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Skin Conductance Fear Conditioning Impairments and Aggression: A Longitudinal Study

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Abstract

Autonomic fear conditioning deficits have been linked to child aggression and adult criminal behavior. However, it is unknown if fear conditioning deficits are specific to certain subtypes of aggression, and longitudinal research is rare. In the current study, reactive and proactive aggression were assessed in a sample of males and females when aged 10, 12, 15, and 18 years old. Skin conductance fear conditioning data were collected when they were 18 years old. Individuals who were persistently high on proactive aggression measures had significantly poorer conditioned responses at 18 years old when compared to others. This association was not found for reactive aggression. Consistent with prior literature, findings suggest that persistent antisocial individuals have unique neurobiological characteristics and that poor autonomic fear conditioning is associated with the presence of increased instrumental aggressive behavior.

Keywords

Electrodermal; fear conditioning; aggression; longitudinal

Introduction

Antisocial Behavior and Fear Conditioning

Adolescent aggression constitutes a problem of significant clinical and social concern. Although aggression does not represent a diagnosis in its own right, it occurs in numerous psychopathological disorders, including oppositional defiant disorder, conduct disorder, antisocial personality disorder, borderline personality disorder, and intermittent explosive disorder (van Goozen, Fairchild, Snoek, & Harold, 2007; Vitiello & Stoff, 1997). Thus

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research focusing on aggression may provide a useful way to increase insight into the underlying psychopathological processes involved in these disorders. Increasing evidence suggests that neurobiological mechanisms are important in explaining individual differences in antisocial, aggressive behavior (Patrick, 2008; Raine, 2002).

One of the key concepts in theories of aggressive/antisocial behavior is reduced classical fear conditioning. Fear conditioning is a form of Pavlovian conditioning through which individuals learn the significance of previously neutral stimuli through a process of association. In a typical classical conditioning paradigm, a neutral, non-aversive tone (CS) is presented to the subject, followed a few seconds later by either a loud tone or an electric shock (UCS). The key measure is the size of the skin conductance response elicited by the CS after a number of CS-UCS pairings. The larger the response to the CS after pairing with the UCS, the better the conditioning. Failing to form the CS-UCS association, as measured by an absence of increased responsivity to the reinforced conditioned stimulus (CS+) followed by the UCS, compared to a control stimulus not followed by an aversive event (CS-), may indicate a lack of anxiety or anticipatory fear, which in turn may give rise to antisocial acts in some individuals.

Empirical studies have consistently shown that poor skin conductance fear conditioning is associated with aggressive and antisocial behavior in children and adult populations (Birbaumer et al., 2005; Fairchild, Stobbe, van Goozen, Calder, & Goodyer, 2010; Fairchild, van Goozen, Stollery, & Goodyer, 2008; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Rothmund et al., 2012). For example, one longitudinal study has revealed that poor skin conductance conditioning at age 3 years is linked to more aggressive behavior at age 8 years and also predisposes individuals to criminal behavior at age 23 years (Gao, Raine, Venables, Dawson, & Mednick, 2010a, 2010b). Additionally, increased conditioned responses, as well as high autonomic arousal and orienting, distinguished adolescents who desisted from crime by age 29 from those that did not (Raine, Venables, & Williams, 1995; Raine, Venables, & Williams, 1996), suggesting a protective role of these mechanisms against antisociality.

Given that the amygdala is critically involved in fear conditioning (Büchel, Morris, Dolan, & Friston, 1998), poor fear conditioning has been considered as a proxy for amygdala dysfunction, especially in studies on young children when brain imaging data are lacking (Fairchild et al., 2008; Gao et al., 2010a). Other brain circuits, including the insula, the anterior cingulate, and the orbitofrontal cortex, have also been found to be involved in fear conditioning (Büchel et al., 1998). Brain imaging studies on adult psychopaths and children and adolescents with callous-unemotional traits are beginning to document the structural or functional deficits in these regions involved in fear conditioning (Blair, 2007; Jones, Laurens, Herba, Barker, & Viding, 2009; Koenigs, 2012; Marsh et al., 2008; Yang, Raine, Colletti, Toga, & Narr, 2010). In sum, poor conditioning may play a significant role in the development of aggressive and antisocial behavior.

