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Behaviorally-inhibited temperament is associated with severity of PTSD symptoms and faster eyeblink conditioning in veterans

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Abstract

Prior studies have sometimes demonstrated facilitated acquisition of classically-conditioned responses and/or resistance to extinction in post-traumatic stress disorder (PTSD). However, it is unclear whether these behaviors are acquired as a result of PTSD or exposure to trauma, or reflect pre-existing risk factors that confer vulnerability for PTSD. Here, we examined classical eyeblink conditioning and extinction in veterans self-assessed for current PTSD symptoms, exposure to combat, and the personality trait of behavioral inhibition (BI), a risk factor for PTSD. 128 veterans were recruited (mean age 51.2 years; 13.3% female); 126 completed self-assessment, with 25.4% reporting a history of exposure to combat and 30.9% reporting severe, current PTSD symptoms (PTSS). PTSD symptom severity was correlated with current BI ($R^2=0.497$) and PTSS status could be predicted based on current BI and combat history (80.2% correct classification). A subset of the veterans ($n=87$) also completed eyeblink conditioning. Among veterans without PTSS, childhood BI was associated with faster acquisition; veterans with PTSS showed delayed extinction, under some conditions. These data demonstrate a relationship between current BI and PTSS, and suggest that the facilitated conditioning sometimes observed in PTSD patients may partially reflect personality traits such as childhood BI that pre-date and contribute to vulnerability for PTSD.

Keywords

behavioral inhibition; eyeblink; classical conditioning; learning; post-traumatic stress disorder (PTSD); veterans

INTRODUCTION

In the wake of exposure to a traumatic event, some individuals develop post-traumatic stress disorder (PTSD), and others may experience varying degrees of sub-clinical PTSD symptoms, including heightened emotional responses to and avoidance of reminders of the

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trauma. The wide range of PTSD symptom severity among individuals exposed to similarly stressful traumatic events (Pitman et al. 1987; Orr et al. 1993; Shalev et al. 1993) suggests that pre-existing vulnerability factors modulate an individual's risk to develop PTSD. Several vulnerability factors have been identified, including genetic polymorphisms (Binder et al. 2008; Amstadter et al. 2009), female sex (Tolin and Foa 2006), prior exposure to highly stressful situations such as combat, rape or other trauma (e.g., North and Smith 1990; Davidson 2000; Seng et al. 2009), brain and physiological abnormalities (Pitman et al. 2006; Liberzon and Sripada 2008) and various sociobehavioral variables including personality traits (Engelhard et al. 2006; Gil and Caspi 2006; Qouta et al. 2007). As one example, behavioral inhibition (BI) is a temperamental tendency for avoidance of or withdrawal from unfamiliar social and nonsocial situations (Fox et al. 2005). This tendency develops early in life and is relatively stable over development (Degnan and Fox 2007). Individuals with high BI are at increased risk for PTSD (Fincham et al. 2008; Kashdan et al. 2009) as well as other anxiety disorders (Hirshfeld et al. 1992).

Diathesis models of PTSD emphasize the dynamic interaction of pre-existing vulnerabilities and environmental interactions. An influential theory of PTSD (Pitman 1988; Pitman et al. 2000) suggests that many PTSD symptoms reflect learned associations in which cues (conditioned stimuli or CSs) present at the time of the trauma (unconditioned stimulus or US) come to evoke emotional responses (conditioned responses or CRs) similar to those provoked by the event itself. According to this model, individual differences in the speed of acquisition of the CR, and in extinction of the CR when the CS is no longer paired with the US, could promote individual differences in the development and maintenance of PTSD symptoms.

Consistent with this view, many studies have reported facilitated acquisition of CRs and delayed extinction of CRs in individuals with PTSD, compared to non-PTSD controls (e.g., Grillon and Morgan 1999; Orr et al. 2000; Peri et al. 2000; Blechert et al. 2007; Burriss et al. 2007; Wessa and Flor 2007; Jovanovic et al. 2010a), although other studies have found no such effects (e.g., Ayers et al. 2003; Orr et al. 2006; Vythilingam et al. 2006; Ginsberg et al. 2008a; Ginsberg et al. 2008b). The variability of results may partially reflect different experimental design and/or different parameters such as stimulus duration and intensity.

Such between-group studies do not address the question of whether observed abnormalities in associative learning are acquired symptoms of PTSD, emerge as a consequence of exposure to stressful events, or pre-date PTSD and exposure to trauma. In the latter case, strong learning of avoidant and emotional responses to trauma-related cues and resistance to extinction of these responses might bias an individual to develop PTSD if exposed to traumatic events. The few studies that have attempted to address this question have yielded discrepant results. For example, a prospective study of classical conditioning in firefighter recruits found that degree of delayed CR extinction, assessed during firefighter training, was significantly correlated with PTSD symptom severity following subsequent exposure to a traumatic event (Guthrie and Bryant 2006), suggesting delayed extinction pre-dates trauma exposure in vulnerable individuals. On the other hand, a study of monozygotic twins discordant for combat exposure found that combat-exposed participants with PTSD showed normal extinction of a conditioned fear response, compared with both their non-combat-exposed, non-PTSD co-twins and with combat-exposed, non-PTSD participants, although extinction recall was deficient on the following day (Milad et al. 2008), thus, this study suggests that delayed extinction does not pre-date PTSD. These studies did not take into account pre-existing vulnerabilities, such as temperament, which might help account for the discrepant findings.

Here, we consider a complementary approach to understanding the relationship between vulnerability factors and associative learning, by testing acquisition and extinction of classically-conditioned responses in veterans self-assessed for behavioral inhibition (BI), history of exposure to combat, and current PTSD symptom severity. If facilitated conditioning in PTSD partially reflects preexisting vulnerabilities such as high BI, then it should be evident in veterans with high BI but without PTSD. Alternatively, if facilitated learning emerges only as a symptom of PTSD and/or as a consequence of exposure to trauma, then it should be evident only in veterans with severe PTSD symptoms and/or history of exposure to combat.

