7.50)[a]
Model 2: BMI + smoking + season + days in training	2.71 (1.10–6.68)[a]	*Reference*	3.09 (1.28–7.48)[a]
Model 3: model 2 + perceived stress	2.66 (1.08–6.59)[a]	*Reference*	3.07 (1.27–7.42)[a]
12 weeks			
Model 1: unadjusted (Sleep duration)	1.55 (0.81–2.98)	*Reference*	1.78 (0.95–3.33)
Model 2: BMI + smoking + season + days in training	1.57 (0.81–3.02)	*Reference*	1.77 (0.94–3.34)
Model 3: model 2 + perceived stress	1.52 (0.79–2.94)	*Reference*	1.78 (0.94–3.36)

BMI = body mass index. 7-9 h is considered as reference. [a] = $P < 0.05$; [aa] = $P < 0.01$.

7.5 Discussion

This study extends upon the findings presented in **Chapter 6** by demonstrating that poor sleep quality is associated with increased RTI risk, irrespective of sleep duration. We showed that recruits reporting very poor sleep quality (PSQI ≥ 7) during civilian life were almost twice as likely to suffer an RTI during military training compared to those reporting good sleep quality (PSQI < 5) (**Figure 12**). Notably, this association persisted after adjustment for known RTI risk factors including BMI, smoking status, season, days in training, and perceived stress. We also showed that short (< 7 h) sleepers during civilian life were more susceptible to RTI during training (**Figure 13A**). However, the association between short sleep duration during civilian life and RTI during training was only present in those reporting poor sleep quality; good sleep quality provides protection against infection (**Figure 13B**). Furthermore, very poor sleep quality during the first month of training was associated with over a threefold increased risk of subsequent RTI, however, this association was attenuated following adjustment for perceived stress. This attenuation likely reflects the shared variance between poor sleep quality and elevated stress during the demanding early weeks of training and may indicate that perceived stress partially mediates rather than simply confounds the sleep quality–RTI association. Finally, recruits with enduring poor sleep quality across both civilian life and early training had a sixfold greater risk of RTI compared to recruits reporting consistently good sleep quality (**Figure 14**). Importantly, improvements in sleep quality from civilian life to the first month of training were associated with protection against RTI. Collectively, these findings highlight that subjective sleep quality, more so than sleep duration, is a critical determinant of immune resilience, especially during periods of increased physical and psychosocial stress.

The current findings are consistent with and extend those of prior studies linking sleep with immune function and infection susceptibility. Whereas **Chapter 6** demonstrated that good PSQ protected against the heightened risk of respiratory infection during sleep restriction, the present study did not observe an independent effect of sleep restriction on infection risk. Instead, we found that good sleep quality provided protection against infection in those sleeping short or long durations. The absence of an independent effect of sleep restriction in the present study may reflect methodological and contextual differences between the two cohorts. In **Chapter 6**, sleep was assessed at a single time point based on the night immediately preceding the medical assessment, providing a precise measure of acute sleep restriction (mean reduction of 2.1 h from civilian life). In contrast, sleep in this study was assessed using the PSQI, which reflects typical sleep over the preceding month, and so this approach captured

habitual rather than acute changes in sleep and may have attenuated the apparent magnitude of sleep restriction (mean reduction of 0.8 h from civilian life). However, taken together, these studies support the notion that how individuals perceive their sleep may be as important, if not more so, than the number of hours slept in determining immune health.

The mechanisms underlying the observed associations likely reflect the multifaceted impact of poor sleep quality on neuro-endocrine immune modulation. As discussed in Chapter 6, poor PSQ is associated with reductions in slow-wave sleep (SWS) and dysregulation of the HPA axis, resulting in a disrupted circadian cortisol rhythm (Backhaus et al., 2004). This dysregulation may impair innate and adaptive immune responses, including diminished T-cell activation and antibody production, therefore potentially compounding susceptibility to infection (Besedovsky et al., 2019; Lange et al., 2011). Conversely, improvements in PSQ may enhance immune health through restoration of sleep architecture and normalisation of HPA axis activity.

The current findings presented include several strengths to further provide confidence in the results. Recruits presenting with subjective common cold symptoms or CRP values > 10 mg/L (indicative of acute infection) (Pearson et al., 2003) at the start of training or were diagnosed with a RTI within one week of arrival were deemed not healthy and subsequently excluded from analyses to mitigate reverse causation. Regression models were adjusted for variables known to be associated with increased RTI risk, including BMI (Harpsoe et al., 2016), smoking (Arcavi & Benowitz, 2004), season (Heikkinen & Jarvinen, 2003), and perceived stress (Cohen et al., 1999; Cohen et al., 1991), and further adjustment to account for the number of days spent in training. The present findings are further strengthened by the agreement between subjective and objective sleep assessments (**Figure 11**). Actigraphy-derived sleep efficiency corresponded closely with self-reported sleep quality, supporting the reliability of perceived sleep measures in this population.

Several limitations should be acknowledged. First, while PSQI provides a validated measure of global sleep quality, it remains a subjective tool and the inclusion of polysomnography would have allowed for more precise characterisation of sleep stages. However, we complemented self-reported sleep measures with actigraphy, which demonstrated good agreement with PSQ, thereby supporting the validity of the subjective assessments used in this study. Second, the study sample comprised predominantly young, healthy males of similar

SES, which may limit generalisability to other populations such as older adults, females, or civilians experiencing chronic sleep disturbances. However, it should be noted civilians embarking on a military training course affords a unique opportunity to examine the association between sleep and RTI in a healthy, pre-screened population under standardised conditions. Finally, future studies should confirm the infectious origin of physician diagnosed RTI by performing pathological analysis on throat swabs.

In conclusion, this study provides novel findings that poor sleep quality, rather than short sleep duration, is a stronger and more consistent predictor of respiratory infection risk in young adults undergoing military training. Improvements in sleep quality were associated with protection against infection, highlighting the importance of targeting sleep quality, and not merely sleep duration, in interventions aimed at reducing infection susceptibility.

Recommendations for the MOD deriving from this chapter's findings:

1. **Provide all recruits with a sleep hygiene pack prior to the start of training.** Given that poor sleep quality in civilian life independently predicts RTI risk during Phase 1, a simple and scalable prevention strategy would be to send all recruits a standardised sleep hygiene pack in the weeks before they commence training. This could include a fitted eye mask, ear plugs, and a brief evidence-based guidance leaflet covering practical strategies to improve sleep quality. Normalising sleep as a performance and health priority before recruits arrive at training would complement the in-training interventions recommended across this thesis and could begin to improve sleep quality during the period when civilian life sleep is most predictive of subsequent training health outcomes.
2. **Recognise that adequate sleep duration does not guarantee immune resilience.** A key finding of this chapter is that sleep quality predicts RTI risk independently of sleep duration; recruits sleeping the recommended 7–9 hours but reporting very poor sleep quality were at over twice the RTI risk of those sleeping the same duration with good quality. New guidance should be given to recruits, training staff, and medical personnel that explicitly highlights that restorative, good quality sleep matters as much as sleep length.

CHAPTER 8 – Poor sleep increases the risk of acute musculoskeletal injury

8.1 Abstract

We prospectively examined the association between sleep and MSKI in military recruits undertaking basic training, whereby 970 young adults commencing Phase 1 12-weeks of basic military training consented to participate. Civilian-life sleep duration and quality were assessed at baseline using the PSQI. At training weeks 5 and 12, wrist-worn actigraphy and repeat PSQI quantified sleep-wake indices, sleep disturbances and perceptions of sleep quality during training were assessed. Physician determined MSKI during training, classified as either traumatic or overuse, was extracted from medical records. Logistic regression analyses were adjusted for lifestyle and psychosocial factors, training exposure and aerobic fitness. Poor sleep quality (global PSQI ≥ 7) in civilian life was reported in 24%, with sleep duration decreasing from 8.0 ± 1.5 h in civilian life to 6.3 ± 0.9 h during training. A total of 208 recruits suffered an MSKI during training, of which half were traumatic, with 93% of MSKI cases resulting in time lost from training and were associated with failure to complete the course (OR = 1.7, 95% CI 1.2–2.3, $P < 0.01$). In civilian life, poor sleep quality was not associated with MSKI during training, but short sleepers (< 6 h) were more likely to suffer MSKI, particularly traumatic injuries occurring in the first month of training (Model 3: OR = 3.13, 95% CI 1.31–7.46, $P < 0.05$) and across the full training phase (Model 3: OR = 2.29, 95% CI 1.12–4.65, $P < 0.05$). During the first month of training, very poor sleep quality (PSQI ≥ 7) was associated with nearly threefold increased risk of traumatic MSKI after week 5 (Model 3: OR = 2.82, 95% CI 1.23–6.46, $P < 0.05$). Recruits with enduring very poor sleep quality across both civilian life and the first month of training were over four times more likely to suffer traumatic MSKI after week 5 (Model 3: OR = 4.54, 95% CI 1.43–14.36, $P < 0.05$). There was no association between sleep quality and MSKI in recruits who improved their sleep quality during training ($P > 0.05$). In conclusion, poor sleep quality and short sleep duration were associated with increased risk of MSKI during military training, particularly traumatic injuries.

