EFFECTS OF WORK-RELATED SLEEP RESTRICTION ON ACUTE PHYSIOLOGICAL AND PSYCHOLOGICAL STRESS RESPONSES AND THEIR INTERACTIONS: A REVIEW AMONG EMERGENCY SERVICE PERSONNEL

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Abstract
Emergency work can expose personnel to sleep restriction. Inadequate amounts of sleep can negatively affect physiological and psychological stress responses. This review critiqued the emergency service literature (e.g., firefighting, police/law enforcement, defense forces, ambulance/paramedic personnel) that has investigated the effect of sleep restriction on hormonal, inflammatory and psychological responses. Furthermore, it investigated if a psycho-physiological approach can help contextualize the significance of such responses to assist emergency service agencies monitor the health of their personnel. The available literature suggests that sleep restriction across multiple work days can disrupt cytokine and cortisol levels, deteriorate mood and elicit simultaneous physiological and psychological responses. However, research concerning the interaction between such responses is limited and inconclusive. Therefore, it is unknown if a psycho-physiological relationship exists and as a result, it is currently not feasible for agencies to monitor sleep restriction related stress based on psycho-physiological interactions. Sleep restriction does however, appear to be a major stressor contributing to physiological and psychological responses and thus, warrants further investigation.

Key words:
Sleep, Cytokines, Stress, Cortisol, Mood, Psycho-physiological

INTRODUCTION AND REVIEW OBJECTIVES

Inadequate sleep quality and quantity is a common problem in modern society, which, in turn, can negatively affect psychological and physiological functioning [1]. Evidence suggests that periods of partial and total sleep deprivation/restriction can impair immune function (e.g., above and below normal pro- and anti-inflammatory cytokine levels) [2,3], hormone secretion (e.g., higher and flatter diurnal cortisol levels) [4,5] and instigate adverse psychological changes (e.g., symptoms of anxiety and

depression) [1,6–9]. Furthermore, an increasing body of evidence has demonstrated a link between sleep restriction and negative long-term physical and mental health outcomes [2,10–16]. For instance, inadequate or disrupted sleep has been associated with cardiovascular and metabolic diseases [10,12,14–16], and depression [6–8]. Worldwide, cardiovascular diseases (CVDs) are the leading cause of death [17], while depression remains the leading cause of disability [18]. The links that exist between widespread chronic, long-term negative health outcomes and sleep, underscore the need to examine and characterize acute psychological and physiological stress responses to sleep restriction and deprivation.

To fully evaluate and understand the relationship between acute sleep restriction and physical and mental health, an integrated approach that takes into account both acute psychological and physiological responses to this stressor should be considered. While the understanding of how stress affects psycho-physiological responses and their interaction is still limited, specific findings suggest that psychological health and well-being may influence physiological processes and vice versa [19–21]. Indeed, evidence suggests that an increase in stress exposure simultaneously induces both physiological (i.e., higher and flatter diurnal cortisol levels and/or abnormally high or low cytokine levels) and psychological changes (i.e., mood and behavioral disturbances), and that these responses can be positively or negatively correlated with one another [22–25].

Under normal circumstances, cytokines and glucocorticoids (e.g., cortisol) form a feedback loop, whereby stress elicits the release of pro-inflammatory cytokines which activate the hypothalamic pituitary adrenal (HPA)-axis and results in the release of cortisol and anti-inflammatory cytokines [26–29]. In turn, cortisol and anti-inflammatory cytokines negatively feedback to suppress and regulate the inflammatory response [26–29]. However, exposure to intense or prolonged stress can disrupt this feedback loop

causing an enhanced/up-regulated inflammatory state and HPA-axis disturbances [28,30,31].

These maladaptations are typically associated with negative physiological (e.g., CVD and metabolic syndrome) and psychological health outcomes (e.g., depression) [28,32,33], and in chronic situations, may be underlying these stress-related diseases [30,31,34,35]. For instance, elevated levels of sleep regulating cytokines interleukin (IL)-6, IL-1β and TNF-α have been positively associated with CVD [36–38], metabolic syndrome [39] and depression [40,41]. Furthermore, higher, flatter diurnal cortisol patterns have been related to depression [42,43]. Other distinctive parts of the cortisol stress response such as elevated morning cortisol levels measured in plasma have also been positively associated with CVD and metabolic syndrome related features (e.g., glucose intolerance, insulin sensitivity, hypertension, atherosclerosis) [44–46]. Despite these associations, the exact direction and magnitude of a 'normal' acute change in cytokine, cortisol and mood responses to different periods of sleep restriction is largely unknown, as is the degree to which these acute stress responses are quantifiable risk factors to health [47]. Accordingly, an integrated approach that combines assessment of multiple responses and their interactions may help evaluate and assist in contextualizing the significance of these stress responses [24,25,28,48,49] to sleep restriction.

One of the most common causes of inadequate sleep are work-related factors [50]. For instance, early start times and shift work can cause a misalignment to the circadian rhythm of physiological functions [50]. Reduced sleep opportunities, as a result of extended work hours, long commutes, overtime and being on-call can also disrupt sleep [50]. In addition, exposure to environmental (e.g., light and noise), physical (e.g., intense physical work) and/or psychological (e.g., critical decisions, life threatening situations) work-related stressors can disrupt the circadian rhythm and prevent adequate

sleep [14,50,51]. Emergency (e.g., firefighting, law enforcement/police, some emergency medical services) and defense force services (e.g., military, navy, army) are a unique group of occupations in which personnel is exposed to non-standard scheduling of work hours whilst completing physical work demands on a daily basis [52–58]. For instance, emergency personnel can perform long hours of intermittent physically intense work (up to 15-h) with little rest between consecutive shifts, which can last up to a week [55,56,59–61].

Furthermore, some personnel have reported that the constant readiness (i.e., hyper-vigilance) to respond to an emergency alarm felt while on-duty can transfer to the off-duty environment [62]. This state of hyper-vigilance in combination with excessive work hours and exposure to other occupational stressors, could place personnel of these physically demanding occupations at an increased risk of suffering from inadequate sleep. Indeed, a higher prevalence of sleep disturbances has been reported among firefighting, police, paramedic and military personnel when compared to other occupations [60,61,63–66]. For the purpose of this review, further mention of 'emergency services' or 'emergency personnel' will refer to firefighting, police, paramedic/ambulance and defense force personnel, unless stated otherwise.

The high prevalence of adverse long-term health outcomes (e.g., CVD, metabolic syndrome and depression) associated with sleep restriction reported among these occupations [67–70] is of further concern for emergency services. For instance, Courtney et al. [68] have found paramedic personnel in Australia to have a higher prevalence of sleep-related mental health outcomes (i.e., depression and anxiety) when compared to community samples. Furthermore, the findings among police have revealed that officers reporting shorter sleep durations had a significantly greater number of metabolic syndrome related factors when compared to non-police workers [71] or officers who received more sleep [72].

