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Child Safety Signal Learning in the Context of Parental Worry, Overprotection,
PTSD, and Depression

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Abstract

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Individuals with anxiety-related psychopathology often have impairments in safety signal learning, which is thought to be a biomarker of anxiety-related disorders, and which manifests in difficulty inhibiting fear to safety cues (e.g., Jovanovic et al. 2010). Accordingly, one way in which parental anxiety-related psychopathology may affect a child's development is by changing the nature of pivotal, early parent-child learning experiences related to identifying what is safe and what is not. This study used the well-established conditional discrimination (AX+/BX-) paradigm to explore safety signal learning in 8-11 year-old children in relation to parental worry, overprotection, PTSD, and depression. This paradigm assesses fear-potentiated startle (FPS) to conditioned stimuli (CS) that are either paired with an aversive air puff to the throat (AX+), or

that are never paired with the air puff (BX-). FPS to the threat cue (A) presented in conjunction with the safety cue (B) in the absence of the air puff (AB) serves as the critical test of safety signal learning. Dependent variables were FPS to the AX+ and AB trials, as well as retrospective expectancy ratings of whether the air puff had been paired with each CS. Children whose parents were higher in overprotective parenting had higher FPS to the safety transfer cue (AB) than to the safety cue, suggesting impaired safety signal learning. Additionally, higher parental depression was associated with lower initial child FPS to both the danger and safety cues, suggesting a blunting effect of parental depression on child fear responding. By contrast, children whose parents reported higher symptoms of worry did not differ from children whose parents reported lower symptoms of worry in FPS to the danger, safety, or safety transfer cues. Parental PTSD was also not strongly associated with child fear responding or inhibition. Further, children whose parents were lower and higher in worry or overprotective parenting did not differ in cognitively discriminating among the danger, safety, and safety transfer cues. This study provides valuable information about fear inhibition during the middle childhood years, suggesting that impairments in safety signal learning are specifically related to overprotective parenting. Overprotective parenting may thus be a viable target for the prevention and treatment of anxiety-related disorders in children.

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DEDICATION

This dissertation is dedicated to my daughter, Ayelet, who fills my life with so much love and joy and who gives me countless opportunities to make use of my behaviorist training.

Ayelet, the hardest thing by far about writing this dissertation was all the extra time that I had to spend away from you in order to write it. I hope that you will read this some day though and that it will inspire you to reach high and strive for whatever it is that makes you happy, no matter how challenging the journey may be. I love you more than words can say and am so lucky to be your ima.

INTRODUCTION

Fear is an evolutionarily programmed emotion with a clearly advantageous function in situations that threaten survival (Beckers, Krypotos, Boddez, Effting, & Kindt, 2013). The same emotion becomes maladaptive, however, when present in situations that are unlikely to be dangerous (Lissek, Pine, & Grillon, 2006). Accordingly, a growing body of evidence suggests that elevated fear responding in the context of cues that signal safety may be a potential mechanism contributing to the onset and maintenance of anxiety-related psychopathology, such as generalized anxiety disorder (GAD) and posttraumatic stress disorder (PTSD). Indeed, safety signals, defined as cues that predict the absence of threat, are typically reliably detected (Jovanovic et al., 2005). However, deficient safety signal learning may interfere with effective functioning and corrective learning processes that allow for the formation of non-fearful associations (Craske, Kircanski, Zelikowsky, Mystkowski, Chowdhury, & Baker 2008), thus paving the way for the emergence of debilitating and pervasive anxiety and worry.

Safety signal learning impairments also likely exacerbate the symptoms of anxiety-related psychopathology, making the world seem more threatening than it actually is. For example, an individual with PTSD may respond fearfully to the sound of a starter pistol at a track meet, failing to utilize the many cues around that assure safety. Indeed, impairments in fear inhibition have been found across the age span in individuals with high levels of trait anxiety (Haddad, Pritchett, Lissek, & Lau, 2012; Gazendam, Kamphuis, & Kindt, 2013; Haaker et al. 2015), anxiety disorders (Reeb-Sutherland et al., 2009; Waters, Henry, & Neumann, 2009) including GAD (Lissek et al., 2014) and panic disorder (Lissek et al., 2010), and PTSD (Grillon & Morgan, 1999; Jovanovic et al., 2009; 2010). This inability to appropriately inhibit fear in the presence of non-threatening stimuli may be the result of exaggerated amygdala activity coupled

with weakened inhibitory prefrontal control of the amygdala, both of which are consistently highlighted in neurobiological models of pathological anxiety and worry (e.g., Martin, Ressler, Binder, & Nemeroff, 2009) and PTSD (e.g., Jovanovic & Ressler, 2010). Reduced safety signal learning may also partially be explained by alterations in genetic polymorphisms that have been found to be associated with impaired fear inhibition (Wendt et al., 2015). It may also be that impairments in fear inhibition reflect the overgeneralization of conditioned fear from threatening to non-threatening stimuli (Lissek et al., 2010; 2012), possibly resulting from the catastrophic “what if’s” of worry that are often present in anxiety-related psychopathology (Lissek et al., 2014). Regardless of the mechanism, the findings of a recent meta-analysis of elevated fear responding to conditioned safety, but not danger, cues in individuals with anxiety disorders, including PTSD (Duits et al., 2015), highlight the centrality of impaired fear inhibition in anxiety-related psychopathology.

Such impairments in fear inhibition can be assessed in the laboratory using a number of different tasks, including the conditional discrimination (AX+/BX-) paradigm (e.g., Myers & Davis, 2004; Jovanovic et al., 2009). This paradigm assesses conditioned fear responses elicited when a startle probe is presented in conjunction with one of two compound stimuli; either the conditioned exciter, AX+, or the conditioned inhibitor, BX-. Whereas A in compound with X predicts an aversive air puff directed to the larynx (i.e., the unconditioned stimulus; US), B in compound with X signals the absence of the US. After repeated AX+ and BX- trials, a startle probe is presented in the presence of A (the threat signal) in compound with B (the safety signal). This safety signal transfer trial (AB) occurs in the absence of the US, serving as the critical test of safety signal learning.

Unlike healthy individuals who show less fear responding to AB than to AX+ due to the

association B has with safety, individuals with PTSD (Jovanovic et al., 2010) and those with elevated PTSD symptoms (Jovanovic et al., 2009) do not show this decrease in fear responding, as assessed via fear-potentiated startle (FPS; Blumenthal et al., 2005). In addition, in contrast to healthy controls and individuals who have depression or who are low in PTSD symptom severity, those with PTSD (Jovanovic et al., 2010) and elevated PTSD symptoms (Jovanovic et al., 2009) fail to discriminate between AX+ and BX- trials. Interestingly, highly symptomatic individuals with PTSD do not differ from psychiatrically healthy controls in their trial-by-trial expectancy ratings of whether AX+, BX-, and AB predict the US (Jovanovic et al., 2009). This dissociation between startle reactivity and expectancy ratings suggests that although individuals with PTSD fail to respond appropriately to safety signals physiologically, they are able to consciously differentiate between threatening and non-threatening cues.

Notably, despite the similarities (Post, Feeny, Zoellner, & Connell, 2015; Watson, 2005) and high comorbidity (Kessler, Chiu, Demier, & Walters, 2005) between anxiety-related psychopathology and other distress-related disorders, such as depression, inappropriate fear responding to safety signals appears to be unique to anxiety-related psychopathology. In fact, studies consistently show a blunting effect of depression on fear responding to threat in anxious individuals (Lang & McTeague, 2009; Melzig, Weike, Zimmermann, & Hamm, 2007). Similarly, those with stand-alone depression (Jovanovic et al., 2010) and depressive symptoms (Dibbets, Broek, & Evers, 2014) do not differ from psychiatrically healthy controls in their responding to threat or safety cues. Although Jovanovic and colleagues also found that individuals with comorbid PTSD and depression show greater fear responding to safety signals compared to those with depression or without a psychiatric diagnosis (Jovanovic et al., 2010), these findings are likely the result of elevated PTSD symptom severity in the co-occurring PTSD

and MDD group (Jovanovic, Kazama, Bachevaliar, & Davis, 2012). Indeed, anxiety-related symptom severity aside, depression is generally thought to activate compensatory, defensive mechanisms aimed at reducing the aversiveness of unpleasant circumstances in patients (Grillon & Baas, 2003), thus leading to attenuated fear responding in individuals with anxiety disorders and co-occurring depression. Further, as noted above, fear responding does not appear to differ between those with and without depression alone. Impairments in fear inhibition may therefore be a useful tool for distinguishing anxiety-related psychopathology from depression.

Whether these impairments in fear inhibition are a cause or a consequence of anxiety-related psychopathology is not yet known; but is an important line of inquiry that could potentially inform future preventative efforts. Safety signal learning impairments have been found not only in individuals with elevated levels of anxiety, worry, and PTSD, as noted above, but also in adolescents (Craske et al., 2009) and seven-year-old children (Barker, Reeb-Sutherland, & Fox, 2014) at-risk for developing anxiety-related disorders. Furthermore, evidence suggests that decreased inhibition of fear may predict the onset of symptoms of both anxiety (Buss, Davis, Kiel, Brooker, Beekman, & Early, 2013; Craske et al., 2012; Jovanovic et al., 2014; Reeb-Sutherland et al., 2009) and PTSD (Pole et al., 2009). Together, these findings lend support to the hypothesis that deficient fear inhibition may be implicated in the development of persistent anxiety and worry.