8.2 Introduction

MSKI represent a major concern in both athletic and military populations, where the physical and occupational demands of training place individuals at heightened risk. In the United States, approximately 8.6 million sport-related injuries are reported annually, underscoring their significant public health impact (Sheu et al., 2016). Within the military, MSKI are the primary cause of lost training days and contribute substantially to attrition and reduced operational effectiveness (Bullock et al., 2010). A range of factors have been identified as determinants of MSKI. These include non-modifiable characteristics, such as age, sex and previous injury, alongside modifiable factors such as body composition, fitness and training load (Bahr & Holme, 2003; Meeuwisse, 1994). Identifying modifiable contributors is critical for the development of targeted, evidence-based prevention strategies. Sleep has recently emerged as a potentially important, yet comparatively underexplored, determinant of injury risk (Dobrosielski et al., 2021).

Athletes and soldiers are particularly vulnerable to disrupted sleep due to high training demands, irregular schedules and frequent transitions in environment or time zone. Mechanistic pathways linking inadequate sleep duration or poor sleep quality to injury risk are multifactorial. Sleep loss impairs cognitive performance and motor control, slowing reaction time and reducing protective neuromuscular responses during load-bearing activity (Avedesian et al., 2022; Dobrosielski et al., 2021; Wilke & Groneberg, 2022). Concurrently, disrupted sleep alters endocrine function and compromises tissue repair, potentially undermining musculoskeletal integrity (Dobrosielski et al., 2021). Evidence suggests these mechanisms may be especially pertinent to traumatic injuries, with retrospective analyses linking poor sleep quality to elevated risk of acute MSKI (Lee, Chung, & Kim, 2021).

Despite growing recognition of sleep as a risk factor, prospective evidence remains sparse. A recent systematic review identified a lack of controlled cohort studies, particularly in military settings where the burden of MSKI is high (Dobrosielski et al., 2021). While short sleep duration has been linked with elevated injury risk in athletic settings (Luke et al., 2011; Milewski et al., 2014; von Rosen et al., 2017), findings regarding sleep quality remain inconsistent (Grier et al., 2020; Ruan et al., 2020). The present study aimed to address these gaps by prospectively evaluating the role of sleep in predicting MSKI among 970 young adults undergoing Basic military training, a 12-week programme of fundamental military instruction delivered under controlled conditions. Basic military training offers a unique opportunity to

investigate the prospective relationship between sleep and MSKI under standardised living and working conditions (e.g., housing, diet and physical activity), in adults facing the challenges associated with relocating to a new living and working environment alongside the physical and psychological demands of the course. We hypothesised that both short sleep duration and poor sleep quality would be associated with increased risk of traumatic MSKI, and that improvements in sleep quality across training would exert a protective effect.

8.3 Methods

8.3.1 Study design and participants

Study design and participants are outlined in **Chapter 3, section 3.1 – 3.3.**

8.3.2 Procedures

A total of 1,188 healthy recruits (98% male) provided written informed consent to participate in the study (**Figure 15**). Procedures are detailed in **Chapter 3, section 3.3**.

Psychosocial and lifestyle measures were assessed in private using online surveys (LimeSurvey, Hamburg, Germany) via an electronic tablet. Sleep quality and duration in the previous month was assessed at week 1 and one-month later with the widely used PSQI (see **Chapter 3, section 3.7** for details) (Buysse et al., 1989). Actigraphy was used to obtain objective measures of sleep at the start of training (see **Chapter 3, section 3.7** for details).

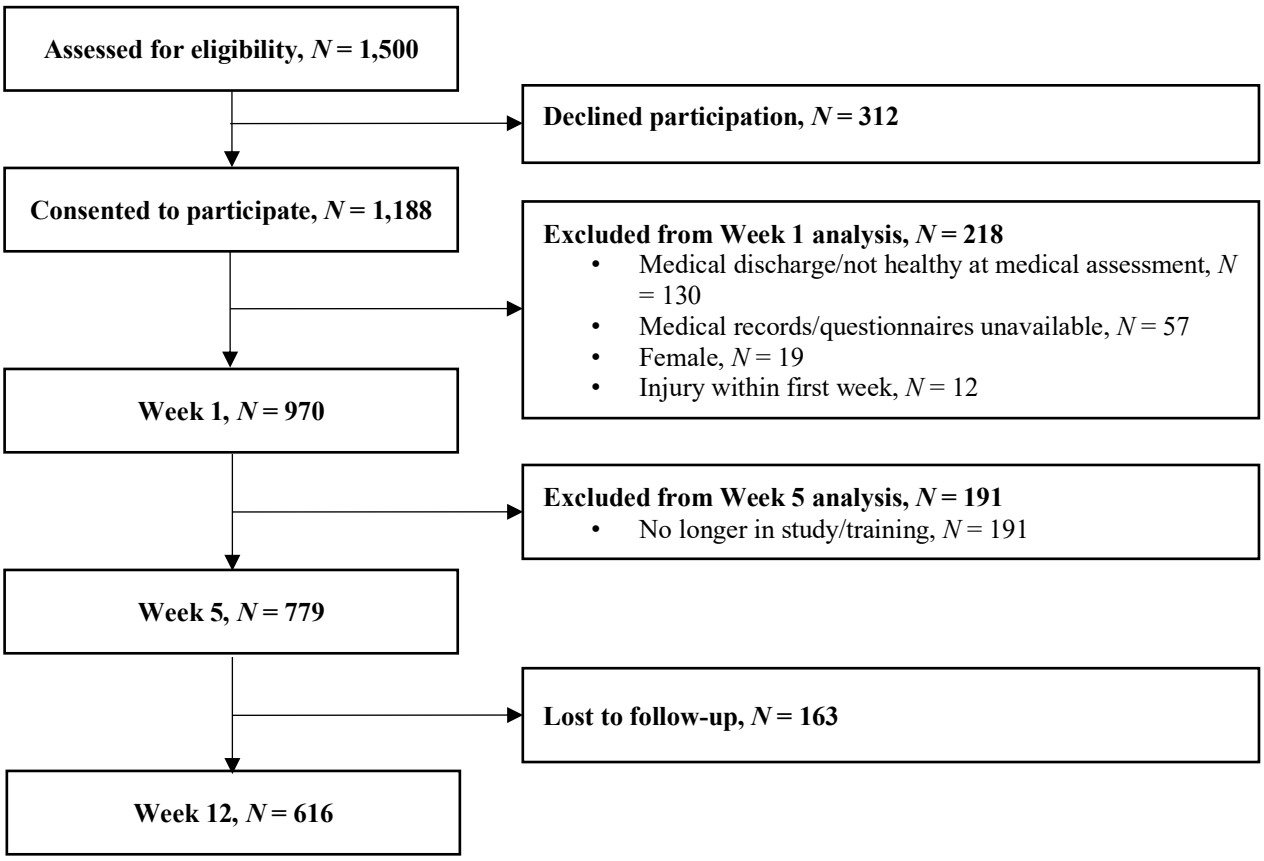

Figure 15. *Flowchart outlining recruits assessed for eligibility, consented and available at the start and during the training course.*

8.3.3 Outcomes

The primary outcome of this study was MSKI during military training. MSKI diagnosis is described in **Chapter 3, section 3.11**.

The physician's medical evaluation in week 1 ensured that the recruit was fit to commence training, mitigating against the risk of reverse causation (i.e., existing injury influencing the sleep assessments). To provide further confidence regarding reverse causation, at the statistical analysis stage, we also excluded MSKI cases suffered in close proximity to sleep assessments. In addition, to ensure a valid comparison against non-injured participants in analyses isolated to traumatic or overuse injuries, recruits who experienced overuse injuries were excluded when examining traumatic injuries and vice versa.

8.3.4 Statistical analysis

All analyses were conducted using SPSS 28 (IBM, Armonk, NY, USA) with statistical significance set at $P < 0.05$. Descriptive data are presented as mean ± SD for continuous variables or absolute numbers and percentages for categorical variables (**Table 20**), with one-independent t-test or chi-squared analysis used for comparison of descriptive data stratified by recruits that completed training versus those lost to follow up (**Table 21**). Logistic regression was used to examine the association between MSKI and likelihood of completing the training course.

A small proportion of PSQI data at week 5 were missing, assumed missing at random, with 17% (N = 166) of recruits missing PSQI data e.g. due to administrative issues with questionnaires. To avoid a potential bias, multiple imputation was carried out using predictive mean matching (50 iterations and 40 imputed datasets), in line with recommendations (Graham, 2009). After accounting for participants who withdrew from Army training and the study by week 5, PSQI collected at week 1 and all variables collected at week 5 were included in multiple imputation, including the outcome variable RTI incidence.

To examine the association between sleep and MSKI, sleep quality and sleep duration were assessed separately as continuous and categorical data. Sleep quality was categorised using global PSQI scores < 5 (good sleep quality), 5 or 6, and ≥ 7 (poor sleep quality); ≥ 7 is a threshold recognised to identify clinically significant sleep complaints (Buysse et al., 2007). Sleep duration was categorised into < 6 h, 6 -< 7 h, 7 – 9 h and > 9 h. Hierarchical logistic regression analysis was used to examine the association between poor sleep and MSKI. Furthermore, we examined the association between each of the individual PSQI components and MSKI. We next examined the association between sleep quality and MSKI in recruits reporting enduring sleep i.e., those reporting poor sleep quality at both week-1 and week-5, and whether improved sleep quality during the first month of training was associated with protection against MSKI. An improvement in sleep quality was defined as a leftward shift of at least one classification in sleep quality e.g., from poor sleep quality in week-1 (PSQI ≥ 7) to good sleep quality by week-5 (PSQI < 5).