Given the high prevalence of sleep restriction [61,63,64] and negative sleep-related health outcomes reported among emergency personnel [67–70], it is important to understand how work-related sleep restriction affects personnel's acute psychological and physiological stress responses. Therefore, the 1st part of this review will identify and critique any gaps in the available occupational-based literature that has investigated the effects of sleep restriction on acute hormonal, inflammatory and psychological (i.e., mood, anxiety levels, perceived stress) responses among personnel of emergency services. This review is focused on understanding how personnel respond to modest periods of chronic sleep restriction (i.e., 1–7 nights) that could reflect a single shift, a working week or deployment to a large emergency event (e.g., large bushfires/wildfires). Therefore, research investigating extended periods of reduced sleep (e.g., 8-week military training) will not be examined in this review. Furthermore, the independent influence that night-shifts in emergency work have on stress responses is beyond the scope of this review and therefore, will not be evaluated.

There is growing support in the literature to simultaneously measure multiple responses and their interactions to assess the relevance/importance of stress responses [28,73]. Accordingly, it would be valuable for services to know whether a psycho-physiological approach can help contextualize the significance of acute stress responses to sleep restriction and therefore, assist services to efficiently monitor the acute health of their personnel in the field. For example, if exposure to work-related stressors such as sleep restriction elicits similar or related psychological and physiological responses, then monitoring the health of personnel could be achieved by using self-report measures (e.g., psychological questionnaires). Physiological assessments (e.g., blood samples) could then compliment these measures to provide a more complete picture of the personnel's stress related health.

Therefore, the 2nd part of this review will interrogate the pertinent emergency service-based literature to determine if a psycho-physiological approach can help contextualize the significance of any acute stress responses to assist emergency services monitor the health of their personnel.

While this review intends to provide a comprehensive evidence-based inclusive of various emergency-based occupations, the sleep and stress response research to date has focused mainly on soldiers. Consequently, the balance of literature in this review from each uniform service reflects what is currently available. Furthermore, where emergency-specific research is not available, findings from the wider stress response literature that has investigated periods of sleep restriction similar in length to that demonstrated during emergency work (i.e., 1–7 nights) will be reviewed, and where possible, their transferability to personnel in emergency occupations will be examined.

MATERIAL AND METHODS

Study selection and literature search strategy

This narrative review searched for sleep and stress-related research conducted in emergency-based occupations, the duties of which could be described as physically demanding. Although narrative, the source articles were identified using a systematic search strategy of the global database Ebsco Host to search health-related databases (Academic Search Complete, The Allied and Complementary Medicine Database, CINAHL, Global Health, Health Source (Consumer and Nursing/Academic Editions), MasterFILE, MEDLINE/PubMed, PsycARTICLES, PsycBOOKS, PsycEXTRA, Psychology and Behavioral Sciences, PsycINFO, PsychTESTS and SPORTDiscus) to identify relevant English-language studies published between January 1985 and September 2013.

The occupation-based key words used for the search included: 'firefighters', 'fire fighters', 'fire-fighters', 'police', 'law enforcement', 'paramedics', 'ambulance personnel', 'soldiers', 'navy', 'military' and 'defense force' searched together with sleep and stress response related words of key interest that included: 'sleep deprivation', 'sleep restriction', 'cortisol', 'cytokines', 'mood' and 'psycho-physiological'. Each of the key words mentioned was searched for individually and in conjunction with each other. In addition, relevant articles were identified from the references provided in the original articles retrieved.

The search results were screened and obviously irrelevant or duplicate articles were omitted. Further articles from non-peer-reviewed sources were excluded from the search results. Abstracts and full-texts of the remaining results were then scanned and included in the final review if they met the inclusion criteria outlined in Table 1.

Table 1. Inclusion criteria for the literature review

Inclusion criteria	Explanation
Participants	active duty emergency (e.g., firefighting, police/law enforcement, paramedic/ambulance personnel, rescue workers) or defense force (e.g., army, navy) personnel in physically demanding occupations or healthy adults exposed to periods of sleep restriction similar to emergency personnel (see below)
Period of sleep restriction/deprivation	complete or partial sleep restriction (i.e., < 7 h sleep) from 1–8 consecutive nights
Shift type	single day or consecutive shifts with periods of restricted sleep no specific night shifts
Physiological stress responses	pro- and/or anti-inflammatory cytokines and/or cortisol
Psychological stress responses	a valid and reliable subjective mood, behavior and/or anxiety questionnaire (e.g., Profile of Mood States, Brunel Mood Scale, State-Trait Anxiety Inventory)

RESULTS AND DISCUSSION

The impact of work-related sleep restriction on physiological stress responses among emergency service personnel

Sleep restriction and changes to cortisol

Despite the high prevalence of sleep disturbances reported among police [61,64,65], firefighting [66], paramedic [60] and military personnel [74,75], only one emergency service-based study has investigated the specific effect of restricted sleep on physiological stress responses [76]. Goh et al. [76] have found no significant difference in the overall daily cortisol levels between control (i.e., 8 h sleep) and sleep deprived military personnel (i.e., 1 night of total sleep deprivation). Though there was a significant increase in cortisol levels at 1:30 p.m. on the day after sleep deprivation [76].

Similar results for daily cortisol levels have been found in non-emergency service-based investigations [77–79], indicating that in isolation, a single night of complete sleep restriction may not be a sufficient stressor to significantly affect the overall diurnal release of cortisol among emergency personnel. Determining the isolated (i.e., with no other significant external physical or psychological stressors present) effect that controlled periods of shortened sleep may have on emergency responders' physiological responses is difficult due to their multi-stressor environments (e.g., emergency incidents that can last hours or days and expose personnel to sleep deprivation and physical work) [52,60,61] and consequently, it has not been investigated to a great extent. However, the findings from multi-day military studies may provide some insight into the possible effect restricted sleep opportunities between periods of physical work and military-related demands (e.g., food and water restriction) have on stress responses.

To date, several studies have investigated soldiers' hormonal changes in response to receiving as little as 1–2-h of total sleep across the course of near continuous physical training spanning 3–7 days [80,81]. For instance, Opstad and Aakvaag [81] have reported that the normal circadian variation in the morning and evening cortisol levels on day 1 and 4 of the military training disappeared, indicating an abnormal circadian cortisol release (Table 2). In a more recent study, Opstad [80] employed a high frequency cortisol sampling method to further investigate the effect of a similar 5-day physical training course with minimal sleep (i.e., 1–3-h of total sleep over the course) on military cadets' circadian cortisol release (Table 2). Similar to control conditions (i.e., no physical training and an 8-h sleep opportunity), cortisol levels followed a normal diurnal rhythm on day 1 of the course. However, throughout the rest of the course, mean cortisol levels remained consistently elevated (+130–140%) and over the final 24-h period, the circadian rhythm had almost disappeared [80]. Moreover, 4–5 days after completing the course the circadian cortisol rhythm remained significantly different from the control period [80], adding further support to the possibility of a disrupted circadian cortisol rhythm following consecutive days of sleep restriction during military training. Neither study [80,81], however, controlled for sleep duration or frequency. Instead, the participants slept when possible between training activities and it was estimated by the authors that the participants obtained 1–3-h of total sleep over the course [80,81]. The lack of control over sleep variables (i.e., timing, duration and frequency) limits the ability to make definite conclusions regarding whether sleep duration, frequency and/or rhythm disruption influenced the diurnal dysregulation of cortisol or not.

