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The effect of trauma on stress reactivity in aggressive youth

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ABSTRACT

To address gaps in the literature related to the contribution of childhood trauma on aggression, we evaluated salivary cortisol and heart rate changes to psychological challenge in aggressive children with various degrees of trauma. We hypothesized that traumatized and aggressive youths will exhibit higher responsiveness to an active challenge (Violent film—VF) than aggressive youth with no trauma but will not differ when viewing a Non-Violent film (NVF). A total of 25 children (aged 9–12; M = 15, F = 9) with history of aggression were assessed for trauma exposure. Children viewed the two films in randomized order. Four salivary cortisol and pulse measurements were obtained before (T1), 15 min after the start (T2), at the end (T3), and 15 min following the end of the movie (T4). Repeated measures Analysis of Covariance (ANCOVA) using Film (VF/NVF), Cortisol/Time at T1–T4, Group (Trauma/Non-Trauma), and Film Order were performed with age and gender as covariates. There were significant main effects for Group and Cortisol/Time for the Trauma group showing greater cortisol responsiveness than the Non-Trauma group that was most pronounced during the NVF. These results suggest that aggressive youth with personal history of trauma may exhibit unique biological characteristics, which may have important implications for classification and treatment.

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1. Introduction

Aggressive behaviors are one of the leading causes for referrals for psychiatric evaluation of young children (Peterson et al., 1996). Ensuing in depth assessments of youth with aggression frequently establishes diagnoses like attention deficit/hyperactivity disorder (ADHD) or conduct disorder (CD); however, aggression is not a core feature of ADHD and is not necessarily present in children with CD. Further, childhood traumatization has also been linked to the development of subsequent aggressive behaviors, but similarly, aggression is not a required symptom of childhood posttraumatic syndrome. These observations point out that the relationship between aggression, disruptive behaviors and childhood trauma is not straightforward.

Aggression has been linked to states of baseline physiological under-arousal (Murray-Close et al., 2008) that may translate psychologically into either aversive emotional experiences (Coren, 1999), or experiences of reduced fear (Raine, 2002a, 2002b). In result, affected individuals appear vulnerable to engage in aggressive behaviors because the latter produce physiological arousal that can in turn alleviate aversive emotions. Alternatively, diminished experience of fear may compromise the role of fearful states as a deterrent to physical fighting and aggression. All of the above seem to negatively affect the functioning of the behavioral inhibition system, which guides individual's reactions to challenging situations (Fox et al., 2005). For instance, several studies have shown that while behavioral inhibition appears associated with increased cortisol (King et al., 1998; Blair et al., 2004), behavioral disinhibition, viewed as a tendency to react with boldness and spontaneity to novel situations (Lopez et al., 2004; Hirshfeld-Becker et al., 2007) has been linked to decreased cortisol during psychological challenge (Blair et al., 2004). Not surprisingly, abnormal levels of cortisol responsiveness have been observed in children with ADHD and CD, who are often characterized as behaviorally disinhibited. Attenuated cortisol responses have been reported in children with ADHD upon awakening (Blomqvist et al., 2007) or when exposed to stress such as performing timed cognitive tests (King et al., 1998; Hong et al., 2003) compared to healthy controls. Low hypothalamic–pituitary–adrenal (HPA) axis activity, however, does not seem specific to ADHD; low basal cortisol levels have been linked to physical aggression in children with CD (McBurnett et al., 2000; Pajer et al., 2001), and oppositional defiant disorder with or without comorbid ADHD (Snoek et al., 2004). Low basal cortisol also seems to predict more aggressive behavior in later adolescence in males regardless of diagnosis (Shoal et al., 2003). Additionally, differences in cortisol reactivity have been repeatedly documented in association with aggression in children with CD compared to nonaggressive children, but the directionality of the findings has varied across studies (van Goozen et al., 2000; van de Wiel et al., 2004; Kempes et al., 2008). A recent meta-analysis for both basal cortisol ($k = 72$ studies, $N = 5480$) and for cortisol reactivity to a