Persistent Aggressive Individuals

It has been proposed that aggressive behavior can arise for many reasons, and may be transiently present in an individual. Longitudinal research that identifies a persistent pattern

of aggression across time is a better measure of the aggressive phenotype most likely associated with underlying physiological differences (Raine et al., 2005). However, very few studies have investigated whether persistent antisocial individuals in particular evidence autonomic conditioning abnormalities. To the authors' knowledge, only one study has examined autonomic fear conditioning in persistent vs. transitional antisocial individuals. In a study on 14 to 18 year olds, Fairchild et al. (2008) examined skin conductance conditioning in 54 healthy controls, 43 adolescents with early-onset/persistent conduct disorder (CD) and 28 with adolescence-onset/transitional CD. Both CD groups showed conditioning deficits compared to the control group, and no differences were found between the two subtypes of CD groups. However, a few limitations of that study should be noted. First, participants were classified into CD sub groups based on their retrospective accounts of behavioral problems before age 10. This may be problematic since some participants might have been misclassified. Second, only male participants were included in that study. Based on the prior literature, it is hypothesized that a stronger relationship between fear conditioning deficits and aggression would be found in the persistent aggressive individuals in particular.

Reactive vs. Proactive Aggression

Recently, attention to the distinction between two forms of aggression, reactive and proactive, has increased (Hubbard, McAuliffe, Morrow, & Romano, 2010). Reactive aggression has been characterized as impulsive, hostile, affective, and an uncontrolled angry response to frustration or provocation (Crick & Dodge, 1996), while proactive aggression has been characterized as predatory, controlled, and instrumental in that it is used to obtain rewards beyond harming the victim (Crick & Dodge, 1996; Vitiello & Stoff, 1997). It has been suggested that a general propensity towards impulse control problems contributes to reactive aggression whereas a separate propensity involving deficient empathy and stimulation-seeking tendencies appears to contribute independently to proactive aggression (Patrick, 2008).

Although studies have suggested that these two forms of aggression differ in their phenomenology and neurobiological features (Lopez-Duran, Olson, Hajal, Felt, & Vazquez, 2009; Raine et al., 1998; van Goozen et al., 2007; Vitiello & Stoff, 1997), research linking different subtypes of aggression with psychophysiological correlates has been rare. In one study of school-aged children, those with reactive aggression showed increased heart rate reactivity during a provocation when compared to those with a combination of reactive and proactive aggression (Pitts, 1997). In another study of second graders, reactive aggression was positively related to skin conductance reactivity to a frustration challenge, although no heart rate effect was found (Hubbard et al., 2002). In a pilot study of 36 fourth- and fifth-graders, Hubbard et al. (2010) found that increased skin conductance and heart rate were linked to reactive aggression when provoked, whereas reduced skin conductance and heart rate were associated with high proactive aggression. It has been proposed that reactive aggression is associated with heightened emotional and elevated autonomic arousal (Scarpa & Raine, 1997, 2000), reflecting an autonomic stress response and negative emotionality to which children react with aggressive behavior. In contrast, proactive aggression has been proposed to be linked to hypoarousal (Scarpa & Raine, 1997, 2000). It is believed that

because some antisocial children are hypoaroused, they seek stimulating and risky situations to bring their arousal back to optimal levels (Eysenck, 1997; Raine, Reynolds, Venables, & Mednick, 1997). Alternatively, low arousal may indicate a fearlessness temperament which impairs one's ability to learn from punishment (Scarpa & Raine, 1997). In summary, evidence suggests that reactive aggression is characterized by autonomic hyperarousal, and that conditioning deficits may be specific to proactive aggression, a form of aggression linked to psychopathy (Raine et al., 2006).

The Present Study

In summary, there is considerable heterogeneity within the construct of aggression and few studies have integrated research on the different etiological trajectories to offending, analysis of autonomic responses, and subtypes of aggression. This study aims to address these gaps by investigating whether reactive and proactive aggression are differentially associated with autonomic fear conditioning, and whether this psychophysiological abnormality applies to persistent aggressive individuals compared to individuals whose antisocial behavior is transitional, using a prospective longitudinal design with a community-based sample. Based on prior literature, it was hypothesized that: 1) those with persistently high proactive aggression scores will show impairment in conditioning relative to those without persistent proactive aggression scores; and 2) the association will be specific for the proactive type of aggression; that is, no significant relationship shall be found between fear conditioning and reactive aggression.

