Original Article

CHRONIC STRESS

Chronic Stress
Volume 3: 1–9

DOI: 10.1177/2470547019871901
journals.sagepub.com/home/css
SAGE

The Predictive Value of Early-Life Trauma, Psychopathy, and the Testosterone–Cortisol Ratio for Impulsive Aggression Problems in Veterans

Pauline O. J. Korpel[1,2], Tim Varkevisser[1,2], Sylco S. Hoppenbrouwers[3], Jack Van Honk[4,5], and Elbert Geuze[1,2]

Abstract

Background: In this study, we examined whether early-life trauma, psychopathy, and the testosterone/cortisol ratio predicted impulsive aggression problems in veterans.

Method: A sample of 49 male veterans with impulsive aggression problems and 51 nonaggressive veterans were included in the study. Logistic regression analysis was performed with early-life trauma, primary and secondary psychopathy, and testosterone/cortisol ratio as continuous predictor variables; impulsive aggression status was entered as a binary outcome measure. Correlation analyses were conducted to examine pairwise relations among the predictors.

Results: Results indicated that early-life trauma and secondary psychopathy, but not the testosterone/cortisol ratio or primary psychopathy, were significant predictors of impulsive aggression status.

Conclusions: The current results indicate that early-life trauma and secondary psychopathy are risk factors for impulsive aggression problems among veterans. Future studies are needed to determine the exact causal relations among the variables examined here.

Keywords

impulsive aggression, early-life trauma, psychopathy, testosterone, cortisol, veterans

Received 13 May 2019; Accepted 1 August 2019

Introduction

Problems with anger and aggression frequently occur among veterans. In one study, 11% of U.S. Iraq and Afghanistan veterans were found to have engaged in at least one act of severe violence in the first year after homecoming.[1] In another study, 6.5% of Dutch Afghanistan veterans reported symptoms of hostility one-year post deployment.[2] Although aggression can be instrumental (premeditated), most veterans display the impulsive (reactive) form of aggression.[3] Problems with impulsive aggression have been linked to depression,[4] posttraumatic stress disorder (PTSD),[3] suicidal behavior,[5] and intimate partner violence.[6] Given these serious consequences, it is important to gain a better understanding of the factors that govern impulsive aggression in veterans.

Research has shown that aggression and violence are particularly prevalent in individuals with a history of early-life trauma.[7–10] For instance, Sarchiapone et al.[8] found a significant positive association between measures

[1]Department of Psychiatry, University Medical Center Utrecht, Utrecht, the Netherlands
[2]Brain Research and Innovation Center, Dutch Ministry of Defence, Utrecht, the Netherlands
[3]Thalamus, Centre for Neuropsychiatry and Behavioural Neurology, Wolfheze, the Netherlands
[4]Utrecht University, Utrecht, the Netherlands
[5]University of Cape Town, Cape Town, South Africa

Corresponding Author:
Tim Varkevisser, Brain Research and Innovation Center, Dutch Ministry of Defence, Lundlaan 1, 3584 EZ Utrecht, the Netherlands.
Email: t.varkevisser@umcutrecht.nl

of early-life trauma and lifetime history of aggression in male offenders. In addition, Whitfield et al.[11] investigated data from the Adverse Childhood Experiences Study (consisting of 8629 participants) and discovered that the odds of being a perpetrator of intimate partner violence nearly doubled when individuals had experienced traumatic stress as a child. Traumatic stressors can be defined as life-threatening experiences or violations of bodily or emotional integrity (sexual abuse, emotional/physical punishment, or neglect).[12] Traumatic events in early life can disrupt important developmental processes, such as attachment to primary caregivers and self-regulatory skills.[13,14] Such disruptions may well lead to disturbances in emotion regulation and behavioral inhibition in adulthood. Early-life trauma seems to be quite prevalent among veterans, with 44% of Dutch Afghanistan veterans experiencing one or more forms of physical abuse before the age of 18 years, 6% reporting childhood sexual abuse, and 22% reporting emotional abuse as a child.[15] To our knowledge, however, no study has ever examined the role of early-life trauma in impulsive aggression problems among veterans.

Psychopathy is characterized by a lack of empathy, insensitivity to punishment, and manipulative behavior.[16,17] Psychopathic individuals are more prone to conduct crimes that are violent than nonpsychopathic individuals and the crimes they commit tend to be more sadistic.[18] Woodworth and Porter[19] found that the homicides committed by psychopathic criminals were more often (93.3%) instrumental in nature than the homicides committed by nonpsychopathic criminals, which were more frequently (51.6%) caused by reactive (rather than premeditated) violence. This finding resonates with the idea that psychopathic individuals typically display instrumental (goal-oriented) violent behavior; nevertheless, problems with impulsive (affect-driven) aggression are also a common occurrence among these individuals.[18] In fact, psychopathy is often subdivided into a callous interpersonal component (primary psychopathy, or factor 1) and a factor marked by negative emotionality, frustration intolerance, impulsivity, and antisocial behavior (secondary psychopathy, or factor 2). Research has shown that primary psychopathy may be closely related to instrumental aggression; in contrast, secondary psychopathy is marked by impulsive aggression and antisocial behavior.[20] Psychopathy (like impulsive aggression) has previously been associated with experiencing traumatic events early in life,[21] and a recent meta-analysis recorded significant impairments in threat processing among psychopathic individuals.[22] Although the prevalence of (factor 1 or 2) psychopathy in veterans is currently not known, one small study ($N = 144$) recorded a prevalence of 8.1% for sadistic personality disorder in veterans.[23] No study has ever examined the potential roles of factor 1 and/or 2 psychopathy in impulsive aggression problems among veterans.

