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Anger and aggression problems in veterans are associated with an increased acoustic startle reflex

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ABSTRACT

Anger and aggression are frequent problems in deployed military personnel. A lowered threshold of perceiving and responding to threat can trigger impulsive aggression. This can be indicated by an exaggerated startle response. Fifty-two veterans with anger and aggression problems (Anger group) and 50 control veterans were tested using a startle experiment with 10 startle probes and 10 prepulse trials, presented in a random order and with a random interval between the trials. Predictors (demographics, Trait Anger, State Anger, Harm Avoidance and Anxious Arousal) for the startle response within the Anger group were tested. Increased EMG responses were found to the startle probes in the Anger Group compared to the Control group, but not to the prepulse trials. Furthermore, Harm Avoidance and State Anger predicted the increased startle reflex within the Anger group, whereas Trait Anger was negatively related to the startle reflex. These findings indicate that threat reactivity is increased in anger and aggression problems. These problems are not only caused by an anxious predisposition, the degree of anger also predicts the startle reflex.

1. Introduction

Anger and aggression related problems have widespread consequences, ranging from partner/familial violence to a burden for society. These problems are frequently found in samples of deployed military veterans (Elbogen et al., 2010; Heesink, Rademaker, Vermetten, Geuze, & Kleber, 2015) and appear to be rather chronic (Heesink et al., 2015). In case of a psychiatric disorder with comorbid anger and aggression, treatment outcome is impaired (Forbes et al., 2008).

Definitions of anger and aggression are diverse. In short, aggression refers to behaviour with the intention to harm, whereas anger refers to the emotion (Anderson & Bushman 2002). Often, a distinction is made between the planned, premeditated form of aggression, and unplanned, impulsive aggression. The focus of this study is the impulsive form of aggression, in which anger plays an important role.

Dysregulated anger and aggression are symptoms of many disorders, such as posttraumatic stress disorder (PTSD; McHugh, Forbes, Bates, Hopwood, & Creamer, 2012), depression (Painuly, Sharan, & Mattoo, 2005) and Intermittent Explosive Disorder (IED; Coccaro, 2012). The importance of diagnostic criteria for dysregulated anger has been stated before, in order to recognize and study these problems (Del Vecchio and O'Leary 2004; Morland, Love, Mackintosh, Greene, & Rosen, 2012). Since aggressive behaviour is prevalent in many disorders, it is relevant to study it under NIMH's Research Domain Criteria (RDoC).

RDoC is a dimensional approach in which behavioural measures and neuroscience are integrated (Cuthbert 2014). Within the RDoC framework, impulsive aggression fits into the negative valence domain. Part of the negative valence domain is the acute threat response. It is hypothesized that impulsive aggression is accompanied by a lowered threshold of perceiving situations as

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threatening, and threat activates a biologically predisposed survival mode, including fear and flight reactions as well as anger and fight reactions (Novaco & Chemtob 2002). This defensive motivational system is related to activity in limbic brain structures. Furthermore, the domain of arousal within RDoC is an important construct in aggressive behaviour, as it regulates the processes within the negative valence system. Arousal plays a role in the sensitivity to stimuli. For example, heightened arousal might lead to threat-related vigilance. Dysfunctional anger and aggression have been linked to heightened physiological arousal (Mackintosh et al., 2014).

The startle reflex is a response to a sudden, intense stimulus leading to a rapid muscle contraction. This can be measured by a facial electromyography (EMG) in response to a loud noise. An exaggerated startle reflex can indicate the lowered threshold of perceiving threat, due to the sudden and intense nature of the stimulus that leads to a defensive response. Baseline startle reactivity is influenced by the activity of limbic brain structures (Baas, Milstein, Donlevy, & Grillon, 2006) as the amygdala, the bed nucleus of the stria terminalis (BNST) and the brainstem (Grillon & Baas, 2003). This reflex is thought to initiate the selection of an appropriate response to a certain threat. When the startling stimulus is presented several times, the startle reflex diminishes; this is known as habituation. Furthermore, when a less intense stimulus precedes the startle-eliciting stimulus, the response is inhibited. This prepulse inhibition (PPI) is thought to reflect low-level gating of information processing, or early attentional regulation (Grillon & Baas 2003). Deficits of PPI are an indication of the inability to filter out unnecessary information.