Method

Participants

The data in this study come from the University of Southern California (USC) Twin Study of Risk Factors for Antisocial Behavior (RFAB) and the sample was selected on the basis of being twins. The RFAB is a longitudinal study of the interplay of genetic, environmental, social, and biological factors on the development of antisocial and aggressive behavior from childhood to young adulthood. Participating families were recruited from the Los Angeles community and the sample is representative of the ethnic and socio-economic diversity of the greater Los Angeles area. RFAB is in its fifth wave of data collection and to date four waves of data have been analyzed. The total sample contains 1,569 subjects (780 twins and triplets). On the first assessment (Wave 1) the twins were 9-10 years old ($N = 614$, $mean\ age = 9.59$, $SD = 0.58$). On the second assessment (Wave 2), the twins were 11-13 years old ($N = 445$, $mean\ age = 11.79$, $SD = 0.92$). On the third assessment (Wave 3), the twins were 14-15 years old ($N = 604$, $mean\ age = 14.82$, $SD = 0.83$), and during Wave 4 the twins were 16-18 years old ($N = 504$, $mean\ age = 17.22$, $SD = 1.23$). Informed consent and assent were obtained from all participants. More details on the protocol procedures and zygosity determination can be found elsewhere (Baker, Barton, Lozano, Raine, & Fowler, 2006; Baker, Barton, & Raine, 2002; Baker et al., 2013). Complete data on one or more waves of aggression and wave 4 conditioning were available for 329 participants.

Measures

To measure aggressive behavior in the twins, we used the Reactive and Proactive aggression Questionnaire (RPQ) completed by the twins' caregivers (parent ratings on the twins). Caregiver participation was primarily (> 92%) the biological mothers. The RPQ is a validated 23-item questionnaire designed to measure reactive and proactive aggression in children and adolescents from the age of eight (Raine et al., 2006). The RPQ includes 11 reactive items (e.g., "He/she damages things when he/she is mad"; "He/she gets mad or hit others when they tease him/her") and 12 proactive items (e.g., "He/she threatens and bullies other kids"; "He/she damages or breaks things for fun"). The items in the RPQ have a three-point response format: 0 = never, 1 = sometimes, 2 = often, and scores are summated to assess reactive and proactive aggression. Reactive aggression can range from 0-22 and proactive aggression can range from 0-24. Confirmatory factor analysis using the RPQ in the Pittsburgh Youth Study (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998) has shown an acceptable fit for a two-factor reactive- proactive model that is superior to a one-factor model (Raine et al., 2006). This has also been replicated using the current sample, with a two-factor reactive- proactive model providing a better fit than a one-factor model (Baker, Raine, Liu, & Jacobson, 2008).

Formation of Aggressive Groups

Among the 329 participants who had conditioning data, 21 were missing aggression data on all waves, 2 had only one wave, 58 had two waves, 61 had three waves, and 187 had all four waves of aggression data. Therefore following analyses were conducted among 306 participants who had at least two waves of aggression data. The descriptive statistics for the main study variables are listed in Table 1. As can be seen in the table, the PA measures were highly skewed at each wave. Therefore, a categorical rather than a dimensional approach was first used to analyze the data. Two discrete groups were formed on the basis of whether participants fell into the top 50% cutoffs on two or more waves of Proactive Aggression (PA) measure. Persistently high PA participants ($N = 59$) were defined as those who fell into the top 50% of PA scores at two or more waves, and the other participants were in the comparison group ($N = 247$). Means, SDs, and ranges for the two groups on PA measures are listed in Table 2. High and low PA groups differed significantly on their proactive aggression scores on each wave and on the average of aggression scores across four waves. Groups did not differ on zygosity, race, or sex (Table 2).

Similarly, two groups were formed based on their score on Reactive Aggression (RA) across waves. Persistently high RA participants ($N = 106$) consisted of those who were in the top half of RA scores at two or more waves, and the others were in the comparison group ($N = 200$). See Table 2 for the means, SDs, and ranges for the two groups on all measures. High and low RA groups differed significantly on their reactive aggression scores on each wave and on the average of aggression scores across four waves. Groups did not differ on zygosity, race, or sex (Table 2). Among the 306 participants, 186 were low on both PA and RA, 14 were high on PA but low on RA, 61 were low on PA but high on RA, and 45 were high on both PA and RA.