Studies into the neurobiological basis of aggression have frequently recorded elevated levels of the steroid hormone testosterone, along with reduced levels of cortisol.[24,25] In the seminal work of Dabbs et al.,[26] offenders high in (salivary) testosterone were found to commit crimes that were more violent than offenders low in testosterone, an effect that was significantly stronger when—in addition—cortisol levels were low. This positive relationship between the testosterone/cortisol (T/C) ratio and aggression has since been replicated (albeit in adolescents).[27,28] Furthermore, elevated T/C ratios have been recorded in relation to socially dominant behavior,[29] externalizing personality problems,[30] and—most notably—early-life stressors;[31] the few studies that examined the relationship between T/C ratio and psychopathy have shown mixed results.[32,33] It should be noted, however, that the Vietnam Experience Study,[34] consisting of 4462 army veterans, was not able to replicate the relationship between T/C ratio and aggressive behavior, although the authors did record a positive correlation between testosterone (by itself) and aggression. Hence, additional research on the potential link between T/C ratio and impulsive aggression in veterans is required.

In this study, we further examined the interplay between impulsive aggression, early-life trauma, psychopathy, and plasma T/C ratio in veterans. Logistic regression analysis was used to test the hypotheses that a history of early psychological trauma, secondary but not primary psychopathy, and/or an elevated T/C ratio can significantly predict the presence or absence of impulsive problems in veterans. Pairwise correlations between the predictors of interest were also computed for exploratory purposes.

Method

Participants

The current study was conducted as part of a larger initiative to disentangle the neurobiological and psychological factors underlying impulsive aggression problems in Dutch veterans.[35–38] The sample consisted of 100 Dutch male veterans divided into two groups: impulsive aggression ($N = 49$) and control ($N = 51$). Demographic information of the participants is presented in Table 1. Criteria for inclusion in both groups were as follows: military men in the age range of 18 to 50 years who had been deployed for at least four months. The main criterion for inclusion in the experimental group was the presence of impulsive aggression problems as defined by the research diagnostic criteria for intermittent explosive disorder by Coccaro.[39] The main criterion for inclusion in the control group was the absence of such aggression

Table 1. Demographic information for the impulsive aggression and control groups.

Measure	Impulsive aggression group	Control group	*p*
Age	35.47 ± 6.85	34.57 ± 7.33	.528
Number of deployments	2.29 ± 1.40	2.55 ± 1.60	.385
Duration of deployment (months)	9.68 ± 5.78	12.19 ± 7.20	.091
Number of years since last deployment	7.67 ± 5.47	6.27 ± 2.95	.115
Rank			.091[a]
Enlisted	4 (8.2%)	3 (6%)	
Corporal	8 (16.3%)	12 (24%)	
NCO	16 (32.7%)	14 (28%)	
Officer	1 (2%)	8 (16%)	
Not currently enlisted	20 (40.8%)	13 (26%)	
Education			.434[a]
Higher	9 (18.3%)	16 (31.4%)	
Middle	35 (71.4%)	29 (56.9%)	
Lower	5 (10.2%)	6 (11.8%)	

Note: NCO: Non-commissioned officer.
[a]Determined with a Fisher–Freeman–Halton Test.

regulation problems. Exclusion criteria for both groups were substance abuse and severe neurological disorders. All participants in the impulsive aggression group were following treatment for anger and aggression-related complaints at the Military Mental Healthcare Organization in the Netherlands. The veterans with impulsive aggression problems were recruited via referral by their therapist or from a database of individuals who previously participated in studies at our department (and gave permission to be approached in the future). The nonaggressive control veterans were recruited through advertisement in newspapers and magazines or via the abovementioned database of participants.

Procedures

This study was approved by the Medical-Ethical Committee of the University Medical Centre Utrecht. All participants signed informed consent after receiving a written and verbal description of the study. To determine testosterone, cortisol, and Sex Hormone-Binding Globulin (SHBG) levels, 36 ml blood was collected (venous) between 08:30 and 10:30 A.M. in EDTA vacuum containers that were immediately stored on ice. The plasma samples were centrifuged at 3500 r/min for 12 minutes at 4°C. Afterward, the samples were transferred and stored at a temperature of −80°C. Finally, the questionnaires were administered to the participants.