Furthermore, the participants were also performing physical work and had a substantially restricted daily energy intake during the training course [80,81]. These factors potentially confound the interpretation of these findings, as exposure to physical work and energy restriction has also been found to disrupt normal diurnal cortisol

Table 2. Emergency service based sleep restriction and stress response studies

Reference	Sample	Intervention/ Design	Sleep restriction (SR)	Other stressors	Stress responses	Timing of samples	Main findings
Lieberman et al. [82]	male soldiers (N = 13)	baseline followed by an 84-h lab-based military training	baseline: 4× nights of *ab libitum* sleep training; 7×1 h sleep opportunities (6.2±0.4 h total sleep)	physical work, restricted energy intake, hot environment	POMS	POMS: ~ 8:00 a.m. day 1, 3 and 4 during baseline week and on day 1, 3 and 4 during training	POMS: increase from baseline to training in tension (day 3: 3.4, +74%; day 4: 2.7, +52%; p = 0.01), depression (day 3: 5.4, +216%; day 4: 5.6, +329%; p = 0.02), confusion (day 3: 5.3, +120%; day 4: 7.4, +176%; p < 0.001), fatigue (day 3: 9.4, +145%; day 4: 12.0, +194%; p < 0.001) & anger (day 3: 8.4, +420%; day 4: 9.4, +324%; p = 0.002), vigor (day 3: -3.5, -65%; day 4: -3.4, -57%; p < 0.001)
Lieberman et al. [83]	male army officers (N = 31)	pre-training assessment followed by 53-h training exercise and post-training assessment	pre-training: 1× *ab libitum* sleep (5.3±0.2 h) training: intermittent sleep (3±0.3 h total sleep)	physical work, hot environment and restricted energy intake	Salivary cortisol; POMS	cortisol: pre-training (day 1: 6:00 p.m., day 2: 6:00 a.m., 12:00 p.m., 6:00 p.m.), during training (day 3: 6:00 a.m., 12:00 p.m., 6:00 p.m.) post-training (day 4: 12:00 p.m., 6:00 p.m., day 5: 6:00 a.m.) POMS: pre-training (day 1: 6:00 p.m.), during training (day 4: 12:00 p.m.) post-training (day 5: 5:00 a.m.)	cortisol: decreased from pre-training at 6:00 a.m. (day 2) to day 3 at 6:00 a.m. (-7.7 nmol×l⁻¹, -39%; p < 0.001), day 5 at 6:00 a.m. (-3.6 nmol×l⁻¹, -18%; p < 0.05) increased from pre-training at 6:00 p.m. to post-training at 6:00 p.m. (day 1 vs. 3: 6.1 nmol×l⁻¹, +105%; day 1 vs. 4: 4.4 nmol×l⁻¹, +76%; day 2 vs. 3: 8 nmol×l⁻¹, +207%; day 2 vs. 4: 6.3 nmol×l⁻¹, +164%, p < 0.001) POMS: change from pre- to post-training in tension (2.3, +53%; p < 0.002), depression (3.2, +168%; p < 0.001), confusion (7.8, +252%; p < 0.001), fatigue (17.7, +466%; p < 0.001), anger (2.5, +86%; p ≤ 0.01), vigor (-15.2, -75%; p < 0.001)

Study	Sample	Protocol	Sleep	Stressors	Blood measure	Sampling	Results
Opstad [80]	male military cadets (N = 10)	baseline (1 week prior) followed by 5-day training course and recovery (4–5 days post-training)	intermittent sleep totalling 1–3 h (baseline and recovery: 9–10 h sleep opportunities)	physical work and restricted energy intake	blood serum cortisol	~ 4 h intervals for 24 h a week prior to the course (baseline), on days 1–2, days 4–5 of training and 4–5 days post-training (recovery)	24-h mean cortisol increased from baseline (283 ± 24 nmol×l^{-1}) to days 1–2 (519 ± 30 nmol×l^{-1}; +83%), days 4–5 (556 ± 30 nmol×l^{-1}; 96%), but normal after recovery (287 ± 25 nmol×l^{-1}; significance not reported) circadian cortisol rhythm different from baseline (p = 0.0016) and disappeared days 4–5 morning cortisol on days 1–2 and 4–5 increased above baseline (+130–140%; p < 0.00005)
Lundeland et al. [97]	male military cadets (N = 8)	7-day ranger-training course	intermittent sleep totalling ~ 7 h (1 h/24 h)	physical work and restricted energy intake	blood plasma cortisol and cytokines (LPS-simulated TNF-α, IL-1β, IL-6)	7:00 a.m. baseline and during training on day 3, 5, 7	cortisol: increased from baseline (413 nmol×l^{-1}) to day 3 (505 nmol×l^{-1}) and day 5 (874 nmol×l^{-1}) then decreased on day 7 (631 nmol×l^{-1}; p < 0.05) cytokines: LPS whole-blood: TNF-α increased from baseline (40.2 ng×10^6) to day 3 (125.5 ng×10^6) then decreased to day 5 (56.1 ng×10^6; p < 0.05) IL-1β increased from baseline (68.8 ng×10^6) to day 3 (192.8 ng×10^6) then decreased to day 5 (62.6 ng×10^6; p < 0.05) IL-6 increased from baseline (240.9 ng×10^6) to day 3 (419.8 ng×10^6) then decreased to day 5 (212.1 ng×10^6; p < 0.05)

Table 2. Emergency service based sleep restriction and stress response studies – cont.