In the current study, we considered classical eyeblink conditioning, in which the CS is a tone, the US is an airpuff aimed at the eye that evokes a protective eyeblink, and the CR is an anticipatory eyeblink that occurs after CS onset but before US arrival. Participants were randomly assigned to receive training under delay contingencies, in which the CS and US overlap and co-terminate, or omission contingencies, in which a CR causes omission of the US. Typically, imposition of an omission contingency into eyeblink conditioning results in lower performance, in terms of number of CRs produced (Logan 1951; Massaro and Moore 1967); accordingly, inclusion of an omission group in the current study allowed us to examine whether veterans with high BI, history of combat exposure, and/or severe PTSD symptoms could modulate their responding as a function of CS-US contingency. Following eyeblink acquisition, each participant also received a series of CS-alone trials, during which the eyeblink CR was expected to extinguish, allowing us to determine whether delayed extinction occurred as a function of BI, history of combat exposure, and/or severe PTSD symptoms.

As our measure of BI, we used the Adult and Retrospective Measures of Behavioural Inhibition (AMBI/RMBI), two recently-developed self-report measures that allow assessment of current and childhood inhibited temperament (Gladstone and Parker 2005); however, a relationship between these measures and current PTSD symptom status has not been established. Therefore, a secondary purpose of this study was to determine whether AMBI/RMBI scores were significantly related to presence vs. absence of current, severe PTSD symptoms in veterans.

METHODS

Participants

Veterans (N=128) were recruited from the New Jersey Health Care System (NJHCS), East Orange, NJ. Participants included 111 males and 17 females (13.3%), with mean age 51.2 years (SD 8.0, range 23-65) and mean education 13.4 years (SD 1.7, range 9-20). The group included 94 African-Americans (73.4%), 26 Caucasians (20.3%) and 8 individuals of Mixed/Other Race (6.3%). When asked to identify specific wars or conflicts in which they had served, 44 veterans reported having served during the Vietnam conflict (approx. 1970-1975), 17 during Operation Desert Storm (1991), 7 during Operation Enduring Freedom/Operation Iraqi Freedom (2001+), and 39 during peacetime or no specific conflict; others reported having served during various conflicts including operations in Granada, Panama, and the Middle East. Veterans were not included or excluded based on any medical or psychiatric history, but were asked to self-report current medications; 41 participants (32%) self-reported taking psychoactive medications. While some participants were able to clearly identify the name of their medications, others simply reported the use of medication (e.g., "anxiety meds" or "anti-depressant"). Thus, further analysis regarding medication usage in this sample was not possible.

Participants received payment at the rate of \$30 per hour (maximum of \$60) for their participation in the study. Testing generally occurred between 1000-1200 and 1300-1500; no obvious differences in subject demographics or experimental data were observed as a function of testing time. All participants gave written informed consent before initiation of any experimental procedures; procedures were approved by the NJHCS Institutional Review Board and were conducted in accordance with the Declaration of Helsinki and guidelines established by the Federal Government for the protection of human subjects.

Self-report Measures

After informed consent, participants completed a battery of paper and pencil questionnaires prior to instrumentation for electromyography (EMG) recording. These questionnaires typically required 20-30 min to complete. The package included the Adult and Retrospective Measure of Behavioural Inhibition (AMBI/RMBI; Gladstone and Parker 2005), the Combat Exposure Scale (CES; Keane et al. 1989), and the PTSD Checklist-Military version (PCL-M; Blanchard et al., 1996).

The Adult Measure of Behavioural Inhibition (AMBI) is a 16-item self-report inventory that assesses current tendency to respond to new stimuli with inhibition and/or avoidance, and has also been shown to be a measure of anxiety proneness; individuals scoring from 2 to 15 are classified as “uninhibited” while individuals scoring from 16-32 are “inhibited” (Gladstone and Parker 2005). In addition to a total AMBI score, items group into four subscales derived from factor analysis (Gladstone and Parker 2005): ‘fearful inhibition’ (FI), ‘risk avoidance’ (RA), ‘non-approach’ (NA), and ‘low sociability’ (LS). Items in the FI subscale assess the general tendency to respond with wariness, show hyper-vigilance, and become physically anxious in response to novel social situations (e.g., “Do you intend to withdraw and retreat from those around you?”). Items on the RA subscale assess the tendency to avoid physical risk, adventurous activities, and high social stimulation (e.g., “If physically able, would you enjoy adventure holidays with some element of risk?”). Items in the NA subscale assess willingness to engage others in novel social situations (e.g., “Do you tend to introduce yourself to new people?”). Items in the LS subscale assess preference for solo activities (e.g., “Do you prefer your own company to the company of others?”).

The Retrospective Measure of Adult Inhibition (RMBI) is an 18-item self-report inventory used to assess childhood memories of exhibiting inhibition to the unfamiliar; individuals scoring from 0 to 11 are classified as “uninhibited” while individuals scoring from 12-25 are “inhibited” (Gladstone and Parker 2005). Like the AMBI, there are four RMBI subscales derived from factor analysis (Gladstone and Parker 2005): ‘fearful inhibition’ (FI), ‘risk avoidance’ (RA), ‘non-approach’ (NA), and ‘shyness and sensitivity’ (SS). Items for the first three subscales are similar to those on the AMBI, but target memories of childhood behaviors. The SS subscale assesses self-rated shyness, particularly at school. The RMBI allows respondents to endorse a “do not remember” item. For items where participants endorsed “do not remember,” scores were pro-rated based on responses to the remaining questions on the same RMBI subscale, with reverse scoring taken into account. Any participant who endorsed “do not remember” on more than 50% of the total RMBI items was scored as incomplete.

The Combat Exposure Scale (CES) is a 7-item self-report questionnaire that assesses exposure to stressful military events, with items rated on frequency, duration, and degree of exposure; for example, the question “Were you ever surrounded by the enemy?” can be endorsed at a level of 1=“no” to 5=“more than 12 times.” Total CES score is calculated from a sum of weighted scores; as in prior studies (e.g., Ginsberg et al. 2008a), veterans with a CES score of 0-7 were classified as non-combat while those with a score of 8+ were classified as having history of exposure to combat.

The PTSD Checklist-Military Version (PCL-M) is a 17-item self-report questionnaire that asks about presence and frequency of PTSD symptoms in response to stressful military experiences; symptoms are rated according to how much they have bothered the participant in the past month, on a scale from “Not at all” to “Extremely.” Specific questions correspond to DSM-IV symptom clusters including cluster B (re-experiencing the traumatic event), cluster C (avoidance/numbing), and cluster D (increased arousal). PCL-M scores of 50+ have been shown to be a predictor of PTSD in military samples (Weathers et al. 1993; Blanchard et al. 1996). Accordingly, we also categorized participants according to presence or absence of current, severe PTSD symptoms (PTSS) based on this cutoff.

