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Advancing the allostatic load model in military training research: from theory to application

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Research physiologists use theoretical models to test new empirical relationships between physiological variables and psycho-physiological outcomes and compare observed outcomes with theoretical predictions to support or refute models. Models, while valuable, often focus on a limited perspective as part of a larger reality. In understanding Warfighter health, a more holistic perspective within a model is needed since this population is exposed to a high degree of physical, cognitive, and emotional demands/loads during training throughout a career. Focusing on the physical performance aspects of occupational exposures is important; however, this neglects imperative interrelationships between the psychological and musculoskeletal domains of health, which must be quantified for early in-field prevention of injury, underperformance, or psychological harm. Chronic duration of the physiological stress response may disrupt adaptive mechanisms and result in allostatic load, characterized as a maladaptive biological process by which physiological stability ('allostasis') fails owing to repeated and chronic stress exposure, which can negatively affect physical and cognitive function. It may also increase vulnerability to atypical reductions in occupational physical performance and psychological and musculoskeletal health. The purpose of this review was to (i) summarize empirical research of atypical, negative consequences of military training on physical performance and psychological and musculoskeletal health (ii); reconsider the underlying biological process rendering maladaptive outcomes observed during training by leveraging a 'stress perspective' wherein military training-related stressors perturb stress systems and lead to allostatic load, which may serve as a mechanism by which maladaptation occurs; (iii) summarize the impact of allostatic load quantified by the Allostatic Load Index (ALI) on physical performance, psychological wellbeing, and musculoskeletal health; and (iv) propose the use of valid and reliable commercially-available wearable devices as tools to measure allostatic load by collecting longitudinal cardiometabolic and neurobehavioral (sleep) data during training and determining verifiable signals associated with ALI and maladaptive outcomes. Allostatic load is an evolving model that may be suited to understand the long-term health effects of military training-related stress. There is opportunity to improve our understanding of measurement tools involving wearables to establishing the

relationship between allostatic load and long-term health outcomes in military personnel.

KEYWORDS

stress, regulation, allostasis, wearable, performance, musculo skeletal disorder, psychological distress

1 Introduction

Warfighters are characterized as military service members directly engaged in combat-related roles and/or military operations (Nindl et al., 2017). In the United States (US), Warfighter readiness, lethality, and resilience remain a continued priority in the US Armed Forces as evidenced by the enacted “Military Readiness and Injury Prevention Act of 2019” (S.1860) (Moran and Smith, 2019), the Holistic Health and Fitness (H2F) Initiative of 2020 (U.S. Army Center for Initial Military Training, 2023), the Brandon Act of 2023 to advance mental health supportive programs (Military Health System, 2023), and the Defense Health Agency’s 6-year advancement plan (2021–2027) to leverage fitness wearable devices for measuring and promoting readiness (Cisneros, 2023). Together, such initiatives may define the optimal Warfighter as one who is healthy enough to operate on short notice with or without appropriate recovery, resilient enough to overcome environmental, internal (biogenic, physiological), and external (mechanical, social) stressors while maintaining occupational role performance, and robust against the occurrence of musculoskeletal injury (MSKI) (Moran and Smith, 2019; U.S. Army Center for Initial Military Training, 2023; Military Health System, 2023; Cisneros, 2023).

Pertinent to the operational success of the Warfighter is the execution and completion of military training courses to learn and excel in physical, academic, and tactical skills for deployment, accrue individual military rank, and extend one’s military career (Drain et al., 2015; Caspar et al., 2020; Bartlett et al., 2015). Warfighters spend considerable amounts of time each year enrolled in such courses, ranging between eight and 12 weeks for the majority of courses (Bulmer et al., 2022a; Alemany et al., 2008; Henning et al., 2014; Harman et al., 2008), with specific curricula ranging four to 6-weeks in duration (Edgar et al., 2021). Although their curriculum aims to enhance physical and psychological readiness for deployment (Flanagan et al., 2012), empirical research over 3 decades pinpoints the atypical negative effects of training, including MSKIs (Jones et al., 2010), worsened physical performance (Burley et al., 2018) and lower psychological wellbeing (Bulmer et al., 2022a). Together, these effects contribute to attrition owing to deterred medical or psychological health (Tait et al., 2022; Forse et al., 2024) and financial burden on healthcare systems (Dijksma et al., 2020).

Prevention initiatives within the US Armed Forces that aimed to tackle such negative effects observed during training have implemented deductive approaches wherein accrued findings from national databases of military health records result in advanced programs to address prevention, early identification, and management of negative effects (Cooper et al., 2024). Such programs also aimed to rescue financial security in the military domain (Cooper et al., 2024). Previous research demonstrated beneficial results of programs targeting musculoskeletal (Wardle

and Greeves, 2017), physical fitness (Burley et al., 2020), and psychological health (Adler et al., 2015) when employed independently. However, although holistic health programs, such as the H2F Initiative, which aims to implement preventive care for soldiers due to increasing sleep and mental health concerns, and MSKI rates (Culley and DaLomba, 2025), have been a mainstay within US brigades since 2020, few interventions have assessed its effectiveness on musculoskeletal, physical fitness, and psychological health (Cooper et al., 2024). A 2025 study observed an increase in awareness of H2F in soldiers without assessing its effectiveness on targeted outcomes (Culley and DaLomba, 2025). Hence, as opposed to employing a deductive mechanism that relies on a reactive technique that leads to actionable programs, implementing an inductive mechanism by reconsidering the underlying biological processes that may render negative outcomes, such as allostatic load (McEwen, 1993), may improve our understanding of their development and advance preventive means.

Allostatic load is a theoretical biological framework that outlines a maladaptive biological process wherein physiological stability, known as allostasis (Sterling, 1988) (Figure 1), characterized as the constant dynamism of physiological activity of biological systems that appropriately responds to stressors to maintain homeostasis, fails owing to dysregulated primary mediator activity from chronic stress exposure and reflects the cumulative physiological burden (McEwen BS., 1998). Allostatic load is a stress regulation model that describes how the ‘cost’ of adaptation to chronic stress exposure may result in multi-system dysregulation (Sterling, 1988) (Figure 2A). This model is reflected by the presence of *primary outcomes* or effects, characterized as the degradation of protective mechanisms that mediate physiological stress responses (i.e., desensitization of glucocorticoid receptors) and *secondary outcomes* or effects (Guidi et al., 2020), characterized as consequential physiological responses from primary outcomes reflected by heightened cardiometabolic and altered neurobehavioral (i.e., sleep) health (McEwen, 2006; Logan and Barksdale, 2008; Feigel et al., 2024a), as well as adverse behavioral responses, such as worsened physical performance (Germano et al., 2023), sleep quality (Christensen et al., 2022), self-appraised resilience (Felix et al., 2023), and perceived stress appraisal (McEwen, 2007). *Tertiary outcomes* emerge from secondary outcomes, such as pain syndromes, musculoskeletal disorders, and illnesses (Feigel et al., 2024a; Beckie et al., 2016; Parker et al., 2022) (Figure 2A). Taken together, this model highlights the influence of the environment, individual variation, and brain-body interactions to serve as a process by which chronic stress may result in secondary (i.e., poor fitness) and tertiary outcomes (i.e., MSKI) during training (McEwen BS., 1998; Feigel et al., 2024a).

Allostatic load is measured by its traditional operationalization, the Allostatic Load Index (ALI) (Figure 2B), which comprises

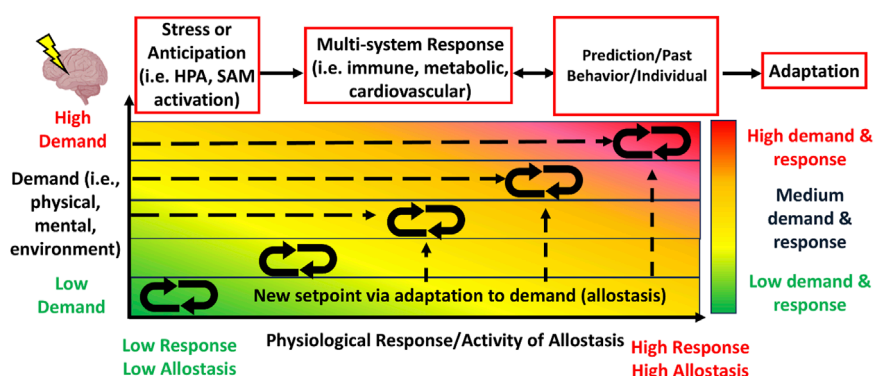


FIGURE 1

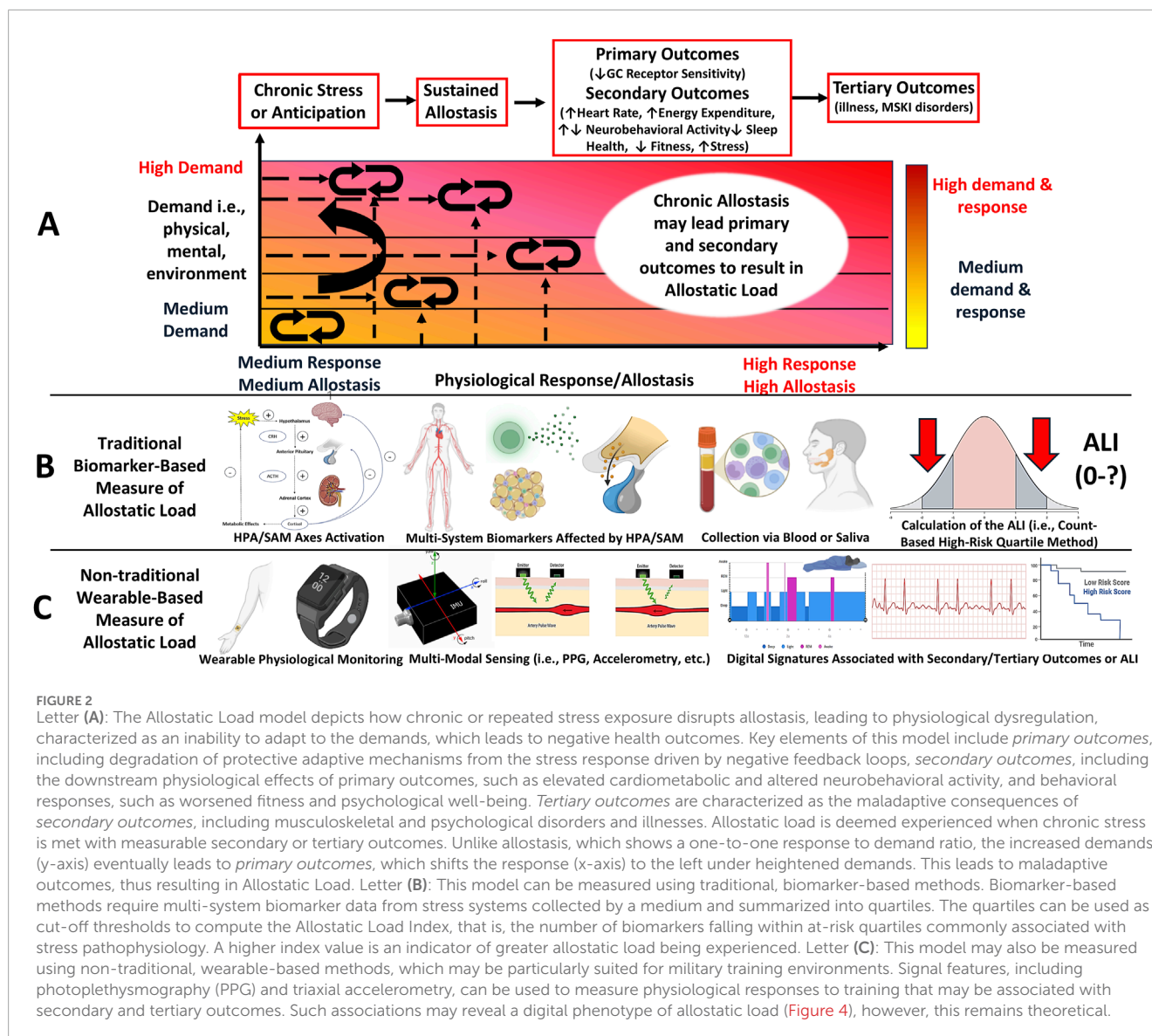
The allostasis model illustrates the constant dynamism of physiological activity of stress-related systems, including activity from the hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medullary (SAM) systems, that serve to appropriately respond to physical, mental or environmental stressors in order to adapt to the physical, mental or environmental demands in a one-to-one ratio. Stress or anticipation of a stressor activates a physiological response to which leads to multi-system (i.e., immune, cardiovascular) responses. The physiological response is mediated and influenced by individual prediction of the stressor, one's past experience or behavior, and individual factors (i.e., age), resulting in adaptation. With each increase in demand requires a met response of the body, which always stays in dynamic flux (McEwen, 1993).

a count-based composite score representing the number of biomarkers affected by chronic psycho-physiological stress across neuroendocrine, autonomic, and immune systems (Juster et al., 2010; Seeman et al., 1997). The ALI score is determined by the number of biomarkers collected with higher scores indicating higher allostatic load (McLoughlin et al., 2020; Carbone et al., 2022) (Figure 2B). The use of the ALI to quantify allostatic load has grown considerably (Juster et al., 2010; Seeman et al., 1997; Geronimus et al., 2006). This has been shown in epidemiological studies (Carbone, 2021; Andrzejak et al., 2023; Bruun-Rasmussen et al., 2024), which have associated ALI with several negative health outcomes, including subclinical risk factors for cardiovascular disease (Logan and Barksdale, 2008), heightened morbidity and mortality rates (Parker et al., 2022; Bruun-Rasmussen et al., 2024), accelerated mechanisms of aging (Seeman et al., 1997), psychological disorders (Beckie et al., 2016; Berger et al., 2019; Berger et al., 2018), physical locomotor dysfunction and musculoskeletal disorders (Germano et al., 2023), reduced muscular strength and postural balance (Germano et al., 2023; Hansen et al., 2016), and worsened psychological wellbeing, such as symptoms of anxiety (Gou et al., 2025; D'Alessio et al., 2020) and depression (Gou et al., 2025) and lower resilience (Felix et al., 2023), sleep quality (Christensen et al., 2022), and perceived stress (Guidi et al., 2020; Beckie et al., 2016; Juster et al., 2010; Geronimus et al., 2006).