On the other hand, a worry induction following discriminative conditioning resulted in enhanced FPS to both threat and safety cues during extinction (Gazendam & Kindt, 2012), suggesting that excessive worry may lead to impairments in fear inhibition. Relatedly, Kindt and Soeter (2014) failed to find a relationship between trait anxiety and fear inhibition and suggested that deficient fear inhibition may more likely be a product of high anxiety states than a biomarker

for the development of anxiety and worry. However, it could be that their findings reflect a protective mechanism of appropriate fear inhibition in those who are high in trait anxiety. More specifically, given that anxiety disorders often emerge during childhood (Craske & Waters, 2005), it is possible that their undergraduate sample passed through childhood relatively unscathed from more debilitating levels of anxiety and worry precisely because they were free from impairments in fear inhibition. Whether inappropriate fear responding in the presence of safety cues is a predictor or an artifact of anxiety-related psychopathology thus remains unclear.

Given that the median age of onset of anxiety disorders is 11 years (Kessler et al., 2005), examining fear inhibition in children during the early- to middle-childhood years could provide valuable information about safety signal learning in childhood. Although there is some evidence suggesting that physiological discrimination between threat and safety cues may be impaired across the board prior to the age of 10 years (Glenn, Klein, Lissek, Britton, Pine, & Hajcak, 2012), irrespective of child anxiety levels (Jovanovic et al., 2014), these results are limited by the cross-sectional nature of the studies in which they were found. Further, additional evidence indicates that children as young as seven years of age can distinguish between threat and safety cues (Barker et al., 2014), as can children between the ages of 8-13 (Gamwell, Nylocks, Cross, Bradley, Norrholm, & Jovanovic, 2015); though males showed better discrimination than females in the latter study. Moreover, when safety signal learning is examined at the cognitive level, children as young as eight years of age are able to distinguish between threat and safety cues (Glenn et al., 2012; Jovanovic et al., 2014; Gamwell et al., 2015). Additional research is needed in order to clarify this discrepancy between physiological and cognitive responding to safety cues in middle childhood.

Efforts to further understanding of safety signal learning during the middle childhood years must give consideration to the concepts of multifinality and equifinality (Sroufe, 1997). Impairments in fear inhibition may result in a number of developmental outcomes (i.e., multifinality) and multiple pathways may lead to the same impairments in fear inhibition (i.e., equifinality). Although inappropriate fear responding to safety signals likely reflects a complex interaction of biological, psychological, and social processes, evidence from literature examining the intergenerational transmission of anxiety-related disorders (e.g., Micco et al., 2009; Starr, Conway, Hammen, & Brennan, 2014) suggests that parenting may be one factor contributing to the transmission of these disorders (Fisak & Grills-Taquechel, 2007; Leen-Feldner, Feldner, Knapp, Bunaciu, Blumenthal, & Amstadter, 2013; van der Bruggen, Stams, & Bogels, 2008), and possibly impairments in fear inhibition (Barker et al., 2014), within families. Several key theories (Edwards et al., 2010; Fisak & Grills-Taquechel, 2007; Lebowitz, Leckman, Silverman, & Feldman, 2016; Rapee, 2009; Starr et al., 2014) elucidate how parental anxiety-related psychopathology may impact children and potentially result in dysfunctional fear responding.

Behavioral inhibition (BI; Rosenbaum et al., 1993; Hirshfeld-Becker et al., 2008) is conceptualized as a temperamental trait characterized by the tendency to respond to novel people, places, and situations with fear (Kagan, 1994). BI may be a precursor for child anxiety (Fox, Henderson, Marshall, Nichols, & Ghera, 2005; Hirshfeld-Becker et al., 2008) and is highly correlated with parental anxiety (Rosenbaum et al., 1993), which in turn, is associated with child anxiety (Beidel & Turner, 1997; Biederman et al., 2006; Burstein, Ginsburg, & Tien, 2010; Chapman, Petrie, Vines, & Durrett, 2012; McClure, Brennan, Hammen, & Le Brocque, 2001). In fact, a recent meta-analysis found that children of parents with an anxiety disorder are approximately 3.91 times more likely to have an anxiety disorder themselves compared to

children of psychiatrically healthy parents (Micco et al., 2009). Relatedly, maternal and child bias for threat are highly correlated (Creswell, Schniering, & Rapee, 2005), and this tendency to interpret events in a threatening manner is thought to play a critical role in anxiety-related psychopathology (e.g., Beck, Emery, & Greenberg, 1985).

Other theories regarding how anxiety-related psychopathology in parents affects children include both genetic and environmental influences (Edwards et al., 2010). Although anxiety likely often results from a complex interplay of genetic and environmental factors, recent, compelling evidence from the rodent literature suggests that fear may, in some cases, be transmitted at the epigenetic level. In a study by Dias and Ressler (2013), two generations of offspring of male mice undergoing odor fear conditioning prior to conception demonstrated not only increased behavioral sensitivity to the paternally conditioned odor but also changes in the neuroanatomical representation for the conditioned odor (Dias & Ressler, 2013). These alterations in olfactory neuroanatomy were also found in mice conceived via *in vitro* fertilization using adult sperm acquired ten days after odor fear conditioning, while modifications in sperm were found not only in odor-conditioned mice but also in their offspring. Collectively, these findings point to an epigenetic mechanism for fear transmission involving modification of gene expression in response to environmental input, though do not preclude fear transmission through social processes.

Notably, theoretical accounts of the origins of child anxiety disorders suggest that environmental influences, particularly parenting, may account for the greatest variance in anxiety during the early-to-middle childhood years, when parental involvement is highest (Connell & Goodman, 2002) and when children have yet to differentiate from their parents (Rapee, 2009). Indeed, empirical support for the importance of shared environmental factors comes from the

findings of twin studies in children with anxiety, which suggest that roughly one-third of the variance in childhood anxiety disorders is accounted for by genetics and that the remaining two-thirds is likely explained by environmental factors and to a small extent, measurement error (e.g., Eley et al., 2003; 2008). More recently, epigenetic explanations have gained increasing attention (Dias, Maddox, Klengel, Ressler, 2015), though efforts to untangle epigenetic mechanisms from the behavioral transmission of anxiety-related processes in humans are still in their infancy.

As noted above, one shared environmental factor that might account for the strong association between parent and child anxiety is the transmission of impairments in fear inhibition from one generation to the next. Although fear and fear inhibition are most often thought to be transmitted in a “top-down” manner from parents to their children, fear-related processes may also be transmitted in a “bottom-up” fashion from children to their parents, or even transactionally (Fisak & Grills-Taquechel, 2007). The theoretical accounts that have been examined the most, however, posit a “top-down” transmission of fear and reduced fear inhibition via parenting and point to early learning experiences including modeling, information transfer, reinforcement of anxious and avoidant behaviors (see Fisak & Grills-Taquechel, 2007, for a review), and parental control (see McLeod et al., 2007, for a review).

Modeling behaviors that may influence the development of anxiety-related pathology have been found to impact children as early as infancy (e.g., Rosnay, Cooper, Tsigras, & Murray, 2006) and include expressing anxious thoughts (e.g., Moore et al., 2004), visibly displaying anxiety (Dubi et al., 2008), and engaging in avoidance behaviors (Silverman, Cerny, & Nells, 1988), all while in the child’s presence. For example, children of parents with panic disorder are more likely than children of healthy controls to make threatening, panic-related interpretations of ambiguous stimuli (Schneider et al., 2002), possibly due to having witnessed their parents’ panic

attacks. In addition, Borelli and colleagues found that higher parental anxiety was associated with greater child-reported reactivity to imagining being in potentially frightening situations, only among children whose parents reported high levels of negative emotion to imagining their children in the same situations (Borelli, Rasmussen, St. John, West, & Piacentini, 2015).

Although parents and children completed the tasks of this study by Borelli and colleagues independently, parents' displays of distress may partially underlie the relationship between parent and child anxiety. Relatedly, in a study of rodents, Debiec and Sullivan (2014) found that maternal fear expressions and odors in the presence of a maternal-, but not pup-, conditioned fear odor resulted in fearful responding in pups and increases in pups' corticosterone levels, supporting both social modeling and epigenetic accounts of fear transmission.

With regard to information transfer, parents may explicitly communicate to their children that objectively safe situations are actually dangerous or may make unnecessarily cautionary statements, such as "be careful," that convey a similar message (e.g., Beidel & Turner, 1998). This overprotective or controlling parenting style is theorized to lead to a sense of decreased self-efficacy in children as well as increased anxiety (Rapee, 2001). In addition, parental rewarding (e.g., giving extra attention) of or assisting in children's anxious and avoidant behaviors (e.g., allowing a child to stay home from school on a test day) may reinforce such behavior, thus encouraging anxious and avoidant coping styles (Rapee, 2002).