Mice genetically predisposed to aggression show an enhanced acoustic startle response and diminished PPI compared to controls (Naumenko, Kozhemyakina, Plyusnina, & Popova, 2014; Sallinen, Haapalinna, Viitamaa, Kobilka, & Scheinin, 1998). In humans with dysfunctional anger and aggression, the acoustic startle reflex has not been studied before in startle trials alone, without other stimuli as affective pictures or faces. Within a healthy population higher levels of aggressiveness as a personality trait were associated with a slower habituation rate (Blanch, Balada, & Aluja, 2014). This habituation is thought to reflect the biological background of judgment and motor or cognitive responses (Blanch et al., 2014).

An enhanced startle response was found in healthy participants during the viewing of angry faces (Dunning, Auriemmo, Castille, & Hajcak, 2010; Springer, Rosas, McGetrick, & Bowers, 2007). These angry face stimuli are classified as threatening stimuli and might therefore elicit a defensive response, measured by an enhanced startle response. Furthermore, angry faces might elicit angry feelings, which in turn can increase startle reactivity. Additionally, hyperactivity of the amygdala was found in a population of Intermittent Explosive Disorder (IED) patients, a disorder characterized by impulsive aggressive behaviour, during aggressive faces viewing (Coccaro, McCloskey, Fitzgerald, & Phan, 2007). These aggressive faces are stated to be a paradigm for social threat. This hyperactivity of the amygdala might be linked to heightened arousal and a lower threshold for perceiving stimuli as threatening.

As stated before, dysfunctional anger and aggression have been linked to heightened arousal. Studying the startle response, including the habituation effect and prepulse inhibition, gives an objective, neurophysiological evaluation of regulation of arousal. It is hypothesized that military men with dysfunctional anger and aggression have an exaggerated startle response and show less PPI. Furthermore, we tested the influence of trait and state anger on the startle response within veterans with anger and aggression problems. In addition, given the link between anger, aggression and anxiety (i.e. Castillo et al., 2014) we also examined whether an exaggerated startle response is linked to underlying anxiety traits.

2. Methods

2.1. Participants

In this study, 52 veterans with dysregulated anger and aggression were included (Anger group). They were recruited via their psychologists/psychiatrists affiliated with Military Mental Health Care Institute and via advertisements in the waiting room and newsletters for veterans. Additionally, fifty control veterans without anger and aggression problems were also included. These were recruited by advertisements or had participated in previous studies. Inclusion criteria for the Anger group were based on the four research criteria for impulsive aggression described by Coccaro (2012): 1) Verbal or physical aggression towards other people occurring at least twice weekly on average for one month; or three episodes of physical assault over a one year period; 2) the degree of aggressiveness is grossly out of proportion; 3) the aggressive behaviour is impulsive (not premeditated); 4) the aggressive behaviour causes either distress in the individual or impairment in occupational or interpersonal functioning (Coccaro, 2012). Inclusion criteria for the Control group were 1) no current DSM-IV diagnosis; 2) no history of pathologic aggressive behaviour.

The Ethics Committee of the University Medical Center Utrecht, The Netherlands, approved this study and all participants signed an informed consent before participation after having received a complete written and verbal explanation of the study. This study was carried out in accordance with the Declaration of Helsinki.

2.2. Interview and questionnaires

The Dutch version of the International Neuropsychiatric Interview (MINI) was used in order to screen for the presence of comorbid psychiatric disorders (Van Vliet, Leroy, & Van Megen, 2000). The complete MINI was administered. In this interview the following current or life-time disorders were screened: depressive disorder, dysthymia, suicidal risk, (hypo)manic disorder, panic disorder, anxiety disorder, agoraphobia, social phobia, obsessive compulsive disorder, PTSD, alcohol or drug dependence and/or abuse, psychotic disorders, anorexia nervosa, bulimia nervosa, generalized anxiety disorder, antisocial personality disorder, somatization disorder, hypochondria, body dysmorphic disorder, pain disorder, attention deficit hyperactivity disorder (ADHD) and adjustment disorder.