Recent work from our group observed an increased ALI score following a 10-week military training course in both sexes (males: +2 ALI (5 out of 8); females: +1 ALI (4 out of 8)), and observed an association with worsened physical performance to support this framework as a useful model outlining a biological process associated with maladaptive training-related outcomes (Feigel et al., 2025a). Since the origination of the ALI in 1997 (Seeman et al., 1997), the number of biomarkers and algorithms used for the ALI has grown (McLoughlin et al., 2020; Carbone et al., 2022). However, this method has limitations for longitudinal study designs (Feigel et al., 2025a; Magtibay and Umapathy,

2023), including obtaining several biomarkers multiple times over a study duration, which may render the ALI a challenging method for military training environments (Feigel et al., 2025a). Therefore, as an alternative approach, recent studies have begun to employ non-traditional, commercial wearable-based methods for its assessment (Magtibay and Umapathy, 2023; Corrigan et al., 2021; Corrigan et al., 2023) (Figure 2C). Wearable-based methods may be a promising approach for allostatic load assessment owing to devices' noninvasive wear and their ability to capture high-resolution physiological time-series data from multiple sensors (Figure 2C). Importantly, such tools may reveal digital signatures and serve as a proxy measures of the ALI (Magtibay and Umapathy, 2023). However, empirical research on this phenomenon remains unexplored. Moreover, although the ALI has been examined in military populations (Feigel et al., 2025a), limitations for wearable-based methods, such as their methodological variability and data quality, should be considered for its feasibility for employment (Feigel et al., 2024a; Magtibay and Umapathy, 2023).

The purpose of this review is to (i) summarize the empirical research of atypical, negative consequences of military training on physical performance and psychological and musculoskeletal health (ii); reconsider the underlying biological process rendering maladaptive outcomes commonly observed during training by leveraging a 'stress perspective' where military training-related stressors, such as energy restriction, physical overtraining, cognitive distress, and sleep deprivation, perturb stress systems and lead to allostatic load, which may serve as a mechanism of training-related maladaptation; (iii) summarize the empirical research of allostatic load quantified by the ALI on physical performance, psychological wellbeing, and musculoskeletal health; and (iv) propose the use of valid and reliable commercial wearable devices as tools to measure allostatic load by collecting longitudinal cardiometabolic and neurobehavioral data during training and determining verifiable signals associated with ALI and maladaptive outcomes. Such findings may reveal a 'digital phenotype' of allostatic load for in-field detection



and introduce a field-expedient method to identify personnel at-risk for maladaptive outcomes that occur during military training courses.

2 Musculoskeletal, physical performance and psychological maladaptation to military training

In the US Armed Forces, across all branches (Army, Navy, Air Force, and Marines), ~5% of all hospitalizations have been attributed to MSKIs, with ~90% classified as ‘non-combat’ MSKIs (Hauret et al., 2010). ‘Non-combat’ MSKIs are characterized as those MSKIs that occur not from ballistic weaponry, improvised explosive devices (IED), parachuting, or vehicular accidents (i.e., helicopter crashes), and remain the leading cause of outpatient medical care in the US Army of active duty personnel, with

two million encounters each year (Molloy et al., 2020a). ‘Non-combat’ designated MSKIs of the upper and lower body regions account for 80% of all observed MSKIs (Molloy et al., 2020a; Molloy et al., 2020b), 50%–75% of those sustained in the lower body, including lumbar/sacral spine, pelvis, and lower extremity, MSKIs (Jones et al., 2010; Hauschild et al., 2018; Lovalekar et al., 2018). Notably, the hip, ankle, and foot account for 14%, 12%, and 12% of all lower body MSKIs, respectively (Hauschild et al., 2018). Importantly, ~60% of limited duty days and 65% of non-deployable Warfighters have been attributed to non-combat MSKIs (Flanagan et al., 2012; Jones et al., 2010). The majority (~85%) (Hauschild et al., 2018) of MSKIs sustained are categorized as ‘mechanical’ owing to external shear forces induced upon the musculoskeletal system, with ~75% categorized as ‘overuse’ owing to the cumulative microtrauma (i.e., repetitive stress) and 10% as non-contact acute trauma (non-ballistic) MSKIs (Jones et al., 2010; Hauschild et al., 2018; Jensen et al., 2019).

Overuse MSKIs observed during military training range in type from bone stress fractures (Molloy et al., 2020a; Molloy et al., 2020b; Koltun et al., 2022), inflammation and pain (Hauret et al., 2010), sprains and strains (Lovalekar et al., 2018; Lovalekar et al., 2023; Lovalekar et al., 2021), and even soft tissue degenerative diseases, such as osteoarthritis (Showery et al., 2016; Knapik et al., 2018). Lovalekar et al. (2023) (Lovalekar et al., 2023) observed a cumulative MSKI incidence of 39.7% in women and 23.1% in men among 736 US Marine candidates ($n = 131$ women), with ~65% categorized as overuse MSKIs (Lovalekar et al., 2023). Overuse MSKIs have been suggested to occur from the rigorous and physically demanding training involved (Allison et al., 2017). Additionally, overuse MSKI is an important cause of attrition, disability, and loss of military readiness (Songer and LaPorte, 2000) and high financial cost (Lovalekar et al., 2018). A 2018 study assessing the cost of such MSKIs among Air Force Special Tactics Operators showed that the total lifetime cost sustained by them during only a 1-year period was US \$1.2 million (Lovalekar et al., 2018). Notably, a 2000 retrospective cohort study describing the MSKI occurrence during a 6-week United States Marine Corps (USMC) Officer Candidates School (OCS) course among 480 candidates ($n = 30$ women) observed a cumulative incidence (one or more MSKIs) of 60.7% (women: 80.0%; men: 59.5%) with overuse MSKIs making up 65.2% of all encounters in men, and 70.3% of encounters in women. Together, overuse MSKIs were responsible for 0.62 and 1.67 lost training days per man and woman, respectively (Piantanida et al., 2000). However, a 2023 retrospective cohort study of OCS candidates undergoing this course reported a lower cumulative injury incidence of 39.7% in women ($n = 52$) and 23.1% ($n = 140$) in men. Although these results show improvement in overuse MSKI rates, further analysis reveals that such MSKIs remained the predominant MSKI type in both sexes (women: 66.2%; men: 65.4%), suggesting that they remain a medical challenge as they were 2 decades ago (Lovalekar et al., 2023).

Although overuse MSKIs remain a prevalent challenge in contemporary training courses, recent evidence also observes atypical reductions in physical performance following training in both sexes (Brock and Legg, 1997; Booth et al., 2006; Tanskanen et al., 2011; Givens et al., 2023a). Physical fitness is emphasized as a critical element for advancement early in military servicemember careers (Agostinelli et al., 2022) that predict the success of military job-task roles (Bartlett et al., 2015; East et al., 2017). This is due to the physically demanding and commonly recurring gender-neutral tasks soldiers are required to perform in ground close combat roles (Nindl et al., 2015; Szivak and Kraemer, 2015). Additionally, the uplift of bans for women preventing enrollment in ground close combat in nations of the North Atlantic Treaty Organization (NATO) has positioned research on the physiological effects of physical training on male and female servicemembers to the forefront (Fitriani and Matthews, 2016; Sterczala et al., 2023; Feigel et al., 2024b). Recent research has shown that lowered fitness during training may pose problems for role performance (Alemany et al., 2008; Allison et al., 2017), and risk of detrainment and lethality during deployment (Pihlainen et al., 2023). Decreases of 15%–20% in aerobic capacity and 10% in maximal muscular strength have been observed in both sexes following training (Häkkinen et al., 1985). Burley et al. found that 15% and 14% of recruits showed a significant decline ($\geq 5\%$) in maximal

muscular strength assessed by a 1 repetition-maximum box lift and local muscular endurance assessed by a maximum number of pushups achieved in 2-min (Burley et al., 2018). Further analysis from this same investigation observed that a total of 7% and 4% of the sample reduced in estimated $\text{VO}_{2\text{peak}}$ and 3.2 km load carriage performance ($\geq 5\%$), respectively (Burley et al., 2018). Similarly, Givens et al. observed no significant improvement in upper body muscular endurance (count: 6 ± 1 vs. 6 ± 1 , $p > 0.05$) or aerobic capacity ($25:14 \pm 0:15$ vs. $24:51 \pm 0:15$, $p > 0.05$) following a 10-week course in female US Marines (Givens et al., 2023a). Although these results also depend upon training length and specificity (Coge et al., 2024), fatigue accumulation (Heilbronn et al., 2023), and motivation (Myllylä et al., 2023), such results demonstrate a divergent performance response to training in cohorts undergoing identical physical training curricula.

In addition to observed atypical reductions in physical performance, previous investigations have observed individuals (10%–25%) (Robinson et al., 2009) demonstrating lower levels of psychological wellbeing during training that can contribute to volitional attrition rates up to 25.8% (Forse et al., 2024). Lower psychological wellbeing during military training (Forse et al., 2024; Beckie et al., 2016; Bulmer et al., 2022b; McFadden et al., 2024a) may be characterized as the independent or combined experience of one or more of the following: (i) lower self-appraised psychological resilience (Forse et al., 2024; Nindl et al., 2018; McFadden et al., 2024b), a dampened degree to which an individual copes in stressful situations or during times of adversity (Connor and Davidson, 2003), (ii) higher perceived stress appraisal, a heightened degree of self-reported perception of situations being particularly stressful (Cohen et al., 1983), and (iii) worsened subjective sleep difficulty, a higher degree to which individuals feel that they struggle to attain a sufficient night of sleep (Bender et al., 2018; Kargl et al., 2024). Recent investigations have observed a link between the aforementioned symptomatology and risk of readiness for deployment (Paxton et al., 2024), and incidence of psychological disorders (Beckie et al., 2016), including post-traumatic stress disorder (PTSD) (Abouzeid et al., 2012). Active duty personnel are likely to worsen symptoms concerning suicidal ideation (OR = 1.90, 95% CI = 1.20–2.90) following training compared to reservists (Robinson et al., 2009). A retrospective cohort study that assessed a battery of psycho-physiological characteristics of 1006 OCS candidates (79.5% male) observed that lower self-appraised resilience was amongst the main predictors of attrition (Forse et al., 2024). Although perceived stress has been shown to improve cognitive focus and motivation to foster effective learning (Ross et al., 2024), chronic, heightened levels of stress can dampen cognitive performance (Ross et al., 2024) and risk discharge (Taylor et al., 2009). Taylor et al. (2009) (Taylor et al., 2009) observed that perceived stress was directly associated with acute stress biomarkers, whereas active coping ability was not, suggesting that perceived stress plays a fundamental role in the physiological stress response (Taylor et al., 2009). Among 202,339 active duty enlisted US Air Force trainees, 50% reported sleep difficulties, with 9% reporting frequent occurrences (“often”, “most of the time”), which served as the strongest predictor of attrition. Further analysis observed that trainees with frequent sleep difficulties were 2.7 times more likely to be discharged (Taylor et al., 2020).

3 Role of chronic stress on maladaptation to military training: an allostatic load perspective

Given that a substantial relative incidence (~60–65%) of overuse MSKIs, physical fitness decrements (~30%), and worsened psychological wellbeing (~10–25%) occur during military training, which may lead to consequences that threaten national security (Nindl et al., 2018; Good et al., 2020) and financial wellbeing of the US military healthcare system (Dijksma et al., 2020; Lovalekar et al., 2018; Pope et al., 1999), it is suggested that observed outcomes may be avoidable by the modification of risks. However, such risks must be delineated before prevention strategies can be implemented. Reductions in physical performance and the incidence of overuse MSKIs during training have been previously attributed to inadequate and excessive physical training stimuli, respectively (Burley et al., 2018), limited post-training recovery (Hansen et al., 2021), excessive mechanical loading on musculotendinous tissues (Feigel et al., 2023) and nutritional deficiencies (Alemany et al., 2008). Reduced psychological wellbeing, in turn, has been previously attributed to excessive or blunted stress responses (Tait et al., 2022; Taylor et al., 2017), introversion, or undesirable personalities (Saxon et al., 2020), childhood adversity (Ee et al., 2023), and a lack of previous training experience (Barrett et al., 2022). Though such factors have been revealed through the use of multi-factorial predictive models and their mitigation via individual interventions (Cooper et al., 2024), there is a lack of a unified physiological factor associated with such outcomes that may serve as the foundation bridging several psychophysical outcomes and reduce the analytical burden in identifying risk factors.