Eyeblink Conditioning

Materials and Apparatus—The eyeblink conditioning apparatus and procedures were as previously described (Beck et al. 2008). Auditory stimuli were produced with Coulbourn Instruments (Allentown, PA) signal generators and passed to a David Clark aviation headset (Model H10-50, Wrochester, MA). Sound levels were verified with a Realistic sound meter (Radio Shack). Airpuffs were produced by pressurizing ambient air to 3.5 psi (Fürgut Industried, Aitrach, Germany), and released through sylastic tubing attached to the boom of the headphones by a computer controlled solenoid valve (Clipper Instruments, Cincinnati, OH). The boom was placed 1 cm from the eye and aimed at it. To transduce the eyelid EMG signal, pediatric silver/silver chloride EMG electrodes with solid gel were placed above and below the left eye, with the ground electrode placed on the neck. The EMG signal was passed to a medically isolated physiological amplifier (UFI, Morro Bay, CA), low pass filtered and amplified 10K. The signal for EMG was sampled at 200 Hz by an A/D board (PCI 6025E, National Instruments, Austin, TX) connected to an IBM compatible computer. Software control of stimulus generation was accomplished with LabView (National Instruments, Austin, TX). For the omission contingency, a time-varying Gabor filter processed the data up to 10 ms prior to US trigger. If a CR was detected during this period, the US trigger was not initiated.

Procedures—Participants were pseudorandomly assigned to the delay and omission groups: for each pair of participants, the first was randomly assigned to one group, and the second was assigned to the other group. Participants were seated in a comfortable chair and fitted with EMG electrodes; they were instructed that the study evaluated responses to tones and airpuffs to the eye, that they were to watch a silent video of their choice (e.g., *Free Willy* with sound muted), and to stay awake. Each participant was then exposed to three airpuff stimuli; these trials served to verify the ability of the participant to produce a UR. Data from participants who failed to produce URs, or failed to remain awake throughout the experiment, were discarded from analysis.

Conditioning then commenced. For participants in the delay group, the CS was a 500-ms 83-dB, 800-Hz pure tone (50 ms rise/fall) that co-terminated with the US, a 50-ms air puff; the inter-trial interval varied between 15 and 25s. For participants in the omission group, all parameters were identical to delay training, except that emitting a conditioned response (CR) at least 40 ms prior to the airpuff prevented US delivery. Five blocks of 12 acquisition trials were presented (total 60 trials). In both delay and omission groups, acquisition trials were followed by 20 extinction trials that were similar to the acquisition trials except that the US was not presented.

Data Processing—Processing of eyeblink responses followed methods previously reported (Beck et al., 2008; Servatius et al., 1998). To determine the occurrence of an eyeblink, EMG activity was first low-pass filtered using lowess filter (Stat-Sci, Tacoma, WA) using a time constant of 0.025 and a smoothing interval of 5. Using the filter values,

activity greater than 0.2 (unitless) corresponds to an eyeblink. An α -response (orienting response) was scored when an eyeblink occurred within 80 ms of CS onset; these responses rarely occur with the present equipment configuration. A CR was scored when an eyeblink was elicited 80 ms after CS onset but before US onset. An unconditioned response (UR) was scored when an eyeblink was produced 0-100 ms after US onset during the three US-alone presentations preceding training.

Data Analysis

Questionnaire scores were analyzed using t-test or univariate analysis of variance (ANOVA) for continuous values and chi-square tests for categorical values. Inter-item relationships and correlations across scores were analyzed using the Pearson correlation coefficient (using Yates continuity correction for 2×2 tables) or Cronbach's α . Stepwise linear regression was used to investigate the ability of questionnaire scores to predict PCL-M scores and stepwise discriminant analysis was used to investigate the ability of categorical values based on questionnaire scores to predict PTSS status.

For eyeblink data, the dependent measure was percent CRs scored within each block of trials and total percent CRs over the entire acquisition or extinction phase. Total scores were analyzed by Pearson correlation to assess relationships with continuously-valued questionnaire scores, and univariate ANOVA to assess relationships with categorical values; scores across the five blocks of acquisition and extinction were analyzed by repeated-measures ANOVA, with post-hoc ANOVA and t-tests as appropriate. As explained below, UR magnitude was included as a covariate in all assessments of acquisition data, and total percent CRs during acquisition was included as a covariate in all assessments of extinction data.

All ANOVAs used type III sum-of-squares, as appropriate for designs where cell sizes are unequal (and some cells may be empty). All tests were two-tailed, with threshold for significance set at 0.050. Where multiple pairwise comparisons were made, Bonferroni correction was used to reduce alpha to protect against risk of increased family-wise error; the corrected alpha is reported in the text only when p-values approach 0.050 but fall short of the corrected alpha.

Because of the low number of females in the current study, and the fact that random assignment led to only three females being assigned to the omission group, we did not consider gender as a factor in the analysis of eyeblink data. When the eyeblink analyses described above were re-run on the subset of data from males only, the general pattern of results was qualitatively similar to that obtained for the complete dataset, although the reduced sample size meant lower power to detect significant differences.

RESULTS

1. Questionnaire Data

CES and PCL-M—Of the total sample (n=128), 2 participants did not complete any questionnaires, and their data were dropped from all further analysis.

Among the remaining 126 participants, 94 (74.6%) were classed as non-combat based on CES scores; the remaining 32 were classed as combat (25.4%). Mean CES score for the non-combat veterans was 1.5 (SD 2.5); for the combat veterans it was 19.6 (SD 8.8).

Mean PCL-M score for the 126 participants was 38.2 (SD 18.7, range 17-85), with participants endorsing "Moderate" or higher for an average of 1.5 cluster B symptoms (SD 2.0), 2.4 cluster C symptoms (SD 2.6), and 2.1 cluster D symptoms (SD 1.9). Scores on all

PTSD symptom clusters were significantly correlated with each other (Pearson's r , all $r > 0.650$, all $p < 0.001$) and with CES score (Pearson's r , all $r > 0.350$, all $p < 0.001$). Total PCL-M scores, as well as cluster B, C, and D scores, were all significantly higher in combat than non-combat veterans (Table 1; independent-samples t -tests, all $p < 0.004$).