However, although certain parenting behaviors may lead children to react fearfully or with anxiety in non-threatening contexts, others may foster more appropriate, non-fearful responses. Nurturing parenting curtails the inhibited behavior of rhesus monkeys bred to be highly reactive (Suomi, 1997); and, in humans, maternal modeling of positive responding to fear-relevant stimuli helps children learn how to appropriately inhibit fear (e.g., Egliston & Rapee,

2007). As discussed above, impairments in fear inhibition may be more strongly linked to anxiety-related disorders than exaggerated fear responding (Duits et al., 2015), underscoring the potential value of parenting behaviors that beget adaptive fear inhibition. Moreover, extrapolating from the findings of Duits and colleagues (2015), the presence of parental anxiety-related psychopathology may not necessarily have any bearing on whether children respond with elevated levels of fear to threatening stimuli, but may instead influence children's tendencies to appropriately inhibit fear in non-threatening circumstances.

Given the association between anxiety-related psychopathology and impairments in fear inhibition, parents with these disorders may be doubly impaired: possessing a deficit in safety signal learning and also failing to model appropriate safety signal responding for their children. These early learning experiences may act in conjunction with, or independently of, a genetic predisposition toward impaired fear inhibition, resulting in deficient safety signal learning in children. Relatedly, parental anxiety-related psychopathology may interfere with children's learning of underlying contingencies associated with threat and safety cues. Specifically, a lack of adaptive experiences involving learning how to differentiate between these cues paired with incomplete cortical maturation of prefrontal inhibitory functioning (Gee et al., 2013) may mean that children of worried, anxious parents lack the contingency awareness that is necessary in order to distinguish between dangerous and non-dangerous cues.

This proposed link between parental anxiety-related psychopathology and child impairments in fear inhibition may also be explained by overprotective parenting, which is characterized by parenting behaviors that limit children's exposures to situations that are construed to be dangerous (Chorpita & Barlow, 1998; Hudson & Rapee, 2004). According to theoretical models of anxiety, such parenting behaviors teach children that the world is a

dangerous place and that they cannot manage challenging situations, thus fostering an avoidant coping style and an increase in anxiety (Manassis & Bradley, 1994; Rapee, 2009). In line with these models, overprotective parenting is associated with maternal anxiety (Clarke, Cooper, & Creswell, 2013), and findings examining the effects of overprotective parenting both longitudinally (Edwards et al., 2010; Rapee, 2009) and experimentally (de Wilde & Rapee, 2008) suggest that overprotective parenting behaviors predict later elevations in child anxiety. Further, a meta analysis examining the association between parenting and child anxiety found that autonomy-granting, which is the opposite of parental control or overprotective parenting, accounted for 18% of the variance in child anxiety (McLeod, Wood, & Weisz, 2007). Similarly, another review found that parental control had a non-trivial, medium-sized effect ($d = 0.58$) on child anxiety (van der Bruggen Stams, & Bogels, 2008). More recently, maternal, but not paternal, overcontrol was found to have an indirect effect on the relationship between maternal and child anxiety symptoms (Borelli, Margolin, & Rasmussen, 2015). From a theoretical perspective, the safety signal learning impairments that are found in individuals with anxiety-related psychopathology likely contribute to the impression that the world is full of danger (Jovanovic et al. 2012), which in parents, may manifest in the form of excessive worry about child safety and overprotective parenting behaviors. Parents with higher symptoms of anxiety-related psychopathology may therefore restrict their children's exposure to perceived threatening situations, thus narrowing opportunities for their children to learn how to distinguish between safe and unsafe circumstances.

In summary, the presence of anxiety-related symptoms in parents may influence whether children respond with or without fear to non-dangerous stimuli. Specifically, higher levels of parental worry, anxiety, and overprotective parenting are likely associated with child

impairments in fear inhibition.

Child safety signal learning in the context of parental psychopathology has, to our knowledge, yet to be studied. This study examined whether parental worry, which has been proposed as a transdiagnostic factor (Kertz, Bigda-Peyton, Rosmarin, Björgvinsson et al., 2012) associated with anxiety, depression, and stress (Olatunji, Broman-Fulks, Bergman, Green, & Zlomke, 2009), is associated with safety signal learning impairments in children. Relatedly, this study also examined whether child safety signal learning impairments are associated with overprotective parenting and parental PTSD and depression. Parents who have children in their middle childhood years (8-11 years old) were recruited. Children completed self-report questionnaires and parents completed self- and parent-report questionnaires, in addition to a clinical interview. Children also completed a safety signal learning paradigm (Jovanovic et al., 2010) with the unconditioned stimulus (US) being an aversive air puff to the larynx and the dependent variable being FPS to pairs of shapes that were repeatedly presented with either an aversive air puff to the throat (i.e., the danger trials, AX+), or that were never paired with the air puff (i.e., the safety trials, BX-). FPS responses were measured in the safety signal transfer trials, during which one shape from the danger trials was paired with one shape from the safety trials in the absence of the air puff (i.e., the AB trials).

Given that anxiety-related psychopathology has consistently been associated with impairments in fear inhibition, but not fear responding (see Duits et al., 2015 for a review), we hypothesized that higher parental worry would predict greater child FPS to the safety signal transfer trials but not to the danger trials, suggesting greater impairments in fear inhibition alone. In addition, given that cortical maturation of prefrontal inhibitory functioning is far from complete during the middle childhood years (Gee et al., 2013) and that parental impairments in

safety signal learning might be associated with few opportunities for children to learn how to distinguish between threatening and non-threatening cues, we hypothesized that higher parental worry would be related to a higher likelihood of children predicting the US in their expectancy ratings, particularly for the safety signal transfer trials. We also hypothesized that higher overprotective parenting would predict greater child FPS to the safety signal transfer trials alone and that higher overprotective parenting would be related to a higher likelihood of children predicting the US in their expectancy ratings, particularly for the safety signal transfer trials. Further, given that overprotective parenting is associated with maternal anxiety (Clarke et al., 2013), we hypothesized that higher overprotective parenting would have an indirect effect on the relationship between higher parental worry and greater child FPS to the safety signal transfer trials. More broadly, we hypothesized that higher parental PTSD symptoms would also predict greater child FPS to the safety signal transfer trials but not to the danger trials. By contrast, given that adults with stand-alone depression (Jovanovic et al., 2010) and depressive symptoms (Dibbets, Broek, & Evers, 2014) do not differ from psychiatrically healthy controls in their responding to either threatening or non-threatening cues, we did not hypothesize a relationship between parental depression and child FPS to any of the trials.

METHOD

PARTICIPANTS

Parent-child dyads ($N = 35$) were recruited from a large, metropolitan community via advertising in newspapers, parenting websites and blogs, school newsletters, flyers that were distributed at large parenting lectures and camp fairs, and flyers that were placed in schools, community centers, mental health outpatient clinics, churches, synagogues, grocery stores, convenience shops, restaurants, and campus buildings.

Eligible parents were between the ages of 18-65, were able to read, write, and speak English, and were the biological parent and primary caregiver of the eligible child, defined as the main person responsible for care and control of the child and for the child's overall health and welfare. Exclusion criteria for parents included a current diagnosis of substance dependence, schizophrenia, bipolar disorder, another disorder with delusional content, or depression that was severe enough to require immediate psychiatric treatment based on the Structured Clinical Interview for DSM-IV (SCID-IV; First et al., 1995).

Eligible children were between the ages of 8-11 years, spoke English, and had normal hearing and normal or corrected-to-normal vision. Exclusion criteria in children included a parent-reported cognitive disability (not including ADHD). These criteria were chosen to allow for broad inclusion while assuring children could appropriately respond to the measures and tasks. In addition, parent-child dyads were excluded if the child self-report and startle data were both missing ($n = 1$) or if the parent interview and questionnaire data were both missing ($n = 1$). This ensured that relationships could be examined between at least some of the parent and child variables. See Table 1 for a summary of inclusion and exclusion criteria.

The mean age of included children was 9.31 years ($SD = 1.08$) and the children were predominantly male (62.9%) and Caucasian (60%). For parents, the mean age was 41.06 ($SD = 6.23$) and the sample was predominantly female (91.4%) and Caucasian (62.9%). Current and lifetime rates of DSM-5 Axis I diagnoses, not including PTSD, in parents were 8.57% and 31.43%, respectively. The rate of lifetime trauma-exposure was 25.71%. Among trauma-exposed parents, index DSM-5 Criterion A trauma exposures reported were sexual assault ($n = 1$), non-sexual assault ($n = 1$), childhood sexual assault ($n = 3$), childhood non-sexual assault ($n = 1$), and

unexpected death of a loved one ($n = 3$). The mean time since index trauma among these parents was 15.16 years ($SD = 12.98$).

PARENT MEASURES

Parental measures were chosen to assess for inclusion and exclusion criteria, continuous measures of key constructs (e.g., worry, PTSD, depression, parenting style), and potential moderators.