To assess whether this sample of 306 youth was representative of the initial population ($N = 1,569$), comparisons were made between those contained in the study and the rest of the sample on antisocial and demographic (sex, race, SES) measures. Groups did not differ on sex ($\chi^2(1) < 1$), SES ($t(1196) = -0.624, p = .533$), wave 1 RA ($t(1245) = -0.973, p = .331$), wave 2 RA ($t(872) = -1.482, p = .139$), wave 3 PA and RA (PA, $t(1174) = -0.564, p = .573$; RA, $t(1174) = -0.374, p = .709$), and wave 4 PA and RA (PA, $t(583) = 0.974, p = .330$; RA, $t(583) = 0.805, p = .421$). However, those contained in this study scored slightly lower on wave 1 PA ($t(532.786) = -2.036, p = .042, d = -0.130$) and wave 2 PA ($t(405.961) = -2.295, p = .022, d = -0.175$) than the rest of the original sample. Finally, slightly more Hispanics were included in the current study ($\chi^2(5) = 14.998, p = .01$).

Skin Conductance Recording and Quantification

Skin conductance was recorded with a bioamplifier manufactured by the James Long Company (Caroga Lake, NY), with a low-pass filter set to 10 Hz. Silver-silver chloride electrodes were attached to the first and second fingers using the volar surface of the distal phalanges on the non-dominant hand (Dawson, Schell, & Filion, 2007, figure 7.3). The conducting medium was K-Y lubricating jelly, surrounded by an adhesive electrode collar (1 cm in diameter). The signal was digitized at a sampling rate of 512 Hz, and responses elicited by the stimuli were scored through the James Long Company software. A rise in skin conductance level was judged a response if it occurred within a window of 1 – 4 seconds after stimulus onset. Additionally, the slope in skin conductance level was required to exceed the baseline slope (at stimulus onset) by a minimum of 0.05 $\mu\text{S}/\text{second}$. Skin conductance response amplitude was defined as the peak change in skin conductance occurring within seven seconds of response initiation. If no response was detected, then the magnitude for that particular trial assumed a value of zero rather than being omitted. We decided against confining our analyses to nonzero values (i.e., amplitudes) in order to exploit the full set of observations (Isen, Raine, Baker, Dawson, Bezdjian, & Lozano, 2010; Tuvblad, Gao, Isen, Botwick, Raine, & Baker, 2012).

Fear Conditioning Task

The fear conditioning task consisted of presenting the subject with a neutral tone 8.0 s in duration (the conditioned stimulus, CS+) followed by a picture of an attacking dog and a 0.5 s burst of 105 db white noise (the unconditioned stimulus, UCS), presented through earphones, and a second neutral tone (the CS-) not followed by these negative stimuli. The CS+ and CS- were 800 and 1200 Hz, respectively. Five CS+ and five CS- trials were presented in random order while skin conductance responses were recorded. Orienting responses (ORs) to CS+ and CS- were measured on each trial as the square root of the magnitude of the largest increase in skin conductance beginning within 1-4 s after CS onset (Prokasy & Kumpfer, 1973), and unconditioned responses (UCRs) were measured on each reinforced trial as the square root of the magnitude of the largest response beginning within 1-4 s after UCS onset. For each participant the average square root OR magnitudes to the CS+ and the CS- were computed, and the measure of conditioning was taken as the difference between the average square root response to CS+ and to CS-. Mean values of UCR magnitude were also computed. At the end of conditioning task participants were asked whether they were aware of the type of tone (high pitched or low pitched) that was paired

with the aversive picture of the dog and the loud noise, or whether they felt that the pairing was not systematic, or whether they could not tell which was paired, and how certain they were about their answer. Subjects who selected the correct tone and indicated that they were “fairly certain” of their choice were considered aware of the CS– UCS relationship.

Finally, the initial orienting response (OR) was scored for each participant. This was the average of the skin conductance responses to the first two CSs and indicates general orienting responses before any associative learning occurred.