Apparatus and Materials

Questionnaires. Early-life traumatic events were measured with the Dutch Early Trauma Inventory-Self Report (ETI-SR).[40,41] This self-report measure consists of 27 dichotomous items on the severity and frequency of traumatic events before the age of 18 years. The ETI-SR consists of four subscales: General Trauma (11 items), Physical Punishment (5 items), Emotional Abuse (5 items) and Sexual Abuse (6 items). The ETI-SR has good construct validity ($r = .37$–$.47$ correlation with Clinician Administered PTSD Scale; Blake et al.[42]) and internal consistency ($\alpha = .70$–$.87$).[43] The internal consistency of the Dutch version of the ETI-SR in the sample of Rademaker et al.[41] was comparable to the correlations reported by Bremner et al.[40] for the Physical Abuse ($\alpha = .76$) and Emotional Abuse ($\alpha = .83$) subscales; the Cronbach's alpha for the General Trauma ($\alpha = .48$) and Sexual Abuse ($\alpha = .53$) scales was somewhat lower.

Psychopathy was assessed via the Levenson Self-Report Psychopathy Scale (LSRP).[44] The LSRP consists of 26 items with statements on personality and behavioral traits associated with psychopathy. The statements are rated on a four-point Likert-type scale (*disagree strongly, disagree somewhat, agree somewhat,* and *agree strongly*). The scale is divided into a primary psychopathy (factor 1) and secondary psychopathy (factor 2) subscale. Primary psychopathy is characterized by callous, manipulative, and selfish tendencies with a lack of guilt or empathic response. Secondary psychopathy is marked by impulsivity, frustration intolerance, and a lack of long-term goals. Miller et al.[45] found LSRP-1 to be related to an antisocial interpersonal style (low agreeableness and conscientiousness) and LSRP-2 to negative emotionality and disinhibition (high neuroticism and low conscientiousness). Brinkley et al.[46] reported the LSRP to have sufficient construct validity ($r = .35$ correlation with Hare's Psychopathy-Checklist)[47] and good internal consistency ($\alpha = .85$ for total LSRP).

Plasma. Testosterone, SHBG, and cortisol levels were determined using electrochemiluminescence immunoassay on a Modular-E170 (Roche Diagnostics GmbH, Mannheim, Germany). Bioactive testosterone (BAT) was calculated using total testosterone and SHBG levels via the Vermeulen method.[48]

Research Design and Statistical Analyses. Group-level statistical analyses for the demographic variables were conducted via independent-samples *t* tests and Fisher–Freeman–Halton tests (for nonparametric data). The significance of ETI, LSRP-1, LSRP-2, and T/C ratio as predictors of impulsive aggression status was assessed via multiple logistic regression, with the control group as reference category. Pearson correlations coefficients were

computed to examine pairwise relations among the predictors of interest. An alpha level of .05 (two-sided) was employed for all statistical tests reported here. All analyses were conducted using IBM-SPSS Statistics 22 and MatLab 2017b.

Data and Code Availability Statement

The data that support the findings of this study are available upon reasonable request from the corresponding author (T. V.).

Results

Group Characteristics

The independent-samples t tests revealed significant group differences in ETI ($t(98) = 4.24$, $p < .001$), LSRP-1 ($t(98)$, 2.54, $p = .013$), and LSRP-2 ($t(98) = 10.01$, $p < .001$) scores in the impulsive aggression, relative to the control group. ETI scores were higher in the aggression group ($M = 6.33$) than in the control group ($M = 2.90$). In addition, both LSRP factor 1 and 2 scores were higher in the impulsive aggression group ($M = 30.20$, $M = 22.65$, respectively) than in the control group ($M = 26.53$, $M = 15.02$, respectively). No significant difference in T/C ratio was observed between the impulsive aggression ($M = 2.45$) and control group ($M = 2.24$) ($t(98) = 1.26$, $p = .211$). Six participants received antidepressant treatment (escitalopram ($n = 2$), citalopram ($n = 1$), venlafaxine ($n = 1$), and sertraline ($n = 2$)), which is known to influence cortisol levels[49]; however, there seemed to be no difference in the T/C ratio of participants who received antidepressant treatment versus those who did not ($p = 121$). Hence, these participants were not omitted from any subsequent analyses.

Twenty-nine of the 49 veterans in the impulsive aggression group had a diagnosis for one or more comorbid psychiatric disorders at the time of assessment, as determined with the Mini-International Neuropsychiatric Interview.[50] The diagnoses included major depression ($n = 17$), bipolar disorder ($n = 2$), panic disorder ($n = 3$), social anxiety disorder ($n = 3$), specific phobia ($n = 1$), generalized anxiety disorder (GAD) ($n = 3$), obsessive–compulsive disorder ($n = 1$), PTSD ($n = 16$), alcohol abuse or dependence ($n = 4$), drug abuse or dependence ($n = 1$), antisocial personality disorder ($n = 1$), and attention-deficit hyperactivity disorder ($n = 3$). Only one of the 51 veterans in the control group had a comorbid psychiatric diagnosis (GAD).

Correlation Analyses

Exploratory correlation analyses were conducted to assess pairwise relations between early-life trauma (ETI), psychopathic (LSRP factors 1 and 2), cortisol, BAT, and T/C ratio. The results of these analyses are presented in Table 2. A significant medium and positive correlation was observed between early-life trauma and LSRP-2 ($r = .302$, $p < .01$). As expected, a significant positive large-to-medium correlation was recorded between LSRP-1 and LSRP-2 ($r = .492$, $p < .01$).

