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RESEARCH ARTICLE

Age differences in allostatic load among veterans: The importance of combat exposure

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Abstract

The current study examinedage differences in allostatic load among nonveterans, noncombat veterans, and combat veterans. Participants included 280 individuals from the Midlife Development in the United States (MIDUS) survey, including 164 veterans ($n = 48$ combat veterans; $n = 116$ noncombat veterans) and 116 nonveterans. Age differences in allostatic load were similar among nonveterans and noncombat veterans, $B = 0.002$, $SE = .011$, $p = .878$, with older adults showing higher levels of allostatic load than their comparatively younger counterparts. Among combat veterans, however, a different pattern emerged. In this group, levels of allostatic load were similar across age, seemingly due to higher levels of allostatic load among younger combat veterans, $B = -0.029$, $SE = .014$, $p = .031$, $\eta_p^2 = .022$. Results reveal the importance of considering combat exposure when examining health outcomes of military veterans, particularly in the context of age.

Military service has been linked to improved physical health among younger veterans but worse health among older veterans (Landes et al., 2017; MacLean, 2013; Wilmoth et al., 2010). The reasons for this are multifactorial. At the outset, individuals who serve in the military tend to be healthier and more strongly integrated into a social network (Wilmoth et al., 2019; Wolf et al., 2013). Yet, a shift occurs in later life, with research indicating that older veterans report poorer health outcomes than nonveterans due to factors such as poorer health behaviors (Wilmoth et al., 2010, 2019). Furthermore, there is evidence that differences in health outcomes between veterans and nonveterans vary by war era, race/ethnicity, and level of combat exposure (Landes et al., 2017, 2018; Maclean & Edwards, 2017). Military service is also associated with more frequent stressor exposure (Pflanz & Sonnek, 2002), and, because there is a biological toll each time the stress response is initiated (McEwen, 2006), the effects could accumulate over time, leading to poorer health (Institute of Medicine, 2008). This biological toll often occurs before the onset of disease and could be one pathway through which poorer health among veterans emerges over time. To this aim, the current study examined whether age differences in allostatic load (AL)—a measure of biological dysregulation—varies according to military status (i.e., nonveterans, noncombat veterans, and combat veterans).

A substantial body of research has demonstrated poorer health outcomes, commonly assessed through specific documented diagnoses or point-in-time global measures of self-reported health, for veterans versus nonveterans (for a summary, see Wilmoth et al., 2019). Researchers have also explored whether veteran–nonveteran differences in health outcomes vary across the life course due to countervailing aspects of military service (MacLean, 2013; Wilmoth et al., 2019). Military service members appear to have an early- to midlife health advantage due to two factors: a "healthy soldier effect" and a "military capital effect." The healthy soldier effect results from individuals being screened for health problems before entry into military service, leading to a positive health selection effect that extends at least into the early part of postservice veteran life (Wilmoth et al., 2019; Wolf et al., 2013). The military

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capital effect refers to the multiple benefits that result from military service, such as health-promoting behaviors, long-lasting, robust social networks, military-related occupational skills that translate into improved civilian employment opportunities, and postservice health care and social benefits (Kleykamp, 2007; MacLean, 2013; Sheehan & Hayward, 2019; Wilmoth et al., 2019). These factors may partly explain why veterans have higher ratings of self-reported health, fewer comorbidities at 50 years of age (Wilmoth et al., 2010), and lower mortality risk at younger ages (Landes et al., 2017) than nonveterans.

The veteran health and mortality advantage that is present in early- to midlife, however, does not appear to be as strong, or present at all, among those who served longer, were exposed to combat or environmental hazards, acquired a service-connected disability, or served during a particular war era (Landes et al., 2017, 2018, 2019; Porter et al., 2019; Sheehan et al., 2015; Taylor et al., 2015; Wilmoth et al., 2010, 2018). In addition, the early life health advantage some veterans experience does not appear to persist into later life (e.g., Wilmoth et al., 2010). Findings from several studies have indicated that health declines more rapidly among veterans compared to nonveterans, resulting in a health and mortality disadvantage that is apparent by 60–70 years of age and extends into older age (Landes et al., 2017; Liu et al., 2005; London & Wilmoth, 2006; Wilmoth et al., 2010). This shift in health outcomes is attributed to a military hazard effect (Wilmoth et al., 2019). Compared with nonmilitary occupations, military service typically involves exposure to training or combat-related risk factors or hazards, inclusive of possible exposure to toxic chemicals, that can result in an increased chance of incurring physical and psychological harm (Aldwin et al., 1994; Ardelt et al., 2010; Bullman & Kang, 2000; Schnurr & Spiro, 1999; Taylor et al., 2015; Wilmoth et al., 2019). In addition, health behaviors one develops in response to military culture or required activity levels during their time in service, such as smoking and high caloric intake, often extend postservice and may have detrimental effects on long-term health (London et al., 2017; Teachman & Tedrow, 2013). Moreover, interpersonal trauma, such as sexual harassment or assault, which are experienced disproportionately by female service members, may have long-term effects on health (O'Brien & Sher, 2013; Suris & Lind, 2008). The cumulative effect of illness, injury, or trauma exposure during one's time in service, combined with the continuation of adverse health behaviors, can result in poorer health outcomes and increased mortality risk for veterans later in life (Landes et al., 2019; Wilmoth et al., 2019).

The explanations provided for the possible health crossover for veterans, from advantage to disadvantage, are plausible. It is logical to think that the cumulative effect of exposure to physical and psychological hazards, as well as postservice negative health behaviors, may lead to poorer health outcomes later in life. Yet, beyond the more severe outcomes of injury, illness, or negative health behaviors, occupational stress related to military service may be more severe compared with most other occupations (Adler et al., 2004; Campbell & Nobel, 2009). Although military service members experience typical occupational stressors, such as work overload, role uncertainty, interpersonal conflicts, and organizational constraints, even when not they are deployed or engaged in combat, they also face the increased stress of knowing that due to unexpected shifts in geopolitics, they could be deployed at any time (Adler et al., 2004; Campbell & Nobel, 2009). A primary stressor of being deployed or anticipating deployment is concerns of being shot at, injured, or killed (Kulka et al., 1990); however, beyond the stress related to an increased risk of harm to oneself, current or anticipated deployments also involve stress due to separation from family members, suboptimal living conditions, loss of freedom, exposure to wounded or dead individuals, and concerns about one's ability to complete mission requirements such as killing (Institute of Medicine, 2008; McEwen et al., 2012).