To measure anger and aggression, Dutch versions of the State-Trait Anger Expression Inventory-revised (STAXI-2; Hovens, Rodenburg, & Lievaart, 2015) and the Aggression Questionnaire (AQ; Meesters, Muris, Bosma, Schouten, & Beuving, 1996) were used. The STAXI-2 consists of 57 items on a 4-point Likert scale and is divided into two subscales; State Anger and Trait Anger. The AQ consists of 29 items on a 5-point Likert scale and is divided into 4 subscales; Physical Aggression, Verbal Aggression, Anger and Hostility.

The Mood and Anxiety Symptom Questionnaire (MASQ) Anxious Arousal subscale was used to measure current anxiety-specific symptoms (Watson et al., 1995). This scale consists of 17 items on a 5-point Likert scale. The Harm Avoidance subscale of the short version of the Temperament and Character Inventory (TCI; Cloninger, Svrakic, & Przybeck, 1993) was included to measure fearful, anxious personality characteristics and consists of 15 items on a dichotomous scale.

Exposure to childhood trauma was assessed using the Dutch short-form self-report version of the Early Trauma Inventory (Bremner, Bolus, & Mayer, 2007; Rademaker, Vermetten, Geuze, Muilwijk, & Kleber, 2008). This checklist assesses exposure to traumatic experiences (general trauma, physical abuse, emotional abuse and sexual abuse) before the age of 18 years using 27 dichoto-

mous. The total score represents the number of different traumatic events experienced.

The Posttraumatic Stress Disorder Symptom Checklist (PSS; Foa, Riggs, Dancu, & Rothbaum, 1993) was used to measure PTSD symptoms. The PSS consists of 23 items on a 4-point Likert scale.

2.3. Experiment

The experiment consisted of 20 startle probes of 50 ms bursts of white noise of ~105 dB. Half of the probes were preceded by a 20 ms prepulse of ~70 dB with an inter-stimulus interval of 120 ms. During the experiment, background noise of ~60 dB was presented. All noises were delivered through headphones. The first probe was always a stand-alone startle probe, whereas all the other probes were randomly presented with an inter-trial interval of 15–25 s. The total duration of the experiment was 6.66 min. The participants focused on a white fixation cross on a dark grey background while sitting in a chair. They were instructed that they would hear some sounds through the headphones. Furthermore, they were asked to sit as still as possible and try not to laugh or talk during the experiment. The experiment was part of a larger study, which examined psychological and biological aspects of aggression in veterans.

2.4. Startle measures

Eye-blink responses were measured with two 6 mm silver chloride (AgCl) cup skin surface electrodes filled with high conductive recording gel. The electrodes were placed below the lower left eyelid in line with the pupil and separated by 15–20 mm (Blumenthal et al., 2005). A Biopac MP150 system with a sampling rate of 1000 Hz recorded the EMG signals.

2.5. Pre-processing

EMG responses were pre-processed using AcqKnowledge software version 4.3. An offline filter was used for the raw EMG signal. To filter out the high and low artifacts a Finite Impulse Response (FIR) band-pass filter of 28–500 Hz was used. To filter out electrical noise, an Infinite Impulse Response (IIR) band stop filter was used. Next, the Root Mean Square (RMS) was calculated from the raw signal. The maximum value of the squared EMG signal within the time window between 20 and 120 ms after the startle probe onset was extracted from the data (Blumenthal et al., 2005). Furthermore, a baseline mean EMG response of two seconds before the onset of the startle probe was subtracted from the maximum value. This window of 2 s was divided into epochs of 100 milliseconds. Per epoch we measured the variation, and when this exceeded 0.0115 mV, we excluded this epoch from the baseline measurement in order to exclude artifacts as blinking or other movements. After visual inspection of the data, the most discriminative criteria were selected based on manual rejection of the trials. This baseline correction gives us the advantage of a large interval while constraining the risk of including artifacts.