Following Weathers et al.'s (1993) cutoff of 50+ as indicating severe current PTSD symptoms (PTSS), 39 of our 126 veterans were classed with PTSS (30.9%); this included 13 of the 32 combat veterans (40.6%) but only 20 of the 94 non-combat veterans (21.3%), a statistically significant difference (Yates-corrected chi-square $\chi^2 = 14.48$, $df = 1$, $p < 0.001$). Among the 17 females in the current study, 10 had PTSS (58.8%) compared with only 29 of 109 males (26.6%), a statistically-significant difference (Yates-corrected chi-square $\chi^2 = 5.72$, $df = 1$, $p = 0.017$).

AMBI/RMBI—All 126 participants responded to all AMBI questions, except for one participant who failed to enter a response to question 3 (“Do you tend to become quiet?”). Three participants endorsed “do not remember” responses on all RMBI questions, and so their total RMBI and RMBI subscales could not be calculated. These three participants were accordingly included in analysis of AMBI but not RMBI data. Among the remaining 123 participants, only four endorsed more than one “do not remember” responses on RMBI. RMBI item response rates ranged from 92.7% (question 3: “Were you reluctant to go to school on your first day or the first day after holidays?”) and 93.5% (question 4: “Did you prefer parties with crowds of children rather than small gatherings?”) to 100% (8 questions total).

Mean AMBI/RMBI total scores and subscale scores for the veteran sample are shown in Table 2. Both AMBI and RMBI were significantly higher in individuals with PTSS (Table 1; independent-samples t -tests, all $t > 2.75$, all $p < 0.010$). Although Gladstone & Parker (2005) reported higher Risk Avoidance in females, the gender difference was not significant in the current sample (males RMBI: $M = 2.8$, $SD = 1.4$; AMBI: $M = 3.4$, $SD = 1.4$; females RMBI: $M = 3.4$, $SD = 1.5$; AMBI: $M = 4.1$, $SD = 1.3$; independent-samples t -tests, all $t < 1.8$, $0.050 < p < 0.010$), nor were there significant gender differences on any other AMBI/RMBI scores or subscales (all $t < 1.0$, all $p > 0.300$).

Internal consistency of AMBI and RMBI total and subscale scores was estimated using Cronbach's α , with reverse scoring for individual questions taken into account. For the sixteen questions comprising AMBI total score, Cronbach's $\alpha = 0.841$; for individual AMBI subscales, inter-item reliability was high for non-approach, fearful inhibition, and low sociability subscales (with α ranging from 0.588 to 0.793) but lower for risk avoidance ($\alpha = 0.249$). Similarly, inter-item reliability was high for the eighteen questions comprising RMBI total score ($\alpha = 0.816$) as well as for the non-approach, fearful inhibition, and shyness and sensitivity subscales (α ranging from 0.600 to 0.722) but lower for risk avoidance ($\alpha = 0.233$).

In the current sample, AMBI and RMBI scores were highly correlated (Pearson's $r = 0.559$, $p < 0.001$). Within AMBI, all four subscale scores were significantly correlated (all $p < 0.008$); correlations ranged from $r = 0.242$ (non-approach vs. risk avoidance) to $r = 0.644$ (fearful inhibition vs. low sociability). Within RMBI, the non-approach, fearful inhibition, and shyness and sensitivity subscales were all correlated with each other (all $r > 0.600$, all $p > 0.001$) but risk avoidance was not correlated with any of the other subscales (all $r < 0.150$, all $p > 0.300$).

Based on the cutoffs in Gladstone and Parker (2005)'s original validation article, 62 of 123 (50.4%) veterans in the current sample were “inhibited” based on RMBI scores and 69 of

126 (54.8%) veterans in the current sample were “inhibited” based on AMBI scores. There were no significant differences in gender distribution or history of combat exposure among individuals classed as inhibited vs. uninhibited on AMBI or RMBI (all $\chi^2 < 1.00$, all $p > 0.500$). Among veterans classed as “inhibited” based on RMBI, 26 had PTSS (41.9%) compared with only 12 of those classed as “uninhibited” (19.7%), a significant difference (Yates-corrected chi-square, $\chi^2 = 7.14$, $df = 1$, $p = 0.008$). Similarly, among those classed as “inhibited” based on AMBI, 29 had PTSS (42.0%) compared with only 10 of those classed as “uninhibited” (17.5%; Yates-corrected chi-square, $\chi^2 = 8.76$, $df = 1$, $p = 0.003$).

Predicting PTSD Symptoms Based on Combat History and BI—Stepwise linear regression on PCL-M scores, with factors of total AMBI, total RMBI, and CES score revealed that PCL-M scores could be significantly predicted by a two-variable model including AMBI ($\beta = 0.501$) and CES ($\beta = 0.443$). This model could account for significant variance in PCL-M scores ($R^2 = 0.497$; $F(2, 120) = 59.27$, $p < 0.001$); addition of RMBI into the model did not account for significant additional variance ($p > 0.050$). When the regression was repeated replacing AMBI/RMBI total scores with the eight subscale scores, the best prediction was produced by a three-factor model including CES ($\beta = 0.415$) and two AMBI subscales: Fearful Inhibition ($\beta = 0.306$), and Low Sociability ($\beta = 0.221$). This model could also account for significant variance in PCL-M scores ($R^2 = 0.473$; $F(3, 119) = 35.31$, $p < 0.001$); addition of the remaining AMBI and RMBI subscales into the model did not account for significant additional variance (all $p > 0.050$).

Univariate ANOVA on PCL-M total score, with factors of AMBI and RMBI (“inhibited” vs. “uninhibited”) and combat history (exposed vs. non-exposed) revealed significant main effects of combat ($F(1, 115) = 31.98$, $p < 0.001$) and AMBI ($F(2, 115) = 8.72$, $p = 0.004$) with no main effect of RMBI and no interactions (all $F < 1.00$, all $p > 0.400$). Specifically, veterans with a history of exposure to combat had higher PCL-M scores than non-combat veterans (Figure 1A) and veterans classed as inhibited (on AMBI or RMBI) had higher PCL-M scores than uninhibited veterans (Figure 1B). Considering scores on PCL-M cluster B, C, and D symptoms, the results were similar (Table 1): significant main effects of combat history and AMBI (all $F > 4.00$, all $p < 0.050$) with no effect of RMBI and no interactions (all $F < 3.0$, all $p > 0.050$).

As noted above, 30.9% of the veterans in our sample were classed with PTSS, meaning that they scored 50 or higher on PCL-M. Stepwise discriminant analysis, using *a priori* probability of 30.9%, with independent variables of AMBI, RMBI and CES scores, found that a model including AMBI (standardized coefficient 0.762) and CES (standardized coefficient 0.710) correctly classified 21 of 39 PTSS cases (53.8% sensitivity) and 80 of 87 non-PTSS cases (92.0% selectivity) for an overall 80.2% correct classification. Addition of RMBI to the model did not significantly increase predictive power (at tolerance-to-enter/remove 0.050).