Recognizing that military service likely leads to levels of unpredictable and uncontrollable stress that are comparatively higher than those associated with nonmilitary occupations, it is plausible that stress-linked alterations in physiological biomarkers are more prevalent among individuals who have served in the military. Few studies, however, have examined the link between military service and physiological biomarkers. Of the studies that have been conducted, most have focused on the link between biomarkers and blast exposure (e.g., Gyorgy et al., 2011; Wang et al., 2017) or the development of posttraumatic stress disorder (PTSD). For example, the authors of one study found that individuals who showed higher levels of norepinephrine shortly after deployment were more likely to develop PTSD 3 months later (Highland et al., 2015). Similarly, findings from another study demonstrated that individuals who showed dysregulated chemokines, which are biomarkers of inflammation, were also more likely to develop PTSD (Zhang et al., 2020). Research also indicates that an underlying cause of "Gulf War Illness" is heightened inflammatory biomarkers (Butterick et al., 2019), further demonstrating that dysregulated biomarkers are linked to worse health in military veterans.

One way to comprehensively assess biomarkers across multiple domains of health is through AL, which is tied to the concept of allostasis. Allostasis refers to the body's ability to meet the demands of a stressor (McEwen, 2001). For example, to flee from a threat, the body must adapt by increasing its heart rate and blood pressure. When the threat is over, the body returns to homeostasis. The dynamic process of responding to a threat and returning

to homeostasis is termed allostasis. Repeatedly engaging in this process can eventually tax organ systems, resulting in cumulative biological dysregulation, or AL (McEwen, 1998). AL has been linked with adverse life events and circumstances, such as lower socioeconomic status (e.g., Seeman et al., 2004), lower levels of social integration (Seeman et al., 2002), and exposure to environmental stressors (Mair et al., 2011). Although suggested as a possible explanation for differences in veteran and nonveteran health (Institute of Medicine, 2008; McEwen et al., 2012), to our knowledge, no studies have compared AL between these two populations. If veterans do experience increased AL, this may provide insight into the differential health patterns observed between veterans and nonveterans across the lifespan. Moreover, because AL exists at a subclinical level, differences could exist even in the absence of clinical disease.

Differences in health outcomes between veterans and nonveterans may be further magnified by combat exposure, as it has been associated with poorer health outcomes among military veterans (e.g., Elder et al., 2016). In a study of United States Civil War veterans, for example, military trauma, including increased exposure to military comrade deaths, was associated with increased morbidity and mortality (Pizarro et al., 2006). Similarly, in a study of World War II veterans, individuals who were exposed to combat showed increased morbidity and mortality 15 years after the war ended (Elder et al., 1997). A study of veterans who participated in the Health and Retirement Study also reported an association between combat exposure and later-life health. However, the link between combat exposure and later-life health was largely due to concomitant traumatic experiences, such as exposure to dead combatants (Taylor et al., 2016). Finally, among individuals deployed between 2001 and 2006, those who saw combat had worsening trajectories of health over a 15-year period than those who did not (Porter et al., 2019).

Findings from studies utilizing veteran-only samples have indicated that combat exposure can lead to particularly pernicious health outcomes. Thus, it is possible that it is not veteran status but rather combat exposure that is associated with higher levels of AL. This hypothesis is supported by findings from two prior studies. Analyzing data from the National Comorbidity Study, Sheffler et al. (2016) reported a higher prevalence of physical health conditions among men who had experienced combat compared with men who had not experienced combat; however, this study did not control for veteran status, preventing discernment of possible health differences between noncombat veterans and nonveterans. Maclean and Edwards (2017) were able to overcome this limitation by using data from the Health and Retirement Study. They report that whereas combat veterans had more life-threatening illnesses than nonveterans, there was no such difference between noncombat veterans and nonveterans.

It is also unclear if typical age-related patterns of AL—that is, higher levels with increasing age—emerge among veterans. On one hand, the heightened stress of serving in the military may result in progressively higher levels of AL with increasing age. On the other hand, normative age-related processes may attenuate differences in AL at later ages that are apparent at earlier ages (Crimmins et al., 2003). If so, differences in levels of AL may be higher among younger veterans but less pronounced with increasing age.

Most previous research on veteran health outcomes has utilized veteran-only samples, with relatively few comparing veteran and nonveteran health (Wilmoth et al., 2019). Among studies that have compared veteran and nonveteran health outcomes, only one accounted for combat status, and none examined differences in AL. The current study addressed gaps in the literature by examining age differences in AL between veterans and nonveterans while accounting for combat status. We expected that the comparatively higher levels of stress during military service, especially for individuals who experienced combat, would lead to differences in AL between veterans and nonveterans, from lowest to highest, in the following order: nonveterans, veterans without combat exposure, and veterans with combat exposure. However, informed by research indicating that levels of AL tend to level off after 60 years of age (Crimmins et al., 2003), we predicted that the effect would be most pronounced at younger ages.

METHOD

Participants and procedure

Data for the current study came from the Midlife Development in the United States (MIDUS) survey, which is an ongoing national longitudinal study that aims to understand factors contributing to mental and physical health across adulthood. The MIDUS study includes a main project, consisting of a telephone interview and self-administered questionnaires, as well as multiple subprojects (Radler, 2014). Wave 1 was collected in the mid-1990s, and Wave 2 was collected approximately 10 years later. Participants from Wave 1 included noninstitutionalized, English-speaking adults who were recruited using random digit dialing (RDD) from phone numbers found in telephone banks (Radler, 2014). Because the collection of physiological biomarkers was added during Wave 2, all analyses in the current study used Wave 2 data (for information on MIDUS participants at Wave 2, see Radler & Ryff, 2010). Wave 2 participants (N = 5,555) consisted of five groups: individuals who were contacted through RDD (n = 2,257);

twins (n = 1,484) and siblings (n = 733) of the RDD group; oversamples from metropolitan areas (n = 489); and an African American sample from Milwaukee (n = 592). Only participants who completed the main survey (i.e., 2004–2006) and the Biomarker Project (i.e., 2004–2009) were retained for the present analyses (n = 1,255).

The Biomarker Project was designed to collect a comprehensive set of biomarkers from a subsample of MIDUS participants. All Wave 2 MIDUS participants were invited to participate in this subproject. Of the larger sample, 1,255 individuals chose to participate and 1,243 had useable data. Participants underwent an intensive, 2-day physical examination at one of three general clinical research centers (GCRCs), which were located at the University of California Los Angeles (UCLA), the University of Wisconsin, and Georgetown University. During these examinations, participants provided anthropometric data and samples of blood, saliva, and urine, from which biomarkers were obtained. All samples were collected and processed according to standardized instructions (for details, see Love et al., 2010). All procedures were approved by the Institutional Review Board of the University of Wisconsin and UCLA. Individuals who participated in the Biomarker Project were similar to those who did not participate with regard to age, race, sex, income level, and marital status; however, participants who completed the Biomarker Project were more likely to have a college degree (Love et al., 2010).