Logistic Regression Analysis

Logistic regression analysis was performed to assess the significance of ETI, LSRP factors 1 and 2, and the T/C ratio as predictors of impulsive aggression status; the control group was used as reference category for this analysis, and all predictors were entered into the model simultaneously (entry method). A test of the model against a constant-only model proved statistically significant ($\chi^2(4, N = 100) = 74.44$, $p < .001$). A Nagelkerke's R^2 of .700 indicated a moderate relationship between predicted and observed group membership. Prediction success with ETI, both LSRP factors, and T/C ratio as predictor variables was 83% (79.6% aggression, 86.3% control). The Wald criterion indicated that ETI ($p = .036$) and LSRP-2 ($p < .001$) were significant (positive) predictors of impulsive aggression problems. LSRP-1 and

Table 2. Correlations among T/C ratio, Cortisol, Testosterone, ETI, LSRP-1, and LSRP-2.

	1. T/C ratio	2. Cortisol	3. BAT	4. ETI	5. LSRP-1	6. LSRP-2
1. T/C ratio						
2. Cortisol	−.662[a]					
3. BAT	.593[a]	.132				
4. ETI	.158	−.067	.167			
5. LSRP-1	−.073	−.063	−.117	.146		
6. LSRP-2	.098	−.048	.081	.302[a]	.492[a]	

Note: BAT = bioactive testosterone; ETI = Early Trauma Inventory; LSRP = Levenson Self-Report Psychopathy; T/C: testosterone/cortisol.

[a]Correlation is significant at the.01 level (two-tailed).

the T/C ratio were not significant predictors of impulsive aggression problems ($p = .179$ and $p = .537$, respectively). The Exp(B) value indicated that when ETI was raised by one unit (corresponding to one traumatic experience), the odds of being in the impulsive aggression group increased by a factor of 1.21. This indicates that participants were 21% more likely to be classified in the impulsive aggression group, with each trauma they experienced. The odds ratio of LSRP-2 was 1.73, indicating that for each unit increase in LSRP-2 score, participants were 73% more likely to be classified in the impulsive aggression group. For an overview of the binary multiple logistic regression results, see Table 3. The addition of psychiatric comorbidity as a nuisance variable influenced the above results insofar as that it rendered the predictive value of ETI nonsignificant ($p = .10$), while comorbidity itself became a significant predictor of impulsive aggression status ($p = .04$).

A second logistic regression model was fit in order to determine whether the interactions between T/C ratio and ETI, T/C ratio and LSRP-1, and ETI and LSRP-1 would significantly enhance the model fit compared to the (above) model that assessed only the main effects of the predictors of interest. T/C ratio, ETI, LSRP-1, and LSRP-2 were entered simultaneously in the first block, while the interactions terms T/C ratio × ETI, T/C ratio × LSRP-1, and ETI × LSRP-1 were entered simultaneously in the second block. No significant improvement in model fit was observed when adding these interaction terms to the model ($\chi^2(3,\ N = 100) = 1.648$, $p = .649$).

Discussion

The aim of the current study was to examine the interplay between impulsive aggression, early-life trauma, psychopathy, and plasma T/C ratio in veterans. Logistic regression analysis indicated that early-life trauma and secondary psychopathy, but not primary psychopathy or the T/C ratio, were significant predictors of impulsive aggression status (see Table 2). Moreover, a significant positive correlation was observed between early-life trauma and secondary psychopathy (see Table 3).

The results obtained here build on a growing literature that appreciates the negative influence of early-life trauma on psychological functioning in later life. In the current sample, the number of potentially traumatic events during childhood significantly predicted impulsive aggression problems in veterans. This replicates findings from previous studies that demonstrated a significant positive link between measures of early-life trauma and impulsive aggression in nonveteran populations.[7–10] We also recorded a significant positive correlation between early-life trauma and psychopathy factor 2, a construct marked by problems with impulsive aggression;[20,21] no significant correlation was observed between early-life trauma and factor 1 psychopathy. We hypothesize that childhood traumatic experiences may lead to an insecure attachment style that disrupts the development of normal emotion regulation and coping skills, thus reinforcing maladaptive relational patterns and ultimately contributing to the development of impulsive aggression problems in adulthood.[13,51] Alternatively, the direction of effect may be reversed, such that parents may react negatively to aggressive traits in children leading to rejection and/or child abuse. The small and nonsignificant correlation between early-life trauma and factor 1 psychopathy scores is difficult to explain. Perhaps that an innate callousness and ability to navigate and manipulate social situations, the core features of factor 1 psychopathy might protect someone against the exposure to potential trauma as a child (or make one less susceptible to the influence thereof). Additional research is needed that formally test this hypothesis.