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stressor ($k=29$ studies, $N=2601$) failed to find a relationship between these cortisol measures and externalizing behaviors in adolescents (Alink et al., 2008). Conversely, low resting heart rate seems to be the best-replicated biological correlate of aggression in children and adolescents, which may reflect reduced noradrenergic functioning and a fearless, stimulation-seeking temperament (Raine, 2002a, 2002b; Ortiz and Raine, 2004; Lorber, 2004). However, some argue that heart rate variability is different for different aggression types so that disinhibited or reactive aggression appears linked to decreased heart rate variability whereas premeditated aggression seems associated with increased heart rate variability (Scarpa et al., 2010).

Of particular interest to the topic of this paper is the relationship between childhood traumatization and the consequential development of aggression, which has been documented in youth who either experienced (Connor et al., 2003; Hazen et al., 2006) or witnessed abuse (Hazen et al., 2006; Chemtob et al., 2008). These trauma-related consequences may further persist over the course of development, supported by the finding that physically abused children are significantly more likely to be arrested for both non-violent and violent offenses as adolescents (Lansford et al., 2007). Also of interest is the observation that irritability in adult trauma survivors, which is similar to "reactive aggression" in children, is one of the diagnostic criteria of posttraumatic stress disorder (PTSD). However, as important as it is to investigate the interaction between psychosocial and biological processes in the development and maintenance of aggression (Raine et al., 1997; Raine, 2002a, 2002b) the purported biological pathways leading from childhood trauma to aggression are complex and remain poorly understood. Trauma in children alone may result in heightened basal cortisol levels and cortisol reactivity (Carlson and Earls, 1997; Cicchetti and Rogosch, 2001a; Gunnar et al., 2001, 2009). These effects of traumatization on the relation between aggression and HPA system have been linked to more pronounced dysregulation of cortisol among aggressive children with history of trauma (Cicchetti and Rogosch, 2001a, 2001b). This thesis is further supported by findings that the combination of heightened cortisol reactivity to provocation and experiences of victimization (physical abuse and community violence exposure) is associated with high levels of aggression (Raine, 2002a, 2002b; Scarpa and Ollendick, 2003) and that adverse parenting, family conflicts, and acute life events may contribute to an increased cortisol responsiveness in children with ADHD and externalizing problems (Freitag et al., 2009). One study that specifically examined the association between relational and physical aggression and maltreatment reported results suggesting that physiological correlates of aggression may be different for these two forms of aggression and may also differ among maltreated vs. nonmaltreated youths (Murray-Close et al., 2008). Less is known about the link between traumatization, aggression and heart rate changes in children outside of one study suggesting that increased heart rate seems to predict later development of PTSD in children exposed to traumatic injury (Kassam-Adams et al., 2005). Taken together, these reports clearly show that the relationship between traumatization, aggression, and cortisol and heart rate responsiveness is not one-dimensional and requires further investigations. While it appears that physiological under-arousal is more consistently associated with childhood aggression in particular, the effects of childhood traumatization on the association between aggression and biological correlates such as cortisol and heart rate responsiveness remain controversial.

This interplay between traumatic exposure and disruptive behaviors poses the question as to whether aggressive behaviors in childhood that are underpinned by trauma may represent a biologically distinct subgroup. Given the fact that traumatic stress has a strong influence on both the HPA and autonomic nervous system, illustrated by altered responsiveness of cortisol and heart rate (Gunnar and Quevedo, 2008; Chrousos, 2009), it is reasonable to examine if particular changes in these biological measures may differentiate aggressive youths with prior trauma exposure from aggressive counterparts with no trauma. Toward this purpose we designed an experimental paradigm that followed well established methodology for the assessment of physiological responsiveness to *active* and *neutral* stimuli indexed by the changes in salivary cortisol secretion and heart rate. The primary goal of this study was to collect preliminary data on measures of HPA and autonomic nervous system responsiveness to an *active* (Violent film) and *neutral* (Non-Violent film) psychological challenge in aggressive children with various degrees of trauma (i.e. *Trauma* vs. *Non-Trauma* youths). This protocol tested the hypothesis that traumatized and aggressive youths will exhibit higher levels of responsiveness to the active psychological challenge (Violent film) than aggressive youth with no trauma whereas the responsiveness to the neutral psychological challenge (Non-Violent film) will not differ between the two groups. We did not examine a group of nonaggressive children as we wished to focus on subtypes within a specific clinical group of young children (i.e. aggressive youth with and without trauma).