Of the 1,255 participants who completed the Biomarker Project, 890 answered the Armed Forces questions. Participants who did not complete these questions were more likely to be younger, $t(1253) = 4.7$, $p < .001$; White, $\chi^2(1, N = 1{,}253) = 34.37 = 34.37$, $p < .001$; and female $\chi^2(1, N = 1{,}253) = 34.37 = 22.15$, $p < .001$, than those who did. Nonveterans represented 81.1% of the original sample (n = 722) and veterans represented 19.1% of the sample (n = 170). Among veterans, 29% (n = 48) experienced combat and 71% (n = 120) did not.

As described in the Measures section, participants were grouped according to their veteran status as combat veterans, nonveterans, and noncombat veterans. Combat veterans ranged in age from 40 to 82 years, nonveterans ranged in age from 36 to 82 years, and noncombat veterans ranged in age from 36 to 83 years. To ensure that the results were not driven by younger participants in the nonveteran and noncombat veteran samples, individuals younger than 40 of age were excluded from all analyses. This resulted in a total of eight participants being removed (n = 4 nonveterans, n = 4 noncombat veterans). In addition, because the nonveteran sample was much larger than the veteran sample, we culled a smaller age- and gender-matched subset of nonveterans (n = 116) using propensity-scored matching. The final sample included 116 nonveterans, 116 noncombat veterans, and 48 combat veterans. Although the pattern of results did not differ when analyses were conducted on the full sample (N = 890) versus the matched sample (n = 280), all reported analyses were conducted on the matched sample for consistency. Participants were, on average, 61.5 years old (SD = 10.72) at the time of data collection, and most were male (85.0%). The majority of participants were White (81.1%), and 46.4% of the sample had earned a bachelor's degree or higher (see Table 1 for a list of sample characteristics by veteran status).

MEASURES

Biomarkers and AL

AL was calculated using biomarkers of seven physiological systems that were collected during each participant's overnight GCRC visit. Indicators of cardiovascular functioning included resting pulse rate and systolic and diastolic blood pressure. Indicators of hypothalamic–pituitary–adrenal (HPA) axis activity included serum dehydroepiandrosterone sulfate and overnight urinary cortisol. Indicators of glucose metabolism included fasting glucose, glycosylated hemoglobin, and insulin resistance. Indicators of inflammation included C-reactive protein, fibrinogen, IL-6, intracellular adhesion molecule-1, and e-Selectin. Indicators of lipid metabolism included body mass index, high- and low-density lipoprotein cholesterol, triglycerides, and waist-hip ratio. Indicators of parasympathetic nervous system functioning included heart rate variability, including high and low spectral power, the standard deviation of R–R (i.e., heartbeat to heartbeat) intervals, and root mean square of successive differences. Indicators of sympathetic nervous system functioning included overnight urinary epinephrine and norepinephrine. Because the number of biomarkers assessed per system varied from two to six per system, system risk scores were obtained.

To obtain individual system risk scores, values for each biomarker were divided into quartiles, and the proportion of biomarkers for which the participant scored in the high-risk range (i.e., upper or lower quartile depending upon whether high or low values conferred a larger health risk) was calculated. Risk scores, which ranged from 0 to 1 for each system, were then added together for a minimum AL score of 0 and a maximum score of 7. Using this method to compute AL scores ensures that all systems were represented equally regardless of the number of biomarkers assessed. This method has been validated (Gruenwald et al., 2012) and used in previous research (e.g., Brooks et al., 2014; Friedman et al., 2015; Hamdi et al., 2016).

TABLE 1 Demographic characteristics of the sample subgroups

Variable	Nonveterans ($n = 116$)			Noncombat veterans ($n = 116$)			Combat veterans ($n = 48$)		
	M	*SD*	%	*M*	*SD*	%			
Age	59.6*	10.3		63.3	10.8		61.6	10.9	
BMI (kg/m^2)	29.3	5.5		29.5	4.7		29.4	5.8	
Chronic conditions	1.8	1.6		2.0	1.5		1.7	1.3	
Race									
White			79.3			86.2			79.2
Other			20.7			13.8			20.8
Educational attainment									
Less than high school			6.9			6.0			0.0
High school/GED			20.7			16.4			18.8
Some college			26.7			30.2			35.4
College degree or higher			45.7			47.4			45.8
Sex									
Male			73.3***			91.4			97.9
Female			26.7***			8.6			2.1
Smoking history									
No			62.1***			39.7			27.1
Yes			37.9***			60.3			72.9
Prescription medication									
No			23.3			18.1			20.8
Yes			76.7			81.9			79.2

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

History of service in the armed forces

To assess participants' history of service in the U.S. Armed Forces, individuals were asked whether they had ever entered the Armed Forces as well as whether they had ever experienced combat. Participants who replied "no" to both questions were classified as nonveterans, participants who answered "yes" to the Armed Forces question but "no" to the combat question were classified as noncombat veterans, and participants who answered "yes" to both questions were classified as combat veterans.

Covariates

Covariates in the final model included sex (categorized as male or female, as no other descriptions were available in the dataset), race (categorized as White or other because of the small number of minority participants in the sample), educational attainment (categorized as some high school or less, a high school diploma or GED, associate's degree or some college, or bachelor's degree or higher), smoking history (categorized as never smoked or smoked), body mass index (BMI; continuous and mean-centered), prescription drug use (categorized as yes or no), and number of chronic health conditions, subsumed into 18 categories: lung conditions (i.e., asthma, bronchitis, emphysema, other lung conditions or tuberculosis); bone-related conditions (i.e., arthritis and backaches); digestive conditions (i.e., stomach trouble, indigestion, diarrhea or constipation); HIV/AIDS; autoimmune disorders; high blood pressure; diabetes; neurological problems; history of heart trouble; stroke; trouble with mouth, teeth or gums; history of cancer; thyroid disease; hay fever; bladder-related conditions; gall bladder problems; migraines; and hernia. Chronic conditions were winsorized at five or more conditions.