2. Eyeblink conditioning

Study completion rates and group assignment—Of the $n = 126$ veterans who completed the AMBI, CES, and PCL-M questionnaires, a complete eyeblink conditioning data set was obtained from $n = 87$. For one of these participants, RMBI could not be calculated, due to endorsement of “do not remember” responses on over 50% of items; this participant’s remaining questionnaire scores and eyeblink data were included in the analysis.

Conditioning data from the remaining participants ($n = 36$) were unusable. Specifically, 15 participants fell asleep one or more times during the 1-h eyeblink testing session; another 19 participants failed to exhibit any eyeblink URs, even after the experimenter made several

attempts to reposition the headset. Data from the remaining participants were lost due to poor signal quality (noise from ambient electrical fields interfering with EMG signal).

Those participants who completed the eyeblink conditioning study did not differ from those who did not on any measures including gender distribution (Yates-corrected chi-square test, $\chi^2=0.410$, $df=1$, $p=0.522$), age, education (years), or questionnaire scores (independent-samples t-tests, all $p>0.100$). Data from the participants who failed to complete the eyeblink study were discarded from further analysis.

The remaining $n=87$ participants had been randomly assigned to the delay ($n=43$) and omission ($n=44$) groups. There were no significant differences between participants in the delay vs. omission groups on age, education, PCL-M scores, CES scores, or AMBI/RMBI scores (independent-samples t-tests, all $p>0.050$). The groups did however differ in gender distribution (Yates-corrected chi-square test, $\chi^2=4.62$, $df=1$, $p=0.032$), with 10 females assigned to the delay group but only 3 assigned to the omission group. There were no differences between delay and omission groups in distribution of individuals with history of exposure to combat, BI, or PTSS (Yates-corrected chi-square tests, all $p>0.200$).

Unconditioned Eyeblink Responding—An ANOVA on UR magnitude, with factors of training group, history of combat exposure, AMBI, RMBI, and PTSS, revealed no significant main effects or interactions (all $F<2.60$, all $p>0.050$). There were no significant correlations between UR magnitude and any AMBI/RMBI subscale or PTSD symptom cluster (Pearson's r , all $r<0.20$, all $p>0.050$). However, there were significant correlations between UR magnitude and total eyeblink CRs during the acquisition phase of eyeblink conditioning (Pearson's $r=0.301$, $n=87$, $p=0.005$). The relationships between UR magnitude and age, and between age and total CRs, both fell short of significance (all $r<0.200$, all $p>0.050$). Accordingly, UR magnitude but not age was included as a covariate in subsequent analyses of eyeblink acquisition data. Total percent CRs during extinction were correlated with total CRs during acquisition ($r=0.543$, $n=87$, $p<0.001$) but not with age or UR magnitude (all $r<0.200$, all $p>0.050$). Accordingly, percent CRs during acquisition was included as a covariate in subsequent analysis of eyeblink extinction data.

Acquisition and Extinction—Total percent eyeblink CRs during acquisition was significantly correlated with RMBI scores (Pearson's $r=0.216$, $n=87$, $p=0.045$) but not AMBI scores ($r=0.185$, $p=0.086$); neither AMBI nor RMBI scores were correlated with extinction CRs (all $r<0.100$, all $p>0.500$). Of the eight AMBI and RMBI subscales, none correlated significantly with acquisition or extinction CRs (all $r<0.200$, all $p>0.050$). There was no significant difference in total percent acquisition or extinction CRs in veterans with vs. without a history of combat exposure (independent-samples t-tests, all $t<1.50$, all $p>0.100$). Total percent CRs during acquisition and extinction were not significantly correlated with PCL-M total scores or scores on any of the three PTSD symptom clusters (all $r<0.250$, all $p>0.050$).

Figure 2A shows eyeblink responding across the five blocks of acquisition conditioning in the delay and omission groups. A repeated-measures ANOVA on mean percent CRs over the five acquisition blocks, with factors of group (delay vs. omission), RMBI (inhibited vs. uninhibited), AMBI (inhibited vs. uninhibited), PTSS (with vs. without), and history of exposure to combat (combat vs. non-combat), and covariate of UR magnitude, revealed a significant within-subjects effect of block ($F(4,248)=4.03$, $p=0.003$), a block x group interaction ($F(4,248)=2.84$, $F=0.025$) and a three-way interaction between block, RMBI and PTSS ($F(4,248)=3.33$, $p=0.011$); no other effects or interactions were significant (all $p>0.050$). Figure 2B shows the interaction between block, RMBI and PTSS: specifically, there was no difference in responding across blocks between inhibited vs. uninhibited

veterans with PTSS (repeated-measures ANOVA, $F(1,56)=4.55$, $p=0.037$); but among non-PTSS veterans, those with inhibited temperament made more CRs than those with uninhibited temperament ($F(1,56)=4.55$, $p=0.037$).

Figure 3A shows eyeblink responding across the five extinction blocks in the delay and omission groups; given that the delay group had reached higher levels of responding during acquisition (compare Figure 2A), they extinguished at a steeper rate than the omission group. A repeated-measures ANOVA on these data, with factors of group, AMBI, RMBI, PTSS, and combat history, and covariate of total percent CRs during acquisition, confirmed this main effect of group ($F(1,62)=6.29$, $p=0.004$), as well as significant interactions between AMBI and RMBI ($F(1,62)=6.29$, $p=0.015$), between RMBI and group ($F(1,62)=4.48$, $p=0.038$), between block, AMBI and combat history ($F(4,248)=4.62$, $p=0.001$) and between block, combat history, PTSS, and group ($F(4,248)=3.44$, $p=0.009$); no other effects or interactions approached significance (all $p>0.100$). The AMBI x RMBI interaction was due to much higher responding during extinction in the small number ($n=6$) of AMBI-uninhibited, RMBI-inhibited veterans ($M=45.8\%$ CRs, $SD 44.3\%$) compared to the other cells (M range 29.4-30.9% CRs, $SD 25.4-27.5\%$); this difference did not survive post-hoc testing (univariate ANOVA, $F(3,83)=0.62$, $p=0.605$), probably due to the low sample size in that cell. The group x RMBI interaction was due to significantly higher responding during extinction among RMBI-inhibited individuals in the omission group (Figure 3B; independent-samples t-test, $t(42)=2.14$, $p=0.039$) but not in the delay group ($t(41)=1.36$, $p=0.183$). The block x AMBI x combat history was due to higher responding during block 1 of extinction AMBI-inhibited non-combat veterans than in AMBI-uninhibited non-combat veterans (Figure 3C; independent-samples t-test, $t(57.7)=2.11$, $p=0.047$); there was no such effect of AMBI in combat veterans ($t(22)=0.024$, $p>0.500$). To investigate the block x combat history x PTSS x group interaction, separate post-hoc tests were conducted on extinction data from the delay and omission groups. In the omission group, veterans with PTSS gave significantly more CRs during extinction blocks 1 and 2 (Figure 4A; independent-samples t-tests, all $t>2.00$, all $p<0.050$) but not during blocks 3-5 (all $t<1.5$, all $p>0.100$); the interaction with combat did not survive post-hoc analysis (all $p>0.050$). There was no significant effect of PTSS, nor any interaction with combat history, in the delay group (Figure 4A).