2. Methods

2.1. Participants

Subjects were recruited through fliers posted at the Mount Sinai Child and Adolescent Psychiatry Outpatient Clinic and by word of mouth. Study procedures were approved by the Mount Sinai Institutional Review Board. The legal guardian for each child gave written informed consent and the children provided written assent to a third party unrelated to the study. Each child received a $50 gift check for completion of the study protocol.

We identified 30 children ages 8–12, who have exhibited patterns of aggressive behavior, indicated by school suspension due to engaging in physical fighting. Of these 25 children completed the experimental protocol and provided data included in the analyses. All subjects had clinical evaluations, which were not apart of the research protocol and consisted of two to three visits during which the child and at least one parent/caretaker were interviewed by a psychiatrist, who also collected collateral information from the school. The reports from these evaluations were available for review. Since the recruitment for the study was based on the presence of aggression and trauma exposure and *not* on the presence of a particular diagnosis no specific diagnostic instruments (e.g. Kiddie-SADS) were used as part of the protocol.

Subjects' personal history of exposure to traumatic stress varied from no trauma to moderate and severe trauma (e.g. domestic violence, sexual abuse, loss of a biological parent). All subjects had been clinically diagnosed with ADHD, for which they continue to receive pharmacological treatment (e.g. stimulants $n=23$, atomoxetine $n=2$) at the time of the study. However, ADHD was not an inclusion criterion for the study. Children who had clinical diagnoses of major depressive disorder, psychotic disorder, bipolar disorder, pervasive developmental disorder and precocious puberty, who were obese or were receiving antipsychotic treatment for aggression were excluded.

2.2. Procedure

The subjects' personal history was obtained through a psychiatric interview of the child and the primary caretaker (mother $n=22$, father $n=1$, grandmother $n=2$). In order to further assess and quantify children's aggressive behaviors the caretakers completed the Child Behavior Checklist (CBCL) (Achenbach, 1991), which provides scores on eight behavioral domains, including aggression. ADHD severity was assessed by parent ratings on the Conners' ADHD Rating Scale (Conners, 1997), which provides age adjusted scores for hyperactivity and inattention. To assess traumatic stress exposure, the caretaker filled out: i) the Codington Life Events Scale (Coddington, 1972) for the purpose of inquiring about the occurrence of traumatic experiences within the last 12 months; and ii) the Posttraumatic Stress Reaction Index (PTSRI) Parent version (Rodriguez et al., 1998), which queries the parent about the occurrence of traumatic experiences in the child and the nature of the reactions to them, following the DSM criteria for PTSD. Children also completed the PTSRI, Child version. Caretakers and children were not aware of each other's responses on the trauma-related instruments.

Two clinicians performed a full review of the subjects' history in relation to trauma including PTSRI total score, subscale scores for Hyper-vigilance, Avoidance and Arousal, traumatic events reported on the Codington Life Events Scale, nature of the trauma, whether the trauma was reported by both parent and child vs. parent or child alone, and time since exposure. Subjects were placed in dichotomous groups [e.g., *Trauma* ($n=10$) or *Non-Trauma* ($n=15$)]. Subjects were assigned to the Trauma group if the following conditions were met: 1) there had to be an agreement between the child and the parent about the nature of the trauma experienced by the child as reported on the PTSRI, and 2) at least one of the PTSRI reports had to provide a score of 30 or above. The nature of the trauma for the children in the Trauma group was determined to be mainly related to personal loss (death of a parent) or physical/sexual abuse. Although these