Data analysis

Before conducting the analyses, all variables were examined for distributional properties, outliers, and out-of-range values. To minimize the effect of extreme values, outliers were winsorized at the 99th percentile. Analyses were conducted in SAS (Version 9.4), using generalized estimating equations (GEE) in Proc Gen Mod. GEE was chosen over traditional general linear models because it allows for the analysis of correlated data, necessary in the current study due to the inclusion of siblings and twins (for details on how to conduct a GEE, see Liang & Zeger, 1986). Because there was no evidence of skewness or kurtosis in the outcome variable and no

TABLE 2 Allostatic load among nonveterans and veterans (N = 280)

Variable	Model 1 (Main effects only)		Model 2 (Main effects and interaction)		Model 2 (Main effects, interaction, and covariates)	
	B	*SE*	*B*	*SE*	*B*	*SE*
Intercept	1.922*	0.076	1.928*	0.076	2.063*	0.234
Veteran status (ref. = nonveterans)						
Veterans	−0.033	0.114	−0.026	0.114	−0.071	0.110
Age	0.035*	0.005	0.030*	0.007	0.037*	0.007
Age x Veteran Status (ref. = nonveterans)						
Age x Veterans			0.012	0.011	0.007	0.011
Sex (ref. = Female)					−0.285	0.155
Race (ref. = White)					−0.048	0.159
Educational attainment (ref. = bachelor's degree or higher)						
Less than high school degree					0.396	0.262
High school diploma/GED					0.386*	0.138
Associate's degree/some college					0.204	0.122
Smoking (ref. = never smoked)					0.004	0.116
Body mass index (kg/m^2)					0.059*	0.011
Chronic conditions					0.041	0.039
Medication use (ref. = none)					−0.105	0.134

Note: $N = 280$.
*$p < .05$, **$p < .01$, ***$p < .001$.

data transformations were necessary, the distribution was specified as normal, and the link function was specified as identity. The outcome variable in all models was AL, and the explanatory variables included age, veteran status, and their interaction. Covariates included sex, race, educational attainment, BMI, smoking history, current medication use, and chronic conditions.

RESULTS

In the first set of analyses, we examined AL differences among veterans ($n = 164$) and nonveterans ($n = 116$), with the veteran group inclusive of both combat veterans and noncombat veterans, as this is how most previous research comparing veterans and nonveterans has been conducted. Three nested models were run: The first model examined main effects only; the second model examined main effects and the interaction between veteran status and age; and the third model examined main effects, the interaction between veteran status and age, and all covariates (see Table 2). The results of the final model revealed a significant effect of age such that, consistent with previous research, age was associated with increased levels of AL, $B = 0.037$, $SE = .007$, $p < .001$. However, no significant effect was detected for veteran status, $B = -0.071$, $SE = .110$, $p = .515$, or the Veteran Status x Age interaction, $B = 0.007$, $SE = .011$, $p = .497$. Thus, when not controlling for combat status, no differences in AL emerged for veterans compared to nonveterans.

The second set of analyses were identical to the first except that veteran status was stratified into three groups: nonveterans, noncombat veterans, and combat veterans (see Table 3). The results of the final model revealed a significant interaction between age and veteran status. Age differences in AL were similar among nonveterans and noncombat veterans, $B = 0.002$ $SE = .011$, $p = .878$, but combat veterans showed a different pattern than nonveterans, $B = -0.029$, $SE = .014$, $p = .031$, $\eta_p^2 = .022$. To further probe this interaction, predicted AL scores at plus or minus 1 standard deviation above and below the mean were calculated. As can be seen in Figure 1, age differences in AL were larger for nonveterans and noncombat veterans than they were for combat veterans, an effect that appears to be driven by higher levels of AL among relatively younger combat veterans.

Because the combat veteran group was smaller than the other two groups, one concern is that the pattern of results differed because the combat veteran group was underpowered. To examine this possibility, we compared simple slopes in our sample of 280 participants as well as in a reduced, propensity-scored, age- and gender-matched sample of 141 all-male participants (47 combat veterans, 47 noncombat veterans, and

TABLE 3 Allostatic load among nonveterans, noncombat veterans, and combat veterans

Variable	Model 1 (Main effects only)		Model 2 (Main effects and interaction)		Model 2 (Main effects, interaction, and covariates)	
	B	*SE*	*B*	*SE*	*B*	*SE*
Intercept	1.889***	0.085	1.903***	0.085	2.033***	0.222
Veteran status (ref. = nonveterans)						
Noncombat veterans	0.050	0.120	0.025	0.120	0.063	0.117
Combat veterans	−0.007	0.177	−0.017	0.170	0.074	0.152
Age	0.035***	0.005	0.042***	0.009	0.045***	0.009
Age x Veteran status (ref. = nonveterans)						
Age x Noncombat Veterans			−0.002	0.012	0.002	0.011
Age x Combat veterans			−0.039**	0.015	−0.029**	0.014
Sex (ref. = Female)					−0.312*	0.154
Race (ref. = White)					−0.033	0.160
Educational attainment (ref. = bachelor's degree or higher)						
Less than high school degree					0.364	0.257
High school diploma/GED					0.354*	0.139
Associate's degree/some college					0.182	0.120
Smoking (ref. = never smoked)					0.006	0.113
Body mass index					0.058***	0.010
Chronic conditions					0.042	0.038
Medication use (ref. = None)					−0.123	0.135

Note: $N = 280$.
*$p < .05$, **$p < .01$, ***$p < .001$.

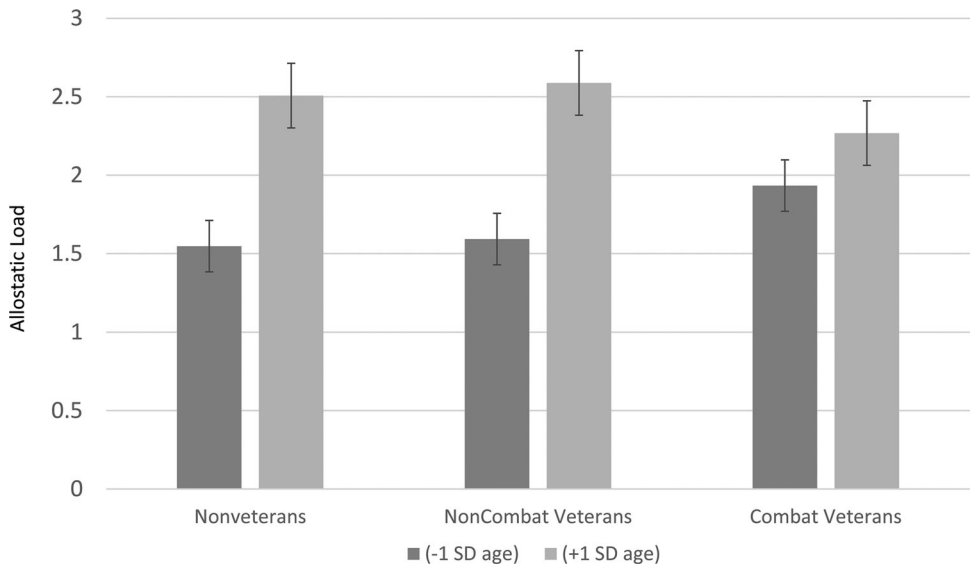

FIGURE 1 Age differences in allostatic load among nonveterans, noncombat veterans, and combat veterans

47 nonveterans). We excluded female participants in these more stringent analyses because only one female reported combat exposure. The results indicated that age-related patterns of AL among combat veterans differed from noncombat veterans in both the original matched sample, estimate = −.038, $p = .008$, and in the reduced sample, estimate = −.040, $p = .046$. Similarly, patterns between combat veterans and nonveterans differed in the original, matched sample, estimate = −.039, $p = .009$, and in the reduced sample, estimate = −.040, $p = .013$.