Statistical Analyses

Several 2 (high vs. low PA group) $\times$ 2 (high vs. low RA group) $\times$ 2 (sex) analyses of variances (ANOVAs) were conducted to examine the skin conductance conditioning, UCR, and OR differences between groups. If group differences between the UCR or OR had been found, further analyses would be conducted to examine if any conditioning differences between aggression level groups might have been due to their differences in responsiveness to aversive stimuli or general orienting differences. Chi-square tests were used to examine group differences on the awareness of the CS-UCS pairing. In addition, to account for the dependence of one twin's scores on his or her co-twin's scores, multilevel modeling using the PROC MIXED routine was also conducted (Singer & Willett, 2003). In addition, a one-way ANOVA was conducted to examine if the 4 groups (high PA/high RA, high PA/low RA, low PA/high RA, and low PA/low RA) differed significantly on conditioning, UCR or OR. Finally, for exploratory purposes, we also counted the number of times, from 0 to 4, that a participant was above the group median on PA or RA, and used these numbers to predict wave 4 conditioning in multiple regression equations. An α level of .05 was used to evaluate the significance of all regression analyses. Effect sizes were reported using Cohen's d (Cohen, 1988) and partial η^2 .

Results

Descriptive Statistics

The correlation coefficients between skin conductance conditioning and UCRs at wave 4, aggression measures at four waves are listed in Table 1. All aggression measures were significantly correlated with each other. Skin conductance conditioned response magnitudes at wave 4 were significantly negatively correlated with waves 1 and 4 proactive aggression ($ps < .05$). Initial ORs were significantly negatively associated with waves 3 and 4 reactive aggression ($ps < .05$).

Skin Conductance Conditioning, UCRs and Aggressive Groups

A 2 (high vs. low PA group) $\times$ 2 (high vs. low RA group) $\times$ 2 (sex) ANOVA revealed that the high PA group had significantly poorer skin conductance conditioning than the low PA group ($F(1, 302) = 4.418, p = .037$, partial $\eta^2 = 0.017$). The high and low RA groups didn't differ on conditioning, $F(1, 302) = 1.172, p = .28$. The interaction effect between RA and PA grouping was not significant, $F(1, 302) = 1.887, p = .17$. No significant effect involving sex was found, $F(1, 302) < 1$. No main effect or interaction effects were found for the UCRs (all F 's < 1) or initial ORs (all F 's $< 2.7, ps > .30$). There were no significant effects

involving sex (all F 's < 1). Finally, groups did not differ on their awareness of the CS-UCS pairing ($\chi^2(1) = 0.182, p = .461$ for proactive aggression groups; $\chi^2(1) = 0.395, p = .678$ for reactive aggression groups). Means and SDs are listed in Table 2 and also see Figure 1 for group comparisons on conditioning. Since no main or interaction effect involving sex was found significant, following analyses were conducted among the overall sample. One-way ANOVA revealed that the four groups differed significantly on their conditioning, $F(1, 302) = 4.119, p = .007, \eta^2 = .039$. Post-hoc comparisons with Bonferroni corrections showed that the high PA/high RA group ($M = -0.028, SD = 0.207$) had significantly lower conditioning than the low PA/low RA group ($M = 0.087, SD = 0.244, d = -0.51$), and the low PA/high RA group ($M = 0.130, SD = 0.238, d = -0.71$). The high PA/low RA group ($M = 0.062, SD = 0.194$) did not differ from any of the other three groups. No group differences were found for UCR or OR ($p > .23$).

Multiple Regressions

The number of times a participant was above the group median on PA across the 4 waves was significantly associated with conditioning at wave 4, $F(1, 304) = 4.923, p = .027, \beta = -0.126, SE = 0.011, t = -2.219$. In contrast, the number of times a participant was above the group median on RA was not associated with conditioning, $F(1, 304) < 1, p = .516, \beta = -0.037, SE = 0.010, t = -0.651$.