Potential confounding factors on the association between poor sleep and MSKI include BMI (Hruby et al., 2016), smoking (Robinson et al., 2016), season (Carswell et al., 2023), number of days spent in training (to account for exposure bias), aerobic fitness determined by 1.5 mile

run time, a commonly used military test of endurance fitness capacity (Carswell et al., 2018; Robinson et al., 2016). For each logistic regression analysis, after checking assumptions, model 1 investigated the unadjusted association with MSKI. Model 2 was adjusted for BMI, smoking, and season; model 3 included model 2 plus adjustment for training exposure and aerobic fitness.

8.4 Results

8.4.1 Sample characteristics and MSKI incidence and impact

Demographic and lifestyle data for the $N = 970$ recruits who commenced training are presented in **Table 20**. Accounting for selection bias due to loss to follow-up, demographic and lifestyle behaviours were comparable in those who completed the course and those lost-to-follow up apart from depressive symptoms and perceived stress in the previous month (**Table 21**). A total of 208 recruits suffered a MSKI during the 12 weeks of Phase 1 training, of which half were traumatic injuries (**Table 22**). The detrimental impact of suffering a MSKI during training was highlighted as MSKI was associated with failure to complete the course (model 1: OR = 1.7, 95% CI 1.2–2.3, $P < 0.01$) and 93% of MSKIs resulted in time lost from training (10.9 ± 12.7 d; **Table 22**).

8.4.2 Sleep quality and duration in civilian life and during military training

The mean PSQI score in civilian life was 4.9 ± 2.6 with 24% reporting very poor sleep quality (**Table 23**). Surprisingly, there was no influence of military training on sleep quality despite a reduction in sleep duration from 8.0 h ± 1.5 h in civilian life to 6.3 h ± 0.9 h in the first month of training; this was due to increased sleep pressure during training improving sleep latency and improving sleep efficiency (**Table 23**). However, the proportion of recruits reporting daytime dysfunction doubled by week-5 compared to week-1. The remaining PSQI components were unchanged in the first month of training (**Table 23**). In total, 21% (N = 162 of 779) improved their sleep quality classification between civilian life and the first month of training.

Table 20. *Descriptive data and musculoskeletal injury incidence in the population that commenced training.*

		Total sample
Population that commenced training		***N* = 970**
Demographics		
	Age (y)	21 ± 4
	Ethnicity, White [*N* (%)]	820 (85)
Education		
	UK level 3 qualification or higher [*N* (%)]	369 (38)
	No formal qualifications [*N* (%)]	163 (17)
Anthropometrics		
	Height (m)	1.77 ± 0.07
	Body mass (kg)	76.0 ± 11.5
	BMI (kg/m^2)	24.2 ± 3.2
Lifestyle behaviours		
	Current smoker [*N* (%)]	251 (28)
	Alcohol user [*N* (%)]	674 (70)
Season of enrolment		
	Winter-Autumn [*N* (%)]	777 (80)
Physical fitness		
	1.5 mile run time (s)	569 ± 51
Psychological		
	Depressive symptoms (range 0–30)	5.0 ± 3.9
	Perceived stress (range 0–40)	11.7 ± 5.8

Values presented as mean ± SD unless otherwise stated. BMI = body mass index. Depressive symptoms were assessed using the Centre for Epidemiologic Studies Depression Scale-10 item and perceived stress was assessed using the 10-item Perceived Stress Scale.

8.4.3 The association between civilian life sleep and MSKI risk during training

Logistic regression analysis showed short sleep duration in civilian life was associated with increased MSKI during training, (Model 3: OR = 2.29, 95% CI 1.12–4.65, $P < 0.05$). The association between sleep duration in civilian life and MSKI during training was stronger when isolating traumatic injuries, whereby recruits who reported sleeping < 6 h in civilian life were over three times more likely to suffer a traumatic injury in the first four weeks of training (Model 3: OR = 3.13, 95% CI 1.31–7.46, $P < 0.05$, **Figure 16** and **Table 24**) and over twice as likely to suffer a traumatic injury across the full 12-week course (model 3: OR = 2.29, 95% CI 1.12–4.65, $P = 0.02$, **Table 24**).

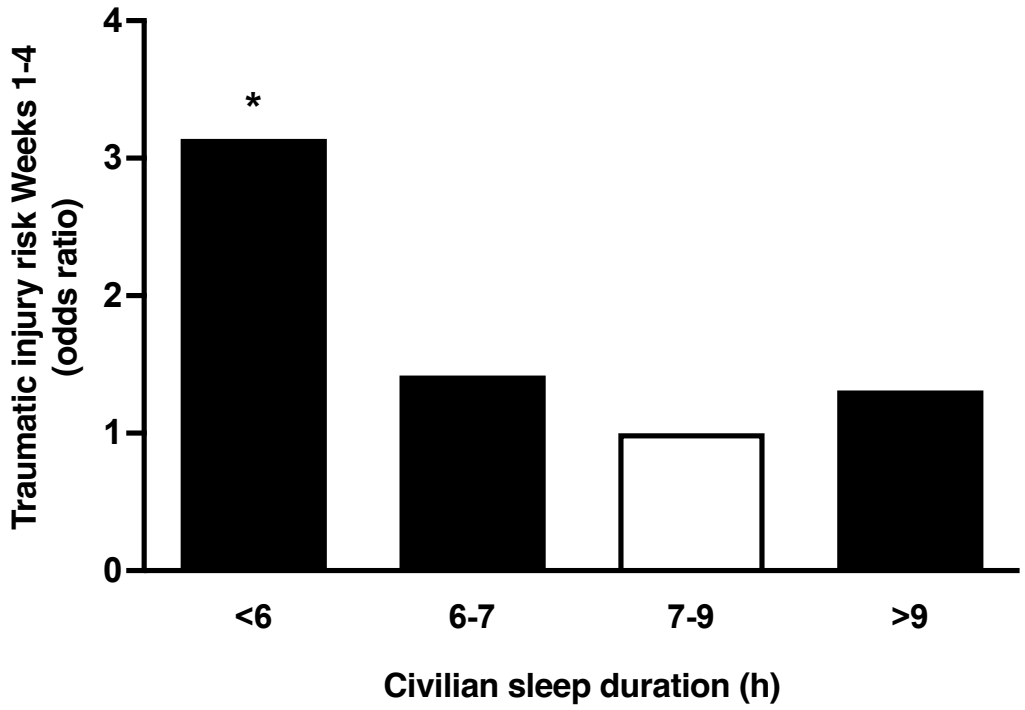

Figure 16. The influence of sleep duration in civilian life on traumatic musculoskeletal injury risk. Odds ratios presented are from unadjusted analyses. 7-9 h is considered as reference. * *P < 0.05 vs reference.*

8.4.4 The association between poor sleep quality in the first month of training and MSKI risk after week-5

During the first month of military training when sleep opportunity was reduced and sleep pressure increased (**Table 23**), poor sleep quality in the first month of training was associated with an increased risk of MSKI after week-5 (Model 3: OR = 1.98, 95% CI 1.1–3.6, $P = 0.03$). The association between poor sleep quality in the first month of training and MSKI during training was stronger when isolating traumatic injuries (**Figure 17A**, **Table 25**). Importantly, individuals with the poorest sleep quality had objectively shorter sleep (**Figure 17B**) and reported greater daytime dysfunction during the first month of training (**Figure 17C**).

We next examined whether the association between poor sleep and MSKI was stronger in those who reported enduring poor sleep in both civilian life and during the first month of training. Recruits who reported enduring very poor sleep quality were over four times more likely to suffer a traumatic MSKI after week 5 compared with those reporting enduring good sleep quality (Model 3: OR = 4.54, 95% CI 1.43–14.36, $P < 0.05$). In contrast, there was no association between sleep quality and MSKI in recruits who improved their sleep quality classification in the first month of training compared with civilian life (Model 3: OR = 2.78, 95% CI 0.91–8.52, $P > 0.05$). These findings became stronger when examining traumatic injuries (**Figure 18A**, **Table 26**. Importantly, individuals with the poorest sleep quality had objectively shorter sleep (**Figure 18B**) and reported greater daytime dysfunction during the first month of training (**Figure 18C**).