Owing to an overall 12.5% rise in the recruitment rate of the US Armed Forces in the fiscal year of 2024 (US Department of Defense, 2025), structured military training programming functions to enable large masses of individuals to face similar external physical and psychological stress exposures, particularly under current gender-integrated physical training doctrine (Lovalekar et al., 2023). However, previous evidence reports a vast difference in the relative physiological response to stress, which may be detrimental to personnel experiencing greater stress exposures than their peers (Burley et al., 2018; Forse et al., 2024; O'Leary et al., 2018). Given research observing individualized maladaptive outcomes during training (Burley et al., 2018; Forse et al., 2024; Lovalekar et al., 2023), it is important to consider chronic activation of the physiological stress response perturbed by military training stressors, including energy restriction/deficits, sleep restriction/deprivation, physical overtraining, and cognitive distress, that may provoke allostatic load as an important contributor. The following section reviews the physiological response on neuroendocrine, immune, autonomic systems, its response to military training-related stressors and how these responses contribute to allostatic load.

3.1 The physiological stress response

Stress is defined as a constellation of events consisting of an external (i.e., environmental, psychosocial, mechanical) or internal

(i.e., physical, biogenic) stimulus, whether actual or perceived, that precipitates a reaction in the brain followed by a physiological response nonspecific and specific to the stimulus to maintain homeostasis (Selye, 1950; Selye, 1976; Goldstein and Kopin, 2007; Nicolaides et al., 2015; Sher et al., 2020). In turn, *homeostasis* is characterized as the physiologic stability between interdependent biological systems (Billman, 2020). As such, stress encompasses an integrated definition to create a three-pronged construct: (i) a stressor (stimulus), (ii) a stress perception (detection and interpretation in the brain), and (iii) a stress response involving the activation of physiological fight-or-flight and neuroendocrine systems that serve as an adaptive mechanism to maintain homeostasis (Finnell et al., 2017; Gianaros et al., 2017; McEwen and Gianaros, 2011; Cohen et al., 2016; Schneiderman et al., 2005).

The physiological stress response involves the integration of different brain regions and neuronal circuits responsible for the detection and interpretation of physical, psychological, or environmental stressors and the pro-survival and adaptive mechanisms that follow (McEwen and Gianaros, 2011; Cohen et al., 2016). Though diverse stressors engage distinct brain regions for processing and interpretation (Gianaros et al., 2017; McEwen and Gianaros, 2011), the initiation of the stress response involves the activation of the hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medullary (SAM) axes for multi-system (i.e., immune, metabolic, cardiovascular) effects (Sher et al., 2020; Miller et al., 2007). Common military training stressors, including energy deficiency or restriction, physical overtraining, cognitive stress, and sleep deprivation/restriction (Figure 3A), have shown to activate the HPA axis and release its neuroendocrine factors for downstream influence on immune and autonomic activity (see Sections 3.2–3.5) by utilizing three primary structures (Figure 3B). The three structures that respond to such stressors include the paraventricular nucleus of the hypothalamus (PVN), the anterior pituitary gland, and the adrenal cortex. The PVN computes and integrates neuronal and humoral inputs to activate a specialized group of cells that control the level of activation of the HPA axis, including the regulation, synthesis, and secretion of corticotropin-releasing hormone (CRH) into the hypophyseal portal vasculature, which serves as a series of veins connecting two venous capillary beds for the transporting and exchanging of hormones between the hypothalamus and the anterior pituitary gland. The release of CRH and its subsequent binding to cognate receptors on corticotropes of the anterior pituitary can trigger the release of adrenocorticotrophic hormone (ACTH) into the general circulation to bind to melanocortin-2 receptors on the surface of adrenal zona reticularis and the fasciculata cells of the adrenal gland. This binding triggers the release of glucocorticoids, including cortisol and dehydroepiandrosterone (DHEA), to the circulation (Figure 3B) (Nicolaides et al., 2015; Korte et al., 2005).

Cortisol, the primary glucocorticoid in humans (Oyola and Handa, 2017), binds to ubiquitous mineralocorticoid and glucocorticoid receptors on various body tissues to promote appropriate metabolic responses to environmental perturbations, including glycogenolysis and mobilization of free fatty acids for increased energy availability for expenditure and promote pro- and anti-inflammatory cytokine activation (Herman et al., 2012). DHEA, the glucocorticoid antagonist, serves to prevent excessive systemic inflammation and protects the neurologic machinery

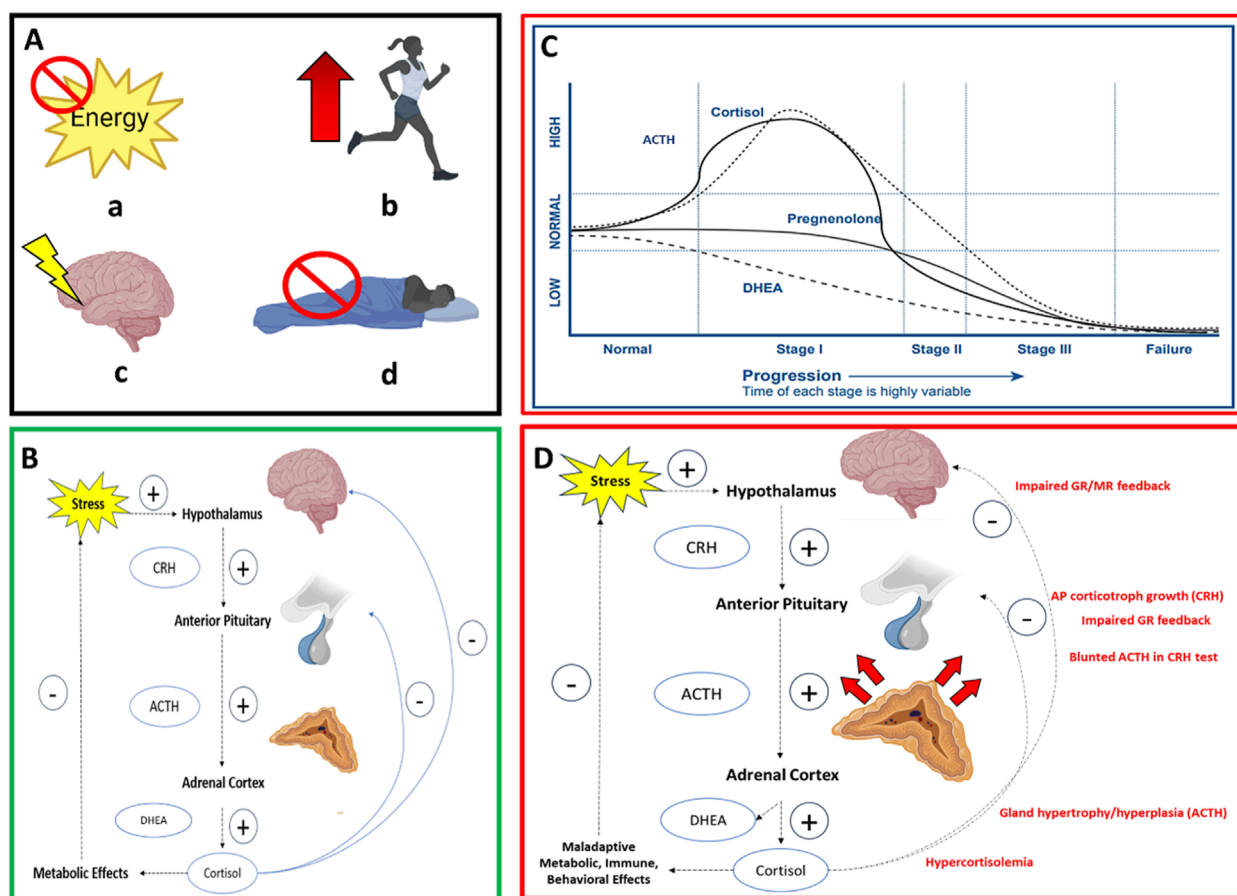


FIGURE 3

Box A illustrates four common military training-related stressors (a) energy restriction/deficiency (b) physical overexertion (c) cognitive stress (d) sleep restriction/deficiency, which are coined the Stressor Pool; Box B illustrates the HPA axis with negative feedback inhibiting CRH release from the hypothalamus and ACTH from the anterior pituitary gland. Box C illustrates the first model (Model 1) of HPA axis dysfunction and one mechanism of allostasis (Model 2) where initial hypercortisolism results in reduced glucocorticoid and mineralocorticoid receptor sensitivity on cells of the hypothalamus and anterior pituitary that impairs negative feedback and renders CRH and ACTH as growth factors. This results in larger functional masses capable of greater binding affinity to maintain the stress response over weeks.

(i.e., glucocorticoid or mineralocorticoid receptors) from damaging effects of excess cortisol (Lennartsson et al., 2012; Lennartsson et al., 2022). Glucocorticoid receptor binding of hormones mediates an adaptive, negative feedback response inhibiting further stress hormone production at all levels of the HPA axis (McEwen and Gianaros, 2011; Herman et al., 2012) (Figure 3B).

In cohesion with the neuroendocrine response to stress, the immune system is triggered by the presence of inflammation reflected by circulating pro- and anti-inflammatory cytokine concentrations that mediate HPA axis activity via receptors in tissues associated with the axis (Kargl et al., 2024). Cytokines include mediators of the interleukin family (i.e., interleukin-6), tumor necrosis factor- α (TNF- α) (Turnbull and Rivier, 1995), and c-reactive protein (CRP). CRP, a systemic marker of inflammation, is closely linked to HPA activity, meaning that when CRP concentrations rise, the HPA axis also tends to activate, which can lead to increased cortisol and DHEA production (Sproston

and Ashworth, 2018). When the immune system is triggered, pro-inflammatory cytokines are released, which can concomitantly stimulate the HPA axis to produce a bi-directional relationship with cortisol and DHEA. Anti-inflammatory cytokines also increase from this response as a negative feedback mechanism (i.e., interleukin-10) to counteract excessive pro-inflammatory effects (i.e., reactive oxygen species) (Kargl et al., 2024).

The physiological stress response also involves the activation of SAM axis owing to its neuronal synaptic circuitry relying on neurotransmitter communication between higher regions of the brain to peripheral receptors (Sapolsky et al., 2000). SAM activation involves the release of enzymes driven by sympathetic nervous system activity, such as salivary α -amylase (sAA), which is triggered by the release of norepinephrine and epinephrine and bind to β -adrenergic receptors in the salivary glands for its production. sAA is a reliable indicator of SAM activity (Nater and Rohleder, 2009), and has previously been used as a biomarker to assess the

SAM response in ambient and military settings (Arhakis et al., 2013; Habersaat et al., 2018; Ishitobi et al., 2010; Klaus et al., 2019). The following sections aim to review empirical research of randomized controlled trial or observational cohort study designs that evaluated the influence of individual stressors commonly experienced during military training, including energy restriction or deficiency, physical overtraining or exertion, cognitive distress, and sleep deprivation or restriction, on the key drivers of allostatic load, including activity of the primary mediators of neuroendocrine, immune and autonomic systems (McEwen BS., 1998). Studies of healthy adults that assessed individual stressors and avoided confounding of additional stressors were included. Each summary included biomarkers commonly used in allostatic load research based on previous reviews (Juster et al., 2010; Carbone et al., 2022) to be consistent in the reporting of mediators across studies.

3.2 Role of energy deficit/restriction on the primary mediators of allostatic load

Energy restriction and/or deficits are commonly experienced during military training courses, with previous research reporting average deficits of 9.7 MJ/day over 8-days of military training (Alemany et al., 2008) and deficits ranging between 1000 and 4000 kcal d⁻¹ over a 61-d US Army training course (Henning et al., 2014). Energy restriction or deficits during training have been attributed to restricted feeding times (Koltun et al., 2023), food choices, and periods of practiced energy restriction to simulate operations (Friedl et al., 2000). Koltun et al. observed that negative eating behaviors, such as energy restriction and deficits, are associated with worsened military health outcomes, including MSKI risk in both sexes (Koltun et al., 2023). A synopsis of the influence of energy deficit or restriction on activity of primary mediators of allostatic load in healthy, non-obese individuals can be shown in Table 1. From this synopsis, there are two main findings from the literature that may be drawn (Alemany et al., 2008; Henning et al., 2014; Ruffing et al., 2022; Degoutte et al., 2006; Huovinen et al., 2015; Buffenstein et al., 2000; Pasiakos et al., 2011) (Table 1).

First, it is observed that neuroendocrine, autonomic, and immune biomarker concentrations depend on the relative severity (i.e., intensity) and duration of the energy restriction or deficit, such that the magnitude and length of the deficit propagates an inverted “U-shaped” curve where substantial restrictions or deficits in severity and/or prolonged duration promote acute increases in end-product concentrations followed by dampened responses with concentrations falling below baseline (Alemany et al., 2008; Henning et al., 2014; Ruffing et al., 2022; Degoutte et al., 2006; Buffenstein et al., 2000; Pasiakos et al., 2011). Energy deficits exemplified by active individuals consuming 4 MJ d⁻¹ (<1000 kcal d⁻¹) less than normal over 1 week (Degoutte et al., 2006) demonstrated significant increases in ACTH (+~30%, $p < 0.05$), cortisol (+~20%, $p < 0.05$) and DHEA (+5%, $p < 0.05$) and reductions in DHEA:Cortisol ratio (-20%, $p < 0.001$) from baseline (Degoutte et al., 2006). Over 2 days of near complete energy restriction where healthy individuals consumed less than 10% estimated calorie requirements, Pasiakos et al. (2011) (Pasiakos et al.,

2011) observed a significant decrease in circulating cortisol (-70%, $p < 0.001$) and an upregulation of DHEA (+68%, $p < 0.001$) from baseline suggested to be driven by an inverse relationship with lower concentrations of leptin (Pasiakos et al., 2011). Likewise, Pritchard et al. (1999) (Pritchard et al., 1999) reported an 86% increase in dehydroepiandrosterone-sulfate (DHEA-S) following a 3-month energy deficit in healthy adult men (Pritchard et al., 1999). In contrast, however, Alemany et al. (2008) (Alemany et al., 2008) observed a significant reduction in DHEA concentrations following an 9.7 MJ d⁻¹ energy deficit over an 8-day military training exercise (Alemany et al., 2008). However, as this training included additional stressors, this finding should be interpreted with caution.