Reference	Sample	Intervention/ Design	Sleep restriction (SR)	Other stressors	Stress responses	Timing of samples	Main findings
Gundersen et al. [94]	male military cadets (N = 8)	7-day ranger-training course	intermittent sleep totalling ~7 h (1 h/24 h)	physical work and restricted energy intake	blood plasma cortisol and cytokines (TNF-α, IL-1β, IL-10, IL-1ra, IL-6); LPS-simulated (TNF-α, IL-1β)	7:00 a.m. baseline and during training on day 2, 4, 7	cortisol: increased from baseline (599±34 nmol×l⁻¹) to day 2 (1 072±82 nmol×l⁻¹, +79%) cytokines: IL-1ra increased from baseline (207±15 pg×ml⁻¹) to day 7 (841±437 pg×ml⁻¹, +306%; p < 0.05) IL-6 increased from below detection at baseline to day 2 (10.6±1.3 pg×ml⁻¹) and 4 (6.8±1.6 pg×ml⁻¹, p < 0.05) LPS whole-blood: TNF-α increased from below detection at baseline to day 7 (150.1±25.3 pg×ml⁻¹; p < 0.05) IL-1β increased from baseline (8.9±2.8 pg×ml⁻¹) to day 7 (45.2±6.3 pg×ml⁻¹, +408%; p < 0.05)
Slaven et al. [106]	male and female police officers (N = 391)	reported on sleep quality and depression symptoms	n.a.	n.a.	CES-D; PSQI	CES-D: once to assess depressive symptoms in 1 week PSQI: once to assess sleep quality and behavior in 1 month	correlation between CES-D and PSQI global score, and PSQI components (data not reported; p < 0.001) CES-D increased across increasing quintiles of PSQI for males (4.72, 5.60, 7.88, 12.67, 12.65; p < 0.001) and females (5.53, 6.21, 13.08, 10.88, 12.63, p = 0.001)
Bøyum al. [93]	male military cadets (N = 87)	5–7-day training course	intermittent sleep totalling 2–3 h	physical work and restricted energy intake	blood plasma cytokines (IL-1α, IL-6, IL-1β, IL-2, IL-3, IL-4)	between 6:00–7:00 a.m. each day	12–20% decrease in IL-6 on days 4–7 of training (p < 0.05) no significant change in other cytokines

Study	Sample	Protocol	Sleep	Stressor	Measure	Timing	Results
Goh et al. [76]	male military personnel (N = 14)	control night followed by single night of SR	control: 1×8 h sleep SR: 1×40 h	no	salivary cortisol	control and SR day 1: 8:00 a.m., 1:30 p.m., 6:00 p.m., 9:00 p.m., 12:00 a.m. control day 2: 8:00 a.m., 1:30 p.m., 6:00 p.m. SR day 2: 3:00 a.m., 6:00 a.m., 8:00 a.m., 1:30 p.m., 6:00 p.m.	cortisol increased at 13:30 h after SR (16.56 nmol×l⁻¹; +200%; p < 0.01) normal circadian cortisol rhythm
Opstad and Aakvagg [81]	male military cadets (N = 11)	5-day training course while receiving high or low calorie intake; follow-up 11 days post-training	training; intermittent sleep totalling 1–2 h follow-up: not reported	physical work and restricted energy intake	blood serum cortisol	Training: 6:30–7:30 a.m. (except day 4: 9:00–10:00 a.m.) and 6:00–7:00 p.m. follow-up: 6:30–7:30 a.m. and 6:00–7:00 p.m.	cortisol decreased from day 2 to 4 in both groups (low calorie: –220 μmol×l⁻¹, –31%; high calorie: –200 μmol×l⁻¹, –33%; sig and change not reported, estimated from graphs provided), and normal diurnal pattern had disappeared on days 1 and 4

n.a. – non-applicable; CES-D – Center for Epidemiologic Studies Depression Scale; IL – interleukin; OR – odds ratio; PSQI – Pittsburgh Sleep Quality Index; POMS – Profile of Mood States; LPS – lipopolysaccharide; SR – sleep restriction.

levels [84,85]. Therefore, which stressor or combination of stressors has the greatest effect on the participants' cortisol response remains to be determined.

Although both Opstad [80] and Opstad and Aakvaag [81] have observed a dysregulated cortisol response, different discrete parts of the cortisol circadian cycle were investigated in each study. For instance, in the more recent study, Opstad [80] has reported that daily cortisol secretion increased significantly over the 5-day training course, while the earlier study [81] reported a decline in the morning (i.e., 8:00 a.m.) cortisol production. Both increases and decreases in cortisol level have been demonstrated following stress exposure and could indicate allostatic load (i.e., wear and tear) on the endocrine system expressed as either an intensified or suppressed cortisol production [86,87]. The increased acute daily levels of cortisol have been associated with insulin resistance, which could accelerate the progression of type II diabetes, atherosclerosis and hypertension [44,45,88].

Conversely, persons exposed to chronic stress have demonstrated inadequate morning (salivary) cortisol levels one hour after awakening [89]. McEwen and Seeman [90] define an inadequate cortisol response as a form of allostatic load that occurs when the HPA-axis produces too little cortisol in response to a stressor, which as a result, causes immune mediators (e.g., inflammatory cytokines) and other systems that are normally contained by cortisol, to become overactive. Consequently, hyperactivity of these systems can increase the risk of auto-immune and inflammatory disorders (e.g., rheumatoid arthritis and multiple sclerosis) [27,90].

Methodological differences between the studies cited above could have contributed to the conflicting results for cortisol. For instance, single day cortisol sampling implemented by Opstad and Aakvaag [81] provides less stable measures of cortisol when compared to a multi-day sampling assessment [91], such as that adopted by Opstad [80]. Furthermore, the morning rise in cortisol

known as the cortisol awakening response depends closely on awakening time [92]. Therefore, variation in diurnal cortisol demonstrated between these studies could be also due to the differences in the time of cortisol sample collection after awakening. For instance, Opstad [80] and Opstad and Aakvaag [81] collected morning cortisol at 8:00 a.m. and between 6:30 and 7:30 a.m. respectively, yet neither study reported when the participants slept, limiting the ability to take into account what effect awakening time had on the morning cortisol levels in these studies. In addition, cortisol was examined by both Opstad [80] and Opstad and Aakvaag [81] using blood, while Goh et al. [76] used saliva samples. Evidence suggests that a high cortisol response can occur among individuals as a result of drawing blood (i.e., venepuncture) [79]. Therefore, the sampling methods could further explain different findings for cortisol between these studies [76,80,81].

The mixed findings for diurnal cortisol could also be due to differences in the duration and frequency of sleep deprivation and restriction examined. For instance, Goh et al. [76] have reported that a single night of total sleep deprivation had no effect on overall diurnal cortisol levels. Meanwhile, significant changes were reported following extreme periods of sleep restriction endured over consecutive days examined in the military training studies [80,81].

Sleep restriction and changes to cytokines

To date, research has investigated what impact sleep restriction during military operations has on pro- and anti-inflammatory cytokines [93,94]. For instance, Bøyum et al. [93] have investigated IL-6, IL-1α, IL-1β, IL-2 and IL-4 levels among military cadets before and during 5–7 days of a continuous military training combined with sleep (i.e., 2–3-h of total sleep) and calories restriction. Bøyum et al. [93] have found a –12–20% reduction in IL-6 on days 4–7 (p < 0.05) (Table 2) [93], but no

change in any of the other investigated markers. The decline in IL-6 is in contrast to the findings from the modest (3–6-h sleep per night) sleep restriction studies of similar duration, which have demonstrated an increase in daily cytokine levels among healthy adults following sleep deprivation [3,95,96].