As predicted, psychopathy factor 2 (negative emotional, frustration intolerance, antisocial behaviors) but not factor 1 (callous interpersonal) significantly contributed to the prediction of impulsive aggression problems in veterans. This finding is consistent with a previous report by Coccaro et al.,[52] and complements earlier

Table 3. Binary logistic regression model predicting group membership (aggression vs. control) with ETI, LSRP-1, LSRP-2, and T/C ratio as predictor variables.

Predictor	*B*	Standard error	Wald statistic	*p*	Exp *B* (odds ratio)	95% confidence interval	
						Lower	Upper
T/C ratio	0.25	.40	0.38	.537	1.28	0.59	2.78
ETI	0.19	.09	4.40	.036	1.21	1.01	1.44
LSRP-1	−0.08	.06	1.80	.179	0.92	0.82	1.04
LSRP-2	0.55	.12	22.29	<.001	1.73	1.38	2.18

Note: $R^2 = .71$ (Nagelkerke). Model $\chi^2(1) = 75.67$, $p < .001$. ETI: Early Trauma Inventory; LSRP-1: Levenson Self-Report Psychopathy factor 1; LSRP-2: Levenson Self-Report Psychopathy factor 2; T/C: testosterone/cortisol.

work by Reidy et al.[20] who showed that factor 1 (but not factor 2) psychopathy was significantly related to instrumental aggression. Taken together then, our results suggest that impulsive aggression problems in veterans may not necessarily cooccur with high levels of instrumental (premeditated) aggression, but rather that the two forms of aggression represent distinct constructs, even though they may be difficult to distinguish from one another in clinical practice.

Logistic regression analysis revealed that plasma T/C ratio was not a significant predictor of impulsive aggression problems in the current sample of veterans. Also, no significant correlation was observed between T/C ratio and any of the other predictors of interest (ETI, LSRP-1, and LSRP-2). These results are in conflict with findings from earlier studies that found aggression to be associated with high (salivary) testosterone and low cortisol levels.[24–26,28] In line with the present work, however, Mazur et al.[34] previously recorded no significant relation between plasma T/C ratio and aggression in a sample of 4462 army veterans. One notable difference between the earlier studies that *did* report a significant association between T/C ratio and aggression and the current study is that the former (e.g., the article by Dabbs et al.[26] described in "Introduction" section) extracted hormone levels from saliva, whereas here, and in the study by Mazur and colleagues, plasma levels were used. Although saliva and plasma T/C are generally believed to be comparable,[53,54] it is possible that the use of needles elevated the cortisol levels of the veterans who experience blood sampling as stressful,[55] thereby confounding the T/C ratio. We also note that many of the previous studies examined T/C ratio in forensic groups without formally distinguishing impulsive from instrumental aggression. In these forensic samples, instrumental aggression may be relatively more common, whereas we explicitly included veterans with impulsive aggression here. This difference in operationalization may have affected the T/C ratios measured here versus those of earlier studies. Finally, the T/C ratio is influenced by a myriad of factors that we did not control for (nor measure) here, such as caffeine intake, stress, exercise,[56] body fat,[57] nicotine use,[58] time of day,[59] and amount of sleep.[60]

The current findings should be interpreted in light of the following limitations: First, we determined T/C ratios based on a single plasma sample, which likely limited the reliability of our measurement, and increased the variance due to factors such as those described above (stress, body fat, amount of sleep, etc.). By using T/C ratios rather than hormone levels per se, and by sampling at the same time of day across participants, some of this variance might have been accounted for. Nevertheless, repeated hormone measurements are highly recommended for any future endeavor that seeks to examine T/C levels in the context of (impulsive) aggression. Second, in our study, testosterone and cortisol levels were extracted from plasma (venipuncture) rather than from saliva. As mentioned above, saliva and plasma T/C levels are generally believed to be comparable;[53,54] nevertheless, the anticipation stress associated with the use of needles likely influenced the hormone levels measured here, especially cortisol.[55] Third, our analyses were limited to a male-only sample of veterans; the findings reported here may therefore not be generalizable to a female veteran (and/or nonveteran) population. Fourth, since our study was retrospective by design, we could not make any formal claims as to the causal link between (traumatic) exposure to combat and the development of impulsive aggression problems. Likewise, we were unable to disentangle the effects of early versus combat-related trauma in the development of impulsive aggression. It is known that a disadvantaged socioeconomical background, as well as childhood behavioral problems, including delinquent behavior, may increase the likelihood of enlisting in the military.[61,62] Hence, the problems with impulsive aggression experienced by our participants might have already been present before deployment or even before enlistment. We note, however, that all participants had been deployed for at least four months, as per inclusion criterion, during which time they experienced typical war-zone stressors, such as being exposed to enemy fire, or witnessing a colleague or civilian get seriously injured or killed. Fifth, while none of the participants were themselves physically injured during deployment, we did not formally assess actual blast exposure in the present study. Hence, we cannot fully exclude the possibility that traumatic brain injury, a known precursor for the development of impulsive aggression problems, affected the current results.[63]