Similar dose-response relationships are revealed concerning autonomic and immune biomarkers in response to energy restriction or deprivation (Durgeurian et al., 2018; Hennigar et al., 2021). Among competitive weightlifters, Durgeurian et al. observed a significant increase in resting sAA (+364.60%, $p < 0.05$, Cohen's $d = 1.72$) following a 6-day energy restricted diet of 8.4 ± 3.6 MJ d⁻¹ when compared to a control group consuming 15.4 ± 5.0 MJ d⁻¹ (Durgeurian et al., 2018). However, after a 4-week energy restriction protocol with greater absolute severity (<25% of calories from baseline; 1650 ± 911 kcal d⁻¹), Waldman et al. found no significant difference in resting sAA concentrations in firefighters (Waldman et al., 2020). Although Durgeurian et al. showed a conservative energy deficit (~2007.65 kcal d⁻¹) than Waldman et al. (~1650 kcal d⁻¹), the relative change in Durgeurian et al. (-1438 kcal d⁻¹) was more severe than Waldman et al. (-400 kcal d⁻¹), which may have contributed to the results (Table 1). Further, an acute 2-d energy restriction protocol has shown to evoke autonomic nervous system balance toward sympathetic dominance (Solianik and Sujeta, 2018), with more chronic energy deficits demonstrating a blunting in sympathetic activity measured by heart rate variability (Jenkins et al., 2022; Mazurak et al., 2011). During a 72-h sustained combat and training operations (SUSOPS) in active-duty personnel, Hennigar et al. observed a -43% energy deficit (-2047 kcal d⁻¹) in a restricted diet group (2515 ± 171 kcal d⁻¹) compared to an +18% energy deficit in a balanced diet group (5437 kcal d⁻¹ ± 377 kcal d⁻¹; $p < 0.001$). Further analysis observed significant increases following SUSOPS in CRP and IL-6 in both groups, with the restricted group showing a 59% greater increase in CRP than the balanced group ($+2.6 \pm 5.3$ mg L⁻¹, $p < 0.001$) (Hennigar et al., 2021). However, as the SUSOPS also included heavy physical training and sleep restriction (<4 h-night⁻¹), which may confound immune responses (Kargl et al., 2024), these results should be interpreted with caution. Over a 2-year energy restriction protocol, Trevizol et al. observed a significant reduction in IL-6 to indicate improvement in inflammation (Trevizol et al., 2019). Likewise, a 2020 meta-analysis demonstrated that energy-restricted diets reduce CRP concentrations compared to baseline through a considerable length of intervention (≥ 2 months) (Wang et al., 2020). In contrast, a 2022 systematic review observed an increase in circulating inflammatory cytokines in military personnel following field training exercises; however, this result may be confounded by additional stressors (E Silva et al., 2022).

Second, there is a general consensus that a reduction in body mass, as often observed following military training (Friedl et al., 2000; Nindl et al., 1997; Nindl et al., 2012), may serve as an

TABLE 1 Empirical research of randomized controlled trial or observational cohort study designs that evaluated the influence of energy deficiency or restriction (independent variable) on primary mediators of allostatic load (dependent variable) in non-obese, healthy populations.

Author	Population	Methods	Outcome
Ruffing et al. (2022)	21 F Recreationally- Active	2 × 24 h blood sampling pre-post 3-mo ExRx + ER (−15%–60% EA, 5 ExRx-wk ^{−1} 70%–80% HR _{max})	↔C _{24hrAUC} , ↔C _{DaytimeAUC} , ↔C _{mean} , *↑C _{MorningAUC}
Ackerman et al. (2013)	21 F AE vs.18 F EE vs. 20 F NE	Cross-sectional frequent sampling (2300–0700) to assess C (pulse amplitude, mass, half-life and AUC) dynamics	AE: *↑C _{SerumOvernightAUC} AE: *↑C _{SerumOvernightPulseAmp}
Loucks et al. (1985)	9 F Balanced Diet vs. 9 F Restricted Diet vs. 8 F Control Sedentary	Frequent sampling (10-min intervals over 24 h) to assess C and ACTH dynamics following 10 kcal kg ^{−1} ·d ^{−1} for 4-d	Restricted Diet: ↔ACTH _{PulseFreq} , ↔C _{PulseFrequency} , ↑C _{24hUrine}
Laughlin and Yen, (1996)	8 F, AA vs. 8 F, EA Trained Cyclists	Frequent sampling to assess 24-h dynamics of C	AA: *↑C _{Serum24hBasal}
Aleman et al. (2008)	34 M US MOCs	9.7 MJ d ^{−1} over 8-d	*↓DHEA _{SerumFreeBasalMean} , ↔DHEA-S _{SerumFreeBasalMean}
Degoutte et al. (2006)	20 M Judoists	ER (−4 MJ d ^{−1} ; n = 10) or No ER (n = 10) over 7-d	ER: *↑ACTH _{SerumBasalMean} , *↑C _{SerumBasalMean} , *↓DHEA-S/C _{SerumBasalMean} , *↑DHEA-S _{SerumBasalMean}
Henning et al. (2014)	23 M US Rangers	−1000–4000 kcal d ^{−1} over 61-d	↑C _{SerumBasalMean} , ↔DHEA _{SerumBasalMean}
Huovinen et al. (2015)	15 M Athletes	4-wk ER (HWR: −750 kcal d ^{−1} w/≥2 g·kg ^{−1} ·d ^{−1} PRO (n = 8 M) or LWR: −300 kcal d ^{−1} w/≥2 g·kg ^{−1} ·d ^{−1} PRO [n = 7 M])	HWR: ↔C _{SerumBasalMean} , *↑C _{HWRSerumBasalMean}
Hooper et al. (2017)	9 M EHMC vs. 8 M NE	Cross-sectional sample of C dynamics between groups (EHMC: 27.2 ± 12.7 kcal d ^{−1} ·FFM ^{−1} ; NE: 45.4 ± 18.2 kcal d ^{−1} ·FFM ^{−1})	EHMC: ↓C _{SerumBasalMean}
Pasiakos et al. (2011)	12 M, 1 F	48-h ER of <10% est. calorie requirements	*↑DHEA-S _{SerumBasalMean} , *↓C _{SerumBasalMean}
Durquerian et al. (2018)	11 M Competitive Weightlifters	Diet Group (n = 6; −5% body mass over 6-d; 8.4 ± 3.6 MJ d ^{−1}) Control Group (n = 5; ±0% body mass over 6-d; 15.4 ± 5.0 MJ d ^{−1})	Diet Group: *↑SAA _{SalivaBasalMean} , ↔C _{SalivaBasalMean} , ↔DHEA _{SalivaBasalMean}
Waldman et al. (2020)	15 M Firefighters	4-wk ER (−25% of calories from baseline)	↔C _{SerumBasalMean} , ↔CRP _{SerumBasalMean} , ↔SAA _{SalivaBasalMean}
Trevizol et al. (2019)	220 (153 F) Healthy Volunteers	2-y ER (−25% of calories from baseline; n = 145) or Control (n = 75) from CALERIE Trial	ER: *↓IL-6 _{PlasmaBasalMean}
Hennigar et al. (2021)	10 M Active-Duty Military Personnel	72-h SUSOPS w/Restricted Diet (−2047 kcal d ± 920 kcal d or −43% ± 9% energy deficit; n = 6) or 72-h SUSOPS w/Balanced Diet (+689 ± 852 kcal d or +18% ± 20% energy deficit)	Restricted Diet: *↑CRP _{SerumBasalMean} Both Groups: *↑IL-6 _{PlasmaBasalMean} , *↑CRP _{SerumBasalMean}

Note. Bolded values in Outcome column demonstrate a significant result ($p < 0.05$). C = cortisol; DHEA, dehydroepiandrosterone; DHEA-S, dehydroepiandrosterone-sulfate; ACTH, adrenocorticotrophic hormone; CRP = c-reactive protein; IL-6, interleukin-6; SAA, salivary α-amylase; F = female; M = male; MOCs, marine officer candidates; AE, amenorrheic exercisers; EE, eumenorrheic exercisers; NE, non-exercisers; ER, energy restriction; ED, energy deficit; EHMC, exercise-hypogonadal male condition; SUSOPS, sustained combat and training operations; HWR, high-weight-loss group; LWR, low-weight-loss group; PRO, protein; ExRx = exercise training program.

indicator of increased basal HPA activity (Ruffing et al., 2022; Schorr and Miller, 2017; Villanueva et al., 1986). Ruffing et al. (2022) observed a significant correlation between reduced body mass and increased 24-h area-under-the-curve (AUC) cortisol

concentrations ($r = -0.473, p = 0.030$), suggesting that cortisol could respond to reduced chronic energy stores (Ruffing et al., 2022). Additional studies observed increased basal cortisol concentrations among females with anorexia nervosa (Schorr and Miller, 2017;

Misra and Klibanski, 2014) and in exercising females who were in a chronic energy deficit (Villanueva et al., 1986). Third, the hunger signal ghrelin may play a modulatory role in HPA axis activity during energy deficits (Misra et al., 2005). Previous studies observed a positive correlation between ghrelin and cortisol concentrations in females with anorexia nervosa ($r = 0.480$, $p = 0.002$) (Misra et al., 2005), with military-simulated energy restriction observing a negative association between satiety and cortisol ($r = -0.550$, $p < 0.05$) and DHEA-S concentrations ($r = -0.620$, $p < 0.05$) (Pasiakos et al., 2011). Hence, the augmented HPA activity during energy deficit or restriction appears to produce a dose-response relationship associated with deleterious changes in body composition and subjective satiety changes. Taken together, energy restriction or deprivation may serve as a potent driver of the primary mediators of allostatic load (Feigel et al., 2025a).

3.3 Role of physical overtraining on primary mediators of allostatic load

Physical overtraining—physical training conducted beyond one's finite ability to recover adequately between training sessions and supported by reduced physical performance (Pope et al., 2018) - is commonly reported during military training (Nindl et al., 2017; Booth et al., 2006; O'Leary et al., 2018; Chicharro et al., 1998). Previous research reports that external training loads, characterized as the total work performed that contributes to the internal training load (Foster et al., 2017; Impellizzeri et al., 2019), which is defined as the relative physiologic indicator reflecting the psycho-physiological response to external loads (Foster et al., 2017; Impellizzeri et al., 2019), is often operationalized in military training by the total distance covered (O'Leary et al., 2018; Jurvelin et al., 2020; Whittle, 2022). Distances range, on average, between eight and twelve miles per day during initial training courses (Feigel et al., 2024a; Givens et al., 2023a; O'Leary et al., 2018) (Drain et al., 2015; Pihlainen et al., 2023). As a synopsis of the impact of physical overtraining, which may be experienced in military courses (Booth et al., 2006; O'Leary et al., 2018; Chicharro et al., 1998), on the primary mediators of allostatic load, four findings may be drawn (Henning et al., 2014; Roberts et al., 1993; Slivka et al., 2010; Fry et al., 1993; Volek et al., 2004; Cadegiani and Kater, 2017) (Table 2).

First, there is a general consensus that physical overtraining perturbs neuroendocrine activity such that it increases cortisol and DHEA concentrations as short as 1 week of training up to 74 days of a heavy physical training program (Roberts et al., 1993; Slivka et al., 2010; Volek et al., 2004; Cadegiani and Kater, 2017). However, this is not always observed in elite athlete populations owing to differences in physical fitness level and training experience (Slivka et al., 2010; Fernández-Garcia et al., 2002). Second, there are contradictions in results reported by individual variations in the stress response (Fry et al., 1993; Fernández-Garcia et al., 2002). For instance, the occurrence of hypocortisolism during heavy physical training (Fry et al., 1993; Fernández-Garcia et al., 2002) may be indicative of overreaching in some individuals wherein cortisol is blunted and positive adaptations cease (Armstrong et al., 2021). Third, the increase in HPA activity appears to depend on either

heightened volume or intensity alone, but increased HPA activity may also be observed during high-volume, low-intensity training alone (Roberts et al., 1993; Hooper et al., 2017; O'Connor et al., 1989) or periodized high-volume, low-intensity training to low-volume, high-intensity training (Volek et al., 2004). Fourth, these observations appear independent of sex (Szivak et al., 2023a; Szivak et al., 2023b). Further, similar to the role of energy restriction (Table 1), heavy physical training demonstrates a dose-response relationship with increased relative severity or duration inducing a rise in neuroendocrine markers followed by a blunting effect (Fry et al., 1993; Fernández-Garcia et al., 2002; Bouget et al., 2006). However, as mentioned previously, differences in results may be attributed to physical fitness level (Slivka et al., 2010; Fernández-Garcia et al., 2002).