Structured Clinical Interview for DSM-IV (SCID-IV; First et al., 1995)

The SCID is a clinician-administered interview that assesses current and past DSM-IV diagnoses. The SCID-IV has inter-rater reliability of .70 - .94 (First et al., 1995) and was used in the present study to establish presence of diagnoses other than PTSD. 12% of the cases from the current study were rerated for diagnostic reliability. There was good diagnostic agreement for current anxiety disorders ($\kappa = 1.00$, $p_{pos} = 1.00$, $p_{neg} = 1.00$), major depressive disorder ($\kappa = 1.00$, $p_{pos} = 1.00$, $p_{neg} = 1.00$), substance abuse disorders ($p_{pos} = .00$, $p_{neg} = 1.00$), and other diagnoses ($p_{pos} = .00$, $p_{neg} = 1.00$).

PTSD Symptom Scale-Interview for DSM-5 (PSS-I-5; Foa, McLean, Zang, Zhong, Rauch, et al., 2015)

The PSS-I-5 is a 20-item, semi-structured interview that provides a diagnosis and symptom severity score of DSM-5 PTSD symptoms using a 0 (*not at all*) to 4 (*6 or more times a week/severe*) scale, rated for the past month. The PSS-I was used in the present study to measure PTSD severity and diagnoses in trauma-exposed parents. The PSS-I-5 has good convergent validity with the Clinician-Administered PTSD Scale for DSM-5 (Weathers et al., 2013), a widely-used PTSD interview, $r = .72$, and with a well-validated self-report measure of anxiety, $r = .73$ (Foa et al., 2015). In addition, the PSS-I-5 also has discriminant validity with a well-validated, self-report measure of depression, $r = .62$ (Foa, McLean, Zang, Zhong,

Rauch, et al., 2015). Diagnostic reliability was assessed in the current study by rerating 25% of the cases. Inter-rater reliability was high for PTSD severity (ICC = .99).

Trauma Assessment for Adults (TAA; Resnick, Best, Freedy, Kilpatrick, & Falsetti, 1993)

The TAA is a standardized trauma history interview that assesses for prior lifetime exposure to 13 different types of traumatic events. The manner of trauma exposure is assessed for each item using the following scale: 0 (*No*), 1 (*Witnessed*), 2 (*Experienced*), and 3 (*Witnessed and Experienced*). Frequency of trauma exposure is also assessed for each item using the following open-ended question, “*How many times?*” The TAA was used to assess for exposure to traumatic events.

Posttraumatic Diagnostic Scale for DSM-5 (PDS-5; Foa, McLean, Zang, Zhong, Powers, et al., 2015)

The PDS-5 is a self-report version of the PSS-I-5 that includes two questions designed to assess trauma history and to identify the traumatic event that bothers the respondent the most (i.e., the index trauma). The PDS-5 consists of 20 items measuring post-trauma reactions over the past month rated on a 0 (*not at all*) to 4 (*6 or more times a week/severe*) Likert scale. The PDS-5 was used in the present study as a self-report of PTSD symptoms in the trauma-exposed group. It has good convergent validity with the PTSD Checklist-Specific Version, $r = .90$, and the PSS-I-5, $r = .85$ and good discriminant validity with symptoms of depression, $r = .77$, and anxiety, $r = .64$ (Foa, McLean, Zang, Zhong, Rauch, et al., 2015).

Quick Inventory of Depressive Symptomatology, Self-Report (QIDS-SR₁₆; Rush et al., 2003)

The QIDS-SR₁₆ is a 16-item self-report measure that assesses the severity of DSM-IV depressive symptoms over the past week. Items are rated on a 0 to 3 scale, with higher scores indicating greater symptom severity. For example, the energy item is rated using the following scale: 0 (*there is no change in my usual energy level*); 1 (*I get tired more easily than usual*); 2 (*I*

have to make a big effort to start or finish my usual daily activities. For example, shopping, homework, cooking or going to work); 3 (I really cannot carry out most of my usual daily activities because I just don't have the energy). The QIDS-SR₁₆ has good convergent validity with interview-rated measures of depression, $r = .86-.89$, and requires less time than other depression inventories (Rush et al., 2006).

Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990)

The PSWQ is a 16-item self-report measure that assesses the excessiveness and uncontrollability of the trait of pathological worry using a 1 (*not at all typical*) to 5 (*very typical*) Likert scale of how “typical or characteristic” each item is of the respondent. Worry was examined in this largely psychiatrically healthy sample due to its universality and dimensional nature (Heimberg, Turk, & Mennin, 2004), as well as its link with impairments in fear inhibition (Lissek et al., 2014). The PSWQ shows elevations across all anxiety disorders while discriminating between anxiety and depression (Brown, Antony, & Barlow, 1992). It also has good test-retest reliability, $r = .92 - .93$ (Meyer et al., 1990).

Parental Overprotection Measure (OP; Edwards et al., 2008, 2010)

The OP is a 19-item parent report that assesses parents' general tendency to engage in overprotective parenting behaviors that limit a child's exposure to potentially threatening situations. Items are rated on a 0 (*not at all*) to 4 (*very much*) Likert scale. The OP has strong test-retest reliability for mothers and fathers ($r = .79$ and $.77$, respectively; Edwards, Rapee, & Kennedy, 2008) and adequate construct validity, assessed via observer ratings of maternal overprotection during a challenging puzzle task that was given to mothers and their 7-12-year-old children, $r = .25$ (Clarke et al., 2013).

CHILD MEASURES

Key constructs of child internalizing and externalizing symptoms (e.g., anxiety, depression, attention) from both parent and child informants were assessed.

Child Behavior Checklist for Ages 6-18 (CBCL/6-18; Achenbach & Rescorla, 2001)

The CBCL is a 118-item parent-report of child internalizing and externalizing problems over the previous six months. Items are rated on a 3-point Likert scale (0 = *not true*; 1 = *somewhat or sometimes true*; 2 = *very often true or often true*). The CBCL yields a total problems scale, internalizing and externalizing subscales, eight narrow-band syndrome scales (e.g., Withdrawn/Depressed; Aggression), and six DSM-oriented scales (e.g., Anxiety Problems; Conduct Problems). In the present study, the Internalizing and Externalizing subscales were used in order to capture general distress and problem behaviors and values were reported in T-scores. The CBCL Internalizing and Externalizing subscales have good test-retest reliability ($r = .91$ and $.92$, respectively) (Achenbach & Rescorla, 2001).

Revised Child Anxiety and Depression Scale (RCADS; Chorpita et al., 2000)

The RCADS is a 47-item self-report that assesses DSM-IV symptoms of anxiety and depression. The RCADS is comprised of six subscales: separation anxiety disorder, generalized anxiety disorder, obsessive-compulsive disorder, specific phobia, panic disorder, and major depressive disorder. It also yields an Anxiety Total Score (sum of the five anxiety subscales) and a Total Score (sum of the six subscales). In the present study, the anxiety total and depression subscale scores were used as an index of child anxiety and depressive symptoms. The frequency of items is rated on a 0 (*never*) to 3 (*always*) Likert scale. The RCADS anxiety subscales have good convergent validity with the subscales of another widely used measure of child anxiety, $r = .59-.72$, and the RCADS depression subscale also has good convergent validity with a well-validated measure of child depression, $r = .70$ (Chorpita, Moffitt, & Gray, 2005). Finally, the six subscales

of the RCADS have good discriminant validity with child and parent interview ratings of oppositional behavior, $r = -.05-.07$ (Chorpita, Moffitt, & Gray, 2005).

Revised Child Anxiety and Depression Scale- Parent Version (RCADS-P; Chorpita et al., 2000)

The RCADS-P is a 47-item parent report version of the RCADS. Like the RCADS, the RCADS-P is comprised of six subscales: separation anxiety disorder, generalized anxiety disorder, obsessive-compulsive disorder, specific phobia, panic disorder, and major depressive disorder. It also yields an Anxiety Total Score (sum of the five anxiety subscales) and a Total Score (sum of the six subscales). In the present study, the Anxiety Total Score and the depression subscale score were used as an index of parent perceived child anxiety and depressive symptoms, respectively. The frequency of items is rated on a 0 (*never*) to 3 (*always*) Likert scale. The Anxiety Total Score of the RCADS-P has convergent validity with the CBCL DSM-oriented Anxiety Problems scale, $r = .76$, and the depression subscale of the RCADS-P has convergent validity with the DSM-oriented Affective Problems Scale, $r = .83$ (Ebesutani et al., 2011).

UCLA PTSD Reaction Index for Children/Adolescents, DSM-5 (Child Version: UCLA PTSD-RI; Pynoos & Steinberg, 2013)

The UCLA PTSD-RI is a self-report of child trauma exposure and reactions. It consists of a 15-item trauma history screen, a 27-item PTSD assessment, and a four-item assessment of dissociative symptoms. The PTSD and dissociative items are rated on a 0 (*none*) to 4 (*most*) Likert scale of how often the problem has happened in the past month. The previous DSM-IV version demonstrated convergent validity with the PTSD subscale of a well-validated measure of posttrauma symptoms in children, $r = .75$ (Steinberg et al., 2013).