Areas for future research include the use of prospective longitudinal data to determine the extent to which impulsive aggression problems are caused by early- versus late-life (e.g., combat-related) trauma. The potentially confounding effects of comorbid psychiatric disorders (e.g., PTSD) and traumatic brain injury may also be taken into account. Furthermore, the existing literature suggests that the timing of early-life events (e.g., early childhood vs. adolescence), as well as the type of trauma (e.g., physical, sexual, or emotional), may differentially impact the development of (impulsive) aggression in adulthood.[64,65] Such factors might be taken into account by future endeavors that seek to study impulsive aggression problems (or psychopathy) in veterans. Exploration of additional potential mediators, such as attachment styles, self-regulatory competencies, and self-esteem, is also warranted. Lastly, we recommend that future research focus on androgens other than testosterone, such as dehydroepiandrosterone (DHEA) and DHEA sulfate, since previous studies have shown these may be strongly related to aggressive conduct.[66]

In conclusion, the current study indicates that early-life trauma and secondary psychopathy, but not T/C ratio or primary psychopathy, may serve as viable predictors of impulsive aggression problems in veterans. We hope that these results will ultimately aid in the development of novel or more effective prevention and/or treatment strategies for impulsive aggression so that we may better help those afflicted with these debilitating issues.

Acknowledgments

The authors would like to thank all veterans who took part in this study.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the Dutch Ministry of Defence.

ORCID iD

Tim Varkevisser https://orcid.org/0000-0003-3441-9486
Elbert Geuze https://orcid.org/0000-0003-3479-2379

References

1. Elbogen EB, Johnson SC, Wagner R, et al. Protective factors and risk modification of violence in Iraq and Afghanistan war veterans. *J Clin Psychiatry*. 2012; 73(6): e767–e773.
2. Reijnen A, Rademaker AR, Vermetten E, Geuze E. Prevalence of mental health symptoms in Dutch military personnel returning from deployment to Afghanistan: a 2-year longitudinal analysis. *Eur Psychiatry*. 2015; 30: 341–346.
3. Teten AL, Miller LA, Stanford MS, et al. Characterizing aggression and its association to anger and hostility among male veterans with post-traumatic stress disorder. *Mil Med*. 2010; 175(6): 405–410.
4. Painuly N, Sharan P, Mattoo SK. Relationship of anger and anger attacks with depression: a brief review. *Eur Arch Psychiatry Clin Neurosci*. 2005; 255: 215–222.
5. Romanov K, Hatakka M, Keskinen E, et al. Self-reported hostility and suicidal acts, accidents, and accidental deaths: a prospective study of 21,443 adults aged 25 to 59. *Psychosom Med*. 1994; 56: 328–336.
6. Romero-Martínez Á, Lila M, Catalá-Miñana A, Williams RK, Moya-Albiol L. The contribution of childhood parental rejection and early androgen exposure to impairments in socio-cognitive skills in intimate partner violence perpetrators with high alcohol consumption. *Int J Environ Res Public Health*. 2013; 10: 3753–3770.
7. Duke NN, Pettingell SL, Mcmorris BJ, Borowsky IW. Adolescent violence perpetration: associations with multiple types of adverse childhood experiences. *Pediatrics*. 2010; 125: e778–e786.
8. Sarchiapone M, Carli V, Cuomo C, Marchetti M, Roy A. Association between childhood trauma and aggression in male prisoners. *Psychiatry Res*. 2009; 165: 187–192.
9. Yexley M, Borowsky I, Ireland M. Correlation between different experiences of intrafamilial physical violence and violent adolescent behavior. *J Interpers Violence*. 2002; 17(7): 707–720.
10. Lipschitz DS, Morgan CA III, Southwick SM. Neurobiological disturbances in youth with childhood trauma and in youth with conduct disorder. *J Aggress Maltreat Trauma*. 2002; 6(1): 149–174.
11. Whitfield CL, Anda RF, Dube SR, Felitti VJ. Violent childhood experiences and the risk of intimate partner violence in adults assessment in a large health maintenance organization. *J Interpers Violence*. 2003; 18(2): 166–185.
12. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*., 5th ed. Washington, DC: American Psychiatric Association, 2013.
13. Cook A, Spinazzola J, Ford J, et al. Complex trauma in children and adolescents. *Psychiatr Ann*. 2005; 35(5): 390–398.
14. Ford JD, Courtois CA, Steele K, Van der Hart O, Nijenhuis ERS. Treatment of complex posttraumatic self-dysregulation. *J Trauma Stress*. 2005; 18(5): 437–447.
15. Dorresteijn S, Gladwin TE, Eekhout I, Vermetten E, Geuze E. Childhood trauma and the role of self-blame on psychological well-being after deployment in male veterans. *Eur J Psychotraumatol*. 2019; 10: 1558705.
16. Blair RJR. Dysfunctions of medial and lateral orbitofrontal cortex in psychopathy. *Ann N Y Acad Sci*. 2007; 1121: 461–479.
17. Blair RJR. Empathic dysfunction in psychopathic individuals. In: Farrow TFD, Woodruff PWR, eds. *Empathy in Mental Illness*. New York, NY: Cambridge University Press; 2007: pp. 3–16.
18. Porter S, Woodworth M. Psychopathy and aggression. In: C. J. Patrick, ed. *Handbook of Psychopathy*. New York, NY: The Guilford Press; 2006: pp. 481–494.
19. Woodworth M, Porter S. In cold blood: characteristics of criminal homicides as a function of psychopathy. *Artic J Abnorm Psychol*. 2002; 111(3): 436–445.
20. Reidy DE, Zeichner A, Miller JD, Martinez MA. Psychopathy and aggression: examining the role of psychopathy factors in predicting laboratory aggression under hostile and instrumental conditions. *J Res Pers*. 2007; 41: 1244–1251.
21. Graham N, Kimonis ER, Wasserman AL, Kline SM. Associations among childhood abuse and psychopathy facets in male sexual offenders. *Personal Disord Theory Res Treat*. 2012; 3(1): 66–75.
22. Hoppenbrouwers SS, Bulten BH, Brazil IA. Parsing fear: a reassessment of the evidence for fear deficits in psychopathy. *Psychol Bull*. 2016; 142: 573–600.
23. Reich J. Prevalence and characteristics of sadistic personality disorder in an outpatient veterans population. *Psychiatry Res*. 1993; 48(3): 267–276.
24. Montoya ER, Terburg D, Bos PA, Van Honk J. Testosterone, cortisol, and serotonin as key regulators of social aggression: a review and theoretical perspective. *Motiv Emot*. 2012; 36: 65–73.