Fifth, physical overtraining has also been shown to alter inflammatory cytokine and autonomic biomarker concentrations at rest (Table 2). Fry et al. observed significant increases in markers of inflammation, including IL-2 (+183%, $p < 0.001$), following a 10-day, twice-daily, high-intensity interval running protocol in military personnel (Fry et al., 1993). Similarly, Tibana et al. showed significant increases in IL-6 (+99–197%), IL-10 (+14.4–21%) and a reduction in IL10:IL-6 ratio (−7.1%–8.9%) after a 2-day high-intensity functional interval training protocol (Tibana et al., 2018), and Tuan et al. observed significant increases in serum TNF- α (+~100%, $p < 0.05$) following a 3-day intervention of 30-min running sessions at 85% $\text{VO}_{2\text{max}}$ (Tuan et al., 2008). Previous research demonstrated that cytokines, including CRP, increases post-exercise (Tsao et al., 2009), with a peak around 24 h post-exercise (Reichel et al., 2020; Ispirlidis et al., 2008), but does not appear intensity-dependent (Tsao et al., 2009). Excessive resistance training can augment CRP concentrations in which may remain elevated up to 3 weeks (Fatouros et al., 2006). Concerning autonomic markers, Collins et al. (2019) (Collins et al., 2019) observed significant increases in sAA immediately after and 24-h post high-intensity exercise intervention in male athletes (Collins et al., 2019) and Chiodo et al. (2011) (Chiodo et al., 2011) observed significant increases following combat fighting competitions (Chiodo et al., 2011). Only one study to our knowledge investigated the chronic effects of a 10-month heavy physical training on sAA and observed a significant reduction in athletes indicating parasympathetic dominance and potential fatigue (−22%, $p < 0.05$) (Yasuda, 2025). Although sAA following chronic physical training remains largely uninvestigated, previous studies may support this finding by observing initial increases in sympathetic activity from heart rate variability (Plews et al., 2012) with a time-dependent reduction in sympathetic dominance toward parasympathetic dominance (Plews et al., 2012; Hynynen et al., 2006; Uusitalo et al., 1998; Flatt et al., 2017). Together, these data suggest physical overtraining may serve as a driver of the mediators of allostatic load.

3.4 Role of cognitive stress on primary mediators of allostatic load

Cognitive stress is the heightened perception of stress that can occur during military training (Conkright et al., 2022; Eddy et al., 2015) owing to physical training (Eddy et al.,

TABLE 2 Empirical research of randomized controlled trial or observational cohort study designs evaluating the influence of heavy physical training (independent variable) on primary mediators of allostatic load (dependent variable) in non-obese, healthy individuals.

Author	Population	Methods	Outcome
Roberts et al. (1993)	5 M Trained Runners	74-d of +100% training volume (mileage-wk ⁻¹) from baseline with maintained self-reported intensity	[*] ↑C _{SerumBasalMean}
Slivka et al. (2010)	8 M Trained Cyclists	21-d of Cycling Tour (3,211 km) (169 km d ⁻¹ ± 4 km d ⁻¹) equivalent to +418% ± 142% training volume and +167.0 W ± 4.0 W (+47% ± 1%) of pre-training intensity	↔C _{SalivaryBasalMean} [*] ↔T/C _{SalivaryBasalMean}
Fry et al. (1993)	28 M Elite Weightlifters	1-wk of high-volume, full-body resistance training	[*] ↓T/C _{SerumBasalMean} , [*] ↓C _{SerumPostExRxMean}
Volek et al. (2004)	17 M Resistance-Trained	4-week high volume, periodized full-body resistance training (week 1–2: high-volume, moderate intensity; week 3–4: moderate volume, high-intensity)	[*] ↑C _{SerumBasalMean}
Cadegiani and Kater (2017)	14 OTS Athletes, 25 Healthy Athletes, 12 Sedentary Controls	4-week high volume resistance training (5 days-wk ⁻¹)	[*] ↑C _{SerumBasalMean}
Fernández-García et al. (2002)	9 M Cyclists	3-wk “Vuelta a España” Cycling Tour	[*] ↓C _{PlasmaPostBasalMean}
O’Connor et al. (1989)	14 F Swimmers	4-wk of progressive high volume aerobic training (2,000 yd-d ⁻¹ –12,000 yards d ⁻¹)	[*] ↑C _{SalivaBasalMean}
Bouget et al. (2006)	12 F Cyclists	4-d of +122% training volume and intensity	[*] ↑C _{SalivaBasalMean} [*] ↓DHEAS _{UrineBasalMean} /C _{SalivaBasalMean}
Fry et al. (1994)	5 M Military Personnel of the Special Air Services Regiment of Australian Army	10-d twice-daily interval running sessions (morning: 15 × 1 min @18–21 km h ⁻¹ with 2 min rest between repetitions; afternoon: 10 × 1 min @18–21 km h ⁻¹ with 1 min rest between repetitions)	[*] ↑IL-2 _{SerumBasalMean}
Tibana et al. (2016)	9 M CrossFit Trained	2-d of single ‘Workout of the Day’	[*] ↑IL-6 _{SerumBasalMean} , [*] ↑IL-10 _{SerumBasalMean} , [*] ↓IL-10:IL-6 _{SerumBasalMean}
Tuan et al. (2008)	12 M Trained Runners	3-d of 30 min running exercise @85% VO _{2max}	[*] ↑TNF-α _{SerumBasalMean}
Halsen et al. (2003)	8 M Trained Cyclists	2-wk intensified cycling (14 ± 5 h-wk ⁻¹ equal to +100% training volume of identical proportions of training intensity distribution from baseline)	↔TNF-α _{PlasmaBasalMean} [*] ↔IL-6 _{PlasmaBasalMean}
Yasuda (2025)	21 F National-Level Athletes	10 months of volleyball training and competition (2–2.5 h d ⁻¹ for 6 days wk ⁻¹)	[*] ↓SAA _{SalivaBasalMean}
Chiodo et al. (2011)	16 (6 F) Taekwondo Athletes	1-d of Youth Taekwondo Competition	[*] ↑C _{SalivaBasalMean} [*] ↑SAA _{SalivaBasalMean}
Collins et al. (2019)	21 M Team Sport Athletes	1 x Acute High-Intensity Functional Interval Training Session	[*] ↑SAA _{SalivaBasalMean}

Note. Bolded values in Outcome are significant ($p < 0.05$). C = cortisol; DHEA, dehydroepiandrosterone; DHEA-S, dehydroepiandrosterone-sulfate; T/C = testosterone to cortisol ratio; F = female; M = male; MOCs, marine officer candidates; AE, amenorrheic; EE, eumenorrheic; ER, energy restriction; ED, energy deficit; IL-2, interleukin-2; IL-10, interleukin-10; TNF-α, tumor-necrosis-factor-α; SAA, salivary α-amylase; W = watts; OTS, overtraining syndrome.

2015), negative energy balance (Beckner et al., 2023), sleep deprivation (Passi et al., 2022), environmental conditions, and decision-making tasks (Conkright et al., 2022; Ben-Avraham et al., 2022). Newly recruited soldiers to mandatory military service

face challenging psychological demands on a daily basis (Schei, 1994; Mažeikienė et al., 2021), including separation from family and friends, unpredictable and uncontrollable demands on their time, intense routines, and operating in a space laden with laws

and hierarchies to contribute to cognitive stress (Boermans et al., 2013). Together, these factors can negatively affect the mental health of soldiers and increase the risk for attrition (Tait et al., 2022; Pope et al., 1999). Reduced cognitive performance may be reflected by cognitive fatigue (Eddy et al., 2015; Main et al., 2023), which can impact the ability to maintain an alert and attentive state and risk poor operational performance (Passi et al., 2022). Hockey (1997) (Hockey, 1997) observed that fatigue, in low controllability and high environmental demand situations, is related to HPA and SAM activation owing to direct engagement with an acute stressor (i.e., active problem-focused coping) (Hockey, 1997). Suarez and Perez found increased HPA activity in urban combat training when physical activity remained at a low level (Suárez and Pérez, 2013). Further analysis revealed cognitive stress caused by uncertainty regarding the location of threats, which led to fatigue and impaired post-combat information processing ability (Suárez and Pérez, 2013).

Table 3 summarizes a representative set of investigations (Henning et al., 2014; Lennartsson et al., 2012; Lennartsson et al., 2022; Budde et al., 2010; Schoofs and Wolf, 2011; Kudielka et al., 2004; Salvador et al., 2003) that assessed the influence of cognitive stress driven by the performance of cognitive tasks under time or duty constraints in occupational, classroom, or laboratory settings. Results of these studies observed significant increases (Lennartsson et al., 2012; Schoofs and Wolf, 2011; Kudielka et al., 2004; Salvador et al., 2003) or no change (Henning et al., 2014; Budde et al., 2010) in cortisol, DHEA, DHEA-S, or ACTH in response to a battery of cognitive assessments, including military training-related tasks (Conkright et al., 2022). Acute cognitive stress increases HPA activity among both sexes (Lennartsson et al., 2012; Schoofs and Wolf, 2011; Kudielka et al., 2004), whereas chronic cognitive stress can lead to blunted HPA activity (Miller et al., 2007). One meta-analysis observed a time-dependent HPA activity response with increased time since the onset leading to blunted morning and daily cortisol concentrations (Miller et al., 2007). However, it should be mentioned that Miller et al. observed that cognitive stress responses can be modulated by the nature (physical, social, traumatic), presence (morning, afternoon), emotional involvement (shame vs. loss), and controllability of the stressor (uncontrollable, controllable), which may affect neuroendocrine biomarker activity (Miller et al., 2007).

Considering the influence of cognitive stress on immune and autonomic system function (Table 3), there is evidence of elevated circulating inflammatory markers owing to recurrent daily stressors (Gouin et al., 2012; Vineetha et al., 2014). Among a group of 53 chronic caregivers (56 months $\pm$ 44 months) for dementia (≥ 5 h wk^{-1}), Gouin et al. observed significantly higher concentrations of CRP and IL-6 compared to non-caregiving controls (Gouin et al., 2012). A systematic review on the influence of chronic occupational stress (i.e., employment, burnout and exhaustion, caregiver stress) identified elevated CRP concentrations than control groups (Johnson et al., 2013). However, previous research shows varied sAA responses to chronic stress (Habersaat et al., 2018; Giessing et al., 2020; Wingefeld et al., 2010; Unno et al., 2013; Wood et al., 2021; Juster et al., 2011) despite robust increases during acute stress (Man et al., 2023; Knauff et al., 2021; Chacko et al., 2022; Teixeira et al., 2015). Individuals with

chronic stress disorders demonstrate increases (Tanaka et al., 2012; Tanaka et al., 2013) or decreases in sAA (Teixeira et al., 2015; Wolf et al., 2008) during acute stressor tasks, but dampened basal sAA concentrations (Klaus et al., 2019; Wolf et al., 2008). Together, these data suggest cognitive stress may also perturb the mediators of allostatic load.

3.5 Role of sleep restriction or deprivation on primary mediators of allostatic load

Majority of military training studies observe that soldiers achieve less than the nightly recommended sleep duration of 7–8 h per night (Givens et al., 2023a; Kargl et al., 2024; Hansen et al., 2021), with most studies observing 4–6 h of sleep per night on average (Edgar et al., 2021; Givens et al., 2023a; Kargl et al., 2024; Taylor et al., 2020). Among a representative set of investigations assessing acute (1–2 nights) partial (4 h night^{-1}) and total (0 h night^{-1}) sleep deprivation on neuroendocrine function (Table 4), there is a general consensus of increased HPA activity across studies (Guyon et al., 2014; Balbo et al., 2010). Additional studies assessing the influence of semi-chronic (4 nights) or chronic (5+ nights) find blunted HPA activity owing to “psychological deactivation” or fatigue (Åkerstedt et al., 1982) and reduced HPA sensitivity (van Dalfsen and Markus, 2018). However, chronic short sleepers (<5 h night^{-1}) have been shown to have elevated cortisol concentrations compared to normal sleepers, suggesting that the downregulation of the HPA axis may fail to occur in some individuals (Balbo et al., 2010).

Investigations on the influence of sleep disturbance, defined as interruptions during periods of sleep, and deprivation has shown to alter immune and autonomic responses (Kargl et al., 2024; Irwin et al., 2016). A 2016 systematic review and meta-analysis (N = 72 studies) on the association between sleep disturbance, sleep duration, and inflammation in adults observed that sleep disturbance was associated with higher concentrations of CRP (ES 0.12; 95% CI = 0.05–0.19) and IL-6 (ES 0.20; 95% CI = 0.08–0.31), with shorter sleep duration, but not the extremity of short sleep, was associated with higher CRP (ES 0.09; 95% CI = 0.01–0.17) but not IL-6 (ES 0.03; 95% CI: –0.09–0.14). However, neither sleep disturbances nor sleep duration was associated with TNF- α (Irwin et al., 2016). Among salivary markers, recent literature suggests that sAA may be a cross-species marker of sleep deprivation in tactical (i.e., first responder) and military populations (Lindsey et al., 2025) and individuals with sleep disorders (Thieux et al., 2024). Pajcin et al. observed the influence of 2-d TSD (50-h) on sAA and observed a significant diurnal profile wherein concentrations increased throughout the morning and afternoon ($p < 0.001$) and steadily declined in the evening and early-morning ($p < 0.001$). These results suggested that sAA may be sensitive to the diurnal rhythm for arousal and tracking the diurnal drive for alertness (Pajcin et al., 2017) (Table 4). However, there does not appear to be a consensus as to the direction of sAA with subjective feelings of sleep disturbance and repercussions of sleep debt (i.e., sleepiness and cognitive performance) (Thieux et al., 2024).