UCLA PTSD Reaction Index for DSM-5 (Parent Version: PTSD-RI; Pynoos & Steinberg, 2013)

The PTSD-RI was adapted to create a parent-report of child trauma exposure and reactions,

given that the DSM-IV version of the measure included a parent-report adaptation. The PTSD-RI consists of a 15-item trauma history screen, a 27-item PTSD assessment, and a four-item assessment of dissociative symptoms. The PTSD and dissociative items are rated on 0 (*none*) to 4 (*most*) Likert scale of how often the problem has happened in the past month. The previous DSM-IV parent version of the PTSD-RI demonstrated convergent validity with a well-validated parent-report measure of trauma-related symptoms in children, $r = .75$ (Wherry, Corson, & Hunsaker, 2013).

MATERIALS, DESIGN, AND PROCEDURE

Apparatus

Acoustic startle probes were generated using a stimulator module and calibrated using a digital sound level meter. The unconditioned stimulus was an aversive air puff that was generated from a tank of compressed air attached to a solenoid valve to ensure precise delivery of the 250-ms air puff (Berg & Balaban, 1999). Children wore a camelback hydration backpack, retrofitted for delivery of the air puff through nylon tubing to the larynx: a set-up that has been used previously (e.g., Borelli, Sbarra, Crowley, & Mayes 2011). Psychophysiological data was collected using Biopac's MP150 Data Acquisition System (Biopac Systems, Inc.) running Acknowledge software (version 4.2). An electromyography module (EMG100C) with gain set to 5000, low pass filter set to 500 HZ, and high pass filter set to 10 HZ was used to collect EMG data in the range of -10 to 10 volts. A Dell desktop computer running E-Prime Professional (Version 2) was used to present the experimental stimuli.

Acoustic Startle

A background white noise of 70 dB [A] SPL was delivered continuously throughout the session. The startle probe was a 108 dB [A] SPL, 40-ms burst of broadband noise with a near-

instantaneous rise time, similar to probes used in other child studies (e.g., Borelli, et al., 2011; Jovanovic et al., 2011). All acoustic stimuli were delivered binaurally through high fidelity professional reference headphones (KRK systems, KNS-6400) within a sound-attenuated room.

AX+/BX- Stimuli

The CSs were pairs of shapes presented on a computer monitor. The AX+ shape pair was always paired with the US, while the BX- shape pair was never paired with the US. The AX+ and BX- stimuli consisted of a set of two green, orange, or black shapes (star, triangle, or square) presented centrally on a monitor. The AX+ and BX- stimuli included one unique cue (A, the danger cue; or B, the safety cue) and one common cue (X). The fear inhibition test stimulus (AB) was never paired with the US and consisted of the unique elements of the previously conditioned stimuli (i.e., the danger and safety cues). See Figure 1 for an example of the conditioned stimuli. Shape pairs were counterbalanced across participants with cues differing in both color and shape. Based on the work of Jovanovic et al. (2009, 2010), the shapes within each pair were presented simultaneously for 6 s. On AX+ trials, the CS co-terminated with a 250-ms air puff (US), which was preceded by 500 ms by a 40-ms startle probe. By contrast, the BX- and AB trials terminated immediately after presentation of the startle probe, and therefore BX- and AB were never paired with the US. The aversive stimulus (US) was a 250-ms air puff directed to the larynx with an intensity of 80 psi.

After a 1-min acclimation period consisting of background noise, the test session began with a habituation phase consisting of six startle probes to attenuate initial startle reactivity. The conditioning and testing phases followed. The acquisition phase consisted of three blocks, each of which included four trials of AX+, four trials of BX-, and four startle probes presented in the absence of any stimuli [noise alone (NA) trials], for a total of 12 trials per block. The testing

phase included a block of four NA trials and four AB trials. Trial types were randomly intermixed within blocks and inter-trial intervals were randomized throughout the phases, ranging from 9 to 22 s. The entire session lasted less approximately 15 min.

Retrospective Expectancy of the Air Puff

Following Grillon and Morgan (1999) and Jovanovic et al. (2009), expectancy was assessed via three button response keys. At the end of the safety signal transfer trials, children were asked for each of the three CS shape pairs if they had expected the shape pair to be followed by the air puff. Children were instructed to press “1” for “No” (safe); “2” for “Yes” (threat); and “3” for “I don’t know” (uncertain). These values were then centered such that expectancy of the air puff was scored as 1 for a response of “Yes,” -1 for a response of “No” and 0 for a response of “I don’t know.”

Procedure

Potentially eligible and interested parent-child dyads called the study telephone line. Parents were prescreened on the telephone for probable PTSD, resulting from a DSM-5 Criterion A traumatic event that occurred at least 6 months prior, or lack thereof. PTSD symptoms were assessed carefully over the phone using a screening questionnaire adapted from the PSS-I-5. Parent-child dyads that were potentially eligible and interested came to the laboratory, where they were guided through informed consent and assent procedures. Parents were interviewed using the SCID-IV. Parents reporting a history of trauma exposure were also interviewed using the PSS-I. Meanwhile, children completed self-report questionnaires and screens for auditory and visual impairment. Specifically, children completed an auditory test during which they were required to correctly identify at least eight out of ten different tones at varying decibels. Visual acuity was tested using a Snellen Eye Chart (Snellen, 1862) from a viewing distance of 20 feet.

These auditory and visual screens were performed in order to ensure that children could detect the auditory and visual stimuli presented in the AX+/BX- paradigm. Eligible children then completed the AX+/BX- paradigm while their parents completed self-report questionnaires. Finally, children and parents were debriefed and compensated with \$20 per hour and a “Child Scientist” t-shirt.

DATA RECORDING, REDUCTION, AND RESPONSE DEFINITION

The eyeblink response was measured via electromyography (EMG) of the right orbicularis oculi muscle, following guidelines for startle blink research (Blumenthal et al., 2005). Two 5 mm Ag-AgCl electrodes were used; one positioned 1 cm below the pupil of the right eye and the other placed 1 cm below the lateral canthus (Berg & Balaban, 1999). EMG activity was sampled at 1000 Hz and was amplified, rectified, and smoothed prior to digitizing (Blumenthal et al., 2005). EMG peak magnitude was transformed to microvolts (μV) and converted to T-Scores, as recommended by Blumenthal and colleagues, by standardizing the raw EMG peak magnitude values for each participant as follows: z score value = $(\text{raw magnitude value} - M_{\text{all raw values}}) / SD_{\text{all raw values}}$; T-score value = $(z \text{ score value} \times 10) + 50$.

Valid eyeblink responses were those that started 20-150ms after the onset of the startle probe and peaked within the same window of time (Blumenthal et al., 2005). Eyeblink responses were considered invalid when elevated levels of EMG activity were present without returning to baseline in the 500ms before the startle probe, or when highly variable EMG activity was present both before and after a given trial. Missing psychophysiological data from 12 trials (out of 880 trials) was replaced with the mean values from the other trials of the same conditioned stimulus within the given block. Nonresponses, defined as trials with no discernible blink responses or with responses $< 0.10 \mu\text{V}$ within the 20-150ms window were included in the analyses in order to

account for possible effects of habituation. Outliers, defined as values falling more than three standard deviations above or below the sample mean, were winsorized by replacing them with the mean of the other trials of the same conditioned stimulus within the given block. A total of 15 trials were classified as outliers. Participants with $\geq 30\%$ of trials rejected due to artifacts or related problems ($n = 5$), nonresponses ($n = 3$), or outliers ($n = 0$) were excluded from the psychophysiological analyses. Data from participants who requested to abort the experiment were also excluded ($n = 4$). In addition, five participants did not initiate the psychophysiological experiment due equipment failure ($n = 1$), uncorrected vision problems ($n = 1$), or parental exclusion ($n = 3$). Thus, the final sample size for FPS was 20.

The parent and child symptoms of dyads that were and were not included in the FPS analyses were compared and were generally similar. Overprotective parenting was higher among those without FPS data ($M = 38.15$, $SD = 14.33$) than among those with FPS data ($M = 28.21$, $SD = 11.41$), $t(30) = 2.18$, $p = .04$, $d = 0.77$. Parent-reported child depressive symptoms were also higher among those without FPS data ($M = 5.89$, $SD = 4.29$) than among those with FPS data ($M = 2.81$, $SD = 2.37$), $t(23) = 2.33$, $p = .03$, $d = 0.89$. Additionally, parent-reported internalizing symptoms were higher among those without FPS data ($M = 57.00$, $SD = 11.01$) than among those with FPS data ($M = 46.59$, $SD = 7.67$), $t(23) = 2.75$, $p = .01$, $d = 1.10$.

Based on recommendations for analyzing EMG data (Blumenthal et al., 2005), mean values for startle magnitude to the NA trials of each of the three conditioning blocks and the transfer test were calculated by averaging T-score values for startle reactivity to the probe on each NA trial within the given block. These numbers served as a baseline from which to calculate average FPS, which served as the dependent variable, for each CS type within each block. The following equation (Jovanovic et al., 2010) was used to determine FPS to AX+ and

BX- within each conditioning block and to AB within the testing phase: *Percent Startle*

Potentiation = 100 x (startle magnitude during CS trials – NA startle magnitude within the same block as the CS).