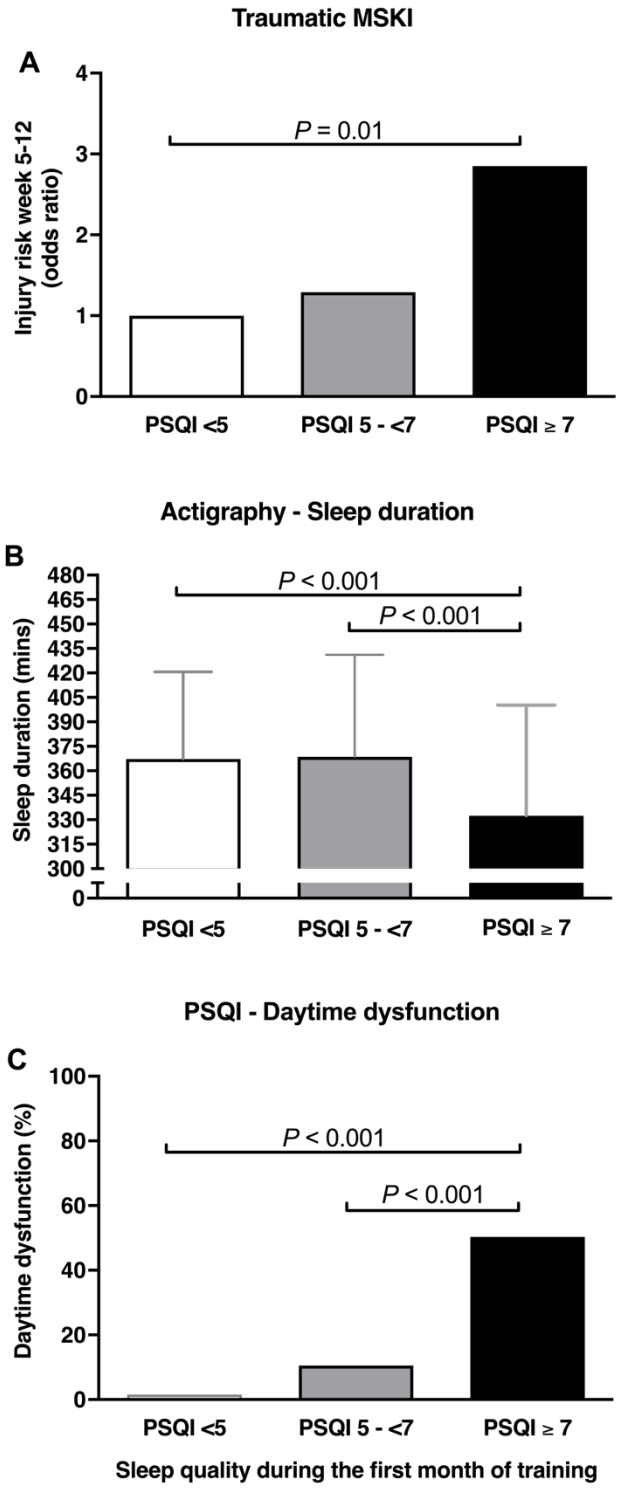

Figure 17. *The influence of global sleep quality in the first month of training on subsequent traumatic musculoskeletal injury risk (A). Panels B and C show objectively assessed total sleep time and the proportion of recruits reporting Daytime dysfunction stratified by sleep quality scores. Sleep quality was categorised using the global Pittsburgh sleep quality index scores < 5 (good sleep quality), 5–6 (poor sleep quality) and ≥ 7 (very poor sleep quality); ≥ 7 is a threshold recognised to identify clinically significant sleep complaints. Good global sleep quality (< 5) considered as reference. In panel A, odds ratios presented are from unadjusted analyses.*

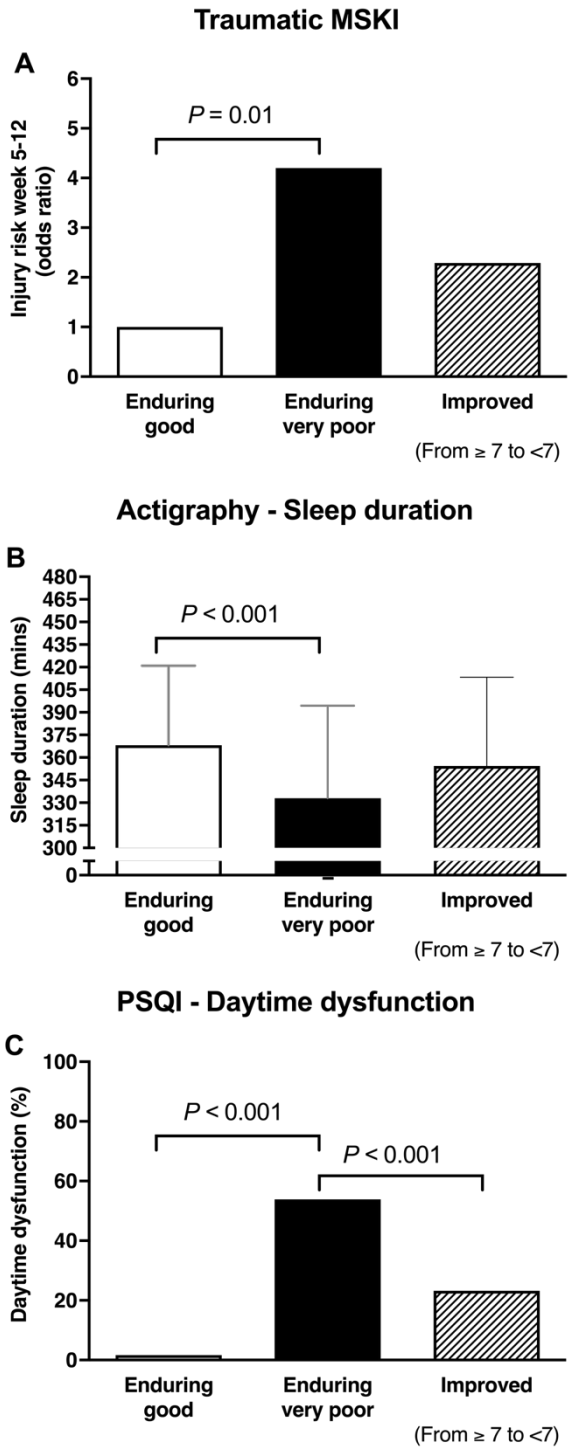

Figure 18. *The prospective influence of enduring global sleep quality (civilian life and first month of training) on traumatic musculoskeletal injury risk (A). Panels B and C show objectively assessed total sleep time and the proportion of recruits reporting Daytime dysfunction stratified by enduring sleep quality scores. 'Enduring good' was defined as consistently reporting a global Pittsburgh sleep quality index score or < 5 in civilian life and in the first month of training. 'Enduring very poor' was defined as consistently reporting a global Pittsburgh sleep quality index score or ≥ 7 in civilian life and in the first month of training. 'Improved' sleep quality was defined as a leftward shift of at least one classification in sleep quality during training. Odds ratios presented are from unadjusted analyses. Enduring good global sleep quality (< 5) considered as reference.*

Table 21. *Descriptive data in the population that commenced training and were lost to follow-up.*

	Completed training	Lost to follow-up
Demographics		
Age (y)	21 ± 4	21 ± 4
Ethnicity, White [*N*(%)]	506 (82)	314 (89)
Education		
UK level 3 qualification or higher [*N*(%)]	252 (41)	117 (33)
No formal qualifications [*N*(%)]	100 (16)	63 (18)
Anthropometrics		
Height (m)	1.77 ± 0.07	1.77 ± 0.07
Body mass (kg)	77.0 ± 11.3	74.3 ± 11.8
BMI (kg/m^2)	24.5 ± 3.1	23.8 ± 3.3
Lifestyle behaviours		
Current smoker [*N*(%)]	167 (27)	93 (26)
Alcohol user [*N*(%)]	438 (71)	236 (67)
Season of enrolment		
Winter-Autumn [*N*(%)]	491 (80)	286 (81)
Physical fitness		
1.5 mile run time (s)	565 ± 49	576 ± 55
Psychological		
Depressive symptoms (range 0–30)	4.4 ± 3.3	6.1 ± 4.7*
Perceived stress (range 0–40)	10.8 ± 5.3	13.4 ± 6.3*

Values presented as mean ± SD unless otherwise stated. BMI = body mass index. Depressive symptoms were assessed using the Centre for Epidemiologic Studies Depression Scale-10 item and perceived stress was assessed using the 10-item Perceived Stress Scale. * $P < 0.05$ *vs.* population that completed training.

Table 22. *Musculoskeletal injury incidence in the across the full 12-week training phase.*

	Commenced training	**Present at training week 5**	**Completed training**
	***N* = 970**	***N* = 779**	***N* = 616**
Full 12 weeks of phase 1 training			
Any injury [*N*(%)]	208 (21)	166 (21)	119 (19)
Traumatic injury [*N*(%)]	96 (46)	79 (48)	55 (46)
Overuse injury [*N*(%)]	112 (54)	87 (52)	64 (54)
Injuries causing time lost from training [*N*(%)]	193 (93)	153 (92)	110 (92)
Time lost from training (d)	10.9 ± 12.7	11.4 ± 13.4	10.5 ± 12.2
Traumatic injury time lost (d)	12.1 ± 11.4	13.4 ± 12.0	12.7 ± 11.7
Overuse injury time lost (d)	10.2 ± 9.4	10.4 ± 10.0	9.0 ± 9.6
First month of phase 1 training			
Any injury [*N*(%)]	86 (9)	66 (8)	36 (6)
Traumatic injury [*N*(%)]	43 (50)	34 (51)	17 (47)
Overuse injury [*N*(%)]	43 (50)	32 (49)	19 (53)
Injuries causing time lost from training [*N*(%)]	83 (96)	64 (97)	36 (100)
Time lost from training (d)	11.2 ± 8.9	12.0 ± 9.4	10.7 ± 8.8
Traumatic injury time lost (d)	11.2 ± 9.3	12.2 ± 9.7	10.7 ± 8.7
Overuse injury time lost (d)	11.3 ± 8.6	11.8 ± 9.2	10.7 ± 9.1

	Commenced training	Present at training week 5	Completed training
	***N* = 970**	***N* = 779**	***N* = 616**
Weeks 5-12 of phase 1 training			
Any injury [*N*(%)]	113 (11)	100 (13)	83 (14)
Traumatic injury [*N*(%)]	48 (42)	45 (45)	38 (46)
Overuse injury [*N*(%)]	65 (58)	55 (55)	45 (54)
Injuries causing time lost from training [*N*(%)]	101 (89)	89 (89)	74 (89)
Time lost from training (d)	12.0 ± 13.3	12.6 ± 14.0	12.2 ± 13.8
Traumatic injury time lost (d)	12.1 ± 13.4	12.6 ± 13.6	12.1 ± 12.9
Overuse injury time lost (d)	9.0 ± 10.0	9.1 ± 10.6	8.3 ± 10.0

Values presented as mean ± SD unless otherwise stated.