Multilevel Modeling

Multilevel modeling controlling for the dependence between twins' scores was conducted for conditioning, UCR magnitude, and initial ORs separately. High PA groups had significantly lower conditioning than the low PA groups ($F(1, 246.12) = 9.639, p = .002, \beta = -0.428$), but no significant PA group effects were found for the UCR magnitude ($F(1, 259.497) = 1.864, p = .173$) or initial ORs ($F(1, 255.082) < 1, p = .554$). High and low RA groups did not differ on conditioning ($F(1, 218.257) = 2.365, p = .125$), the UCR magnitude ($F(1, 248.260) < 1, p = .464$), or initial ORs ($F(1, 227.664) = 1.542, p = .216$).

Discussion

In this longitudinal study, reactive and proactive aggression were assessed in male and female participants repeatedly when they were aged 10, 12, 15, and 18 years old and skin conductance conditioning was recorded when they were 18 years old. We found that individuals with persistent proactive aggressive behavior had reduced fear conditioning, but this was not seen in individuals with persistent reactive aggression. These findings are the first to be conducted using a prospective longitudinal design in both male and female participants to detect main effects of group on autonomic conditioning.

Participants from the high and low proactive aggression groups demonstrated equally large skin conductance responses to the unconditioned stimulus and to the orienting stimulus, suggesting a selective deficit in emotional learning. They were also equally likely to be aware of the CS-US contingency (see Table 2), indicating that the conditioning deficits were not due to cognitive learning impairments. It is proposed that individuals who have poor conditioning are less likely to avoid situations, contexts, and events that are linked to future

punishment (Raine et al., 1996). Our findings suggest that this emotional learning impairment is associated with a more severe instrumental and predatory type of aggressive behavior.

As mentioned above, brain imaging studies on adult psychopaths and children and adolescents with callous-unemotional traits have documented the structural or functional deficits in brain regions involved in fear conditioning (Blair, 2007; Jones et al., 2009; Koenigs, 2012; Marsh et al., 2008; Yang et al., 2010). Although imaging studies specifically designed to contrast a group of persistent antisocial individuals with a group who are not are still lacking, the present findings are broadly consistent with a neurodevelopmental perspective on antisocial behavior (Raine, 2008), and suggest that neural substrates of fear conditioning, including the amygdala, are impaired in persistent antisocial individuals.

Consistent with our hypothesis, high and low RA groups did not differ on conditioning. This finding provides further evidence that not all antisocial individuals have conditioning deficits (Scarpa & Raine, 1997, 2000). Indeed, conditioning deficits were found to be associated with persistent proactive aggression in particular, although the effect size for the significant difference in conditioning between the high and low PA groups were not large ($d = 0.47$). This lack of fear and inability to learn from punishment is associated with delayed development of conscience and risky decision making, which in turn relates to more predatory and instrumental aggressive behavior. In contrast, autonomic hyperarousal and negative emotionality are believed to be associated with impulsive and hostile type of aggression (Scarpa & Raine, 1997, 2000). These different patterns seem to be in line with twin research demonstrating that varying genetic and environmental etiologies underlie reactive and proactive aggression (Baker et al., 2008). Specifically, the stability in reactive aggression from ages 10 to 13 years was due to genetic and non-shared environmental influences, whereas the continuity in proactive aggression was primarily genetically mediated (Tuvblad, Raine, Zheng, & Baker, 2009). In addition, brain imaging studies have suggested that different brain mechanisms are involved in these two types of aggression (Raine et al., 1998). Taken together, findings support a distinction between youth reactive and proactive aggression, and suggest that the pathway to each type of aggression may be unique.

One limitation of the study is that only questionnaire-based measures of aggression through parent report were used. Furthermore, relatively little is known about the extent to which responses to the fear conditioning task may be generalizable to more naturalistic threatening or punishing situations. However, one recent study found that children older than 11 years are able to generalize their fear learning to situations in which stimuli are perceptually similar to the conditioned danger cue (Glenn, Klein, Lissek, Britton, Pine, & Hajcak, 2012), suggesting that fear conditioning responses may be able to predict behavior in more naturalistic settings, such as at home or school. To the authors' knowledge, no longitudinal studies using observational or laboratory-based methods in naturalistic settings have been conducted, possibly due to the fact that they are time and labor intensive. The second limitation is that we assessed youths from a community setting where the base rate of high-scorers is generally low, although findings are consistent with those of more severely aggressive samples. Third, only 329 out of an initial sample of 1,569 participants had full

data to complete this study. Finally, in the current study fear conditioning was only assessed on wave 4, therefore the hypothesis that reduced fear conditioning, reflecting fearlessness, increased risk taking or deficits in avoidance learning, would place these youth at risk for future aggression cannot be directly tested. Nonetheless, our findings seem to suggest that the conditioning-aggression association was stable and robust. Future studies integrating prospective longitudinal assessment of autonomic conditioning, laboratory-based research methods, and multi-informant and objective measures such as official criminal records in a larger sample are needed to replicate these findings.