GENERAL DATA ANALYTIC STRATEGY

Data was screened prior to analysis to check for accuracy of data entry, missing values, and outliers. As noted above, startle data that appeared to be missing at random was replaced using multiple imputation methods (Tabachnick & Fidell, 2006). There was no missing data from the interview, self-, and parent-report measures. In addition, measures were screened for normality, linearity, and homoscedasticity using scatter plots. No data transformations were necessary. No outliers (> 3 SD above the mean) were identified from the interview, self-, and parent-report measures, though as noted above, outliers from the startle data were winsorized to ensure that the results were not disproportionately influenced by children exhibiting extreme startle responses. The Mahalanobis distance procedure was used to check for multivariate outliers and when present, these cases were removed from the relevant multivariate analysis (1 out of 217 data points). No covariates were identified.

Although dichotomizing continuous data is generally not recommended (e.g., MacCallum, Zhang, Preacher, & Rucker, 2002), median splits can reveal important differences that might otherwise be lost, with less risk of type I and II errors than previously thought (Iacobucci et al., 2015). Parental worry and overprotective parenting were therefore divided into low and high groups using median splits in order to maximize variability within our mostly psychiatrically healthy sample. Separate repeated measures analysis of variance (ANOVA), with between-subjects factors of parental worry (low, high) or overprotective parenting (low, high), and within-subjects factors of CS Type (AX+, BX-) and acquisition block (early, middle, late)

examined FPS during acquisition as the dependent variable. Similarly, in order to assess final learning of AX+, BX-, and transfer of that learning to AB, separate repeated measures ANOVA, with between-subjects factors of parental worry (low, high) or overprotective parenting (low, high), and within-subjects factors of CS Type (AX+, BX-, AB) examined FPS during late acquisition and the transfer test as the dependent variables. For all repeated measures ANOVAs, violations of sphericity were corrected using the Greenhouse-Geisser correction.

In addition, in order to assess for discriminative conditioning at the cognitive level, separate repeated measures ANOVA, with between-subjects factors of parental worry (low, high) or overprotective parenting (low, high) and within-subjects factors of CS Type (AX+, BX-, AB) were used to examine retrospective expectancy of the air puff as the dependent variable.

Independent *t*-tests were also used to examine whether children whose parents were low and high in worry and overprotective parenting differed in subjective units of distress (SUDS) prior to the AX+/BX- paradigm and in change in SUDS from before and after the AX+/BX- paradigm.

In order to examine the hypothesis that overprotective parenting would have an indirect effect on the relationship between parental worry and child FPS to the safety transfer cue, we used model 4 of the PROCESS macro for SPSS (Hayes, 2013). We assessed for the presence of an indirect effect even in the absence of a total effect, consistent with research recommendations (Hayes, 2009). Unstandardized indirect effects were computed for each of 10,000 bootstrapped samples, and the 95% confidence interval was computed by determining the indirect effects at the 2.5th and 97.5th percentiles.

Finally, bivariate correlations were conducted in order to examine the relationship between parental PTSD and child FPS, as well as the relationship between parental depression

and child FPS. Bivariate correlations were also conducted in order to examine baseline associations among the parent interview, parent self-report, and parent- and child-report measures, as well as relationships among the child measures and child FPS. These secondary analyses were corrected for multiple comparisons using Holm's step-down procedure (Holm, 1979).

RESULTS

SAMPLE CHARACTERISTICS

Ranges, means, and standard deviations for the parent self-report measures and for the child- and parent-report measures are presented in Tables 2 and 3, respectively. Overall, symptoms for both parents and children were in the minimal to mild range.

BASELINE ASSOCIATIONS AMONG PARENT INTERVIEW, PARENT SELF-REPORT, AND PARENT- AND CHILD-REPORT MEASURES

Bivariate correlations among parental worry, overprotective parenting, PTSD, and depression are presented in Table 4. These parental variables were, for the most part, moderately to strongly associated with one another, though overprotective parenting was not strongly related to clinician-rated PTSD symptoms. Table 5 presents the bivariate correlations among parent-reported and child self-reported symptoms of anxiety, depression, PTSD, SUDS, and internalizing and externalizing symptoms. Parent-reported symptoms were more consistently strongly related to one another (r s ranging from .38 to .88) than child self-reported symptoms (r s ranging from .29 to .75); and parent- and child-reports, with the exception of PTSD symptoms, were only weakly related. Finally, bivariate correlations among parent symptoms and child symptoms, assessed via parent-report (Table 6) and child self-report (Table 7), are presented. Overall, parent self-reported symptoms were moderately to strongly related to parent-reported

child symptoms, but only weakly related to child self-reported symptoms.

LEARNING WITHIN THE CONDITIONAL DISCRIMINATION PARADIGM

Fear-potentiated Startle

Means and standard deviations for FPS to each CS are presented in Table 8. A repeated measures ANOVA, with within-subjects factors of CS type (AX+, BX-) and acquisition block (early, middle, late), examined FPS during acquisition as the dependent variable. There was not a main effect of CS type, $F(1, 19) = .29, p = .60$, but there was a main effect of acquisition block, $F(1.39, 26.46) = 4.13, p = .04$. FPS during early acquisition ($M = 14.74, SD = 15.88$) was significantly higher than FPS during late acquisition ($M = 5.18, SD = 11.36$), $p = .01, d = 0.69$. There was no CS type x acquisition block interaction, $F(2, 38) = .12, p = .89$. In other words, as depicted in Figure 2, children did not demonstrate physiological discrimination between the danger and safety cues, though means were in the expected direction, and FPS decreased over time.

In order to assess final learning of AX+, BX-, and transfer of that learning to AB, a repeated measures ANOVA, with the within-subjects factor of CS type (AX+, BX-, AB), examined FPS during late acquisition and the transfer test as the dependent variables. There was no main effect of CS type, $F(2, 38) = 2.13, p = .13$. Thus, FPS to the critical transfer test (AB) did not differ significantly from FPS to the danger and safety cues, though again as seen in Table 8 and Figure 2, means were in the expected direction.

Expectancy of the Air Puff

Means and standard deviations for retrospective expectancy of the air puff for each CS are presented in Table 8. In order to assess for cognitive learning of the contingencies associated with the conditioned stimuli, a repeated measures ANOVA, with the within-subjects factor of CS

type (AX+, BX-, AB), examined expectancy of the air puff as the dependent variable. There was no main effect of CS type, $F(2, 38) = 1.44, p = .25$, suggesting poor cognitive discrimination among the danger, safety, and the safety transfer cues, though means are in the expected direction. See Table 8 for means and standard deviations.

PARENTAL WORRY

Fear-potentiated Startle

We next examined the hypothesis that higher parental worry would predict greater child FPS to the safety cues but not to the danger cues. In order to assess the effect of parental worry on discriminative conditioning within acquisition, a repeated measures ANOVA, with the between-subjects factor of parental worry (low, high), and within-subjects factors of CS type (AX+, BX-) and acquisition block (early, middle, late), examined FPS during acquisition as the dependent variable. There was not a main effect of parental worry, $F(1, 16) = 1.73, p = .21$, or CS type, $F(1, 16) = .54, p = .47$, but there was a main effect of acquisition block, $F(1.49, 23.88) = 3.70, p = .05$. FPS during early acquisition ($M = 15.30, SD = 3.34$) was significantly higher than FPS during late acquisition ($M = 5.93, SD = 2.83$), $p = .02, d = 3.03$. There was no parental worry x CS type interaction, $F(1, 16) = .02, p = .88$, but there was a trend toward a parental worry x acquisition block interaction, $F(1.49, 23.88) = 3.47, p = .06$. There was no parental worry x CS type x acquisition block interaction, $F(2, 32) = .003, p = 1.00$. A break down of the parental worry x acquisition block interaction revealed that among children whose parents were higher in worry, FPS during early acquisition ($M = 23.31, SD = 3.83$) was significantly higher than FPS during middle acquisition ($M = 7.43, SD = 12.42$), $p = .03, d = 1.73$, and late acquisition ($M = 6.95, SD = 3.68$), $p = .01, d = 4.36$. Among children whose parents were lower in worry, there was no effect of acquisition block. Thus, for both children whose parents were lower and higher

in worry, FPS to the danger cue did not differ significantly from FPS to the safety cue; though among children whose parents were higher in worry, FPS overall was higher in the early rather than the later acquisition blocks.

To examine the main hypothesis in regard to the effect of parental worry on final learning of AX+, BX-, and transfer of that learning to AB, a repeated measures ANOVA, with the between-subjects factor of parental worry (low, high) and the within-subjects factor of CS type (AX+, BX-, AB), examined FPS during late acquisition and the transfer test as the dependent variables. There was not a main effect of parental worry, $F(1, 16) = .20, p = .66$, or CS type, $F(2, 32) = 1.53, p = .23$, nor was there a parental worry x CS type interaction, $F(2, 32) = .01, p = .99$. Thus, among children whose parents were lower or higher in worry, FPS did not differ significantly on the danger and safety trials or on the critical safety signal transfer test.