Table 23. *Global sleep quality and sleep quality components in civilian life, during the first month of training and in the last month of military training in those that completed training.*

	Civilian life	**First month of training**	**Last month of training**
Week of assessment	1	5	12
PSQI			
Global score (0-21) [mean ± SD]	4.9 ± 2.6	5.1 ± 2.3	4.9 ± 3.0
Score ≥ 5 [*N* (%)]	326 (53)	342 (56)	315 (51)
Score ≥ 7 [*N* (%)]	147 (24)	145 (23)	149 (24)
PSQ components			
Average sleep duration (h) [mean ± SD]	8.0 ± 1.5[ab]	6.3 ± 0.9[c]	6.6 ± 1.1[c]
7–9-hour sleep duration [*N* (%)]	397 (64)[ab]	137 (22)[bc]	284 (46)[ac]
Poor sleep quality [*N* (%)]	89 (14)	101 (16)	98 (16)
Poor sleep latency [*N* (%)]	316 (51)[ab]	92 (15)[bc]	140 (23)[ac]
Poor sleep efficiency [*N* (%)]	76 (12)[ab]	21 (3)[bc]	44 (7)[ac]
Sleep disturbance [*N* (%)]	36 (6)[b]	28 (4)	56 (9)[ac]
Daytime dysfunction [*N* (%)]	56 (9)[ab]	98 (16)[c]	90 (15)[c]
Use of sleep medication ≥ 1 a week [*N* (%)]	10 (2)	6 (1)	19 (3)

PSQI = Pittsburgh Sleep Quality Index. Data for perceived sleep quality, sleep disturbance, daytime dysfunction and sleep medication show the proportion scoring '2 or 3' on these components. Data for sleep efficiency shows the proportion scoring '1, 2 or 3'. [a] $P < 0.05$ *vs.* first month of training; [b] $P < 0.05$ *vs.* last month of training; [c] $P < 0.05$ *vs.* civilian life. Data is presented for the sample of participants who were present at 'Week 12', $N = 616$.

Table 24. *Association between civilian life sleep duration and risk of traumatic musculoskeletal injury during military training.*

	Sleep duration in civilian life (hours)			
	7 – 9	< 6	6 - < 7	> 9
Population that commenced training	OR	OR	OR	OR
Traumatic injuries in the first month of training				
Model 1: unadjusted (Sleep duration)	*Reference*	3.39 (1.44–7.97)[aa]	1.29 (0.48–3.49)	1.11 (0.46–2.64)
Model 2: BMI + smoking + season	*Reference*	3.12 (1.31–7.42)[a]	1.26 (0.46–3.43)	1.14 (0.47–2.73)
Model 3: model 2 + aerobic fitness	*Reference*	3.13 (1.31–7.46)[a]	1.26 (0.46–3.46)	1.08 (0.45–2.61)
Traumatic injuries during full training phase				
Model 1: unadjusted (Sleep duration)	*Reference*	2.45 (1.22–4.93)[a]	1.59 (0.83–3.07)	1.25 (0.71–2.22)
Model 2: BMI + smoking + season	*Reference*	2.32 (1.14–4.71)[a]	1.57 (0.81–3.05)	1.27 (0.71–2.25)
Model 3: model 2 + aerobic fitness	*Reference*	2.29 (1.12–4.65)[a]	1.62 (0.83–3.17)	1.22 (0.68–2.18)
Model 4: model 3 + days in military training	*Reference*	2.29 (1.13–4.67)[a]	1.62 (0.83–3.16)	1.22 (0.68–2.18)

BMI = body mass index. 7-9 h is considered as reference. [a] = $P < 0.05$; [aa] = $P < 0.01$.

Table 25. *Association between global PSQI assessed at week-5 and risk of traumatic musculoskeletal injury after week-5.*

	Global PSQI score		
	Good (< 5)	Poor (5 - < 7)	Very poor (≥ 7)
Population present at the end of Phase 1 training	OR	OR	OR
Traumatic injuries occurring after week 5 of training			
Model 1: unadjusted (Sleep quality)	*Reference*	1.29 (0.54–3.12)	2.85 (1.27–6.42)[a]
Model 2: BMI + smoking + season	*Reference*	1.39 (0.57–3.41)	3.02 (1.33–6.88)[aa]
Model 3: model 2 + aerobic fitness	*Reference*	1.33 (0.54–3.30)	2.82 (1.23–6.46)[a]

PSQI = Pittsburgh Sleep Quality Index; BMI = body mass index. Sleep quality was categorised using global PSQI scores < 5 (good sleep quality), 5 - <7 (poor sleep quality, and ≥ 7 (very poor sleep quality); ≥ 7 is a threshold recognised to identify clinically significant sleep complaints [a] = $P < 0.05$; [aa] = $P < 0.01$.

Table 26. *Association between enduring PSQI assessed at week -1 and week-5 and risk of traumatic musculoskeletal injury after week-5.*

	Enduring global PSQI score		
	Enduring Good (< 5)	Enduring very poor (≥ 7)	Improved
Population present at the end of Phase 1 training	OR	OR	OR
Traumatic injuries occurring after week 5 of training			
Model 1: unadjusted (Sleep quality)	*Reference*	4.19 (1.39–12.72)[a]	2.29 (0.79–6.63)
Model 2: BMI + smoking + season	*Reference*	4.82 (1.55–15.02)[aa]	2.84 (0.94–8.53)
Model 3: model 2 + aerobic fitness	*Reference*	4.54 (1.43–14.36)[a]	2.78 (0.91–8.52)

PSQI = Pittsburgh Sleep Quality Index; BMI = body mass index. 'Enduring good' was defined as consistently reporting a global Pittsburgh sleep quality index score or < 5 in the month before training and in the first month of training. 'Enduring very poor' was defined as consistently reporting a global Pittsburgh sleep quality index score or ≥ 7 in the month before training and in the first month of training. 'Improved' sleep quality was defined as a leftward shift of at least one classification in sleep quality during training. [a] = $P < 0.05$; [aa] = $P < 0.01$.

8.5 Discussion

This cohort study provides prospective evidence that poor sleep quality and short sleep duration are associated with increased risk of MSKI in young adults undergoing basic military training. Our findings demonstrate that recruits who reported short sleep duration (<6 h) prior to training were more likely to suffer MSKI, particularly traumatic injuries occurring in the first month of training. Furthermore, poor sleep quality during the first month of training predicted subsequent MSKI, with the strongest associations observed in individuals reporting enduring poor sleep across both civilian life and training. Importantly, recruits who improved their sleep quality during training were not at elevated risk of MSKI, suggesting that sleep health may be a modifiable factor in injury prevention. These findings underscore the importance of promoting sleep health in physically demanding environments and support the integration of sleep monitoring and intervention strategies into military and athletic training programmes.

Our results align with and extend previous literature linking sleep disturbances to injury risk. Retrospective analyses have shown that poor sleep quality is associated with increased risk of acute MSKI (Lee et al., 2021), while short sleep duration has been implicated in elevated injury rates among adolescent athletes (Milewski et al., 2014; von Rosen et al., 2017). However, prospective evidence in adult populations, particularly in military settings, has been lacking (Dobrosielski et al., 2021). The present study addresses this gap by demonstrating that both sleep duration and quality independently predict MSKI risk, with stronger associations observed for traumatic injuries. Importantly, recruits reporting the poorest sleep quality during the first month of training not only had significantly shorter objective sleep duration, as measured via actigraphy, but also reported greater daytime dysfunction. These findings provide mechanistic support for the hypothesis that sleep loss impairs neurocognitive function, including reaction time and motor control (Avedesian et al., 2022; Wilke & Groneberg, 2022), which may compromise protective neuromuscular responses during load-bearing activity. Additionally, disrupted sleep alters endocrine function and tissue repair processes (Dobrosielski et al., 2021; Knutson, Ryden, Mander, & Van Cauter, 2006), potentially undermining musculoskeletal integrity. These mechanisms may be particularly relevant in the context of military training, where recruits are exposed to high physical demands, early morning awakenings and psychological stress, all of which may exacerbate sleep disturbances and injury risk.

Several strengths and limitations should be considered when interpreting these findings. A key strength of this study is its prospective design, which allowed for temporal assessment of sleep and injury outcomes. The controlled nature of military training, where recruits follow standardised schedules, diets, and physical activity routines, minimises confounding and enhances internal validity. Sleep was assessed using both subjective (PSQI) and objective (actigraphy) measures, providing a comprehensive evaluation of sleep health. Furthermore, rigorous steps were taken to mitigate reverse causation, including exclusion of injuries occurring near sleep assessments and stratification of injury types. Regression models were adjusted for known MSKI risk factors, including BMI (Hruby et al., 2016), smoking (Robinson et al., 2016), season (Carswell et al., 2023), training exposure, and aerobic fitness (Carswell et al., 2018; Robinson et al., 2016). However, the study population was male, limiting generalisability to female recruits. Additionally, while the associative design precludes causal inference, the consistency of findings across multiple sleep indices strengthens the plausibility of a causal relationship. Future studies should explore sex differences and examine the efficacy of sleep interventions in reducing MSKI risk.