2.6. Data analyses

First, to test for group differences in startle magnitude and in habituation, data of the 10 startle-alone trials were analysed using a mixed model analysis with a random intercept at the subject level.

Next, an independent samples *t*-test was used in order to test whether the groups differed on prepulse inhibition (PPI). PPI was computed as the percentage reduction of the mean startle response (100 − (mean prepulse/mean startle) * 100). Prior to this analysis, multiple data imputation was performed to deal with missing values. 16.66% of the startle data was missing, due to voluntarily eye blinking or movement within the measuring window. Data-points were imputed when 3 or fewer data-points were missing for a subject. For each subject, the mean was then computed from the available data-points and the imputed data. This was done in 28% and 22% of the subjects, for the trials without and with prepulse, respectively. When more than three data-points were missing for one subject, the mean startle response was imputed directly. In both the trials with and without prepulse, this was done in 17% of the subjects. The imputation was done using predictive mean matching algorithm of the Multivariate Imputation by Chained Equations (MICE) package in R (Van Buuren & Groothuis-Oudshoorn 2011), with 50 multiple imputations using 50 iterations.

In addition, using a linear regression, predictors for the mean startle response were tested per group. This analysis was performed within the imputed dataset. Age, education, early trauma, PSS score, trait anger, state anger, anxious arousal and harm avoidance were used in this model.

All analyses were performed using R software.

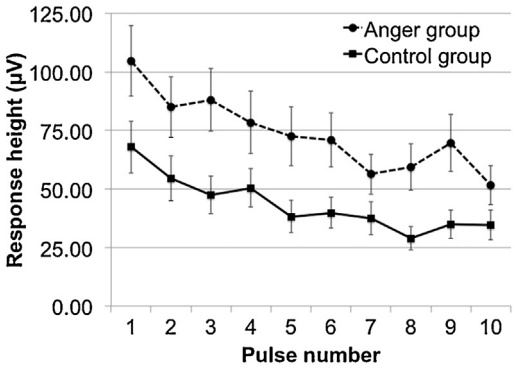

Fig. 1. The average startle response of the Anger and the Control group on the 10 startle pulses.

2.7. Sensitivity analysis

In order to evaluate the effect of the multiple data imputation, the linear regression was also performed as a complete-case analysis, i.e., including only the cases with completely observed startle responses. In 25 participants in the Anger group all startle responses were completely observed.

3. Results

3.1. Demographics

The demographics and characteristics of the two groups are depicted in Table 1. The groups did not differ significantly in age, education and number of deployments. The Anger group scored significantly higher on the anger and aggression measures; the STAXI-2 and the AQ (Table 1).

3.2. Startle reactivity and habituation test

The mixed model analysis in the startle-alone trials revealed a main effect of Time (Estimate = −4.41, SE = 0.95, $t = -4.66$, $p < 0.001$) and a main effect of Group (Estimate = 52.57, SE = 18.19, $t = 2.89$, $p < 0.01$), demonstrating higher EMG responses in general for the Anger group. No interaction effect of Group*Time was found (Estimate = −2.29, SE = 1.32, $t = -1.74$, $p = 0.08$), indicating no differences in change over time between the two groups. The data are depicted in Fig. 1.

Table 1
Demographics and characteristics of the Anger and the Control group.