Expectancy of the Air Puff (US)

The hypothesis that higher parental worry would be related to a higher likelihood of children predicting the US in their expectancy ratings, particularly for the AB trials, was next examined. A repeated measures ANOVA, with the between-subjects factor of parental worry (low, high) and the within-subjects factor of CS type (AX+, BX-, AB) examined expectancy of the air puff as the dependent variable. There was no main effect of parental worry, $F(1, 16) = .18, p = .68$, or CS type, $F(2, 32) = 2.93, p = .07$, and no parental worry x CS type interaction, $F(2, 32) = .30, p = .75$. Thus, children whose parents were lower and higher in worry did not differ in cognitively discriminating among the danger, safety, and transfer test cues.

Subjective Distress

We next conducted independent *t*-tests in order to examine whether higher parental worry would be related to higher child-reported subjective units of distress (SUDs). Children whose parents

were higher in worry ($M = 16.33$, $SD = 16.73$) did not differ from children whose parents were lower in worry ($M = 16.00$, $SD = 16.20$) in pre-task SUDs, $t(15) = -.04$, $p = .97$. Similarly, children whose parents were higher in worry, ($M = 2.86$, $SD = 19.07$) did not differ from children whose parents were lower in parental worry ($M = -7.13$, $SD = 20.22$), in change in SUDs from pre-to-post-task $t(13) = -.98$, $p = .35$.

OVERPROTECTIVE PARENTING

Fear-potentiated Startle

The hypothesis that higher overprotective parenting would predict greater child FPS to the safety cues but not to the danger cues was next examined. In order to assess the effect of overprotective parenting on discriminative conditioning within acquisition, a repeated measures ANOVA, with the between-subjects factor of overprotective parenting (low, high), and within-subjects factors of CS type (AX+, BX-) and acquisition block (early, middle, late), examined FPS during acquisition as the dependent variable. There was not a main effect of overprotective parenting, $F(1, 17) = .61$, $p = .45$, or CS type, $F(1, 17) = .59$, $p = .45$, but there was a trend toward a main effect of acquisition block, $F(1.36, 23.20) = 3.25$, $p = .07$. Specifically, FPS during early acquisition ($M = 14.29$, $SD = 16.18$) was significantly higher than FPS during late acquisition, ($M = 5.36$, $SD = 11.64$), $p = .03$, $d = 0.63$. Among children whose parents were higher in overprotective parenting, there was no effect of acquisition block. There was no overprotective parenting x CS type interaction, $F(1, 17) = .03$, $p = .86$, no overprotective parenting x acquisition block interaction, $F(1.36, 23.20) = .72$, $p = .45$, and no overprotective parenting x CS type x acquisition block interaction, $F(2, 34) = 1.57$, $p = .22$. Thus, for both children whose parents were lower and higher in overprotective parenting, FPS to the danger cue did not differ

significantly from FPS to the safety cue, though FPS did decrease from early to late acquisition at a trend level.

To examine the main hypothesis in regard to the effect of overprotective parenting on final learning of AX+, BX-, and transfer of that learning to AB, a repeated measures ANOVA, with the between-subjects factor of overprotective parenting (low, high), and the within-subjects factor of CS type (AX+, BX-, AB), examined FPS during late acquisition and the transfer test as the dependent variables. There was not a main effect of overprotective parenting, $F(1, 17) = .17$, $p = .69$, or CS type, $F(2, 34) = 1.91$, $p = .16$, but there was an overprotective parenting x CS type interaction, $F(2, 34) = 3.63$, $p = .04$. Among children whose parents were higher in overprotective parenting, FPS to AB ($M = 13.84$, $SD = 14.76$) was significantly higher than FPS to BX- ($M = -1.60$, $SD = 10.35$), $p = .03$, $d = 1.21$. Among children whose parents were lower in overprotective parenting, there was no effect of trial type. See Figure 4. Thus, children whose parents were higher in overprotective parenting demonstrated impaired transfer of fear inhibition from the safety cue to the safety transfer cue during the critical test of safety signal learning, whereas children whose parents were lower in overprotective parenting did not.

Expectancy of the Air Puff (US)

We next examined whether higher overprotective parenting would be related to a higher likelihood of children predicting the US in their expectancy ratings, particularly for the AB trials. A repeated measures ANOVA, with the between-subjects factor of overprotective parenting (low, high), and the within-subjects factor of CS type (AX+, BX-, AB), examined expectancy of the air puff as the dependent variable. There was not a main effect of overprotective parenting, $F(1, 17) = 0.00$, $p = 1.00$, or CS type, $F(2, 34) = 2.13$, $p = .13$, nor was there an overprotective parenting x CS type interaction, $F(2, 34) = .17$, $p = .84$. Thus, children whose parents were lower

and higher in overprotective parenting did not differ in cognitively discriminating among the danger, safety, and safety transfer cues.

Subjective Distress

We next conducted independent *t*-tests in order to examine whether higher overprotective parenting would be related to higher child-reported SUDS before completing the AX+/BX- paradigm and to higher change in SUDS from pre-to-post AX+/BX-. Children whose parents were higher in overprotective parenting ($M = 19.50, SD = 14.80$) did not differ from children whose parents were lower in overprotective parenting ($M = 11.25, SD = 16.20$) in pre-task SUDS, $t(16) = -1.13, p = .28$, though means are in the expected direction. Similarly, children whose parents were higher in overprotective parenting ($M = 25.69, SD = 9.08$) did not differ from children whose parents were lower in overprotective parenting ($M = 9.59, SD = 3.39$) in change in SUDS from pre- to -post-task $t(14) = -.74, p = .48$. Thus, children whose parents were low and high in overprotective parenting generally reported similar levels of distress.

INDIRECT ANALYSIS OF OVERPROTECTIVE PARENTING ON THE RELATIONSHIP BETWEEN PARENTAL WORRY AND CHILD SAFETY SIGNAL LEARNING

We next explored the hypothesis that overprotective parenting would have an indirect effect on the relationship between parental worry and child FPS during the fear inhibition transfer test using bootstrapping procedures. The bootstrapped unstandardized indirect effect was .01, and the 95% confidence interval ranged from -.60 to .32. Thus, the indirect effect of overprotective parenting on the relationship between parental worry and child FPS during the critical test of fear inhibition was not statistically significant.

PARENTAL PTSD AND DEPRESSION

Parental PTSD

In order to examine the secondary hypothesis that higher parental PTSD would predict greater FPS to the safety cues but not to the danger cues, bivariate correlations between parental PTSD and child FPS to each CS (AX+, BX-, AB) during each acquisition block (early, middle, late) and during the fear inhibition transfer test were conducted. As seen in Table 9, parental PTSD (interview and self-report) was not strongly related to child FPS to the danger or safety cues during any of the three blocks of acquisition. Additionally, parental PTSD was not strongly associated with child FPS in the critical safety signal transfer test.

Parental Depression

Next, in order to examine the relationship between parental depressive symptoms and FPS to the threat and safety cues, bivariate correlations between parental depressive symptoms and child FPS to each CS (AX+, BX-, AB) during each acquisition block (early, middle, late) and during the fear inhibition transfer test were conducted. As seen in Table 9, higher parental depression was strongly related to lower child FPS to both the danger and safety cues during the first block of trials. However, parental depression was not strongly related to child FPS to the danger or safety cues during middle or late acquisition, nor was it strongly associated with FPS in the critical safety signal transfer test. Therefore, although higher parental depression was related to an initial blunting of child FPS, parental depression was not closely associated with child FPS following early acquisition.

OTHER FACTORS POTENTIALLY RELATED TO CHILD FEAR- POTENTIATED STARTLE TO DANGER, SAFETY AND SAFETY TRANSFER CUES

Child Gender

In order to examine the effect of child gender on FPS to the danger and safety cues, a repeated measures ANOVA, with the between-subjects factor of gender (male, female), and within-subjects factors of CS type (AX+, BX-) and acquisition block (early, middle, late), examined FPS during acquisition as the dependent variable. There was not a main effect of child gender, $F(1, 18) = .03, p = .87$, CS type, $F(1, 18) = .40, p = .53$, or acquisition block, $F(1.53, 27.51) = 2.37, p = .11$. There was no child gender x CS type interaction, $F(1, 18) = .20, p = .66$, but there was a child gender x acquisition block interaction, $F(1.53, 27.51) = 5.77, p = .01$. Among boys, FPS during early acquisition ($M = 19.40, SD = 16.56$) was higher than FPS during middle acquisition ($M = 4.60, SD = 8.89$), $p = .02, d = 1.11$, and late acquisition ($M = 4.03, SD = 12.74$), $p = .02, d = 1.04$. By contrast, among girls, FPS did not decrease significantly over time. There was no child gender x CS type x acquisition block interaction, $F(2, 36) = 1.01, p = .38$.