In conclusion, this study provides compelling evidence that sleep in the month before commencing military training predicts MSKI risk in the first month of training. Furthermore, poor sleep quality and short sleep duration are associated with increased risk of MSKI during basic military training. The findings highlight the importance of sleep as a modifiable risk factor and suggest that improvements in sleep quality may confer protection against injury. These insights have important implications for military and athletic populations, where injury prevention is critical to performance and operational readiness. Practitioners should consider incorporating sleep health strategies into training programmes, including education on sleep hygiene, monitoring of sleep patterns and targeted interventions for individuals at risk. Future research should investigate the mechanisms linking sleep to MSKI and evaluate the impact of sleep-focused interventions on injury outcomes across diverse populations and settings.

Recommendations for the MOD deriving from this chapter's findings:

1. **Provide recruits with a pre-training sleep hygiene pack to reduce injury risk.** As recommended in Chapters 6 and 7 in the context of respiratory infection, providing all recruits with a standardised sleep hygiene pack before commencing training is equally supported by the MSKI findings of this chapter. Recruits sleeping fewer than six hours in

civilian life were over twice as likely to suffer a traumatic MSKI across Phase 1 training, and any improvement in sleep duration or quality achieved before arrival to training could meaningfully reduce this risk.

2. **Brief training staff on sleep as an injury risk factor.** The stronger association of poor sleep with traumatic rather than overuse injuries is consistent with sleep deprivation impairing reaction time and motor control, both of which are critical during the high-intensity physical activities of Phase 1 training including load carriage, obstacle courses, and weapons drills. Training staff and physical training instructors should be made aware that poor sleep increases risk of acute injury. Simple awareness of this mechanism could support more informed decisions about training intensity and supervision during periods of known sleep disruption.

CHAPTER 9 – General Discussion

9.1 Summary of main findings

This thesis examined the influence of psychosocial and lifestyle factors on illness and injury in British Army recruits commencing military training. Across the empirical chapters (**Chapters 4 – 8)**, consistent evidence emerged that both early-life and current behavioural factors significantly affect adult health outcomes during periods of intense physical and psychological stress.

Chapter 4 demonstrated that childhood adversity prevalence was 47% in this military cohort and was a robust predictor of RTI during training in adulthood. Recruits with a history of childhood adversity were substantially more likely to suffer a physician-diagnosed RTI during training, even after controlling for demographic and lifestyle confounders. Importantly, this relationship was driven specifically by multiple adversities and by abuse-related adversities (physical, emotional, or sexual abuse), whereas a singular adversity event or neglect-related adversities (physical or emotional neglect) showed no independent association with RTI risk. Recruits reporting the most severe childhood adversity (CTQ severity score quartile 4) were nearly two and a half times more likely to suffer RTI compared to those with the lowest adversity levels. These findings reinforce theoretical models suggesting that early adverse experiences become biologically embedded into adulthood (Elwenspoek et al., 2017).

Building upon these findings, **Chapter 5** examined whether psychological resilience factors could moderate the relationship between childhood adversity and RTI. Optimism emerged as a crucial protective factor. Recruits who commenced training with a history of childhood adversity but showed increases in optimism during the first month of training exhibited no elevated RTI risk compared to those without childhood adversity. In stark contrast, recruits with childhood adversity who experienced decreases in optimism were over five times more likely to suffer a subsequent RTI. These findings support Cohen's stress-buffering hypothesis (Cohen & Wills, 1985), indicating that positive psychological changes can offset biological vulnerability stemming from childhood adversity.

Sleep emerged as another crucial determinant of health across multiple studies. **Chapter 6** demonstrated that sleep restriction (defined as an individualised reduction in sleep duration

of ≥2 hours from civilian life) significantly increased URTI risk during the first four weeks of training and across the full 12-week course. However, this association was moderated by PSQ. Recruits with sleep restriction who reported good PSQ were at no greater risk of URTI compared to non-sleep-restricted recruits with good PSQ, despite experiencing similar magnitudes of sleep reduction. In contrast, sleep-restricted recruits reporting poor PSQ were twice as likely to suffer URTI. Findings revealed a significant interaction between sleep restriction and PSQ on URTI susceptibility, whereby the association between sleep restriction and raised URTI risk was only observed in those with poor PSQ. These findings remained robust after adjustment for sex, BMI, smoking, season, mood disturbance, and both long and short civilian sleep duration.

Chapter 7 extended these findings by demonstrating that poor sleep quality, irrespective of sleep duration, was associated with higher RTI incidence throughout the 12-week Phase 1 training period. Recruits reporting very poor sleep quality (PSQI ≥ 7) in civilian life were twice as likely to suffer RTI during training compared to those reporting good sleep quality (PSQI < 5), independent of sleep duration. Importantly, the association between short sleep duration (<7 h) and RTI was only present in recruits reporting poor sleep quality; short sleepers with good sleep quality showed no elevated infection risk compared to those sleeping the recommended 7-9 hours with good quality. Furthermore, very poor sleep quality during the first month of training predicted over a threefold increased risk of subsequent RTI, and recruits with enduring poor sleep quality across both civilian life and early training had a nearly sixfold greater risk compared to those reporting consistently good sleep quality. Critically, recruits who improved their sleep quality from civilian life to training were protected against RTI, demonstrating that sleep quality changes are meaningfully associated with infection risk. This distinction between sleep duration and sleep quality provides important nuance: although duration is well-established as a determinant of immune function, the subjective experience of sleep may be equally or more influential on immune health. Moreover, the agreement between subjective sleep quality ratings and actigraphy-assessed sleep efficiency provided validation for the subjective sleep measures used throughout the thesis.

Finally, **Chapter 8** demonstrated that poor sleep also elevated the risk of MSKI during Phase 1 training. Recruits who reported sleeping <6 hours per night in civilian life were over twice as likely to suffer a traumatic MSKI during the full 12-week course, and this association was

particularly strong for injuries occurring in the first month of training. Poor sleep quality during the first month of training also predicted subsequent MSKI risk, with the strongest associations observed for traumatic rather than overuse injuries. Recruits reporting enduring poor sleep quality across both civilian life and early training were nearly three times more likely to suffer MSKI after week-5 compared to those with consistently good sleep quality. Importantly, individuals with the poorest sleep quality had objectively shorter sleep duration as measured by actigraphy and reported greater daytime dysfunction, providing mechanistic support for the hypothesis that sleep loss impairs neurocognitive function, reaction time, and motor control, thereby compromising protective neuromuscular responses during physical activity.

Collectively, these studies provide evidence that both early-life psychosocial experiences and current behavioural factors shape health outcomes during military training. Childhood adversity elicits long-term vulnerability to infection through mechanisms that may include biological embedding of stress reactivity, while optimism and sleep quality emerge as protective, modifiable factors that can enhance immune health and reduce injury risk.

9.2 Integration of findings and theoretical implications

9.2.1 Biological embedding of childhood adversity.

The observed associations between childhood adversity and RTI risk during adulthood support models of biological embedding, whereby early stress exposure alters stress-sensitive biological pathways in ways that persist into adulthood (Danese & McEwen, 2012; Miller et al., 2011). The sequential regression approach observed in **Chapter 4**, which adjusted for perceived stress and poor sleep quality as potential mediator variables, provides important insight into how these pathways operate. Perceived stress and poor sleep quality during training partially accounted for the overall adversity–RTI association, suggesting that for some recruits, the elevated infection risk associated with childhood adversity operates partly through heightened stress reactivity and its downstream effects on sleep and immune function, consistent with evidence that adversity-exposed individuals perceive subsequent stressors as more threatening and report higher psychological stress in daily life (Dougherty et al., 2004; LoPilato et al., 2020). Consistent with this mechanism, **Chapter 4** demonstrated that recruits with the most severe childhood adversity (CTQ severity score quartile 4) reported higher perceived stress at the start of training (39% vs. 23% in the lowest adversity quartile) and showed dramatic increases in stress during the first month (67% reporting moderate-high stress at week-5 compared to 38% in the lowest adversity group). This pattern suggests that childhood

adversity not only predisposes individuals to baseline immune dysregulation but also amplifies their physiological and psychological responses to new stressors, creating a vulnerability to infection. However, the associations for multiple adversities and abuse-related adversities specifically remained robust after full adjustment for perceived stress and sleep quality, indicating that for these more severe and abuse adversity exposures, the pathway to immune vulnerability extends beyond stress reactivity to encompass more direct biological mechanisms. This is most likely via CTRA which is activated by abuse-related adversities through chronic HPA axis and sympathetic nervous system dysregulation rather than through perceived stress alone (Cole, 2019). The finding that CRP did not differ across adversity severity groups and that adjusting for CRP did not attenuate associations supports this interpretation, indicating that the primary mechanism for abuse-related adversity operates through cellular immune dysregulation rather than systemic inflammation.

Developing further on this, the finding that abuse-related adversities, but not neglect-related adversities, increased RTI risk (**Chapter 4**) is theoretically significant and aligns with dimensional models of adversity that distinguish between threat-related (abuse) and deprivation-related (neglect) experiences (McLaughlin et al., 2014). Abuse-related adversities produce stronger and more persistent HPA axis and sympathetic nervous system activation than neglect, driving the CTRA pattern described above, whereas neglect-related adversities primarily affect cognitive and social development rather than stress-immune pathways (Elwenspoek et al., 2017; McLaughlin et al., 2014). The differential impact of abuse versus neglect on infection risk observed in this thesis suggests that the biological consequences of childhood adversity are not uniform but depend on the specific nature of the adverse experience.