TABLE 3 Empirical research of randomized controlled trial or observational cohort study design evaluating the influence of cognitive stress (independent variable) on primary mediators of allostatic load (dependent variable) in non-obese, healthy individuals.

Author	Population	Methods	Outcome
Budde et al. (2010)	23 M, 17 F	Letter Digital Span and d2-test	↓C _{SalivaryPostMean}
Schoofs and Wolf (2011)	39 M, 44 F	TSST or Placebo-TSST	M: *↑C _{SalivaryPostTSSTMean} ; F: *↑C _{SalivaryPostTSSTMean}
Kudielka et al. (2004)	102 M and F	TSST	*↑ACTH _{PlasmaPostMean} ; *↑C _{SalivaryPostMean}
Lennartsson et al. (2012)	20 M, 19 F	TSST	*↑C _{SerumPostMean} ; *↑ACTH _{PlasmaPostMean}
Lennartsson et al. (2012)	20 M, 19 F	TSST	*↑DHEA _{SerumPostMean} ; *↑DHEA-S _{SerumPostMean}
Salvador et al. (2003)	17 M Judoists	Pre-competition vs. practice salivary C dynamics	*↑C _{SalivaryPreMean}
Gouin et al. (2012)	55 Caregivers, 77 Controls	Cross-sectional blood sample	Caregivers: *↑CRP _{SerumBasalMean} , *↑IL-6 _{SerumBasalMean}
Giessing et al. (2020)	1 M Police Officer	3-week frequent saliva sampling (directly after waking, 30 min later, 6-h later, before bed) during work hours	↔SAA _{SalivaBasalMean}
Wingenfeld et al. (2010)	215 (168 F) Nurses	13-h frequent sampling (07:00 h, 11:30 h, 17:30 h, 20:00 h) on a working day during an early shift	M: *↑SAA _{SalivaBasalMean} , F: *↑SAA _{SalivaBasalMean}

Note. Bolded values in Outcome are significant ($p < 0.05$). C = cortisol; ACTH, adrenocorticotrophic hormone; DHEA, dehydroepiandrosterone; DHEA-S, dehydroepiandrosterone-sulfate; F = female; M = male; TSST, trier social stress test; SAA, salivary α-amylase; CRP = c-reactive protein; IL-6, interleukin-6.

TABLE 4 Empirical research selected owing to randomized controlled trial or observational cohort study design evaluating the influence of sleep deprivation (independent variable) on primary mediators of allostatic load (dependent variable) in non-obese, healthy individuals.

Author	Population	Methods	Outcome
Blumert et al. (2007)	9 M Weightlifter	1-d x TSD	↔C _{SerumBasalMean} , ↔C _{SerumPostExRxMean} , ↔T/C _{SerumBasalMean} , ↔T/C _{SerumPostExRxMean}
Guyon et al. (2014)	13 M Adults	2-d x PSD (4 h-night ⁻¹)	*↑ACTH _{PlasmaBasalMean} ; *↑C _{SerumBasalMean} , ↔ACTH _{PlasmaPulseFreqMean} ; ↔C _{SerumPulseFreqMean} ; *↑ACTH _{PlasmaMorningMean} , ↔C _{SerumMorningMean} ; *↑C _{SalivaryNightMean} , *↑C _{SerumNightMean} ; ↔ACTH _{PlasmaNightMean}
Minkel et al. (2014)	14 M, 12 F Adults	1-d TSD (n = 12) vs. Normal (n = 14)	*↑C _{SerumBasalMean} ; *↑C _{SerumPostTSSTMean}
Akerstedt et al. (1980)	12 M Adults	2-d TSD	*↓C _{PlasmaBasalMean} ; *↓DHEA-S _{PlasmaBasalMean}
Pajcin et al. (2017)	12 (5 F) Adults	2-d TSD	*↓SAA _{SalivaBasalMean}

Note. Bolded values in Outcome are significant ($p < 0.05$). M = male; F = female; TSD, total sleep deprivation; PSD, partial sleep deprivation.

3.6 Mechanism of allostatic load from military training-related stressors

Although military training-related stressors do not occur in isolation during training (Nindl et al., 2018; Friedl et al., 2000), and exhibit bi-directional effects, such as sleep deprivation affecting subjective feelings of psychological stress (Schwarz et al., 2018), the above findings may reveal a pattern of the influence of stressors on primary mediators

of allostatic load by demonstrating acute increases and chronic reductions in biomarker concentrations during stress (Tables 1–4). Chronic stress can lead to one or more forms of HPA axis dysfunction (Figures 3C,D) and alter immune and autonomic system function, which may serve as a mechanism of allostatic load (Selye, 1950; van Dalen and Markus, 2018; Karin et al., 2020) as biomarker concentrations may fall within ‘at-risk’ bounds to support the computation of the ALI (Juster et al., 2010).

HPA axis dysfunction is characterized as a dynamic compensatory response to chronic stress that begins with initial hypercortisolism followed by hypocortisolism (Fries et al., 2005) or diurnal dysrhythmia (McEwen, 2007). The mechanism of HPA dysregulation derives from an evoked HPA axis that engenders an over-responsive system (e.g., hypercortisolism) toward an under-responsive or non-responsive system (e.g., hypocortisolism) (Selye, 1950). Progression from an over to under-responsive system is reflected by one or more forms of HPA axis dysregulation leading to allostatic load (McEwen, 2007; Juster et al., 2011) (Figures 3C,D). Model 1 is one form that illustrates the dynamics of neuroendocrine biomarker responses broken down into three stages when under the influence of chronic stress. These three stages illustrate the trajectory of biomarker concentrations and emulate Han's Selye General Adaptation Syndrome (GAS) theory (Selye, 1950) (Figure 3C). The first stage of biomarker activity is the "Alarm Stage", described as an acute, adaptive response to a stressor. This can also be illustrated by the allostasis model (Figure 1), where a response appropriately meets a demand observed by increased cortisol, DHEA, and ACTH concentrations (Korte et al., 2005). If the stress continues, however, the 'Resistance Stage' occurs, which is characterized by chronic activation of the physiological stress response ("chronic allostasis") as observed by heightened biomarker concentrations. Notably, this stage risks degradation of protective negative feedback mechanisms (i.e., deterioration of glucocorticoid receptor sensitivity) or *primary outcomes*, as shown in the allostatic load model (Figure 2A). Finally, if the stress continues, the "Exhaustion Stage" occurs, which is characterized by a reduction in circulating biomarker concentrations that can signal allostatic load (Sher et al., 2020). Taken together, Model 1 reports that under chronic stress, ACTH will drive cortisol production and deplete DHEA production that leads to reduced cortisol production toward hypocortisolism in the Exhaustion Stage (Stephens and Wand, 2012). Hence, this model benefits the prediction of neuroendocrine biomarker trajectories and downstream immune and autonomic biomarker trajectories to complement the aforementioned results of empirical research on common military training-related stressors (Tables 1–4). Hence, Model 1 may serve as one mechanism leading to allostatic load (McEwen BS., 1998; Sher et al., 2020).

A second mechanism of allostatic load is shown in Model 2 (Figure 3D). Model 2 purports that HPA dysfunction and the downstream effects of immune and autonomic function arise from changes in the total functional masses of the HPA hormone-secreting glands including the adrenal cortex and anterior pituitary (Karin et al., 2020). Karin et al. reported that this mechanism occurs from an initial hypercortisolism followed by reduced DHEA production and glucocorticoid receptor and mineralocorticoid receptor sensitivity of the cells in the hypothalamus and anterior pituitary that impair the negative feedback loop of the HPA axis (Karin et al., 2020). Reduced cell receptor sensitivity with stress hormone production may render the hormones of the HPA as growth factors for the glands in the axis where excessive secretion of CRH can drive pituitary corticotroph cell growth and ACTH can drive adrenal gland hypertrophy/hyperplasia to result in larger functional masses capable of greater binding affinity to maintain the stress response over weeks. Consequently, this form of HPA axis dysfunction can lead to a similar over-to-under responsive neuroendocrine, immune, and autonomic nervous system activity

(Henning et al., 2014; Kargl et al., 2024; Karin et al., 2020) as observed in response to military training-related stressors (Tables 1–4). Hence, Model 2 may serve as a second mechanism of allostatic load. Owing to the role of military training-related stressors on allostatic load, the next section summarizes the impact of allostatic load quantified by ALI on physical performance and psychological and musculoskeletal health, in non-obese, healthy adults and, where available, military personnel.

4 Impact of allostatic load on physical performance, psychological, and musculoskeletal health

4.1 Physical performance

Previous evidence observes that ALI is negatively associated with physical performance outcomes assessed by a battery of maximal strength and balance assessments (Germano et al., 2023; Hansen et al., 2016). Among 1101 healthy volunteers (65–74 years), Germano et al. observed ALI was inversely associated with score on the Short Physical Performance Battery (SPPB) score ($\beta = -0.234$, $p < 0.001$), with indirect effects evidenced between age and socioeconomic status (Germano et al., 2023). Similarly, among 5467 healthy volunteers (48–62 years), Hansen et al. found that the ALI mediated the association between education and physical performance (chair rise ability, postural balance, sagittal flexibility) and muscle strength (jump height, trunk extension, and flexion, handgrip strength) and accounted for 2%–30% of the total effect among women (Hansen et al., 2016). As a secondary analysis of data from the MacArthur Studies of Successful Aging study, Seeman et al. observed step-wise reductions in similar physical performance outcomes with every 1-unit increase in ALI (Seeman et al., 1997) and during a follow-up of 7 years (Seeman et al., 2001). Seeman et al. also found the ALI outperformed predicting physical dysfunction to a greater degree than its individual sub-components, suggesting its benefit of determining risk of physical performance decline (Seeman et al., 2001). However, these investigations were conducted among older (mid to late-life) adult populations (Germano et al., 2023; Hansen et al., 2016). Nevertheless, recent research has linked high ALI with worsened physical performance in younger (<40 years) populations and in military personnel (Feigel et al., 2025a; Hastings et al., 2022). Feigel et al. observed a significant negative association between change (Δ) in ALI from baseline and change in physical performance in men from elements of the USMC Physical Fitness Test (PFT) (Δ Pullups: $\beta = -0.88$, $R^2 = 0.60$, 95% CI: -1.55 , -0.21 ; Δ Push-Pull PFT Score: $\beta = -2.87$, $R^2 = 0.60$, 95% CI: 4.99 , -0.75 ; Δ Total PFT Score: $\beta = -3.48$, $R^2 = 0.58$, 95% CI: -5.76 , -1.19) to suggest a potential role of chronic stress on military physical performance (Feigel et al., 2025a). Data from the National Survey of Midlife Development in the United States (N = 2055, 26–86 years) demonstrate a negative association with ALI and physical function (grip strength: $\beta = -0.11$, 95% CI: -0.15 , -0.07 ; gait speed: $\beta = -0.20$, 95% CI: -0.24 , -0.16) (Hastings et al., 2022). Although military training studies lack use of the ALI, previous research demonstrates an association between altered neuroendocrine and autonomic hormone profiles following training and worsened physical fitness characteristics,

such as muscular power and strength (Szivak et al., 2018), which are important attributes for occupational task performance (Feigel et al., 2024b). Further evidence suggests that increased inflammatory cytokine concentrations can hinder muscle protein synthesis of lean muscle mass (Miller et al., 2022) and negatively influence upper and lower-body muscular strength (Sharma Ghimire et al., 2023). Hence, further research of ALI on physical performance in in-training personnel is warranted.

4.2 Psychological wellbeing

Previous epidemiological studies demonstrate a positive association between ALI score and symptoms of reduced psychological wellbeing in healthy individuals (Guidi et al., 2020), including anxiety (Gou et al., 2025; D'Alessio et al., 2020), depression (Gou et al., 2025; D'Alessio et al., 2020), and perceived stress (Guidi et al., 2020; Beckie et al., 2016; Juster et al., 2010; Geronimus et al., 2006) and a negative association with resilience (Felix et al., 2023), all of which are reported during military training (Forse et al., 2024; Bulmer et al., 2022b; Taylor et al., 2009; Guo et al., 2021). However, cognitive reappraisal was indirectly associated with lower ALI, whereas the tendency to use emotion suppression was indirectly associated with greater ALI (Ellis et al., 2019). Sleep health has been tied to allostatic load from a systematic review demonstrating a positive relationship between chronic sleep difficulty level and ALI (Christensen et al., 2022). Guidi et al. observed poorer objective and subjective sleep quality was associated with ALI in four separate investigations (Guidi et al., 2020). A 2022 systematic review and meta-analysis by Christensen et al. observed a significant negative association between sleep health, characterized by sleep duration and sleep quality (i.e., greater time in restorative sleep stages as compensation) on ALI among epidemiological studies. However, it was also reported that sleep may be bi-directional where poorer sleep quality may contribute to ALI if sleep health is not improved with intervention (Christensen et al., 2022). Interestingly, recent work from our group observed a significant negative association between Δ in sleeping difficulty level and Δ ALI by the end of a 10-week military training course in both sexes (Δ SD: $\beta = -1.25$ to -0.56 , $R^2 = 0.35$ to 0.82 , $p < 0.001$ – 0.046) (Feigel et al., 2025a). Further research into the influence of ALI on psychological wellbeing outcomes in military training populations is also warranted.