DISCUSSION

Given that avoidance reflects a learned association between implicit or explicit cues, individual differences in vulnerability to anxiety disorders such as PTSD may at least in part reflect differences in associative learning. This study was designed to investigate whether differences in associative learning and extinction might be related to PTSD symptom severity, history of combat exposure, or BI. In fact, total conditioned responding during acquisition was correlated with childhood, but not current, behavioral inhibition; extinction was delayed in veterans with PTSS regardless of current or childhood BI, although BI did interact with both combat history and training group to affect extinction. A secondary objective of the study was to determine whether the AMBI/RMBI scale, a self-report measure used to assess current and childhood BI, was associated with PTSD symptoms in veterans; the results demonstrated that current BI and PTSS were correlated, although PTSS status was best predicted by a combination of current BI and combat exposure measures. We discuss these findings further below.

AMBI/RMBI and PTSD Symptoms

Consistent with other studies that have demonstrated increased vulnerability to PTSD in veterans exposed to combat, and in females, we found a higher incidence of PTSS in combat

veterans, and in females, in the current sample. PTSS was also more prevalent in participants who self-reported current behavioral inhibition based on AMBI. Further, within this veteran sample, a model including the individual's history of exposure to combat and presence/absence of current BI could predict PTSS status with slightly over 80% accuracy. To our knowledge, this study is the first documenting a relationship between self-reported current BI and current PTSD symptom severity.

Although current and retrospective BI were highly correlated in this sample, retrospective BI (based on RMBI scores) did not account for additional variance in PCL-M scores beyond what was already accounted for by current BI (based on AMBI scores). This is broadly consistent with the conclusion of Gladstone and Parker (2005), in their original validation article, that AMBI is more useful as a predictor of contemporaneous clinical outcomes.

This study also provides some initial normative data for AMBI/RMBI scores on a veteran sample. Mean AMBI (17.0) and RMBI (12.7) in the current sample were higher than those reported in the original validation paper (Gladstone and Parker 2005) for healthy adult controls (AMBI 12.0, SD 4.7; RMBI 8.7, SD 6.1) but lower than those reported for patients with clinical anxiety (AMBI 19.4, SD 6.3; RMBI 15.0, SD 8.7). The cutoff for high inhibition (AMBI 16+, RMBI 12+), based on median split of the data in the validation paper, nevertheless resulted in only slightly more than half of all veterans in the current sample being classed as “inhibited” based on each scale. This suggests that, even though the veteran means are higher than those in the validation sample, the incidence of “uninhibited” vs. “inhibited” temperament (based on either current or childhood behavior) is comparable.

Internal consistency of AMBI/RMBI questionnaires, and correlations between AMBI/RMBI subscale scores, were similar to those reported by Gladstone & Parker (2005); however, that paper also reported gender differences, with females showing higher Risk Avoidance than males on both AMBI (4.0 vs. 3.3) and RMBI (3.1 vs. 2.5). There was no significant gender difference in the current sample, although the means were similar to those observed in the validation study, suggesting that the lack of a significant difference in the current sample may have been due to the low inclusion of females, although there may also be unique characteristics of the female veteran population. It is also worth noting that the internal consistency of the Risk Avoidance subscales was relatively low in both the current sample and in the Gladstone & Parker report, which could contribute to inconsistent findings.

A final note on the questionnaire data is that, although the majority (94 of 126) veterans were classed as non-combat based on CES scoring criteria, the mean PCL-M score for non-combat veterans was still over 30, as shown in Table 1, and 20 non-combat veterans met criteria for current, severe PTSD symptoms (PCL-M score 50+). Clearly, non-combat veterans can and do report experiencing PTSD symptoms related to their military service. Given that even individuals with subthreshold PTSD symptoms are at risk for other medical and psychiatric disorders – including but not limited to subsequent development of full-blown PTSD (Yarvis and Schiess 2008; O'Donnell et al. 2009) – this is an important population for continued study.

Eyeblink Acquisition

Although current BI was most strongly correlated with degree of current PTSD symptoms, childhood BI was significantly correlated with acquisition of eyeblink CRs. This was particularly true in veterans without PTSS; as Figure 2B shows, veterans with PTSS tended to learn quickly regardless of BI. The fact that there was no main effect of PTSS in the current sample may just reflect the relatively small number of PTSS veterans. Still, the absence of a main effect of PTSS on learning in the current study is broadly consistent with several prior studies documenting no effects of PTSD on acquisition of classically-

conditioned CRs (e.g., Ayers et al. 2003; Orr et al. 2006; Vythilingam et al. 2006), although these prior studies considered patients with a clinical diagnosis of PTSD, whereas our study included both combat and non-combat veterans with PTSS assessed by self-report.

The fact that childhood (RMBI), not adult (AMBI), temperament was associated with learning speed suggests that faster learning is associated with a pre-existing character trait, rather than having been acquired later in life through normal aging or via exposure to combat or other stressors, although as mentioned above, we cannot rule out the possibility that life experiences modify an individual's self-report of retrospective measures. There was no increase in UR magnitude as a function of RMBI, suggesting that increased sensitivity to the US is not a sufficient explanation for the relationship between childhood BI and learning. The relationship between high childhood BI and learning is also broadly consistent with animal models of anxiety vulnerability: specifically, rats bred to show high behavioral inhibition, assessed through open field behavior and other behavioral and physiological characteristics, also show facilitated acquisition of conditioned eyeblink responses (Ricart et al. 2011).