TABLE 4 Simple slope comparisons of the original, matched sample (N = 280) and the reduced sample (n = 141)

	Matched sample		Reduced sample	
Variable	***B***	***SE***	***B***	***SE***
Intercept	1.610***	0.097	1.680***	0.162
Veteran Status (ref. = nonveterans)				
Noncombat veterans	−0.075	0.240	−0.075	0.240
Combat veterans	0.035	0.137	0.164	0.243
Age	0.042***	0.009	0.044***	0.011
Age x Veteran Status (ref. = nonveterans)				
Age x Noncombat Veterans	−0.002	0.012	−0.000	0.019
Age x Combat veterans	−0.039**	0.015	−0.040*	0.016
Simple slopes				
Combat veterans	0.003	0.012	.004	0.012
Noncombat veterans	0.041***	0.008	.044**	0.016
Nonveterans	0.042***	0.009	.044***	0.011
Slope comparisons				
Combat veterans vs. nonveterans	−0.039**	0.015	−0.040*	0.016
Noncombat veterans vs. nonveterans	−0.002	0.012	−0.000	0.019
Combat veterans vs. noncombat veterans	−0.038**	0.014	−0.040*	0.020

Note: Full sample: $N = 280$; reduced sample: $n = 141$.
*$p < .05$, **$p < .01$, ***$p < .001$.

However, patterns did not differ among noncombat veterans and nonveterans in the original, matched sample, estimate = −.002, $p = .900$, or the reduced sample, estimate = −.000, $p = .997$ (see Table 4). We, therefore, conclude that our findings are not an artifact of an underpowered combat veteran sample.

Subclinical biomarker alterations could be the result of clinical disease. As such, we next used Poisson regression to determine if the interaction between age and veteran status was associated with the number of chronic health conditions a participant reported. When compared with nonveterans, no significant associations between age and chronic health conditions emerged for noncombat veterans, $B = -0.014$, $SE = .014$, $p = .185$, or combat veterans, $B = 0.007$ $SE = .013$, $p = .621$.

DISCUSSION

The current study examined whether combat exposure and age were differentially associated with AL in veterans, noncombat veterans, and nonveterans. The results revealed that patterns of AL were similar among nonveterans and noncombat veterans, with both groups showing higher levels of AL with increasing age. However, among combat veterans, AL levels were similar across age groups, seemingly driven by higher levels of AL among relatively younger combat veterans. These findings speak to the necessity of considering combat exposure when assessing health outcomes among veterans, particularly in the context of age.

Our results regarding higher levels of AL among younger combat veterans may help to explain why previous findings have demonstrated age-related shifts in health outcomes among veterans (e.g., Landes et al., 2017). On the surface, younger combat veterans may have no clinical manifestations of disease, such as chronic health conditions or physical health symptoms. At a subclinical level, however, their comparatively higher levels of stress could result in alterations of physiological biomarkers, which could accumulate over time and result in adverse health outcomes in later life (for a review, see Juster et al., 2010; Piazza et al., 2010). This hypothesis is supported by research indicating that although chronic health problems are more common in later life, precursors to these conditions appear earlier in the lifespan (e.g., Barzilay et al., 2007). For example, before a formal diagnosis of heart disease, there are often warning signs and symptoms, such as high cholesterol and elevated blood pressure (Bogers et al., 2007). Thus, a chronic condition often begins developing years before it manifests clinically. This same line of reasoning could provide one explanation for age-related shifts in veteran health outcomes.

The present results also illustrate the importance of taking combat exposure into account when comparing health outcomes between veterans and nonveterans. In our first set of analyses, wherein we compared patterns of AL between nonveterans and veterans, inclusive of

noncombat and combat veterans, no significant differences emerged. Due to data limitations, this would be the endpoint of analysis for most prior studies that have examined differences in veteran and nonveteran health. However, this approach fails to consider that combat veterans may show different profiles because they often experience higher levels of stress and trauma exposure than noncombat veterans (Institute of Medicine, 2008). It was only after we separated the veteran group into combat veterans and noncombat veterans—a strategy that is impossible in many data sets that examine veterans and nonveterans—that differences emerged. This lends support to research revealing that combat exposure has a particularly pernicious effect on physical health (e.g., for review, see Aldwin et al., 2018; Benyamini & Solomon, 2005; Levy & Sidel, 2009; Sheffler et al., 2016). It also supports MacLean and Edwards' (2017) assertion that mixed findings in previous literature may be due to researchers comparing veterans to nonveterans without separating veterans based on combat exposure.

From a theoretical perspective, our findings align with Schnurr and Green's (2004) integrative model on the direct and indirect effects of trauma exposure. According to this model, several factors occur to explain why traumatic experiences are associated with adverse health outcomes. Continual physiological responses, similar to the AL model, are one of the indirect pathways this model posits. In this vein, the current findings may provide a snapshot of the subclinical repercussions of combat exposure.

Although AL is not a disease, per se, individuals with chronic health conditions tend to have higher levels of AL (e.g., Mattei et al., 2010). As such, one concern is that AL is simply a proxy for poorer health. Follow-up analyses, however, indicated that the interaction between age and veteran status did not predict chronic health conditions. Thus, although younger combat veterans in the present sample showed elevated levels of AL, they did not report more chronic health conditions. This then begs the question: Why did the older combat veterans in the sample show levels of AL similar to age-matched noncombat veterans and nonveterans? One possible reason could be differential mortality. Previous findings have indicated that combat veterans are more likely to experience earlier mortality than noncombat veterans (e.g., Elder et al., 1997). Thus, it is possible that the older combat veterans in the current sample were a healthier group than would be expected.