Furthermore, Gundersen et al. [94] have found IL-6 levels to significantly increase from baseline to days 2 (10.6 ± 1.3 pg×ml^{-1}) and 4 ($+6.8\pm1.6$ pg×ml^{-1}) (Table 2) during a 7-day training course that comprised almost identical sleep restriction periods and physical work intensities to those investigated by Opstad et al. [80,81]. However, by completion of the course, IL-6 had returned to baseline levels [94]. In contrast, IL-1ra levels have been reported by Gundersen et al. [94] to increase throughout the training course ($+306\%$; p < 0.05) (Table 2). Furthermore, the pro-inflammatory cytokines TNF-α (from below detection to 150.1 ± 25.3 pg×ml^{-1}; p < 0.05) and IL-1β ($+408\%$; p < 0.05) were found to increase from baseline to completion of the training course (Table 2) [94].

Using an almost identical training protocol, Lundeland et al. [97] have investigated cadets' IL-6, TNF-α and IL-1β levels in LPS-simulated whole blood (Table 2). Findings for IL-6 were similar to those of Gundersen et al. [94], whereas TNF-α and IL-1β levels were also found to increase from baseline to day 3 ($+212\%$ and $+180\%$, respectively), then decreased to day 5 (-55% and -68%, respectively; p < 0.05) [97]. The increase and then the decrease in cytokine levels could indicate either an adaptation to the training course or possibly, failure to continue the same workload, however, this is difficult to determine with no performance data presented [97].

Interestingly, Bøyum et al. [93] have observed contrasting findings for IL-6 when compared to the studies by Gundersen et al. [94] and Lundeland et al. [97]. The decline in IL-6 observed by Bøyum et al. [93] over a short duration (i.e., 4–7 days) of activity was unexpected and suggested

by the authors [93] to be a result of plasma expansion due to excessive water intake. This particular cytokine does however, have both anti- and pro-inflammatory actions in the immune response [98]. Therefore, the significant reduction in IL-6 could indicate possible dysregulation of the immune system.

While no emergency service research has examined the effect of sleep restriction on inflammatory cytokines in a controlled setting, the findings from the wider stress response literature indicate that pro-inflammatory cytokines IL-6, IL-1β, IL-1ra and TNF-α significantly increase or decrease from baseline following single [77,99] as well as multiple nights [100] of complete and partial sleep restriction among healthy subjects. For instance, a single 40-h period of total sleep deprivation caused an $+89\%$ increase from baseline in TNF-α levels at 5:00 p.m. (p < 0.01) and $+95\%$ increase at 8:00 p.m. among healthy men (p < 0.05) (Table 3) [77]. A similar period (i.e., 40 h) of sleep deprivation investigated by Frey et al. [99] has also been found to induce a significant increase from baseline in the morning and afternoon levels of IL-1β (p < 0.05) (Table 2). Similar to Bøyum et al. [93], Frey et al. [99] have also found that severe sleep deprivation resulted in a decrease in IL-6 levels throughout most of the day (p < 0.05) (Table 3).

Conversely, Vgontzas et al. [100] have reported that the 24-h secretion of IL-6 increased when sleep was restricted to 6-h per night for one week (p < 0.05) (Table 3). Additionally, Vgontzas et al. [100] have found that a multi-day sleep restriction period was associated with an increased 24-h secretion of TNF-α in men only (p < 0.01) (Table 3). As previously mentioned, the inconsistent findings for IL-6 levels, could in part be due to the pro- and anti-inflammatory roles of this cytokine [98]. Furthermore, the variable findings for IL-6 indicate that this marker responds differently to a short period of total sleep deprivation (i.e., 40-h) compared to a week of modest sleep restriction [99,100].

Table 3. Healthy general population based sleep deprivation and stress responses studies

Reference	Sample	Intervention/ Design	Sleep restriction (SR)	Other stressors	Stress responses	Timing of samples	Main findings
Frey et al. [99]	healthy men and women (N = 19)	3 baseline days and nights, followed by single SR period and recovery sleep	baseline: 3×8 h sleep opportunities SR: 1×40 h SR recovery: 1×8 h sleep opportunity	no	salivary cortisol and blood plasma cytokines (IL-1β, IL-1ra and IL-6) subjective stress (visual analog scale)	cortisol: every 1 h beginning 2 h into SR period and ending after 38 h cytokines: every 30 min throughout SR period subjective stress: every 2 h	cortisol: no significant change subjective stress: increase at 10:00 a.m. (12 mm, +41%), 2:00 p.m. (10 mm, +33%), 4:00 p.m. (10 mm, +38%), 8:00 p.m. (11 mm, +35%), 10:00 p.m. (20 mm, +80%; all p < 0.05) all magnitude changes estimated from graphs provided cytokines: IL–1β increased at 9:00 a.m. (0.25 pg×ml^{-1}, +125%), 2:00 p.m. (0.20 pg×ml^{-1}, +114%), 3:00 p.m. (0.43 pg×ml^{-1}, +213%) and 4:00 p.m. (0.33 pg×ml^{-1}, +245%; p < 0.05) IL-1ra increased at 9:00 a.m. (35 pg×ml^{-1}, +23%), 10:00 a.m. (30 pg×ml^{-1}, +21%), 11:00 a.m. (20 pg×ml^{-1}, +13%), 7:00 p.m. (40 pg×ml^{-1}, +24%) and 12:00 a.m. (30 pg×ml^{-1}, +16%; p < 0.05) IL-6 decreased at 9:00 a.m. (−0.2 pg×ml^{-1}, −12%), 10:00 a.m. (−0.6 pg×ml^{-1}, −40%), 11:00 a.m. (−0.7 pg×ml^{-1}, −44%), 12:00 p.m. (−0.8 pg×ml^{-1}, −50%), 1:00 p.m. (−0.4 pg×ml^{-1}, −29%), 2:00 p.m. (−0.3 pg×ml^{-1}, −21%), 3:00 p.m. (−0.2 pg×ml^{-1}, −13%), 4:00 p.m. (−0.1 pg×ml^{-1}, −6%), 5:00 p.m. (−0.4 pg×ml^{-1}, −31%) and 6:00 p.m. (−0.2 pg×ml^{-1}, −13%; p < 0.05)