4.3 Musculoskeletal health

Gallagher and Barbe (2022) examined the role of allostatic load on musculoskeletal disorders, characterized as injury, dysfunction, or impairment to the muscle, ligament, tendon, or bone, during occupational settings (Gallagher and Barbe, 2022). Their findings suggested that musculoskeletal disorders, including overuse MSKIs, may result from impaired tissue repair mechanisms driven by mechanical stress. These impairments, in turn, are influenced by underlying inflammatory, autonomic, and neuroendocrine dysfunctions associated with allostatic load, which is precipitated by chronic psychological and physical stress. Overuse MSKIs are comprised of inflammatory and degenerative conditions in

musculoskeletal tissues involving muscles, tendons, ligaments, and peripheral nerves (Edwards, 2018). The authors found that physical work risk factors for overuse MSKIs included high force demands, repetitive work, adoption of non-neutral postures, and repeated heavy lifting, with a combination of high psychosocial work demands during work settings (da Costa and Vieira, 2010), which are common attributes experienced during military training courses (Vaara et al., 2022). Additional psychological risk factors included perceived stress at work, psychological job demands, and low job control (Bongers et al., 1993; Bongers et al., 1993; Deeney and O'Sullivan, 2009). Together, the authors purported that the presence of psychological and physical stress may lead to allostatic load and overuse MSKI owing to a slower-than-normal healing response in the tissue. This may result in faster damage development in musculoskeletal tissues and higher overuse MSKI risk (Gallagher and Barbe, 2022).

Epidemiological studies demonstrate relationships between ALI and musculoskeletal disorders (Guidi et al., 2020; Mori et al., 2014). Among 703 healthy men and women (median age: 56), mixed-effects linear regression controlling for clustering within families and adjusted for age, gender, race/ethnicity, body mass index, menopausal transition stage, childhood socioeconomic status, adult finances, education level, and study center, each standard deviation increment in ALI was associated with between 0.10 and 0.11 standard deviation decrements in lumbar spine bone mineral density (all $p < 0.05$) (Mori et al., 2014). Symptom frequency and intensity were associated with higher ALI among chronic fatigue syndrome patients compared to controls (Maloney et al., 2006; Goertzel et al., 2006). Further research on the influence of ALI on musculoskeletal health on training personnel is warranted (Feigel et al., 2024a).

5 Future of allostatic load assessment in military training research: consideration of commercial wearable devices for monitoring verified digital phenotypes of allostatic load

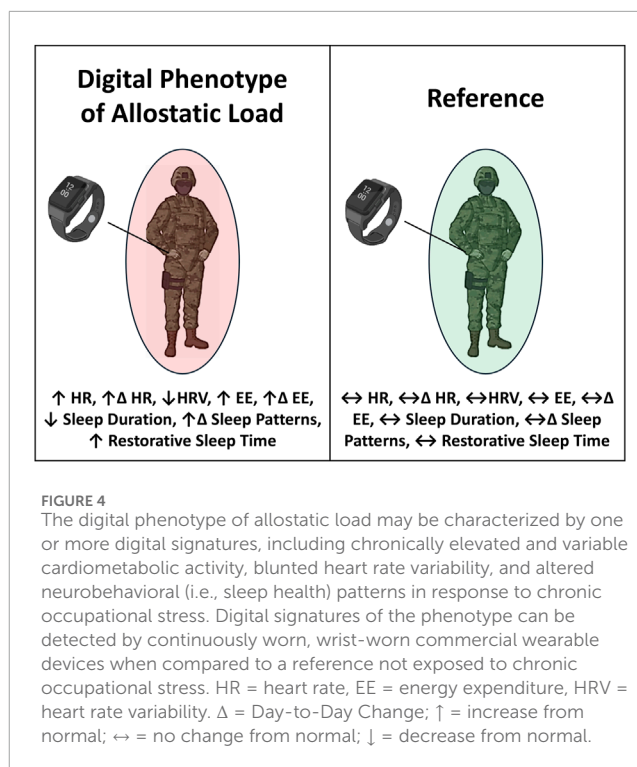
Owing to the influence of military training-related stress on the primary mediators of allostatic load (Table 1–4), and empirical research of allostatic load on physical performance and psychological and musculoskeletal health, there is a growing support for the role of allostatic load on military training-related maladaptive outcomes (Feigel et al., 2024a; Feigel et al., 2025a). However, further empirical research is warranted in this area to support these findings. Therefore, the future of allostatic load monitoring in military training research may direct toward two options for its measurement: the ALI method (Feigel et al., 2025a) or commercial wearable-based methods (Feigel et al., 2024a).

Although the ALI offers the advantages of understanding the biological process underpinning cognitive and physical dysfunction in response to chronic stress when studied under longitudinal study designs, such as using sample-specific biomarker cut-off values for increased specificity (Juster et al., 2011), and selecting biomarkers relevant to the target population and stress exposure (Karlman et al., 2002; Mauss and Jarczok, 2021), the ALI has limitations (McLoughlin et al., 2020; Carbone et al., 2022;

Magtibay and Umapathy, 2023). First, longitudinal tracking of ALI requires more than one blood draw or salivary sample (Figure 2B), which increases participant burden, risk of missing data, and analytical complexity, especially when up to 20 biomarkers are included (Juster et al., 2010). Second, there have been inconsistent ALIs used in the literature comprising different biomarkers (Juster et al., 2010) and algorithms for its computation to limit replication (Carbone et al., 2022). Hence, for occupational populations where conducting repeated ALI assessments presents the logistical challenges of obtaining more than one blood draw or salivary sample, such as military personnel, Magtibay and Umapathy proposed that ubiquitous, commercial wearables (Figure 2C) may overcome challenges (Magtibay and Umapathy, 2023).

Through continuous monitoring, commercial wearable devices use signal features, such as photoplethysmography (PPG) and accelerometry, to capture downstream cardiometabolic and neurobehavioral responses (i.e., sleep architecture or behavior) perturbed by chronic stress (Feigel et al., 2024a; Friedl, 2018). This approach has been suggested to complement the ALI where combinations of wearable-derived signals may define a wearable-derived “digital phenotype” of allostatic load (Feigel et al., 2024a; Corrigan et al., 2021). This phenotype may be characterized by one or more digital signatures, including chronically elevated and variable cardiometabolic activity, reduced heart rate variability, and/or altered sleep architecture—such as increased time spent in restorative sleep stages—in response to chronic occupational stress (Magtibay and Umapathy, 2023) (Figure 4). Our group provided empirical support for this phenotype identified using continuous monitoring of commercial wearable devices in military personnel who experienced tertiary outcomes of allostatic load, including overuse MSKI (Feigel et al., 2024a). Together, these findings, as well as the technological advances in sensors, may support commercial wearable devices as a promising approach (Figure 2C) to detect downstream cardiometabolic and neurobehavioral effects (secondary outcomes; Figure 2A) and assess allostatic load in-the-field (Magtibay and Umapathy, 2023).

Use of physiological time-series data from continuously worn commercial (i.e., wrist-worn, durable, low-burden) wearable technology for allostatic load assessment remains understudied (Feigel et al., 2024a; Corrigan et al., 2021). Using allostasis and allostatic load models as tools for measuring stress responses in occupational settings, Magtibay and Umapathy reported that low-burden, wrist-worn wearable devices within a robust human-machine learning framework could be useful to measure *digital biomarkers* of allostasis, characterized as cardiovascular, metabolic, and behavioral responses to everyday life, and use those signals to detect physiological characteristics of allostatic load, which may take the form of a digital phenotype (Magtibay and Umapathy, 2023) (Figure 4). The authors also reported signals from wearables, such as PPG, triaxial accelerometry, and thermometry, could indicate stress-induced autonomic responses activated by HPA and SAM axes (Magtibay and Umapathy, 2023). Additionally, data features captured by most commercial wrist-worn wearables could be used to determine its relationship with ALI as an alternative proxy of allostatic load assessment in occupational settings (Magtibay and Umapathy, 2023). Software



algorithms could reveal physiologic responses and behavioral tendencies owing to allostatic load, such as altered sleep patterns (Christensen et al., 2022). Previous research suggests that wrist-worn devices may improve participant compliance than waist-worn devices (Kim et al., 2019; Wolpern et al., 2019) and promote continuous monitoring without obstruction or interference during military training (Feigel et al., 2024a; Friedl, 2018; Hinde et al., 2021). As the use of the ALI increases in military training research (Feigel et al., 2025a), the employment of commercial wrist-worn wearable devices (Hinde et al., 2021; Feigel et al., 2025b; Friedl and Looney, 2023) may provide the opportunity to determine verifiable, wearable-derived signals of personnel experiencing allostatic load.

However, an important limitation found in the literature when using wearables to detect allostatic load (Corrigan et al., 2021; Corrigan et al., 2023) is the lack of aligning the signals with the formal definition of allostatic load as proposed by McEwen and Stellar (McEwen, 1993). Based on the definition, the threshold by which allostatic load occurs is not immediately informative with only independent variables (i.e., physiological signals of stress). However, the distinction when allostatic load is experienced may be revealed through the presence of (i) whether maladaptive psycho-physiological outcomes (secondary or tertiary outcomes; dependent variables) are associated with the signals (McEwen, 1993; Magtibay and Umapathy, 2023) or (ii) signals are associated with ALI (Magtibay and Umapathy, 2023). Allostatic load is a biological state where physiological stability fails owing to multi-system dysregulation and occurs concomitantly with secondary or tertiary outcomes (McEwen, 1993). The ALI has been validated through the observation of its association with secondary and tertiary outcomes (Seeman et al., 1997; Seeman et al., 2001), and associated with high ALI scores (i.e., ALI >3 or 4) (Juster et al.,

2010; McLoughlin et al., 2020). Previous studies reveal the utility of commercial wearables in the detection of acute (Chen et al., 2017; Cho et al., 2019; de Vries et al., 2022; Erickson et al., 2022), and chronic occupational stress (O'Leary et al., 2018; Hinde et al., 2021; Erickson et al., 2022; Wyss et al., 2014). However, there have been very few studies that adopted this theory-based ruling to use commercial wearables and determine whether allostatic load is experienced (Feigel et al., 2024a). There remains a lack of research assessing the relation between digital signals and high allostatic load, and with secondary or tertiary outcomes (Feigel et al., 2024a). Such findings may advance the use of commercial, wrist-worn wearables in-the-field for high allostatic load risk detection in military personnel. The next section summarizes the empirical research that used wearable-based physiological signals for evaluating allostatic load detection in the literature thus far (commercial and non-commercial).

5.1 Physiological characteristics of allostatic load

5.1.1 Chronically elevated and variable heart rate

Among the first studies employing wearable devices to evaluate stress system perturbations using allostatic load as a framework, Milosevic et al. developed a research methodology to examine the real-time multi-modal responses to work stress of fifteen nursing volunteers conducting a representative protocol during a workday (Milosevic et al., 2013). They monitored physiological parameters of beat-to-beat blood pressure, heart rate, heart rate variability, respiratory rate, and galvanic skin resistance during a simulated, high-fidelity patient simulator (30-min) intervention (tracheostomy protocol with and without respiratory distress) through the use of garment-worn and electrode-based sensors attached on the chest. Repeated-measures analysis of variance evaluated the physiological changes before, during, and after the protocol, and revealed a significant strain placed on volunteers during the test, owing to a significant rise in blood pressure, heart rate, respiratory rate, and reduction in heart rate variability, with some participants returning to baseline after the test. The authors suggested that this response may be characteristic of allostatic load (Milosevic et al., 2013). However, the ALI, secondary, or tertiary outcomes were not assessed, thus hindering the verification of chronically elevated heart rate and lower heart rate variability as a digital signatures of allostatic load. Recent research from our group, however, observed that these digital signatures measured by valid and reliable commercial wrist-worn devices, were associated with a tertiary outcome of allostatic load including overuse MSKI occurrence (Feigel et al., 2024a). Although the ALI was not assessed from our group in the aforementioned study (Feigel et al., 2024a), this finding may support these signals of the digital phenotype (Feigel et al., 2024a) (Figure 4). Nevertheless, further research on the relationship between these signals and ALI is warranted.

5.1.2 Chronically elevated and variable energy expenditure

Allotaxis and allostatic load cost metabolic energy for responding and attempting to adapt to environmental and

physiological perturbations to maintain homeostasis (Bobba-Alves et al., 2022). Bobba-Alves et al. reported that the transition from allostasis to allostatic load can be defined by an energetic tradeoff wherein allostasis and stress-related energy costs compete with growth, maintenance, and repair mechanisms (Bobba-Alves et al., 2022). However, empirical research using wearable-based energy expenditure as a digital signature to determine whether allostatic load is experienced in personnel remains limited. Givens et al. employed a wrist-worn device for continuous physiological monitoring over a 10-week USMC training course and observed elevated total daily energy expenditures, on average, of 3000 kcal-day⁻¹ (Givens et al., 2023a). Using the allostatic load framework, Feigel et al. observed that male and female personnel who sustained an overuse MSKI had chronically elevated and variable wrist-worn commercial wearable-derived energy expenditure compared to uninjured counterparts, which may help verify this digital signature of allostatic load (Feigel et al., 2024a). To our knowledge, this was the only investigation that used the allostatic load model to evaluate whether wearable-derived energy expenditure was associated with a tertiary outcome. Although these results may suggest that chronically elevated and variable energy expenditures may be digital signatures of the phenotype (Figure 4), further research on the relationship between wearable-derived energy expenditure and ALI is warranted.