In conclusion, this is the first study to demonstrate fear conditioning deficits in individuals with persistent proactive aggression. The deficit is not associated with unresponsiveness to the UCS, low orienting responses, or lack of awareness of the CS-UCS relationship, and is thus not a byproduct of sensory or broad cognitive deficits, but rather reflects a specific deficit in emotional associative learning. These findings help to delineate mechanisms of persistent proactive aggressive behavior, and contribute to our efforts to identify biological markers, such as fear conditioning deficits, that could potentially be further validated as methods for diagnosing the development of the serious predatory subtype of conduct disorder.

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Figure 1.

Skin conductance conditioning for persistently high proactive aggression group and low proactive aggression group, and for persistently high reactive aggression group and low reactive aggression group - illustrating poor fear conditioning in persistently proactively aggressive individuals.

Table 1
Descriptive Statistics For The Main Study Variables, And Correlations Between Skin Conductance Conditioning, Ucrs And Initial Ors At Wave 4, And Aggression Measures At Four Waves

	Proactive Aggression 1 (n = 270)	Proactive Aggression 2 (n = 213)	Proactive Aggression 3 (n = 266)	Proactive Aggression 4 (n = 308)	Reactive Aggression 1 (n = 270)	Reactive Aggression 2 (n = 213)	Reactive Aggression 3 (n = 266)	Reactive Aggression 4 (n = 308)	Conditioning (n = 329)	UCRs (n = 329)	Initial ORs (n = 329)
Proactive Aggression 1	1										
Proactive Aggression 2	.530***	1									
Proactive Aggression 3	.536***	.636***	1								
Proactive Aggression 4	.486***	.424***	.625***	1							
Reactive Aggression 1	.573***	.347***	.332***	.225***	1						
Reactive Aggression 2	.452***	.563***	.400***	.234***	.676***	1					
Reactive Aggression 3	.519***	.477***	.589***	.451***	.626***	.656***	1				
Reactive Aggression 4	.517***	.407***	.544***	.640***	.508***	.557***	.688***	1			
Conditioning	-.136*	-.086	-.108 ^d	-.129*	-.055	-.084	-.016	-.066	1		
UCRs	-.030	-.051	-.043	-.014	-.042	-.053	-.054	-.023	-.003	1	
Initial ORs	-.023	-.075	-.064	-.117 ^d	-.023	-.001	-.128*	-.133*	.023	.120 ^d	
Mean	0.972	0.793	0.920	0.847	7.191	6.241	6.044	5.802	0.077	1.081	0.295
S.D.	1.493	1.794	1.777	1.597	3.799	3.656	3.837	3.608	0.243	0.347	0.352
Median	0	0	0	0	7.0	6.0	6.0	5.75	0.033	1.153	0.204
Skewness	2.16	4.70	4.24	2.63	0.45	0.53	0.83	0.81	0.19	-0.77	1.16
Minimum	0	0	0	0	0	0	0	0	-0.93	0	0
Maximum	10	16	16	10	19	18	21	18	0.79	1.72	1.71

UCRs = Unconditioned Responses; ORs = Orienting Responses.

* Note $p < .05$

*** $p < .001$.

$p < .01$

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Table 2
Descriptive Statistics Of Demographic Variables, Skin Conductance Measures At Wave 4,
And Aggressive Behavior At Each Wave For The Groups