The controlled nature of military training was a significant strength in demonstrating these associations. By standardising living conditions, diet, physical activity, and pathogen exposure, the study design minimised environmental confounding and strengthened the inference that observed associations reflect biological vulnerability rather than differential exposure to pathogens or health-relevant behaviours. This methodological strength suggests that the immune consequences of childhood adversity are sufficiently robust to manifest even in highly controlled environments, underscoring the profound and enduring impact of early-life stress on adult health.

9.2.2 Psychological resilience and the stress-buffering hypothesis

The protective effect of optimism observed in **Chapter 5** provides strong support for Cohen's stress-buffering model, which suggests that psychosocial resources protect health by reducing the appraisal of stressors as threatening and by enhancing coping capacity (Cohen & Wills, 1985). The finding that increases in optimism during the first month of training protected against the elevated RTI risk associated with childhood adversity is particularly striking because it demonstrates that psychological adaptation can offset biological vulnerability stemming from early-life stress.

The mechanisms by which optimism buffers stress and protects immune function are likely multifaceted. First, optimists may engage in more adaptive coping strategies when faced with challenges, such as problem-focused coping and cognitive reframing, which reduce the intensity and duration of stress responses (Carver et al., 1993; Scheier et al., 1994). Second, optimism is associated with lower negative affect and better mood regulation, which may reduce the psychological stress that activates neuroendocrine-immune pathways (Brissette et al., 2002). Third, optimists may exhibit healthier behaviours, including better sleep and greater help-seeking, which independently support immune function (Scheier et al., 1994). Consistent with this third pathway, **Chapter 5** showed that recruits with childhood adversity who increased their optimism reported lower perceived stress and better sleep quality compared to those whose optimism decreased.

Notably, optimism was assessed and shown to change over relatively short timescales (one month), and these changes meaningfully predicted subsequent health outcomes. This finding has important practical implications: it suggests that psychological interventions targeting optimism and positive expectancies may be effective even when implemented during high-stress transitions such as military training. Techniques such as cognitive-behavioural therapy, positive psychology interventions (e.g., best possible self exercises), and resilience training programs have been shown to increase optimism in both civilian and military populations (Joyce et al., 2018; Malouff & Schutte, 2016; Peters et al., 2010; Rozek et al., 2019). Integrating such interventions into military training could represent a scalable, low-cost strategy to reduce infection burden and improve recruit health, particularly among those with adverse childhood experiences.

9.2.3 Sleep as a modifiable determinant of immune and physical resilience

The findings across **Chapters 6, 7, and 8** combine to establish sleep as a critical, modifiable determinant of both infection and injury risk. Importantly, these studies distinguish between sleep restriction, sleep duration and sleep quality, revealing that subjective sleep quality may be as important, if not more so, than the number of hours slept.

The association between sleep restriction and URTI (**Chapter 6**) is consistent with experimental and epidemiological evidence linking insufficient sleep to immune dysregulation (Cohen et al., 2009; Prather et al., 2015). Sleep loss impairs multiple aspects of immune function, including reduced natural killer cell activity, diminished T-cell proliferation, altered cytokine production, and reduced antibody responses to vaccination (Besedovsky et al., 2019). However, the novel finding that good PSQ protected against URTI even during sleep restriction challenges the simplistic notion that sleep duration is the sole determinant of immune health.

Several mechanisms may explain the protective effect of good PSQ. First, good PSQ may reflect better sleep architecture, particularly greater amounts of SWS, which is the most restorative sleep stage and is critical for immune function (Akerstedt et al., 1997; Besedovsky et al., 2019). During SWS, the body exhibits reduced cortisol secretion, increased growth hormone and prolactin release, and enhanced T-cell migration and activation, all processes that support immune memory formation and host defence (Besedovsky et al., 2019; Lange et al., 2011). Individuals who report good PSQ despite sleep restriction may maintain adequate SWS, thereby preserving key immunomodulatory processes. Second, good PSQ may indicate better alignment with circadian rhythms, even if total sleep time is reduced. Circadian misalignment, such as sleeping at the wrong biological time or experiencing irregular sleep-wake schedules, disrupts HPA axis regulation and increases inflammatory markers independent of sleep duration (Wright et al., 2015). Individuals who perceive their sleep as adequate may be better aligned with their endogenous circadian rhythms, minimising the inflammatory consequences of sleep restriction. Third, good PSQ may reflect lower subjective stress and better mood regulation. Poor PSQ is strongly associated with negative affect, anxiety, and perceived stress (Bower et al., 2010; Triantafillou et al., 2019), all of which independently increase infection risk (Cohen et al., 1999; Falagas et al., 2010). Conversely, individuals who perceive their sleep as good may experience less psychological distress, which could buffer against stress-related immune suppression.

The findings in **Chapter 7** further underscore the importance of sleep quality over duration. Recruits sleeping the recommended 7-9 hours per night but reporting very poor sleep quality were over twice as likely to suffer RTI compared to those sleeping 7-9 hours with good quality. Moreover, short sleepers (<7 h) with good sleep quality showed no elevated infection risk, suggesting that good quality sleep may compensate for reduced duration. This pattern was reinforced by the observation that recruits with enduring poor sleep quality (consistently reporting PSQI ≥ 7 in civilian life and during training) demonstrated nearly a sixfold increased RTI risk, whereas those who improved their sleep quality were protected. These findings suggest that interventions targeting sleep quality, such as cognitive-behavioural therapy for insomnia, sleep hygiene education, or stress management, may be as or more effective than interventions focused solely on extending sleep duration.

The extension of these findings to MSKI (**Chapter 8**) reveals that sleep influences not only immune function but also injury susceptibility. The mechanisms linking poor sleep to MSKI likely include impaired neurocognitive function (e.g., slower reaction times, reduced attention, impaired motor control), altered hormonal balance (e.g., reduced testosterone, growth hormone), and compromised tissue repair (Avedesian et al., 2022; Dobrosielski et al., 2021; Leproult & Van Cauter, 2011). Notably, recruits with the poorest sleep quality in **Chapter 8** also had objectively shorter sleep duration and greater daytime dysfunction, suggesting that poor sleep quality co-occurs with insufficient sleep and impaired daytime functioning, factors that increase injury risk through both physiological and behavioural pathways.

9.2.4 Interconnections among childhood adversity, psychological adaptation, and sleep

An important insight emerging from this thesis is that childhood adversity, psychological resilience, and sleep are not independent factors but are interconnected in complex ways. **Chapter 4** demonstrated that recruits with childhood adversity reported higher perceived stress and poorer sleep quality at baseline, consistent with extensive literature linking early-life stress to sleep disturbances in adulthood (Greenfield et al., 2011; Liu et al., 2023). **Chapter 5** showed that recruits with childhood adversity who experienced decreases in optimism also reported the highest levels of perceived stress and poorest sleep quality at week-5, suggesting that psychological maladaptation exacerbates both stress and poor sleep. These interconnections suggest a potential cascade of vulnerability (see **Figure 19**). This integrated model aligns with the allostatic load theory, which proposes that repeated or chronic stress exposure leads to cumulative wear-and-tear on multiple physiological systems, including the

immune, metabolic, cardiovascular, and neuroendocrine systems (McEwen, 1998). Childhood adversity may set the stage for elevated allostatic load by creating biological sensitivity to stress, while poor coping and poor sleep act as additional stressors that further increase allostatic burden.