5.1.3 Altered sleep behavior

Altered sleep behavior is reported as a consequence of allostatic load owing to elevated neuroendocrine, inflammatory, and autonomic nervous system activity experienced from daytime stressors (McEwen, 2006; Christensen et al., 2022; Balbo et al., 2010). Magtibay and Umapathy report that wearables may capture sleep health for allostatic load monitoring (Magtibay and Umapathy, 2023). Fortunately, previous research supports this claim. Using a 23-biomarker ALI, Bei et al. assessed the association of ALI and wearable-derived sleep health via actigraphy, including bed time, rise time, sleep efficiency [ratio of total sleep time to time in bed, multiplied by 100 to yield a percentage], total sleep time, sleep onset latency, wake-after-sleep-onset, and observed that later average bed time ($\beta = 0.15$, SE = 0.02, 95% CI: 0.03, 0.26) and shorter average total sleep time ($\beta = -0.13$, SE = 0.02, 95% CI: -0.24, -0.02) were associated with a higher ALI score, and more variable sleep-onset-latency ($\beta = 0.14$, SE = 0.02, 95% CI: 0.02, 0.26) and wake-after-sleep-onset ($\beta = 0.13$, SE = 0.04, 95% CI: 0.01, 0.26) were associated with ALI (Bei et al., 2017). Martucci et al. observed no significant difference in an 11-biomarker ALI score between patients with insomnia confirmed via actigraphy and normal sleepers (insomnia: 2.5 ± 1 vs. normal: 2.0 ± 1 , $p = 0.200$) (Martucci et al., 2020). However, Feigel et al. found that wrist-worn device-derived absolute and relative time spent in restorative sleep stages, including deep and light stage sleep, was associated with overuse MSKI status during military training (Feigel et al., 2024a). Together, these results may suggest that shorter sleep time, inconsistent sleep patterns, and greater time spent in restorative sleep may serve as signatures of the phenotype (Figure 4).

5.1.4 Blunted heart rate variability

Corrigan et al. (2021) conducted a systematic review on the influence of military training or tactical operator stress on heart

rate variability as a method to assess allostatic load (Corrigan et al., 2021). However, whether the signals were associated with their formal established measurement, the ALI, or secondary or tertiary outcomes were not reported to support the use of heart rate variability as a measure of allostatic load. Nevertheless, the authors revealed an overall reduction in heart rate variability indices in response to acute physical and cognitive stressors, with slower rates of recovery after the completion of acute occupational stressors that was dependent on the magnitude of the stress, as well as chronic stressors observed during nightly heart rate variability assessments (Corrigan et al., 2021). Only one study assessed daily resting heart rate variability in soldiers with markers of stress to provide context to the signal responses (Huovinen et al., 2009). However, without linking the signals with its formal measure or secondary or tertiary outcomes, the definition of allostatic load may be neglected its full use in explaining psycho-physiological maladaptation. Further investigation linking heart rate variability, ALI, and psycho-physical outcomes is warranted. Hence, further research on whether blunted heart rate variability may serve as a digital signature of the phenotype is warranted (Figure 4). A summary of the wearable-derived signals used for allostatic load assessment in the literature, their definitions, and signals often observed in consumer, wrist-worn commercial wearable devices based on Peake et al. (2018), which may be useful for allostatic load measurement in military training environments, is found in Table 5.

6 Limitations and knowledge gaps

As the allostatic load model gains traction in military field training research, several limitations and knowledge gaps remain that must be addressed to support its practical application and clarify its potential role as a mechanism for training-related maladaptation. First, it has been observed that the ALI remains underused in military personnel during training courses (Feigel et al., 2025a). Future research measuring ALI and determining its role on psycho-physical outcomes is warranted. Additionally, future research assessing the linkage between the ALI and wearable-derived signals is warranted to determine whether commercial wearable signals can suit allostatic load measurement in military settings. Further, whether the ALI is associated with different psycho-physical outcomes between sexes (Feigel et al., 2025a), and whether different wearable signals are associated with outcomes and ALI between sexes remains unknown. However, future research on determining which wearable may consider the recommendations provided on Table 5, Friedl (2018), or adopt similar valid wrist-worn models adopted from Feigel et al. (2024a) for “wear and forget” interventions.

Second, wrist-worn commercial wearables produce *in vivo* data that are temporal and dynamic wherein averages of time-series data may neglect the dynamic characteristics of physiological time-series data (Feigel et al., 2024a). These characteristics include linear serial dependencies such as trends, rhythms, and autoregressive dynamics. Non-linear characteristics (Richman and Moorman, 2000), such as changes in complexity (Young and Benton, 2015), are inherent to wearable device data (Gronwald et al., 2021), which can indicate critical increases or decreases in fluctuation (i.e., heart rate) (Gronwald et al., 2020) and can therefore be useful in

revealing the most stressful impacts that stretch allostasis and result in allostatic load. Consideration should be given to how dynamic instruments can provide data to improve our understanding of allostasis and allostatic load. Understanding how allostasis works in everyday life from a dynamic standpoint can help to prevent allostatic load and its consequences.

Third, allostasis and allostatic load research is dominated by population-based designs, such as cross-sectional, case-control or longitudinal studies (Juster et al., 2010; Carbone et al., 2022). Intense within-individual analysis applying time series analysis is very rare and may reveal when and under which circumstances allostatic loading occurs (i.e., reduced physical performance with blunted heart rate variability and high ALI).

Fourth, allostatic load may not be the only model to adopt in explaining military training-related maladaptation (Selye, 1950; Bates et al., 2013). Previous frameworks have been used in military health sectors to bring actionable and measurable risk factors to the forefront of prevention (Bates et al., 2013; Deuster and O'Connor, 2015; Givens et al., 2023b; Jonas et al., 2010). However, such frameworks may not contain biological variables that can quantitatively link a unified mechanism with psycho-physiological outcomes. For example, Bates et al. (2013) introduced the Military Demand-Resource Model (MDR) which, using the Conservation of Resources Theory and the Job Demand Resource Model, was developed for military personnel outside combat roles or for trainees. This model conceptualized how demands, such as information overload, non-combat tasks, and resources (i.e., external: leadership, training; internal: awareness, coping, engagement) interact to affect psychological wellbeing, resilience, and cognitive performance. Although the model addresses psychological aspects and operator performance, it does not consider musculoskeletal health or physical fitness (Bates et al., 2013). Hence, the adoption of allostatic load may be able to provide quantifiable means (i.e., ALI, wearable-assessed signals) to link stress with important military health outcomes for job-role performance. However, the allostatic load model may not be able to explain other non-stress-related military training health outcomes, such as acute traumatic MSKIs from a fall or trauma (Jones et al., 2010). Nevertheless, the allostatic load model may outline a promising biologically-grounded mechanism to explain stress-related health outcomes for future study.

Additionally, Schulkin (2004a) purports that the allostasis and allostatic load models are more advantageous than the more common stress regulation model, homeostasis (Schulkin, 2004a). Homeostasis and allostasis aim to achieve physiologic stability (Schulkin, 2004b). However, each model employs different methods to meet this objective that may render allostasis and allostatic load more favorable for adoption in explaining military training-related maladaptation. Homeostasis leverages negative feedback mechanisms to render setpoints stable (static) and return deviations to pre-stressor and normal levels (Schulkin, 2004a). In contrast, allostasis exemplifies the principle of maintaining stability through constant variation (dynamic) of all the parameters of its internal milieu and appropriately matching them to environmental demands (“stability through change”) to allow for subsequent adaptation (Figure 1). Hence, the allostasis model emphasizes a dynamic rather than static principle to achieve biological setpoints and considers the brain a central component in feedback

TABLE 5 Description of existing wearable-based measures of allostatic load used in the literature and measures that are programmed in modern wrist-worn, commercial wearables from **Peake et al. (2018)**.

Measure	Description	Available in most wrist-worn, commercial wearables
Heart rate (bpm) (Milosevic et al., 2013; Bulmer et al., 2022a)	Rate of cardiac contractility per minute; Rate reflects dominance of autonomic nervous system activity with lower rates indicative of parasympathetic branch activation and higher rates indicative of sympathetic branch activity; Sensitive to HPA and SAM activity from stress. Available in most commercial wearables	Yes
Heart rate variability (Milosevic et al., 2013; Corrigan et al., 2023)	Period (time) between beats (i.e., inter-beat interval duration) measured by frequency or time-domain methods. Nonlinear time series analysis methods (i.e., detrended fluctuation analysis) demonstrate utility in reflecting frequency domain methods to stress. Higher variability has been associated with greater autonomic nervous system balance between branches, with lower variability associated with singular branch dominance that may require heart rate for context. Not available in most commercial wearables	Yes (if No, calculate from time-series data)
Total daily energy expenditure (kcal, rate) (Magtibay and Umapathy, 2023)	Magnitude or rate of total daily calories burned that is estimated using a combination of heart rate, anthropometric, demographic, and activity data captured by wearable devices. Available in most commercial wearables	Yes
Physical activity energy expenditure (kcal, rate) (Magtibay and Umapathy, 2023)	Magnitude or rate of total calories burned during physical activity estimated by wearable devices	Yes
Systolic blood pressure (mmHg) (Milosevic et al., 2013)	Pressure in the arteries when the heart beats and pumps blood throughout the body; Measured by a noninvasive 24-h ambulatory blood pressure monitor worn on the peripheral upper limb to provide a real-time picture of blood pressure fluctuations during the day and evening	No
Diastolic blood pressure (mmHg) (Milosevic et al., 2013)	Pressure in the arteries when the heart relaxes between beats; Measured by a noninvasive 24-h ambulatory blood pressure monitor worn on the peripheral upper limb to provide a real-time picture of blood pressure fluctuations during the day and evening	No
Electrodermal activity (Milosevic et al., 2013)	Changes in the skin's electrical conductance due to sweat gland activity, which is a non-invasive method to assess emotional and physiological arousal to stress; Sensitive to metabolic and neuroendocrine systems; Measured by applying a small current between two electrodes placed on the skin and measuring the skin's resistance or conductance to that current (microsimens). Increased activity is associated with elevated sympathetic nervous system activity	No
Sleep Duration (hh:mm:ss) (Bulmer et al., 2022a; Christensen et al., 2022)	Time period at which one is asleep, with lesser time often associated with indices of allostatic load from biomarker-based measurements	Yes
Sleep Architecture (hh:mm:ss, %) (Christensen et al., 2022)	Time or proportion spent in each sleep stage: Light, Deep, and Rapid-Eye-Movement [REM], with greater time spent in restorative (Deep, REM) stages associated with lower allostatic load index	Yes

regulation, for whole-body adaptation to contexts (Schulkin, 2004b). Indeed, the brain plays a central role in allostasis (McEwen and Gianaros, 2011; Cohen et al., 2016; Korte et al., 2005). The brain controls the mechanisms across systems via the

activity of mediators of allostasis to induce constant variation of parameters in response to stress (McEwen, 2003). Allostasis enforces that the brain designates command by modulating the extent of allostasis via influential factors (experience, memories),

individualization (perception of stress, physical condition of the body) (McEwen B. S., 1998), and re-evaluation of needs by anticipating physiological requirements before the behavior (Drug Addiction, 2025). In contrast, the homeostasis model views each organ system as independent from the brain without any influence of modulation (Schulkin, 2004b).

Together, addressing these gaps and limitations may generate the empirical support needed to advance the allostatic load model to the forefront within military sectors and practice—not only within the US but across international Armed Forces—as an alternative framework for better understanding how military-training-related stress may instigate maladaptive health outcomes. By measuring and testing allostatic load against such outcomes, research physiologists can help develop interventions to mitigate allostatic load and enhance post-training military readiness.

7 Conclusion

Research physiologists use theoretical models to test new empirical relationships between physiological variables and psychophysiological outcomes and compare observed outcomes with theoretical predictions to support or refute models. Allostatic load is a model outlining a biological process whereby physiological stability fails owing to recurrent and chronic stress exposure. This model may be a suitable framework to assess its role on musculoskeletal, physical performance and psychological maladaptation during training. Military-training-related stressors, such as energy restriction, cognitive stress, physical overtraining, and sleep deprivation, disrupts neuroendocrine, immune, and autonomic systems, and contributes to allostatic load as a potential mechanism underlying military training-related maladaptation. However, although epidemiological studies on allostatic load measured by ALI demonstrate relationships with poorer physical performance and psychological and musculoskeletal health, further empirical research in military training populations is warranted to support this model. Owing to the limitations of assessing allostatic load via ALI in occupational settings and longitudinal study designs, future research in military training environments may benefit from wrist-worn, commercial wearable technology as opposed to the ALI to measure downstream cardiometabolic and neurobehavioral responses perturbed by stress systems. However, wearable signals should be verified based on the formal definition of allostatic load by determining whether the signals are associated with the ALI and whether signals are associated with secondary and/or tertiary outcomes. The digital phenotype of allostatic load, characterized by one or more digital signatures, such as chronically elevated and variable cardiometabolic activity and altered neurobehavioral responses, requires further testing before it can be implemented in military training as an “at-risk” indicator. Further research could help military practitioners and leadership identify personnel in need of intervention and combat mitigate allostatic load and prevent maladaptive psycho-physical outcomes.

Data availability statement

The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author.

Author contributions

EF: Conceptualization, Visualization, Investigation, Methodology, Resources, Writing – original draft, Formal Analysis. KK: Methodology, Conceptualization, Writing – review and editing, Supervision, Visualization. ML: Writing – review and editing, Methodology, Supervision, Visualization, Conceptualization. KF: Methodology, Supervision, Visualization, Conceptualization, Writing – review and editing. BM: Project administration, Supervision, Methodology, Writing – review and editing, Resources, Visualization. BN: Resources, Writing – review and editing, Funding acquisition, Visualization, Project administration, Conceptualization, Supervision.

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