Yet another question that arises from our results is why the interaction between combat status and age was not associated with chronic health conditions. One possible explanation is that the chronic condition variable in the current study lacked specificity. The variable consisted of 18 different chronic condition categories, which provides a reasonably comprehensive measure of health. However, it could be that military service, particularly combat service, raises the risk for certain conditions but not others. For example, research has indicated that Parkinson's disease (U.S. Department of Veteran's Affairs, 2016b), respiratory cancers (U.S. Department of Veteran's Affairs, 2016b), and musculoskeletal ailments (Goldberg, 2008) are prevalent among veterans. Thus, had individual conditions been examined, the results may have differed. Unfortunately, the current study lacked the power to examine each condition separately. Future researchers should examine this question in larger samples.

Although the current study took a novel approach in exploring the of age and combat exposure on AL, some limitations should be addressed. First, the sample size was small, limiting our ability to make generalized conclusions. Future research with a larger sample of combat veterans is needed to confirm the present findings. Second, our measure of participants' history of service in the armed forces was limited to two questions: if a person served and if they experienced combat. Not included in the current study were more detailed questions as they relate to the armed service experience, such as the war era during which veterans served; how long they served, how many times they were deployed; and, where applicable, the intensity of the combat exposure they experienced. These factors might distinctly influence the health of veterans years later, and, thus, they should be examined in future research. Third, although our sample ranged in age from 40 to 83 years, the average participant age was 62 years. Although we detected age-related differences in the four decades represented in our sample, future researchers should examine a broader age range to determine at what point these effects begin to emerge. Finally, because our sample size was small, we were unable to conduct cross-wave analyses that would have enabled us to examine mediators of the link between combat status and cumulative biological risk. Future research, with larger samples, should investigate potential mediators in the association between combat status and AL. Similarly, although it was beyond the scope of the current study, future research should also examine whether AL mediates the link between combat exposure and physical health outcomes, such as morbidity and mortality.

These limitations notwithstanding, our findings reveal the importance of taking combat exposure into account when comparing health outcomes between military veterans and nonveterans. They also highlight one mechanism through which the documented age-related shift in the health status of combat veterans may occur. Future work should assess both age and combat exposure when comparing the physical health of military veterans and nonveterans.

OPEN PRACTICES STATEMENT

The study was not formally preregistered. We analyzed data that are not under our direct control; requests to access the data should be directed to https://www.icpsr.umich.edu/web/ICPSR/series/203.

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REFERENCES

Adler, A. B., Castro, C. A., & Britt, T. W. (2006). *Military Life: Operational Stress* (Vol., 2). Greenwood Publishing Group.

Aldwin, C. M., Levenson, M. R., & Spiro, A. (1994). Vulnerability and resilience to combat exposure: Can stress have lifelong effects? *Psychology and Aging*, *9*(1), 34–44. https://doi.org/10.1037/0882-7974.9.1.34

Aldwin, C. M., Park, C. L., Choun, S., & Lee, H. (2018). The impact of military service on stress, health, and well-being in later life. In (A. Spiro III, R. A. Settersten Jr., & C. M. Aldwin Eds.), *Long-Term Outcomes of Military Service: The Health and Well-Being of Aging Veterans* (p. 167–186). American Psychological Association. https://doi.org/10.1037/0000061-010

Ardelt, M., Landes, S. D., & Vaillant, G. E. (2010). The long-term effects of world war ii combat exposure on later-life well-being moderated by generativity. *Research in Human Development*, *7*(3), 202–220. https://doi.org/10.1080/15427609.2010.504505

Barzilay, J. I., Blaum, C., Moore, T., Xue, Q. L., Hirsch, C. H., Walston, J. D., & Fried, L. P. (2007). Insulin resistance and inflammation as precursors of frailty. *Archives of Internal Medicine*, *167*(7), 635–641. https://doi.org/10.1001/archinte.167.7.635

Benyamini, Y., & Solomon, Z. (2005). Combat stress reactions, post-traumatic stress disorder, cumulative life stress, and physical health among Israeli veterans twenty years after exposure to combat. *Social Science & Medicine*, *61*(6), 1267–1277. https://doi.org/10.1016/j.socscimed.2005.01.023

Bogers, R., Bemelmans, W., Hoogenveen, R., Boshuizen, H., Woodward, M., Knekt, P., Dam, R. V., Hu, F., Visscher, T., Menotti, A., Thorpe, R., Jamrozik, K., Calling, S., Strand, B. H., & Shipley, M. (2007). Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: A meta-analysis of 21 cohort studies including more than 300 000 persons. *Archives of Internal Medicine*, *167*(16), 1720–1728. https://doi.org/10.1001/ARCHINTE.167.16.1720

Brooks, K. P., Gruenewald, T., Karlamangla, A., Hu, P., Koretz, B., & Seeman, T. E. (2014). Social relationships and allostatic load in the MIDUS study. *Health Psychology*, *33*(11), 1373–1381. https://doi.org/10.1037/a0034528

Bullman, T. A., & Kang, H. K. (2000). A fifty-year mortality follow-up study of veterans exposed to low-level chemical warfare agent, mustard gas. *Annals Of Epidemiology*, *10*(5), 333–338. https://doi.org/10.1016/S1047-2797(00)00060-0

Butterick, T. A., Trembley, J. H., Hocum Stone, L. L., Muller, C. J., Rudquist, R. R., & Bach, R. R. (2019). Gulf war illness-associated increases in blood levels of interleukin 6 and c-reactive protein: Biomarker evidence of inflammation. *BMC Research Notes*, *12*(1), 816. https://doi.org/10.1186/s13104-019-4855-2

Campbell, D. J., & Nobel, O. B.-Y. (2009). Occupational stressors in military service: A review and framework. *Military Psychology*, *21*(sup2), S47–S67. https://doi.org/10.1080/08995600903249149

Crimmins, E. M., Johnston, M., Hayward, M., & Seeman, T. (2003). Age differences in allostatic load: An index of physiological dysregulation. *Experimental Gerontology*, *38*(7), 731–734. https://doi.org/10.1016/s0531-5565(03)00099-8

Elder, G. H. Jr., Shanahan, M. J., & Clipp, E. C. (1997). Linking combat and physical health: The legacy of world war ii in men's lives. *The American Journal of Psychiatry*, *154*(3), 330–336. https://doi.org/10.1176/ajp.154.3.330

Friedman, E. M., Karlamangla, A. S., Gruenewald, T. L., Koretz, B., & Seeman, T. E. (2015). Early life adversity and adult biological risk profiles. *Psychosomatic Medicine*, *77*(2), 176–185. https://doi.org/10.1097/psy.0000000000000147

Goldberg, K. C. (2008). The epidemiology of health problems in returning operation Iraqi Freedom and Operation Enduring Freedom Veterans: A national and North Carolina-based summary. *North Carolina Medical Journal*, *69*(1), 31–34. https://doi.org/10.18043/ncm.69.1.31