Reference	Population	Study design	Measures		Measures	Sampling	Results
Vgontzas et al. [100]	healthy adults (N = 25)	4 nights of normal sleep followed by 8 nights of partial SR	normal sleep: 4×8 h sleep opportunities SR: 8×6 h sleep opportunities PSG: each night	no	blood plasma cortisol and cytokines (TNF-α and IL-6)	cortisol and cytokines: 24 h sampling (8:00 a.m. to 7:30 a.m. next day) every 30 min on day 4 (i.e., baseline) and day 12 after SR	cortisol: peak morning cortisol decreased after SR (44.14 ± 16.55 nmol×l⁻¹, $p < 0.05$) cytokines: 24-h secretion of IL-6 (0.80 ± 0.3 pg×ml⁻¹; $p < 0.05$) and TNF-α (in men only; 0.26 ± 0.1 pg×ml⁻¹, $p < 0.01$) increased during SR
Thomas et al. [103]	healthy men and women (N = 31)	3 days and nights in a sleep laboratory	adaptation night: 1×7 h sleep opportunity sleep testing: 2×7 h sleep opportunities	no	fatigue (Vitality subscale from Health Survey); LPS cytokines (IL-6 and TNF-α)	cytokines: 11:00 p.m. pre-testing and 8:00 a.m. post-testing fatigue: evening following 1st night of testing	cytokines: evening IL-6 associated with fatigue ($r = 0.17$, $p = 0.05$) evening IL-6 negatively associated with SWS ($r^2 = 0.17$, $p = 0.29$) and positively associated with REM sleep ($r^2 = 0.26$, $p < 0.01$) fatigue: less SWS associated with fatigue the next day ($\beta = 0.55$, $p = 0.02$)
Chennaoui et al. [77]	healthy men (N = 12)	4 night SR experiment (2× baseline nights, 1×SR night and 1× recovery night)	baseline: 2×8 h sleep SR: 1×40 h SR period recovery: 1×8 h sleep PSG: continuous	no	plasma cortisol and cytokines (TNF-α, IL-6)	cytokines and cortisol: every 3 h during day 2 and 4 at 8:00 a.m., 11:00 a.m., 2:00 p.m., 5:00 p.m., 8:00 p.m. and 11:00 p.m.	cytokines: increase from baseline in TNF-α at 5:00 p.m. (+89%, $p < 0.01$) and 8:00 p.m. (+95%, $p < 0.05$) no significant change in IL-6 cortisol: no significant change

Table 3. Healthy general population based sleep deprivation and stress responses studies – cont.

Reference	Sample	Intervention/Design	Sleep restriction (SR)	Other stressors	Stress responses	Timing of samples	Main findings
Wu et al. [104]	healthy men (N = 10)	completed earlier-night SR followed by later-night SR, separated by 10 nights of unrestricted sleep	earlier-night SR: 1×7.4 h sleep, 4×3 h sleep (12:00–3:00 a.m.), 1×7.4 h sleep (recovery) later-night SR: 1×7.4 h sleep, 4×3 h sleep (3:00–6:00 a.m.), $1 \sim 7.4$ h (recovery) PSG: each night during SR	no	serum cortisol, STAI	STAI and cortisol: 7:00 a.m. each morning	STAI: positive correlations between anxiety and SR (earlier-night SR: r = 0.990, p = 0.001; later-night SR: r = 0.946, p = 0.015) anxiety increased after night 1 of SR (Z1 = 7.501, p = 0.006), in both conditions and remained high (Z2 = 12.643, p = 0.000; Z3 = 11.556, p = 0.001; Z4 = 9.682, p = 0.002) cortisol: both conditions caused a decreased morning cortisol from baseline to the 3rd (earlier-night: -102.6 nmol×l^{-1}; later-night: -17.6 nmol×l^{-1}; p = 0.046), 4th (earlier-night: -110.6 nmol×l^{-1}; later-night: -3.03 nmol×l^{-1}; p = 0.010) then returned to baseline on the recovery night negative correlation between cortisol and SR duration (r = -0.955, p = 0.012)
Kajtna et al. [105]	healthy men (N = 22)	completed either SR protocol (N = 9) or normal sleep conditions (N = 13)	SR group: 1×8 h sleep (adaptation), 1×40 h SR period and 1×8 h sleep (recovery) control: not reported PSG: recorded during adaptation and recovery nights	no	BRUMS, serum cortisol	BRUMS: administered in the evenings after adaptation night and SR period cortisol: every 4 h	BRUMS: change in fatigue (4.67, +140%) and vigor (-2.56, -74%; both p < 0.05) following SR cortisol: no significant change no correlation between BRUMS and cortisol

STAI – State-Trait Anxiety Inventory for adults; BRUMS – Brunel Mood Scale; PSG – polysomnography; SWS – slow wave sleep; REM – rapid-eye movement sleep.
Other abbreviations as in Table 2.

Sleep restriction and simultaneous cortisol and cytokine changes

In addition to cytokines, Gundersen et al. [94] have found soldiers' cortisol levels increased from baseline on day 2 (+79%) and day 4 (+74%) of training, while Lundeland et al. [97] have reported increased cortisol levels on day 3 (+22%) and day 5 (+73%). But similarly to IL-6, cortisol had returned towards baseline levels by day 7 (Table 2) [94,97]. The simultaneous increase of both cytokine and cortisol levels is similar to what has been observed among individuals with stress-related illness (e.g., depression, metabolic syndrome, CVD) [28,30,31,101] and therefore, could indicate dysregulation of the bi-directional feedback loop [28,30,31]. However, given that some of these markers returned towards baseline levels by completion of the military training, it is likely that the soldiers' endocrine and inflammatory processes were able to adapt to the stressors of sleep restriction and physical work to prevent adverse outcomes [94].

Similar to other military-based studies in this area [80,81,93], Gundersen et al. [94] have not controlled for sleep duration or frequency and the participants also performed continuous physical work and had a reduced energy intake. Both physical work and energy restriction are stressors capable of causing a change in cytokine [102] and/or cortisol levels [84,85]. Therefore, while these [94,97] and other multi-day military-based studies [80,81,93] provide an insight into the effect sleep restriction may have on personnel's acute cytokine and/or cortisol response, the lack of scientific control demonstrated in available military-based literature [80,81,93,94] clouds the true relationship between the stress of sleep restriction and immune and hormonal responses among emergency service personnel.

Furthermore, the demands investigated in these studies are military-specific. For instance, the extreme sleep restriction endured by military personnel differs somewhat to the moderate, partial sleep restriction civilian emergency service personnel, such as police, firefighters

and paramedics, are typically exposed to [55,60,61]. Yet, given the possible dysregultion of the cytokine and cortisol bi-directional feedback loop, further research should be focused on determining how varying amounts of controlled sleep restriction may affect emergency personnel's hormonal and immunological responses.

While Gundersen et al. [94] and Lundeland et al. [97] have reported that cortisol and cytokine levels were able to recover towards baseline, there is currently insufficient emergency service literature from which one could draw conclusions regarding the optimal recovery time for personnel exposed to sleep restriction. Therefore, further investigation is needed to determine, more specifically, the amount and/or number of recovery sleep(s) required for hormonal and inflammatory markers to recover following various types of emergency work (e.g., firefighting and police work).

Such investigations may assist emergency services in optimizing work/shift structures to minimize negative stress-related health outcomes while still meeting the unique staffing demands (e.g., on-call, response capabilities, and mobilizing for multi-day emergencies) of their organizations.