25. Van Honk J, Harmon-Jones E, Morgan BE, Schutter DJLG. Socially explosive minds: the triple imbalance hypothesis of reactive aggression. *J Pers*. 2010; 78(1): 67–94.
26. Dabbs JM, Jurkovic GJ, Frady RL. Salivary testosterone and cortisol among late adolescent male ffenders. *J Abnorm Child Psychol*. 1991; 19(4): 469–478.
27. Platje E, Popma A, Vermeiren RRJM, et al. Testosterone and cortisol in relation to aggression in a non-clinical sample of boys and girls. *Aggress Behav*. 2015; 41: 478–487.
28. Popma A, Vermeiren R, Geluk CAML, et al. Cortisol moderates the relationship between testosterone and aggression in delinquent male adolescents. *Biol Psychiatry*. 2007; 61: 405–411.
29. Mehta PH, Josephs RA. Testosterone and cortisol jointly regulate dominance: evidence for a dual-hormone hypothesis. *Horm Behav*. 2010; 58: 898–906.
30. Tackett JL, Herzhoff K, Harden KP, Page-Gould E, Josephs RA. Personality × hormone interactions in adolescent externalizing psychopathology. *Personal Disord Theory Res Treat*. 2014; 5(3): 235–246.
31. Simmons JG, Byrne ML, Schwartz OS, et al. Dual-axis hormonal covariation in adolescence and the moderating influence of prior trauma and aversive maternal parenting. *Dev Psychobiol*. 2015; 57(6): 670–687.
32. Glenn AL, Raine A, Schug RA, Gao Y, Granger DA. Increased testosterone to cortisol ratio in psychopathy. *J Abnorm Psychol*. 2011; 120(2): 389–399.
33. Welker KM, Lozoya E, Campbell JA, Neumann CS, Carré JM. Testosterone, cortisol, and psychopathic traits in men and women. *Physiol Behav*. 2014; 129: 230–236.
34. Mazur A, Booth A. Testosterone is related to deviance in male army veterans, but relationships are not moderated by cortisol. *Biol Psychol*. 2014; 96: 72–76.
35. Heesink L, Kleber R, Häfner M, Van Bedaf L, Eekhout I, Geuze E. Anger and aggression problems in veterans are associated with an increased acoustic startle reflex. *Biol Psychol*. 2017; 123: 119–125.
36. Heesink L, Gladwin TE, Terburg D, Van Honk J, Kleber R, Geuze E. Proximity alert! Distance related cuneus activation in military veterans with anger and aggression problems. *Psychiatry Res Neuroimaging*. 2017; 266: 114–122.
37. Heesink L, Gladwin TE, Vink M, van Honk J, Kleber R, Geuze E. Neural activity during the viewing of emotional pictures in veterans with pathological anger and aggression. *Eur Psychiatry*. 2018; 47: 1–8.
38. Varkevisser T, Gladwin TE, Heesink L, van Honk J, Geuze E. Resting-state functional connectivity in combat veterans suffering from impulsive aggression. *Soc Cogn Affect Neurosci*. 2017; 12(12): 1881–1889.
39. Coccaro EF. Intermittent explosive disorder: development of integrated research criteria for diagnostic and statistical manual of mental disorders, fifth edition. *Compr Psychiatry*. 2011; 52(2): 119–125.
40. Bremner JD, Vermetten E, Mazure CM. Development and preliminary psychometric properties of an instrument for the measurement of childhood trauma: the early trauma inventory. *Depress Anxiety*. 2000; 12(1): 1–12.
41. Rademaker AR, Vermetten E, Geuze E, Muilwijk A, Kleber RJ. Self-reported early trauma as a predictor of adult personality: a study in a military sample. *J Clin Psychol*. 2008; 64(7): 863–875.
42. Blake DD, Weathers FW, Nagy LM, et al. The development of a clinician-administered PTSD scale. *J Trauma Stress*. 1995; 8(1): 75–90.
43. Bremner JD, Bolus R, Mayer EA. Psychometric properties of the early trauma inventory-self report. *J Nerv Ment Dis*. 2007; 195(3): 211–218.
44. Levenson MR. Assessing psychopathic attributes in a non-institutionalized population. *J Pers Soc Psychol*. 1995; 68(1): 151–158.
45. Miller JD, Gaughan ET, Pryor LR. The Levenson Self-Report Psychopathy Scale an examination of the personality traits and disorders asociated with the LSRP factors. *Assessment*. 2008; 15: 450–463.
46. Brinkley CA, Schmitt WA, Smith SS, Newman JP. Construct validation of a self-report psychopathy scale: does Levenson's self-report psychopathy scale measure the same constructs as Hare's psychopathy checklist-revised? *Pers Individ Dif. 2001*. 31: 1021–1038.
47. Hare RD. *The Hare Psychopathy Checklist-Revised: Manual*.. Toronto: Multi-Health Systems, Incorporated, 1991.
48. Vermeulen A, Verdonck L, Kaufman JM. A critical evaluation of simple methods for the estimation of free testosterone in serum. *J Clin Endocrinol Metab*. 1999; 84(10): 3666–3672.
49. Manthey L, Leeds C, Giltay EJ, et al. Antidepressant use and salivary cortisol in depressive and anxiety disorders. *Eur Neuropsychopharmacol*. 2011; 21: 691–699.
50. Sheehan D, Lecrubier Y, Sheehan K, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *J Clin Psychiatry*. 1998; 59: 22–33.
51. Riggs SA. Childhood emotional abuse and the attachment system across the life cycle: what theory and research tell us. *J Aggress Maltreat Trauma*. 2010; 19(1): 5–51.
52. Coccaro EF, Lee R, Mccloskey MS. Relationship between psychopathy, aggression, anger, impulsivity, and intermittent explosive disorder. *Aggress Behav*. 2014; 40(6): 526–536.
53. Wang C, Plymate S, Nieschlag E, Paulsen CA. Salivary testosterone in men: further evidence of a direct correlation with free serum testosterone. *J Clin Endocrinol Metab*. 1981; 53(5): 1021–1024.
54. Vining RF, Mcginley RA, Maksvytis JJ, Ho KY. Salivary cortisol: a better measure of adrenal cortical function than serum cortisol. *Ann Clin Biochem*. 1983; 20(6): 329–335.
55. Weckesser LJ, Plessow F, Pilhatsch M, Muehlhan M, Kirschbaum C, Miller R. Do venepuncture procedures induce cortisol responses? A review, study, and synthesis for stress research. *Psychoneuroendocrinology*. 2014; 46: 88–99.
56. Lovallo WR, Farag NH, Vincent AS, Thomas TL, Wilson MF. Cortisol responses to mental stress, exercise, and meals