Gruenewald, T. L., Karlamangla, A. S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., & Seeman, T. E. (2013). History of socioeconomic disadvantage and allostatic load in later life. *Social Science and Medicine*, *74*(1), 75–83. https://doi.org/10.1016/j.socscimed.2011.09.037

Gyorgy, A., Ling, G., Wingo, D., Walker, J., Tong, L., Parks, S., Januszkiewicz, A., Baumann, R., & Agoston, D. V. (2011). Time-dependent changes in serum biomarker levels after blast traumatic brain injury. *Journal of Neurotrauma*, *28*(6), 1121–1126. https://doi.org/10.1089/neu.2010.1561

Hamdi, N. R., South, S. C., & Krueger, R. F. (2016). Does education lower allostatic load? A co-twin control study. *Brain, Behavior, and Immunity*, *56*, 221–229. https://doi.org/10.1016/j.bbi.2016.01.014

Highland, K. B., Costanzo, M., Jovanovic, T., Norrholm, S. D., Ndiongue, R., Reinhardt, B., Rothbaum, B., & Roy, M. J. (2015). Biomarkers of post-deployment resilience among military service members. *Neurobiology of Stress*, *2*, 62–66. https://doi.org/10.1016/j.ynstr.2015.07.001

Institute of Medicine. (2008). *Physiologic, Psychologic, and Psychosocial Effects of Deployment-Related Stress*. The National Academies Press.

Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews*, *35*(1), 2–16. https://10.1016/j.neubiorev.2009.10.002

Kleykamp, M. (2007). A great place to start? The effect of prior military service on hiring. *Armed Forces & Society*, *35*(2), 266–285. https://doi.org/10.1177/0095327X07308631

Kulka, R. A., Schlenger, W. E., Fairbank, J. A., Hough, R. L., Jordan, B. K., Marmar, C. R., & Weiss, S. D. (1990). *Trauma and the Vietnam War Generation: Report of Findings from the National Vietnam Veterans Readjustment Study*. Brunner/Mazel.

Landes, S. D., Ardelt, M., & Landes, A. T. (2018). Exploring the veteran mortality differential: The influence of war era and smoking behavior. In (A. Spiro, R. A. Settersten, & C. M. Aldwin Eds.), *Long-Term Outcomes of Military Service: The Health and Well-Being of Aging Veterans* (pp. 187–206). American Psychological Association.

Landes, S. D., London, A. S., & Wilmoth, J. M. (2019). Service-connected disability and the veteran mortality disadvantage.

Armed Forces & Society, *47*(3), 457–479. https://doi.org/10.1177/0095327X19884721

Landes, S. D., Wilder, J., & Williams, D. (2017). The effect of race and birth cohort on the veteran mortality differential. *Social Science & Medicine*, *179*, 36–44. https://doi.org/10.1016/j.socscimed.2017.02.030

Levy, B. S., & Sidel, V. W. (2009). Health effects of combat: A life-course perspective. *Annual Review of Public Health*, *30*(1), 123–136. https://doi.org/10.1146/annurev.publhealth.031308.100147

Liang, K., & Zeger, S. L. (1986). Longitudinal data analysis using generalized linear models. *Biometrica*, *73*(1), 13–22, https://doi.org/10.1093/biomet/73.1.13

Liu, X., Engel, C. Jr., Kang, H., & Cowan, D. (2005). The effect of veteran status on mortality among older Americans and its pathways. *Population Research and Policy Review*, *24*(6), 573–592. https://doi.org/10.1007/s11113-005-5056-3

London, A. S., Herd, P., Miech, R. A., & Wilmoth, J. M. (2017). The influence of men's military service on smoking across the life course. *Journal of Drug Issues*, *47*(4), 562–586. https://doi.org/10.1177/0022042616678617

London, A. S., & Wilmoth, J. M. (2006). Military service and (dis)continuity in the life course: Evidence on disadvantage and mortality from the health and retirement study and the study of assets and health dynamics among the oldest-old. *Research on Aging*, *28*(1), 135–159. https://doi.org/10.1177/0164027505281572

Love, G. D., Seeman, T. E., Weinstein, M., & Ryff, C. D. (2010). Bioindicators in the MIDUS National Study: Protocol, measures, sample, and comparative context. *Journal of Aging and Health*, *22*(8), 1059–1080. https://doi.org/10.1177/0898264310374355

MacLean, A. (2013). A matter of life and death: Military service and health. In (J. M. Wilmoth & A. S. London Eds.), *Life-Course Perspectives on Military Service* (pp. 200–220). Routledge.

MacLean, A., & Edwards, R. D. (2017). Military service, combat exposure, and health in the later lives of us men. *Longitudinal and Life Course Studies*, *8*(2), 122–137. https://doi.org/10.14301/llcs.v8i2.427

Mair, C. A., Cutchin, M., & Peek, M. K. (2011). Allostatic load in an environmental riskscape: The role of stressors and gender. *Health & Place*, *17*(4), 978–989. https://doi.org/10.1016/j.healthplace.2011.03.009

Mattei, J., Demissie, S., Falcon, L. M., Ordovas, J. M., & Tucker, K. (2010). Allostatic load is associated with chronic conditions in the Boston Puerto Rican Health Study. *Social Science and Medicine*, *70*(12), 1988–1996. https://doi.org/10.1016/j.socscimed.2010.02.024

McEwen, B. S. (1998). Stress, adaptation, and disease. Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *1*(840), 33–44. https://doi.org/10.1111/j.1749-6632.1998.tb09546.x

McEwen, B. S. (2001). Plasticity of the hippocampus: Adaptation to chronic stress and allostatic load. *Annals of the New York Academy of Sciences*, *933*(1), 265–277. https://doi.org/10.1111/j.1749-6632.2001.tb05830.x

McEwen, B. S. (2006). Protective and damaging effects of stress mediators: Central role of the brain. *Dialogues in Clinical Neuroscience*, *8*(4), 367–381. https://doi.org/10.31887/DCNS.2006.8.4/bmcewen

McEwen, B. S., Nasveld, P., Palmer, M., & Anderson, R. (2012). *Allostatic Load: A Review of the Literature*. Australian Government, Department of Veterans Affairs. https://www.dva.gov.au/sites/default/files/allostatic.pdf

O'Brien, B. S., & Sher, L. (2013). Military sexual trauma as a determinant in the development of mental and physical illness in male and female veterans. *International Journal of Adolescent Medicine and Health*, *25*(3), 269–274. https://doi.org/10.1515/ijamh-2013-0061