There was also faster acquisition of eyeblink CRs in the delay than in the omission group; this is consistent with findings from prior eyeblink conditioning studies (Logan 1951; Massaro and Moore 1967). In general, training curves obtained under omission contingencies often replicate those observed under partial reinforcement schedules (Church, 1964), which is often taken to support the position that eyeblink conditioning is truly classical rather than operant in nature (Coleman 1975). The presence of the main effect of group indicates that high-RMBI individuals do not simply produce more CRs under all conditions, but that they and low-RMBI individuals can both modulate their responding as a function of stimulus contingencies.

Eyeblink Extinction

During extinction, participants in the delay group showed fewer CRs (extinguished faster) than the omission group. This is consistent with the partial reinforcement extinction effect (PREE), an increased resistance to extinction that is observed after training with partial reinforcement, as compared to paradigms where reinforcement is present on every trial during training (for review, see Nation and Woods 1980; Flaherty 1985). Among several theories that have been put forward to explain the PREE is the sequential theory of Capaldi (1966); at its simplest, this theory acknowledges that, during acquisition under conditions of partial reinforcement, subjects learn that trials on which the CS is not paired with the US are often followed by trials where the CS and US are paired; thus, on any given trial, the current CS may be paired with the US, even if it was not so paired on the previous trial. Thus, during the early extinction trials, when the CS is no longer followed by the US, the subject is already "trained" to continue responding to the CS despite a train of CS-noUS trials. By contrast, for subjects given acquisition training under delay contingencies, the first CS-noUS extinction trial is a novel event, which is more likely to cause a disruption in responding.

Given this interpretation of the PREE, it is interesting that, in the current study, although there was no effect of PTSS on extinction following delay conditioning, PTSS veterans did show delayed extinction in the omission group. Specifically, during the early extinction blocks, PTSS veterans in the omission group showed strong responding to the CS (Figure 4B). Similarly, there was extinction resistance in the omission group among veterans with high RMBI (Figure 3B). This suggests that there may be individual differences in PREE, such that certain groups – such as those with childhood BI and/or PTSS may be more resistant to extinction following partial reinforcement. Interestingly, persistent responding to stimuli that no longer signal important outcomes has also been proposed as a mechanism

contributing to learned helplessness and to depression (for review, see Nation and Woods 1980), a condition which is highly co-morbid with PTSD (Foa et al. 2006) as well as with subclinical PTSD (Yarvis and Schiess 2008). This raises the question of whether some features of PTSD and depression might reflect the same underlying associative learning mechanisms. To examine this issue further, it would be interesting to assess acquisition and extinction with omission contingencies in patients with depression as well as with both depression and PTSS.

Extinction was also reduced for non-combat veterans with inhibited temperament based on AMBI, although this difference was significant only during block 1 of extinction (Figure 3B). It is not clear why this effect should appear in non-combat but not combat veterans, nor why this interaction involves AMBI rather than RMBI. One difficulty with using the AMBI/RMBI scales is that two measurements are provided, representing current and retrospective inhibition, and although these two measures are generally correlated (as they were in the current study) this correlation is not perfect; in addition, use of two related scales may reduce power of either to demonstrate a significant effect.

Limitations and Future Directions

There are several limitations of the current study, most notably the reliance on participants' memory for retrospective measures (such as RMBI and even CES). There are also limitations related to the current sample, including a small inclusion of females, which hindered investigation of gender differences, and a range of ages and time since combat exposure. Because we were relying on self-report, we were unable to fully investigate the effects of medication, as some individuals were unable to specify their precise medication name or dosage. In addition, although we used CES to assess history of exposure to combat, participants may of course have been exposed to traumatic events unrelated to military service. An important issue in understanding PTSD in veterans is not only psychopathology which develops directly in response to traumatic events experienced during military service (e.g. combat exposure), but also the degree to which prior exposure to combat and other service-related stressors affects veterans' risk for PTSD if exposed to further traumatic events during subsequent civilian life. Although we did not find significant effects of combat history on acquisition or extinction in the current study, with the exception of a combat-AMBI interaction that was significant for only a single block during extinction, this is likely to be due to the fairly low inclusion of combat veterans in the current sample; studies focusing on combat veterans might more clearly address this issue.

Given the fairly low inclusion of combat veterans in the current sample, there was a high rate of PTSS, including over one-fifth of the 94 non-combat veterans. Indeed, non-combat veterans in the current sample reported an average of almost 1 cluster B symptom and more than one symptom each from clusters C and D (see Table 1). This indicates that non-combat veterans may experience severe stressors unrelated to combat at a higher rate than would normally be expected from the general population. Indeed, there is some evidence that individuals who exhibit subclinical PTSD symptoms, but fall short of diagnostic criteria, are at heightened risk of future exposure to trauma, possibly because of an increase in behaviors (e.g. substance abuse) that magnify risk for trauma and/or a decrease in normal psychological processes for recognizing and responding to threat (Orcutt et al. 2002). In addition to increasing risk for development of clinical PTSD, subclinical PTSD poses its own associated health costs; veterans with subclinical PTSD but without a diagnosis of full-blown PTSD are at increased risk for comorbid diseases such as depression, alcohol abuse, poor health, and disability following physical injury (Yarvis and Schiess 2008; O'Donnell et al. 2009). For this reason, although many studies of PTSD in veterans have considered only combat veterans dichotomized as PTSD or non-PTSD based on clinical diagnosis, it is also

very important to study both combat and non-combat veterans and to consider a continuum of PTSD symptom severity, including subclinical as well as clinical PTSD cases.

Despite these limitations, the current study demonstrates that current behavioral inhibition, assessed by self-report using the AMBI, correlates with current, severe PTSD symptoms in veterans; however, AMBI scores alone did not predict PTSS as well as a model that included both AMBI and combat history as predictive variables, reflecting the fact that development of PTSD symptoms depends on exposure to stressors as well as pre-existing vulnerability. On the other hand, retrospective (RMBI) rather than current (AMBI) behavioral inhibition was associated with faster eyeblink conditioning. To the extent that RMBI indexes childhood BI that pre-dates combat exposure in veterans, this finding is consistent with the idea that a bias for faster associative learning may play a role in establishing risk for PTSD, and suggests that the facilitated conditioning sometimes observed in patients with clinically-diagnosed PTSD may be related to pre-existing vulnerability for PTSD, rather than emerging selectively as a consequence of exposure to trauma or development of PTSD symptoms *per se*.