The impact of sleep restriction on mood state among emergency service personnel

In addition to physiological responses, restricted and/or poor quality sleep may also adversely affect emergency workers psychological functioning [31,82,83]. Slaven et al. [106] have investigated self-reported depressive symptoms and sleep quality among police officers who completed the Centre for Epidemiological Studies Depression (CES-D) and Pittsburgh Sleep Quality Index (PSQI) questionnaires. The findings revealed strong correlations between both measures.

For instance, mean CES-D depressive symptom scores increased across increasing quintiles of the PSQI global sleep quality score for males and females (p < 0.001),

indicating that depressive symptoms in male and female officers significantly increased as subjective sleep quality deteriorated [106]. However, the use of self-report measures when investigating sleep may be negatively affected by reporting/recall bias, demonstrated by a propensity to subjectively overestimate sleep length [107]. While it should be noted that the CES-D is highly sensitive to sleep, which is reflected in this study by the strong correlation between depression and sleep among officers, future research would benefit from more objective sleep measures (e.g., activity monitors, polysomnography). Furthermore, the cross-sectional design of this study [106] limits the ability to make causal inferences.

Prospective study designs, multi-day military-based studies have examined soldiers' psychological responses to periods of objectively measured (i.e., activity monitors) sleep restriction and near constant physical work [82,83]. Lieberman et al. [83] have assessed the mood state of soldiers before and after a 53 h continuous physical combat training exercise in which they had 3±0.3 h of total sleep. Using the Profile of Moods States (POMS), Lieberman et al. [83] have reported a significant change from pre- to post-training in each of the mood subscales (i.e., tension, depression, confusion, fatigue, anger and vigor) (Table 2). In a more recent study, Lieberman et al. [82] have assessed soldiers' mood also using POMS during an 84-h laboratory-based military training simulation, which controlled for the length and frequency of sleep opportunities (i.e., 7×1-h sleep breaks) and the type and duration of military activities per day. Over the course, the soldiers had a total of 6.2±0.4 h of sleep, and like their earlier study, all mood subscales significantly worsened over the duration of the training (Table 2). These findings indicate that decrements in mood can persist with 6 h of total sleep while performing an extended (i.e., 84-h) period of physical military work under more controlled laboratory settings.

Both the above mentioned studies [82,83] failed to include a control group. Consequently, it is difficult to determine how much of the reported change in psychological responses were due to the lack of sleep, or a combination of other stressors present (e.g., physical work and energy restriction). Therefore, it is not possible to know which of the stressors, or which combination of stressors is the most damaging. Accordingly, caution should be taken when generalizing findings from multi-day military-based studies to other emergency services (e.g., firefighting, police, emergency medical) routinely exposed to different occupational demands (e.g., only partial sleep deprivation or more intermittent physical work) [52].

Furthermore, these studies [82,83] used wrist-worn activity monitors to determine the participants' sleep duration and frequency. Although suited to field research, caution should be taken when using activity monitors to measure more complex sleep parameters (e.g., sleep architecture) [108]. As a result, it is difficult to determine for certain whether the psychological stress responses observed in these studies are attributed to reduced sleep duration alone or changes to sleep architecture.

Impact of sleep restriction on psycho-physiological stress responses among emergency service personnel

To date only a limited number of studies have investigated psycho-physiological responses to sleep restriction [103,105,109,110] and none has been an emergency service-based study. Thomas et al. [103] have reported that evening stimulated production of IL-6 in healthy adults was weakly associated with subjective feelings of fatigue the next day (r = 0.17; p = 0.05) and have concluded that this relationship was mediated by a reduced amount of slow wave sleep (SWS). Indeed, earlier studies have also reported that increases in circulating levels of IL-6 correlate with decreases in SWS [111,112].

A number of studies have also examined interaction between cortisol and psychological responses to various stressors [25,48], yet only a small number of studies have investigated how sleep restriction, specifically, may affect

the interaction between these responses [105,109,110]. Furthermore, such studies have generally focused on participants with either a sleep disorder or mental illness [109,110] and therefore, were not included in this review. Indeed, to the best of the authors' knowledge, only one study has examined interaction between subjective mood (assessed using the Brunel mood scale) and cortisol levels pre and post a 40-h period of sleep deprivation [105]. However, no correlation has been reported between responses in this study [105]. As such, future research needs to determine whether psycho-physiological relationships exist among healthy emergency responders (free of clinical mental and/or sleep disorders) exposed to acute sleep restriction on the job.

While emergency service research is yet to examine statistical relationships (e.g., correlation) between psycho-physiological changes to sleep restriction, Lieberman et al. [83] have simultaneously investigated soldiers' psychological and physiological responses to sleep loss during simulated combat. As previously described, the soldiers received minimal sleep and had a restricted energy intake while completing a field-exercise that involved almost continuous physical work in an intermittently hot environment (Table 2) [83]. The soldiers completed the POMS questionnaire pre-, during and post-field and cortisol was measured at 6:00 a.m., 12:00 p.m. and 6:00 p.m.

The results demonstrated that the soldiers' mood, including vigor, fatigue, confusion, depression and tension, significantly deteriorated from pre- to post-field (Table 2) [83]. Furthermore, the *post hoc* analysis of cortisol revealed lower levels in the morning and higher levels in the evening during the field-exercise compared to the pre-field levels [83]. In addition, evening cortisol measurements on day 3 and 4 during the combat training were higher than pre-training samples [83]. The low morning and high evening cortisol levels reported by Lieberman et al. [83] could indicate a disrupted cortisol circadian rhythm. Indeed, previous research has demonstrated that

a low awakening cortisol response and high evening cortisol levels have both been associated with negative health outcomes (e.g., depression) [113].

On the other hand, the increase in the evening cortisol levels reported by Lieberman et al. [83] could also indicate that the participants were more active than usual at these times due to the around-the-clock physical work involved in the military training. Consequently, an inadequate (i.e., low) cortisol response in this instance could be problematic and indicate another component of allostatic load [90]. Given how the cortisol response to different stressors can vary (e.g., prolonged and inadequate responses), yet still indicate possible dysregulation of the HPA-axis highlights the need to investigate all aspects of this physiological stress response in future studies.

Moreover, investigating how the cortisol response interacts with other psychological stress responses could help to further contextualize the significance of the response [28]. Indeed the acute psychological and physiological findings by Lieberman et al. [83] may demonstrate that the deterioration in mood during the military training could be associated with a disrupted cortisol response (i.e., low morning and high evening levels). Yet without statistical analysis of the interactions between these markers, it is difficult to determine what the psycho-physiological relationship was (under these conditions).

The lack of a control group is a further limitation of this study [83], making it challenging to determine if the findings were due to sleep restriction, other stressors (i.e., heat stress, energy restriction and physical work) or a combination of these demands. However, this study [83] represents the only available emergency service-based research that examines concurrent changes in the acute psychological and physiological responses to sleep restriction.