Pflanz, S., & Sonnek, S. (2002). Work stress in the military: Prevalence, causes, and relationship to emotional health. *Military Medicine*, *167*(11), 877–882. https://doi.org/10.1093/milmed/167.11.877

Piazza, J. R., Almeida, D. M., Dmitrieva, N. O., & Klein, L. C. (2010). Frontiers in the use of biomarkers of health in research on stress and aging. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *65B*(5), 513–525. https://doi.org/10.1093/geronb/gbq049

Pizarro, J., Silver, R. C., & Prause, J. (2006). Physical and mental health costs of traumatic war experiences among civil war veterans. *Archives of General Psychiatry*, *63*(2), 193–200. https://doi.org/10.1001/archpsyc.63.2.193

Porter, B., Bonanno, G. A., Bliese, P. D., Phillips, C. J., & Proctor, S. P. (2019). Combat and trajectories of physical health functioning in U.S. service members. *American Journal of Preventive Medicine*, *57*(5), 637–644. https://doi.org/10.1016/j.amepre.2019.06.015

Radler, B. T. (2014). The Midlife in the United States (MIDUS) Series: A national longitudinal study of health and well-being. *Open Health Data*, *2*(1), e3. https://doi.org/10.5334/ohd.ai

Radler, B. T., & Ryff, C. D. (2010). Who participates? Accounting for longitudinal retention in the MIDUS national study of health and well-being. *Journal of Aging and Health*, *22*(3), 307–331. https://doi.org/10.1177/0898264309358617

Schnurr, P. P., & Green, B. L. (2004). Understanding relationships among trauma, posttraumatic stress disorder, and health outcomes. In (P. P. Schnurr & B. L. Green Eds.), *Trauma and Health: Physical Health Consequences of Exposure to Extreme Stress* (pp. 247–275). Australian Government, Department of Veterans Affairs, https://doi.org/10.1037/10723-010

Schnurr, P. P., & Spiro, A. (1999). Combat exposure, posttraumatic stress disorder symptoms, and health behaviors as predictors of self-reported physical health in older veterans. *Journal of Nervous and Mental Disease*, *187*(6), 353–359. https://doi.org/10.1097/00005053-199906000-00004

Seeman, T. E., Crimmins, E., Huang, M. H., Singer, B., Bucur, A., Gruenewald, T., Berkman, L. F., & Reuben, D. B. (2004). Cumulative biological risk and socio-economic differences in mortality: MacArthur studies of successful aging. *Social Science & Medicine*, *58*(10), 1985–1997. https://doi.org/10.1016/S0277-9536(03)00402-7

Seeman, T. E., Singer, B. H., Ryff, C. D., Love, G. D., & Levy-Storms, L. (2002). Social relationships, gender, and allostatic load across two age cohorts. *Psychosomatic Medicine*, *64*(3), 395–406. https://doi.org/10.1097/00006842-200205000-00004

Sheehan, C. M., & Hayward, M. D. (2019). Black/white differences in mortality among veteran and non-veteran males. *Social Science Research*, *79*, 101–114. https://doi.org/10.1016/j.ssresearch.2019.02.006

Sheehan, C. M., Hummer, R. A., Moore, B. L., Huyser, K. R., & Butler, J. S. (2015). Duty, honor, country, disparity: Race/ethnic differences in health and disability among male veterans. *Population Research and Policy Review*, *34*(6), 785–804. https://doi.org/10.1007/s11113-015-9358-9

Sheffler, J. L., Rushing, N. C., Stanley, I. H., & Sachs-Ericsson, N. J. (2016). The long-term impact of combat exposure on health, interpersonal, and economic domains of functioning. *Aging & Mental*

Health, *20*(11), 1202–1212. https://doi.org/10.1080/13607863.2015.1072797

Suris, A., & Lind, L. (2008). Military sexual trauma: A review of prevalence and associated health consequences in veterans. *Trauma, Violence, & Abuse*, *9*(4), 250–269. https://10.1177/1524838008324419

Taylor, M. G., Ureña, S., & Kail, B. L. (2016). Service-related exposures and physical health trajectories among aging veteran men. *The Gerontologist*, *56*(1), 92–103. https://doi.org/10.1093/geront/gnv662

Teachman, J., & Tedrow, L. (2013). Veteran status and body weight: A longitudinal fixed-effects approach. *Population Research and Policy Review*, *32*(2), 199–220. https://doi.org/10.1007/s11113-012-9262-5

U.S. Department of Veteran's Affairs. (2016a). *Respiratory cancers and Agent Orange*. https://www.publichealth.va.gov/exposures/agentorange/conditions/respiratory_cancers.asp

U.S. Department of Veteran's Affairs. (2016b). *VA Research on Parkinson's Disease*. https://www.research.va.gov/topics/parkinsons.cfm

Wang, Y., Wei, Y., Oguntayo, S., Wilder, D., Tong, L., Su, Y., Gist, I., Arun, P., & Long, J. B. (2017). Cerebrospinal fluid chemokine (cc motif) ligand 2 is an early-response biomarker for blast-overpressure-wave–induced neurotrauma in rats. *Journal of Neurotrauma*, *34*(4), 952–962. https://doi.org/10.1089/neu.2016.4465

Wilmoth, J. M., Landes, S. D., & London, A. S. (2019). The health of male veterans in later life. *Annual Review of Gerontology and Geriatrics*, *39*(1), 23–48. https://doi.org/10.1891/0198-8794.39.23

Wilmoth, J. M., London, A. S., & Oliver, W. J. (2018). Military service experiences and older men's trajectories of self-rated health. In (A. Spiro III, R. A., Settersten Jr., & C. M. Aldwin Eds.), *Long-Term Outcomes of Military Service: The Health and Well-Being of Aging Veterans* (pp. 125–145). American Psychological Association. https://doi.org/10.1037/0000061-008

Wilmoth, J. M., London, A. S., & Parker, W. M. (2010). Military service and men's health trajectories in later life. *The Journal of Gerontology: Social Sciences*, *65B*(6), 744–755. https://doi.org/10.1093/geronb/gbq072

Wolf, D. A., Wing, C., & Lopoo, L. M. (2013). Methodological problems in determining the consequences of military service. In (J. M. Wilmoth & A. S. London Eds.), *Life-Course Perspectives on Military Service* (pp. 254–274). Routledge.

Zhang, L., Hu, X. -Z., Li, X., Chen, Z., Benedek, D. M., Fullerton, C. S., Wynn, G., Team B., & Ursano, R. J. (2020). Potential chemokine biomarkers associated with PTSD onset, risk and resilience as well as stress responses in us military service members. *Translational Psychiatry*, *10*(1), 31. https://doi.org/10.1038/s41398-020-0693-1

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