However, although inhibited temperament is a risk factor for PTSD, it is neither necessary nor sufficient for PTSD. In the current sample, there are individuals classified as uninhibited on both AMBI and RMBI who nevertheless score above the cutoff for PTSS, as well as individuals with high AMBI/RMBI scores but without PTSS. The fact that PTSS alone did not significantly affect acquisition in the current sample suggests that behavioral inhibition is strongly linked to associative learning, whether or not an individual is subsequently exposed to trauma (e.g. combat) and/or develops PTSS. Thus, inhibited temperament and faster conditioning may be pre-existing factors that provide one (but certainly not the only) pathway to risk for PTSD. On the other hand, the current finding of delayed extinction in PTSS veterans following acquisition under omission contingencies is consistent with the idea that extinction resistance may emerge as an acquired sign following exposure to trauma and/or development of PTSD symptoms; however, milder but significant extinction resistance also appeared in behaviorally-inhibited, non-combat veterans in the current study, suggesting that – at least under some conditions – extinction resistance may reflect risk factors for PTSD, as well as with presence of current, severe PTSD symptoms.

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Figure 1.

(A) PCL-M scores are significantly higher in veterans with a history of exposure to combat ($F(1,117)=36.41$, $p<0.001$). (B) Veterans classed as inhibited (based on either AMBI or RMBI scores) score higher on PCL-M than veterans classed as uninhibited (Univariate ANOVAs, all $F>8.00$, all $p<0.010$). Asterisks indicate significant differences ($p<0.050$). PCL-M=PTSD Checklist - Military version; AMBI/RMBI=Adult/Retropective Measure of Behavioural Inhibition.

Figure 2.

Eyeblick acquisition data. (A) Veterans in the delay group showed more eyeblink conditioned responses (CRs) than in the omission group (repeated-measures ANOVA, $F(1,74)=7.74$, $p=0.007$). (B) There was also a block $\times$ RMBI $\times$ PTSS interaction, such that, among individuals without severe, current PTSD symptoms (noPTSS), those with childhood behavioral inhibition (Inhib) made more CRs than those with an uninhibited temperament (Uninhib, $F(1,56)=4.55$, $p=0.037$).

Figure 3.

Eyeblick extinction data. (A) Given their higher level of responding during acquisition, the delay group showed significantly faster extinction than the omission group (repeated-measures ANOVA, $F(1,62)=6.29$, $p=0.004$). (B) Within the omission group, veterans with uninhibited childhood temperament showed fewer conditioned responses (CRs) than those with inhibited childhood temperament (independent-samples t-test, $t(42)=2.14$, $p=0.039$). (C) Among non-combat veterans, those with inhibited current temperament showed more CRs during the first extinction block than those with uninhibited current temperament (independent-samples t-test, $t(22)=2.11$, $p=0.047$). N is shown at base of each bar in (B) and (C). Asterisks indicate significant difference ($p<0.050$).

Figure 4.

Eyeblick extinction data. (A) There was no difference in acquisition or extinction as a function of PTSS in the delay group (all $p > 0.050$). (B) In the omission group, veterans with PTSS showed more eyeblink conditioned responses (CRs) than those without PTSS during extinction blocks 1 and 2 (independent-samples t -tests, all $t > 2.00$, all $p < 0.050$) but not during blocks 3-5 (all $t < 1.5$, all $p > 0.100$). PTSS=Severe, current PTSD symptoms. Asterisks indicate significant differences ($p < 0.050$).

Table 1

Mean (SD) PCL-M total scores and scores for PTSD cluster B, C, D symptoms in individuals with inhibited vs. uninhibited temperament based on AMBI and RMBI, and with vs. without combat history. Asterisks indicate significant differences (ANOVA with factors of combat history and AMBI/RMBI, with alpha corrected to 0.004 to protect against increased family-wise error) between inhibited/uninhibited and combat/non-combat veterans ($F > 4.00$, $p < 0.004$). PCL-M=PTSD Checklist-Military version, AMBI/RMBI=Adult/Retrospective Measure of Behavioural Inhibition.

	Combat History (n=126)		AMBI (n=126)		RMBI (n=123)	
	Combat (n=32)	Non-combat (n=94)	Inhibited (n=69)	Uninhibited (n=57)	Inhibited (n=62)	Uninhibited (n=61)
PCL-M (total)	53.50* (17.91)	32.97 (16.00)	43.30* (19.56)	31.98 (15.70)	42.13 (18.99)	33.97 (17.18)
Cluster B Symptoms	3.09* (2.02)	0.97 (1.68)	1.97* (2.13)	0.95 (1.67)	1.87 (2.17)	1.13 (1.72)
Cluster C Symptoms	4.41* (2.24)	1.74 (2.32)	3.01* (2.65)	1.70 (2.30)	2.98 (2.71)	1.80 (2.26)
Cluster D Symptoms	3.19* (1.93)	1.76 (1.77)	2.75* (1.81)	1.35 (1.75)	2.63 (1.90)	1.62 (1.76)

Table 2

Mean (SD) AMBI and RMBI total scores and subscale scores for the complete set of 126 veterans and for the subset of 87 veterans who produced useable eyeblink conditioning data. Note that RMBI scores were not available for 3 veterans in the larger sample, due to endorsement of “do not remember” responses for more than 50% of RMBI items; RMBI scores were available for all veterans in the eyeblink sample. AMBI/RMBI=Adult/Retrospective Measure of Behavioural Inhibition.

	Total sample (n=126)	Eyeblink sample (n=87)
RMBI total score	12.7 (6.6)	12.6 (6.7)
Non-Approach (NA)	4.5 (2.9)	4.5 (2.9)
Fearful Inhibition (FI)	2.5 (2.3)	2.5 (2.3)
Risk Avoidance (RA)	2.9 (1.4)	2.7 (1.4)
Shyness & Sensitivity (SS)	2.8 (2.0)	2.9 (2.2)
AMBI total score	17.0 (6.1)	17.2 (6.4)
Non-Approach (NA)	3.2 (1.5)	3.2 (1.6)
Fearful Inhibition (FI)	7.2 (3.4)	7.3 (3.5)
Risk Avoidance (RA)	3.5 (1.4)	3.5 (1.4)
Low Sociability (LS)	3.2 (1.6)	3.3 (1.6)