	High Proactive Aggression	Low Proactive Aggression	<i>p</i>	<i>d</i>	High Reactive Aggression	Low Reactive Aggression	<i>p</i>	<i>d</i>
N	59	247			106	200		
Male	N = 26	N = 113	.816 ^a		N = 50	N = 89	.655 ^a	
(Percentage)	(44.1%)	(45.7%)			(47.2%)	(44.5%)		
Zygosity			.843 ^a				.897 ^a	
MZ males	10	54			25	39		
MZ females	19	69			29	59		
DZ males	8	32			12	28		
DZ females	10	34			15	29		
DZ opposite sex	12	58			25	45		
Race			.345 ^b				.113 ^b	
(Percentage)								
Caucasian	18 (30.5%)	67 (27.1%)			27 (25.5%)	58 (29.0%)		
Hispanic	26 (44.1%)	103 (41.7%)			51 (48.1%)	78 (39.0%)		
Black	1 (1.7%)	25 (10.1%)			5 (4.7%)	21 (10.5%)		
Asian	2 (3.4%)	6 (2.4%)			5 (4.7%)	3 (1.5%)		
Mixed	12 (20.3%)	46 (18.6%)			18 (17.0%)	40 (20.0%)		
Awareness	32 (54.2%)	149 (60.3%)	.461 ^a		61 (57.5%)	120 (60.0%)	.678 ^a	
Initial ORs	0.272 (0.332)	0.302 (0.363)	.566	-0.086	0.255 (0.308)	0.318 (0.379)	.143	-0.182
UCRs	1.029 (0.362)	1.102 (0.338)	.143	-0.208	1.078 (0.381)	1.093 (0.323)	.712	-0.042
Conditioning	-0.007 (0.206)	0.098 (0.243)	.003 ^{**}	-0.466	0.063 (0.238)	0.086 (0.241)	.432	-0.096
Wave 1 proactive aggression	2.305 (1.784) [0, 10]	0.590 (1.124) [0, 6]	< .001 ^{***}		1.613 (1.797) [0, 10]	0.544 (1.027) [0, 5]	< .001 ^{***}	
Wave 2 proactive aggression	2.258 (1.784) [0, 16]	0.315 (0.858) [0, 8]	< .001 ^{***}		1.404 (2.457) [0, 16]	0.293 (0.619) [0, 3]	< .001 ^{***}	
Wave 3 proactive aggression	2.579 (2.841) [0, 16]	0.315 (0.846) [0, 5]	< .001 ^{***}		1.485 (2.363) [0, 16]	0.605 (1.018) [0, 6]	< .001 ^{***}	
Wave 4 proactive aggression	2.475 (1.942) [0, 9]	0.441 (1.153) [0, 10]	< .001 ^{***}		1.377 (1.869) [0, 9]	0.545 (1.283) [0, 10]	< .001 ^{***}	
Wave 1 reactive aggression	9.407 (3.677) [3, 19]	6.514 (3.564) [0, 17]	< .001 ^{***}		9.867 (3.030) [3, 19]	5.369 (3.097) [0, 16]	< .001 ^{***}	
Wave 2 reactive aggression	8.402 (3.753) [2, 18]	5.535 (3.344) [0, 16]	< .001 ^{***}		8.791 (3.018) [2, 18]	4.153 (2.683) [0, 16]	< .001 ^{***}	

	High Proactive Aggression	Low Proactive Aggression	<i>p</i>	<i>d</i>	High Reactive Aggression	Low Reactive Aggression	<i>p</i>	<i>d</i>
Wave 3 reactive aggression	8.973 (4.491) [0, 21]	5.313 (3.123) [0, 17]	< .001 ***		8.729 (3.658) [0, 21]	4.578 (2.899) [0, 13]	< .001 ***	
Wave 4 reactive aggression	8.864 (4.006) [0, 18]	5.098 (3.052) [0, 17]	< .001 ***		8.259 (3.239) [1, 18]	4.535 (3.037) [0, 17]	< .001 ***	
Mean proactive aggression	2.457 (1.815) [0.75, 11.33]	0.516 (0.775) [0, 5]		< .001 ***	1.499 (1.721) [0, 11.33]	0.567 (0.860) [0, 5]	< .001 ***	
Mean reactive aggression	9.004 (3.370) [3.25, 19.33]	5.674 (2.679) [0, 14.25]	< .001 ***		8.957 (2.476) [5.75, 19.33]	4.916 (2.433) [0, 13]	< .001 ***	

Note.

UCRs = Unconditioned Responses; ORs = Orienting Responses.

 $p < .001$,

**
 $p < .01$,

*
 $p < .05$,

^a Chi-square comparison,

^b Fisher's Exact Test.