	Anger (N = 52)		Control (N = 50)		
	Mean	SD	Mean	SD	Statistics
Age	35.26	6.81	34.67	7.30	t (1100) = 0.419, *ns*
Education	4.04	0.64	4.27	0.74	t (1100) = −1.668, *ns*
Number of deployments	2.30	1.37	2.52	1.60	t (1100) = −0.740, *ns*
Years since last deployment	7.73	5.44	6.35	2.96	t (1.99) = 1.606, *ns*
Frequency of aggressive behaviour					
Verbal	4.68	1.39	0.38	1.21	t (1100) = 16.673, p < 0.001
Physical	2.08	1.68	0.00	0.00	t (1100) = 8.950, p < 0.001
Early Trauma	6.38	5.08	2.90	2.56	t (1100) = 4.393, p < 0.001
PSS score	18.36	9.86	2.12	3.39	t (1100) = 11.216, p < 0.001
STAXI-2					
State Anger	23.86	10.99	15.31	1.08	t (1100) = 5.586, p < 0.001
Trait Anger	23.92	6.38	12.83	3.43	t (1100) = 10.998, p < 0.001
Aggression Questionnaire					
Physical aggression	29.64	7.36	18.92	5.35	t (1100) = 8.432, p < 0.001
Verbal aggression	15.26	3.44	11.62	1.68	t (1100) = 6.839, p < 0.001
Anger	25.02	4.71	11.98	3.77	t (1100) = 15.474, p < 0.001
Hostility	24.50	7.71	12.48	4.53	t (1100) = 9.644, p < 0.001
MASQ Anxious arousal	12.12	10.14	1.42	1.94	t (1100) = 7.471, p < 0.001
TCI Harm Avoidance	7.46	4.20	2.75	2.10	t (1100) = 7.209, p < 0.001

Note: Education ranges from 1 to 8. Frequency of aggressive behaviour ranges from 1 to 8. ETI = Early Trauma Inventory; *ns* = not significant; MASQ = Mood and Anxiety Symptom Questionnaire; PSS = Posttraumatic Stress Disorder Symptoms Checklist; SD = Standard deviation; STAXI–2 = State-Trait Anger Expression Inventory revised. TCI = Temperament and Character Inventory, short version.

3.3. Prepulse inhibition

No differences were found in PPI between the Anger group (M = 67.44, SD = 30.45) and the Control group (M = 67.85, SD = 22.51; t = −0.07, p = 0.95).

3.4. Linear regression

To test which variables were significant predictors for a heightened startle response within the Anger group, a linear regression was used. Age, education, early life trauma (before the age of 18), trait anger, harm avoidance, state anger and anxious arousal were tested. The results of this general linear model are depicted in Table 3. Tests to see if the data met the assumption of collinearity indicated that multicollinearity was not a concern (Age, VIF = 1.29; Education, VIF = 1.25; Early Trauma, VIF = 1.25; Trait Anger, VIF = 2.24; Harm Avoidance, VIF = 1.88; State Anger, VIF = 2.04; Anxious Arousal, VIF = 2.20). Lower trait anger, higher scores on harm avoidance and higher state anger were found to be significant predictors of a heightened startle response, whereas age, education, early trauma and anxious arousal were not found to be significant. Furthermore, the same predictors were tested on the mean startle response on the trials with a prepulse. Lower trait anger and higher harm avoidance were also found as significant predictors in these trials (Table 2). In the control group, none of these predictors were found to be significant predictors. The impact of the separate predictors is depicted in scatter plots in the supplemental material.

3.5. Sensitivity analysis

The general linear model was performed within the complete cases, to test the sensitivity of the multiple data imputation. These results are depicted in Table 3. Within the startle trials without prepulse, the predictors trait anger and harm avoidance remained significant, whereas state anger was no longer significant. Furthermore, the PSS score is a significant predictor. In the startle trials with prepulse, trait anger and harm avoidance also remained significant (Table 3).

4. Discussion

This study was carried out to examine whether the startle response in veterans with anger and aggression problems is different from control veterans. Furthermore, predictors of this exaggerated startle response were tested. The main finding of this study is that the startle response is increased in veterans with anger and aggression problems. No difference was found in startle probes preceded by a prepulse. Furthermore, no differences in habituation were found between the groups.