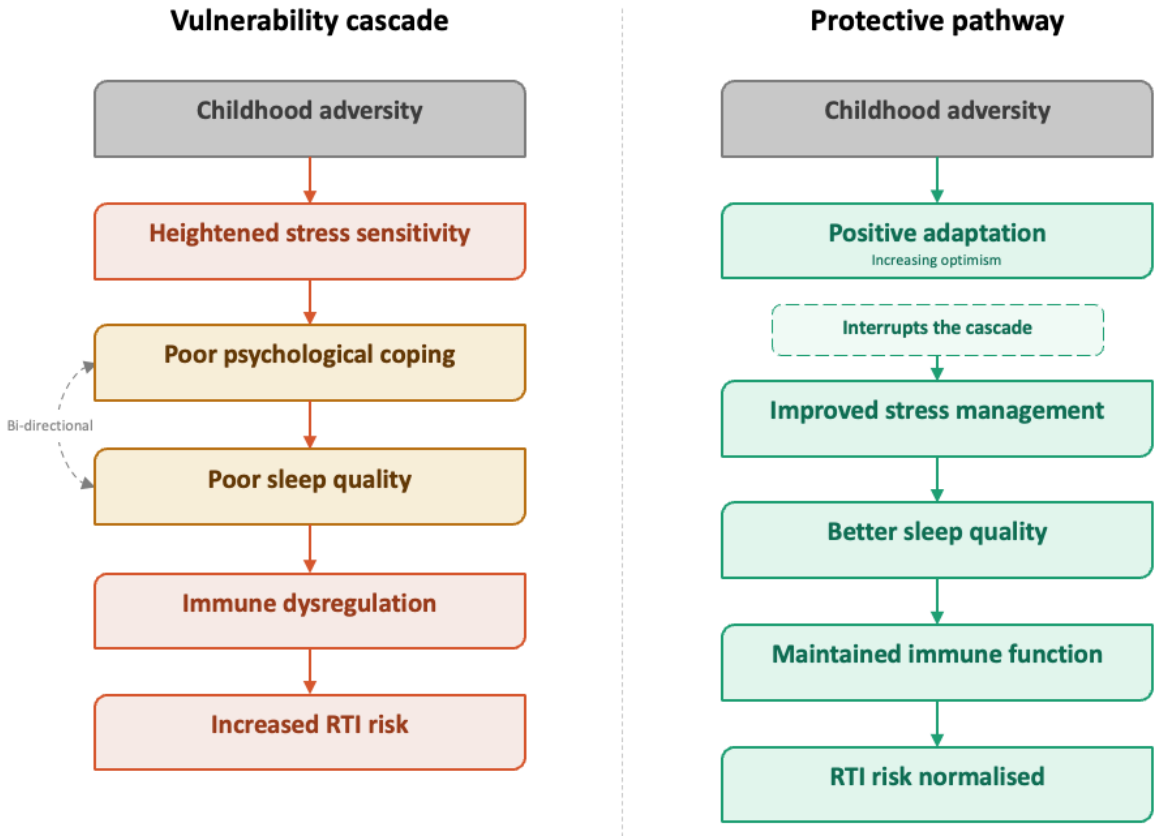

Figure 19. *The integrated vulnerability cascade and protective pathway. Left panel shows the cascade from childhood adversity through heightened stress sensitivity, poor psychological coping, and poor sleep quality to immune dysregulation and elevated RTI risk. The dashed bidirectional arrow reflects the established stress-sleep feedback loop. Right panel shows how positive psychological adaptation, specifically increasing optimism during Phase 1 training, interrupts this cascade, improving stress management and sleep quality and thereby normalising infection risk.*

Importantly, this integrated perspective highlights the potential for multi-component interventions. Rather than targeting only one factor (e.g., sleep or optimism), interventions that simultaneously address psychological resilience, stress management, and sleep hygiene may produce collaborative benefits. For example, resilience training programs that incorporate sleep education and cognitive-behavioural techniques for managing stress may be particularly effective for individuals with adverse childhood experiences.

9.3 Future research directions

The findings of this thesis open several important avenues for future research.

9.3.1 Mechanistic studies

An important next step is to elucidate the biological pathways linking childhood adversity, psychological resilience, sleep, and immune/injury outcomes. Incorporating biomarkers such as diurnal cortisol patterns and immune cell gene expression (e.g., CTRA) would provide mechanistic insights. For example, examining whether recruits with childhood adversity exhibit upregulated CTRA gene expression would advance understanding of biological vulnerability. Similarly, assessing whether improvements in optimism or sleep quality correspond to changes in inflammatory or neuroendocrine profiles would provide evidence for biological mechanisms underlying the protective effects observed in this thesis.

9.3.2 Intervention trials

The modifiable nature of optimism and sleep quality would enable randomised controlled trials to test whether interventions can improve these factors and, critically, whether improvements translate to reduced infection and injury incidence.

For optimism, evidence-based interventions include positive psychology exercises (e.g., best possible self-writing exercises, gratitude journaling), cognitive-behavioural therapy targeting negative attributional styles, and resilience training programs that incorporate optimism-building components (Joyce et al., 2018; Malouff & Schutte, 2016; Peters et al., 2010). These interventions have been shown to increase optimism in civilian and military populations, but their effects on objective health outcomes remain understudied. A trial could randomise military recruits to receive optimism-enhancing training versus standard care and assess effects on RTI incidence, training days lost, and biomarkers of immune function.

For sleep, evidence-based interventions include cognitive-behavioural therapy for insomnia (CBT-I), sleep hygiene education, relaxation training, and environmental modifications (e.g., optimising light exposure, reducing noise) (Irwin et al., 2008). Given the findings in **Chapters 6 and 7** showing that good sleep quality protects against infection even during sleep restriction, interventions that improve sleep quality without necessarily extending sleep duration may be particularly valuable in settings where sleep opportunity is limited (e.g., military training, shift work).

Multi-component interventions that simultaneously target stress management, optimism, and sleep may be especially promising given the interconnections among these factors. For example, a comprehensive resilience training program that includes modules on cognitive reappraisal, optimism cultivation, and sleep hygiene could be tested in recruits with childhood adversity histories. Moderation/mediation analyses could examine whether improvements in optimism and sleep explain intervention effects on health outcomes.

9.3.3 Population diversity and external validity

Future research should extend these findings to more diverse populations. Studies including women are particularly needed, as sex differences in immune function, stress reactivity, HPA axis regulation, and sleep architecture are well-established (Falagas et al., 2007). Women also experience higher rates of certain forms of childhood adversity (e.g., sexual abuse) and certain sleep disorders (e.g., insomnia), suggesting that the associations observed in this thesis may differ by sex (Nelson et al., 2002). Moreover, MSKI incidence is known to be higher in female military recruits, raising the possibility that sleep may be an even stronger risk factor for women (O'Leary et al., 2023). Studies in ethnically diverse populations are also needed to examine whether associations between childhood adversity, optimism, sleep, and health outcomes are consistent across racial and ethnic groups. Cultural factors may influence the expression and reporting of childhood adversity, levels of psychological resilience, and sleep patterns, potentially moderating the observed associations.

9.4 Overall recommendations and potential solutions for the MOD

The findings presented across this thesis have direct and actionable implications for the MOD. Childhood adversity represents a scar that cannot be changed, but with the right identification and support in place, the future for those with adversity can be changed for the better. Psychological adaptation during training and sleep quality, by contrast, are modifiable factors that can be targeted through evidence-based intervention regardless of adversity history. This section synthesises the chapter-specific recommendations into a set of practical proposals.

Firstly, the MOD should explore the feasibility of voluntary, confidential childhood adversity screening at recruitment, while taking into consideration potential ethical concerns. At a population level, routine collection of adversity data across the recruit cohort would allow the Army to build, for the first time, a large-scale understanding of the psychosocial profile of its

recruit population, the prevalence and demographic distribution of childhood adversity across recruitment intakes, and the relationship between early life experience and training health outcomes. This population-level data would provide an evidence base for future health interventions across the military training pipeline. Training staff, medical officers, and welfare personnel should additionally receive brief awareness training regarding the health consequences of childhood adversity. Moreover, a number of ethical concerns in particular must be addressed before any such screening programme is implemented; screening data must be protected from training staff access and held only by medical or welfare personnel, and participation must be voluntary with a clear message that declining does not lead to consequences in career progression or training assessment.

Secondly, before training commences, all recruits should receive a standardised pre-training sleep hygiene pack, including an eye mask, ear plugs, and a brief evidence-based guidance leaflet covering practical strategies for improving sleep quality; consistent sleep and wake times, reducing screen use before sleep and optimising the sleep environment. Given that poor sleep quality in civilian life independently predicts both infection and injury risk during Phase 1, any improvement in sleep quality achieved before arriving at training represents a meaningful reduction in health risk from the beginning of recruits' training journey.

Thirdly, the MOD should develop and evaluate an enhanced resilience training programme, delivered during the first month of Phase 1 training, that simultaneously targets the two modifiable factors identified as most strongly protective against illness and injury in this cohort: optimism and sleep quality. The psychological component of this proposed programme should draw on established techniques shown to increase optimism over short timeframes, including positive goal-setting, best-possible-self exercises, and cognitive reappraisal strategies. The sleep component should deliver evidence-based sleep hygiene education emphasising that sleep quality rather than duration is the primary determinant of immune resilience and injury risk, alongside practical strategies for improving sleep within the constraints of the training environment.

These recommendations form a mutually reinforcing strategy rather than a collection of isolated interventions. Childhood adversity, psychological maladaptation, and poor sleep quality are interconnected vulnerability factors that compound one another across the cascade described in **Figure 19**. Addressing all three in sequence, from recruitment screening through

the first month of Phase 1 training, is likely to be more effective than targeting any single factor in isolation. Investment in evidence-based programmes addressing these modifiable factors represents both a strategic approach to improving health outcomes and reducing operational costs, and a means of ensuring **that individuals' early-life experiences do not irreversibly determine their adult health trajectories,** a principle that sits at the heart of this thesis.

9.5 Conclusions

This thesis provides novel evidence that both early-life experiences and current psychosocial and behavioural factors shape health outcomes during military training. Across five empirical studies involving British Army recruits, several key conclusions emerged:

1. **Childhood adversity is significantly prevalent in military recruits (47%) and elicits long-term vulnerability to respiratory infection in adulthood.** This vulnerability is particularly pronounced for abuse-related adversities and increases in a dose-response manner with adversity severity. These findings support models of biological embedding and highlight the enduring impact of early-life stress on adult immune function.
2. **Optimism is a protective factor that can buffer the negative effects of childhood adversity on infection risk.** Recruits with childhood adversity who showed increases in optimism during training exhibited no elevated infection risk, whereas those whose optimism declined were nearly six times more likely to suffer infection. This supports the stress-buffering hypothesis and demonstrates that psychological resilience can offset biological vulnerability.
3. **Sleep quality is a critical, modifiable determinant of both infection and injury risk.** Good PSQ protected against respiratory infection even during sleep restriction, and poor sleep quality predicted infection irrespective of sleep duration. Similarly, poor sleep quality and short sleep duration significantly increased MSKI risk. These findings challenge the conventional focus on sleep duration alone and highlight the importance of sleep quality.
4. **The effects of childhood adversity, psychological resilience, and sleep are interconnected.** Recruits with childhood adversity reported higher stress and poorer sleep, and those whose optimism declined showed the poorest sleep and highest stress. This suggests that vulnerability factors cluster together and that interventions addressing multiple factors simultaneously may be most effective.

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