following caffeine intake in men and women. *Pharmacol Biochem Behav*. 2006; 83(3): 441–447.
57. Pasquali R, Casimirri F, Cantobelli S, et al. Effect of obesity and body fat distribution on sex hormones and insulin in men. *Metabolism*. 1991; 40(1): 101–104.
58. Steptoe A, Ussher M. Smoking, cortisol and nicotine. *Int J Psychophysiol*. 2006; 59: 228–235.
59. Liening SH, Stanton SJ, Saini EK, Schultheiss OC. Salivary testosterone, cortisol, and progesterone: two-week stability, interhormone correlations, and effects of time of day, menstrual cycle, and oral contraceptive use on steroid hormone levels. *Physiol Behav*. 2009; 99: 8–16.
60. Leproult R, Copinschi G, Buxton O, Van Cauter E. Sleep loss results in an elevation of cortisol levels the next evening. *Sleep*. 1997; 20(10): 865–870.
61. Elder GH, Wang L, Spence NJ, Adkins DE, Brown TH. Pathways to the all-volunteer military. *Soc Sci Q*. 2010; 91(2): 455–475.
62. Teachman J, Tedrow L. Delinquent behavior, the transition to adulthood, and the likelihood of military enlistment. *Soc Sci Res*. 2014; 45: 46–55.
63. Rao V, Rosenberg P, Bertrand M, et al. Aggression after traumatic brain injury: prevalence and correlates. *J Neuropsychiatry Clin Neurosci*. 2009; 21(4): 420–429.
64. Dutton MA, Greene R. Resilience and crime victimization. *J Trauma Stress*. 2010; 23(2): 215–222.
65. Banducci AN, Hoffman EM, Lejuez CW, Koenen KC. The impact of childhood abuse on inpatient substance users: specific links with risky sex, aggression, and emotion dysregulation. *Child Abus Negl*. 2014; 38: 928–938.
66. van Goozen SHM, Matthys W, Cohen-Kettenis PT, Thijssen JHH, van Engeland H. Adrenal androgens and aggression in conduct disorder prepubertal boys and normal controls. *Biol Psychiatry*. 1998; 43: 156–158.