Our results on the heightened startle reflex in veterans with anger and aggression problems correspond to those of studies that show a heightened startle reflex during the viewing of negative pictures. These negative pictures are thought to induce a negative affective state (Grillon & Baas 2003). The current study indicates a lowered threshold for perceiving threatening stimuli in anger and aggression. It has been suggested before that impulsive aggression, originating from anger, is linked to the acute threat response system (Blair 2012). Threatening stimuli can elicit a defensive response as measured by the acoustic startle reflex and in anger and aggression related problems this response is stronger. This indicates a stronger threat-sensitivity for veterans with anger and aggression problems. PTSD is related to a heightened startle response (Holstein, Vollenweider, Jäncke, Schopper, & Csomor, 2010; Pole 2007), and the current study also shows a negative relation between PTSD score and startle magnitude in the complete cases analysis. An overlap between anger and aggression on the one hand and PTSD symptoms on the other hand has been reported before (McHugh et al., 2012; Orth & Wieland 2006; Taft, Vogt, Marshall, Panuzio, & Niles, 2007). The PTSD score is a composite measure of heterogeneous symptoms. The negative relation as found in the current study might most likely be attributed to avoidance/numbing symptoms (see supplemental material).

Furthermore, the increased startle response found in the current study is consistent with findings in mice genetically predisposed to aggression (Sallinen et al., 1998). In that study, mice with altered α_{2C}-adrenoreceptor (α_{2C}-AR) expression show both an increased startle response and more aggressive behaviour. This gives us indications for parallels between animal models and human studies on

Table 2
Predictors for the mean startle response within the Anger group.

Variable	Estimate	Std. Error	*t*	*p*
Intercept	193.73	74.67	2.59	**<0.05**
Age	−1.92	1.49	−1.29	0.21
Education	3.55	15.38	0.23	0.82
ETI	−0.41	1.83	−0.22	0.83
PSS Score	−2.29	1.23	−1.86	0.07
Trait Anger	−4.80	1.67	−2.87	**<0.01**
TCI harm avoidance	7.47	2.52	2.96	**<0.01**
State Anger	2.28	1.07	2.12	**<0.05**
MASQ anxious arousal	0.60	1.36	0.44	0.66

Note: ETI = Early Trauma Inventory; MASQ = Mood and Anxiety Symptom Questionnaire; PSS = Posttraumatic Stress Disorder Symptoms Checklist; TCI = Temperament and Character Inventory, short version. Bold values indicate statistical significance.

Table 3
Results of the sensitivity analysis.

Variable	Estimate	Std. Error	*t*	*p*
Intercept	191.30	83.18	2.30	**<0.05**
Age	−3.31	1.65	−2.01	**<0.05**
Education	17.57	17.35	1.01	0.31
ETI	0.04	2.19	0.02	0.99
PSS Score	−2.77	1.36	−2.04	**<0.05**
Trait Anger	−4.58	1.88	−2.44	**<0.05**
TCI harm avoidance	7.58	2.80	2.70	**<0.01**
State Anger	1.37	1.29	1.06	0.29
MASQ anxious arousal	1.66	1.52	1.09	0.28

Note: ETI = Early Trauma Inventory; MASQ = Mood and Anxiety Symptom Questionnaire; PSS = Posttraumatic Stress Disorder Symptoms Checklist; TCI = Temperament and Character Inventory, short version. Bold values indicate statistical significance.

acoustic startle responses and aggression. Furthermore, it raises the question whether selective α_{2C}-AR agonists might have therapeutic effects in individuals with impulsive aggressive behaviour.

No group differences were found in PPI. Deficits of PPI are an indication of the inability to filter out the unnecessary information and in veterans with aggression related problems this seems not disturbed. PPI deficits have been reported in several disorders such as schizophrenia, PTSD, obsessive compulsive disorder and ADHD (i.e. Braff, Geyer, & Swerdlow, 2001). In the current study the prepulse always had the same intensity and interval between the startle pulses. It would therefore be interesting to test whether PPI differs between patients with anger and aggression problems and controls at other intensities and intervals of the prepulse stimulus. It could be that the current prepulse was not distinctive enough and that other variations in the characteristics of the prepulse do elicit differences between the two groups, as was found to vary in other disorders (Braff et al., 2001; Grillon, Ameli, Charney, Krystal, & Braff, 1992). It has been suggested that PPI deficits in PTSD have their origin in an attentional deficit. Due to distraction by for example thoughts or intrusions, the prepulse might lead to less inhibition compared to controls (Grillon, Morgan, Southwick, Davis, & Charney, 1996). This seems not the case in the current sample, although tests with variations in prepulse characteristics need to further support this.