Laboratory-based sleep studies [99,104] have also demonstrated concurrent physiological and psychological changes similar to those observed in the field by Lieberman et al. [83]. Using a crossover study design, Wu et al. [104]

have investigated the effect of an earlier-night (i.e., sleep from 12:00 to 3:00 a.m.) and later-night (i.e., sleep from 3:00 to 6:00 a.m.) sleep restriction protocol on subjective anxiety (measured using the State-Trait Anxiety Inventory; STAI) and cortisol levels. For each condition, the participants completed an unrestricted baseline sleep, 4 nights of sleep restriction followed by a recovery night and blood samples (for cortisol analysis) were taken each morning (at 7:00 a.m.) [104].

In both conditions, STAI increased from baseline after the 1st night of reduced sleep and then continued to increase each day for the duration of the conditions [104]. Furthermore, positive correlations between STAI and sleep restriction (earlier-night sleep restriction: r = 0.990, p = 0.00; later-night sleep restriction: r = 0.946, p = 0.015) were reported and both sleep periods resulted in the reduced morning cortisol levels [104]. Following the recovery night, cortisol and STAI in both conditions returned towards normal, but only cortisol reached baseline. While this study explored more controlled sleep periods, additional daily samples were needed to provide a more detailed measure of circadian changes in hormonal and mood responses.

Furthermore, the lack of statistical analyses between the psychological and physiological responses limits the ability to investigate any possible psycho-physiological interactions in response to the periods of sleep restriction examined.

In more extreme cases (e.g., natural or man-made disasters such as large bushfires/wildfires), emergency personnel have reported continuous periods of extended wakefulness lasting for more than 24-h [55]. When examined in controlled laboratory conditions, total sleep deprivation was found by Frey et al. [99] to elicit simultaneous physiological and psychological changes among healthy adults. For instance, Frey et al. [99] have reported a simultaneous increase in inflammatory cytokines (IL-1ra, IL-1b) in the morning and evening and higher subjective stress levels

at most time points across the 40-h sleep deprivation period compared with baseline (p < 0.05). However, no significant effects for salivary cortisol levels were detected in this study.

CONCLUSIONS

A single night of sleep deprivation (either partial or full) may not be a sufficient stressor to significantly affect the overall daily release of cortisol among military emergency responders [76]. However, extreme sleep restriction over multiple days of emergency work (e.g., 1–7 h of sleep over 2–7 days) can:
– disrupt the circadian cortisol rhythm [80,81,83],
– disrupt (i.e., above and below baseline or control levels) pro- (i.e., IL-6, TNF-α and IL-1β) and anti-inflammatory cytokine levels (i.e., IL-1ra) [93,94,97],
– elicit adverse psychological responses (i.e., deterioration in mood) [82,83],
– cause a simultaneous increase in both cortisol and cytokine levels (i.e., IL-6) [94,97].

Taken together, this literature informs emergency services that exposure to more than one night of severe work-related sleep restriction experienced between shifts may influence their personnel's acute physiological (i.e., cortisol and cytokine levels) and psychological (i.e., mood) functioning.

The significance (i.e., abnormal or normal) of acute changes in physiological and psychological responses to restricted sleep experienced at work is of further importance to emergency services. Extreme sleep restriction over consecutive days of military training can result in the concurrent deterioration of both mood and disrupted diurnal cortisol levels [83,104]. However, interactions between these responses have not yet been statistically analyzed and therefore, it is difficult to determine, for certain, if a psycho-physiological relationship exists between these markers (under these conditions) among personnel.

Healthy individuals have also demonstrated adverse simultaneous, but not statistically evaluated, changes in multiple physiological (i.e., cortisol and cytokines) and psychological (i.e., STAI) responses following consecutive nights (lasting up to a week) of sleep restriction [100,104]. Conversely, the evidence of psycho-physiological interactions between stress responses following a single night of sleep deprivation remains equivocal [77,99,105]. However, it appears that the release of pro-inflammatory cytokines (i.e., IL-6) positively correlates with feelings of fatigue [103], indicating the negative effect immune dysregulation may have on general well-being.

Moreover, this finding offers some limited empirical support for the use of non-invasive measures of fatigue to evaluate immune function. However, on the whole, evidence investigating psycho-physiological responses to sleep restriction is still very limited and inconclusive. Therefore, it is not currently feasible for uniform services to develop practical means to efficiently monitor the acute stress of their personnel in the field, based on psycho-physiological interactions.

To date, only a limited number of studies have investigated the effect of work-related sleep restriction on acute stress responses among different forms of emergency personnel. In fact, this review of the literature uncovered only one police-based study [31] and no firefighting or emergency medical-related investigations. Meanwhile, the overwhelming majority of the research was focused on multi-day military studies [80–83,93,94,97]. As such, the periods of extreme sleep restriction (i.e., 1–2-h per 24-h) investigated are, in most cases, military specific. Furthermore, military-based studies expose personnel to stressors (such as continuous physical military activities and food and fluid restriction) not commonly experienced during more civilian emergency service work (e.g., firefighting and police work) [55,114].

Consequently, extrapolating findings from the defense occupations with different workloads to more civilian emergency services may under or overestimate the potential stress-related implications and lead to inappropriate advice/recommendations regarding sleep opportunities. Additionally, most military field-based studies have not controlled for sleep duration or frequency and failed to include a control group matched for age, sex or work experience.

As a result, it is difficult to determine how much of the reported change in physiological or psychological responses were due to sleep restriction, other occupational stressors or a combination of stressors and therefore, if sleep restriction (or a combination of stressors) was the most damaging stressor. Regardless, it appears that sleep restriction may be a major occupational stressor contributing to the reported physiological and psychological responses and therefore, warrants further investigation.

FUTURE RESEARCH DIRECTIONS

To further understand the impact of work-related sleep restriction on acute stress responses and to provide emergency services with the knowledge they need to protect the health of their personnel, future research should focus on:
– a wider range of emergency services,
– practical methods to monitor physiological and psychological health of the personnel exposed to work related sleep restriction,
– controlled periods of sleep restriction similar in duration to what is reported/experienced among civilian emergency services,
– concurrent measurement of multiple stress responses and statistical analyses of the psycho-physiological interactions (if any) between these responses,
– all aspects of the cortisol and cytokine response (e.g., normal, inadequate and prolonged responses) that characterize the nature and possible dysregulation of these systems,
– the amount and/or number of recovery sleep(s) required for cortisol and cytokine levels to return to baseline following work-related sleep restriction.

While the acute effect that work-related sleep restriction has on stress remains an important focus, future research could also benefit from longitudinal/follow-up studies to further understand the possible link between this stressor and negative long-term health outcomes. For instance, examining how stress responses recover following exposure to sleep restriction (via post/follow-up testing) during emergency work may provide insights into how acute responses translate into chronic physiological and psychological changes and ultimately, result in adverse long-term health-outcomes.

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