Trait Anger negatively predicted the height of the startle response in the Anger group. Higher trait anger in combination with a lower startle response have been found before, but only to appetitive pictures (Amodio & Harmon-Jones 2011). To negative pictures, no effects were found. This has been attributed to approach-related behaviour associated with Trait Anger (Amodio & Harmon-Jones 2011). Other studies show that higher Trait Anger was associated with higher startle responses (Cook et al., 1991). However, these findings come from a civilian sample without diagnosed anger and aggression problems. In the current control group of veterans, the personality trait of anger did not predict the height of the startle response; it was only a predictor within the Anger group. Participants in the current control group were selected after reporting no problems regarding anger or aggression, leading to lower Trait Anger in the current control group.

The Harm Avoidance personality trait on the other hand, predicted a higher startle response. This has been found before (Cook et al., 1991) and points towards an anxious predisposition underlying an exaggerated startle response in anger and aggression. In anxiety disorders, the startle response is also enhanced (Grillon & Baas 2003). However, the current study shows that the enhanced startle response in the Anger group likely cannot only be explained by an anxious predisposition. State anger also predicted an exaggerated startle response in the Anger group. This is comparable to studies in which negative affective pictures enhance the startle response (Anokhin & Golosheykin 2010). Viewing negative pictures induces a negative affect, which leads to an enhanced startle response. The sensitivity analysis showed that when participants with missing startle responses were excluded, the effect of state anger diminished. This may be explained by the fact that participants with missing data on the startle responses had on average a higher score on state anger. Since we cannot be absolutely certain about missing observations, the effect of state anger should be interpreted with caution.

In this study, participants were included in the Anger group when they reported problems with Anger and Aggression. Although this is not a DSM diagnosis, the Anger group and the Control group showed major differences on the anger and aggression questionnaires. This underlines the discriminant value of these questionnaires. Furthermore, it shows that these questionnaires addressed the anger and aggression problems within the Anger group, demonstrating the serious problems in this group.

The current study only included male veterans. Although anger and aggression are also prevalent in women, the nature and

symptoms can be different and therefore these findings cannot be generalized to a female population. Furthermore, impulsive aggression is not only reported within military populations, also in civilians these problems are frequently reported (van Gelderen, Bakker, Konijn, & Demerouti, 2011). Although the current study therefore carries significance beyond a military sample, it is not possible to generalize to the general population.

In the current study, only the basic acoustic startle response was measured, without inducing a negative or positive affective state. This needs to be disentangled in future research. It would be very informative to test whether inducing a positive affective state in individuals with anger and aggression related problems, would lead to a diminished startle response. This would give some pointers for treatment of these problems. Furthermore, the increased startle response in veterans with anger and aggression problems as seen in the current study might reflect the tendency to overreact in threatening situations. This leads to the speculation whether an increased startle response could provide useful information regarding the selection or exclusion of military personnel for certain duties.

The current study contributes to knowledge regarding the neurophysiological background of anger and aggression. Anger and aggression are associated with an increased startle response. The current study also shows that the increased startle response is the result of a complex interplay between anxious predisposition and state and trait anger. The neurobiological background of this exaggerated fear response needs to be disentangled in future research. For example, using fMRI techniques it could be discovered whether this difference in threat response can be found in corresponding brain areas. Furthermore, it raises questions whether the reaction to threat is a vulnerability factor to develop anger and aggression problems, or a consequence of these problems. It is therefore important to investigate whether the increased startle response remains after successful treatment of aggression or whether it diminishes.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.biopsycho.2016.12.004.

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