To examine the effect of child gender on final learning of AX+, BX-, and transfer of that learning to AB, a repeated measures ANOVA, with the between-subjects factor of child gender (male, female), and the within-subjects factor of CS type (AX+, BX-, AB), examined FPS during late acquisition and the transfer test as the dependent variables. There was no main effect of child gender, $F(1, 18) = .28, p = .60$, or CS type, $F(2, 36) = 2.09, p = .14$, and no child gender x CS type interaction, $F(2, 36) = 1.23, p = .31$. Thus, males and females did not differ in their responding to the danger and safety cues; however, males exhibited a decrease in FPS following early acquisition, whereas females did not.

Child Age

In order to examine whether child age is related to child fear responding or inhibition, bivariate correlations were conducted between child age and FPS to each CS type (AX+, BX-, AB) during each acquisition block (early, middle, late) and during the transfer test. As seen in Table 10,

correcting for multiple analyses, child age was not strongly related to FPS to the danger or safety cues during any of the three blocks of acquisition, nor was it strongly associated with FPS to the safety transfer cue during the transfer test.

Child Anxiety

In order to examine whether child anxiety is related to child fear responding or inhibition, bivariate correlations were conducted between child anxiety (self- and parent-reported) and startle potentiation to each CS (AX+, BX-, AB) during each acquisition block (early, middle, late) and during the transfer test. As seen in Tables 10 and 11, correcting for multiple analyses, child anxiety was not strongly related to FPS to the danger or safety cues during any of the three blocks of acquisition, nor was it strongly associated with FPS to the safety transfer cue during the transfer test. However, although not reaching statistical significance after correcting for multiple comparisons, higher parent-reported child anxiety was strongly associated with lower FPS to the danger cue during middle acquisition, and higher child self-reported anxiety was moderately related to higher FPS to the safety cue during the same acquisition block.

Child Depression, PTSD, and Internalizing and Externalizing Symptoms

In order to examine whether child depression, PTSD, and internalizing and externalizing symptoms are related to child fear responding or inhibition, bivariate correlations were conducted between each of these variables and FPS to each CS (AX+, BX-, AB) during each acquisition block (early, middle, late) and during the transfer test. As seen in Tables 10 and 11, child depression, PTSD, and internalizing and externalizing symptoms were not strongly related to FPS to the danger or safety cues during any of the three blocks of acquisition, nor were they strongly associated with FPS to the safety transfer cue during the safety signal transfer test.

DISCUSSION

Impairments in safety signal learning, or fear inhibition, are thought to be a hallmark of (e.g., Grillon & Morgan, 1999; Jovanovic et al., 2009; Lissek et al., 2010, 2014; Waters, Henry, & Neumann, 2009), and potential risk factor for (e.g., Buss, Davis, Kiel, Brooker, Beekman, & Early, 2013; Craske et al., 2012; Jovanovic et al., 2010, 2014; Jovaonvic & Ressler, 2010; Reeb-Sutherland et al., 2009), anxiety-related psychopathology. This was the first study to examine child safety signal learning in relation to overprotective parenting and parental symptoms of psychopathology, both of which have been implicated in the etiology of child anxiety. Specifically, child fear-potentiated startle (FPS) was examined within a conditional discrimination (AX+/BX-) paradigm in relation to overprotective parenting and parental worry, PTSD, and depression. Children whose parents were higher in overprotective parenting had higher FPS to the safety transfer cue (AB) during the critical safety signal transfer test than to the safety cue (BX-) during the final block of acquisition. In other words, higher levels of overprotective parenting behaviors predicted reduced fear inhibition from the safety cue in the presence of the danger cue. Additionally, higher parental depression was associated with lower initial child FPS to both the danger (AX+) and safety (BX-) cues, indicating that the blunting effects that are typically found for depression on fear responding (Lang & McTeague, 2009; Melzig, Weike, Zimmermann, & Hamm, 2007) transferred to children whose parents reported higher symptoms of depression. By contrast, children whose parents reported higher symptoms of worry did not differ from children whose parents reported lower symptoms of worry in FPS to the danger, safety, or safety transfer cues. Parental PTSD was also not strongly associated with child fear responding or inhibition, though as discussed below, these findings should be interpreted with caution. Collectively, these findings point to potential mechanisms underlying the link between parent and child anxiety-related disorders; enhancing understanding of the

impact that parenting style and parental psychopathology have on a proposed biomarker, specifically impaired safety signal learning, for anxiety-related psychopathology (e.g., Jovanovic & Ressler, 2010) in children. These findings also further elucidate the relationship between child anxiety and fear responding and inhibition during a critical period of child development.

HIGHER OVERPROTECTIVE PARENTING PREDICTED POORER CHILD SAFETY SIGNAL LEARNING

As predicted, children whose parents were higher in overprotective parenting demonstrated impaired transfer of fear inhibition from the safety cue to the safety transfer cue during the critical test of safety signal learning. This finding fits with theoretical models that posit overprotective parenting behavior as a key pathway involved in the intergenerational transmission of anxiety-related psychopathology (e.g., Borelli, Margolin, & Rasmussen, 2015; Lebowitz et al., 2016). According to these models, overprotective parents repeatedly provide their children with signs that caution of danger, even when the chances of risk ahead are low. These messages, whether explicit or implicit, may inflate children's perceptions of risk and increase the likelihood of children staying on the anxious path in front of them. Never straying far from this path, these children may, in turn, later struggle in distinguishing true danger from the non-dangerous signs that they have mistakenly learned to interpret as threatening. This early learning history may act in conjunction with, or independently of, a genetic predisposition toward fearful responding not only to threatening stimuli, but also to stimuli that should signal safety. Specifically, exaggerated amygdala activity, weakened inhibitory prefrontal control of the amygdala, and genetic polymorphisms may contribute to or result from impairments in fear inhibition (Jovanovic & Ressler, 2010). Notably, this failure to inhibit fear to innocuous stimuli is likely most problematic (Craske et al., 2012; Jovanovic et al., 2010), as it may interfere with

effective functioning and prevent the acquisition of new, non-fearful associations. Coupled with a dearth of corrective learning experiences, these impairments in safety signal learning may lead to an internalization of the belief that the world is fraught with danger, which may then contribute to both the emergence and maintenance of child anxiety-related psychopathology (Rapee, 2009). These theoretical models are consistent with findings linking parenting to child anxiety, both cross-sectionally (de Wilde & Rapee, 2008; van der Bruggen Stams, & Bogels, 2008) and longitudinally (Borelli, Margolin, & Rasmussen, 2015; Edwards et al., 2010).

Critically, however, while prior studies have looked for a direct connection between overprotective parenting and child anxiety, the present study is the first to take a timely step backward in order to also examine whether, as theory suggests, children whose parents are overprotective really do inaccurately identify safety cues as threatening. This is especially important given that at least one study failed to find a strong relationship between overprotective parenting and child anxiety (Clarke et al., 2013). It is possible that by focusing solely on child anxiety and its relationship to overprotective parenting, the study by Clarke and colleagues missed more proximal effects of overprotective parenting on children, such as impaired safety signal learning, that may emerge prior to the onset of debilitating symptoms of worry and anxiety.

Interestingly, when examining the relationship between overprotective parenting and child anxiety directly, the current investigation found a strong association between these variables only when child anxiety was measured via parent report. In fact, child anxiety was virtually unrelated to overprotective parenting when the former was examined from the child's perspective. These discrepant results converge with those of prior studies showing divergence in parent and child reports of child psychopathology (e.g., Lagattuta, Sayfan, & Bamford, 2012),

and highlight the importance and utility of multi-informant approaches to child assessment.

By contrast, the finding from the present study of a moderate but non-significant relationship between higher overprotective parenting and higher parental worry and clinician-rated PTSD (Table 4) is inconsistent with previous studies suggesting a more robust link between overprotective parenting and parental symptoms of anxiety (e.g., Clarke et al., 2013; Edwards et al., 2010). However, this discrepancy in findings may be an artifact of the constructs under examination. Whereas prior studies assessed the relationship between overprotective parenting and parental symptoms of anxiety more generally, the present investigation examined overprotective parenting in relation to the more specific construct of parental worry. Although worry consistently demonstrates strong associations with anxiety (e.g., Olatunji et al., 2010), the construct of worry does not capture less cognitive processes (e.g., behavioral avoidance, physiological reactions) of anxiety-related disorders. Alternatively, this discrepancy may stem from the present study's use of a largely non-clinical sample of parents and their 8-11-year-old children as opposed to a sample of parents of preschoolers (Edwards et al., 2010) or children with anxiety disorders (Clarke et al., 2013). Indeed, it may be that the strength of the relationship between overprotective parenting and parental anxiety varies with the age and anxiety level of one's child. Similarly, it may also be that the relationship between overprotective parenting and parental anxiety becomes most evident in parents reporting more clinically significant levels of anxiety.

Ironically, the lack of a strong relationship between overprotective parenting and parental worry in the present study potentially magnifies the importance of the finding of greater safety signal learning impairments in children whose parents were higher in overprotective parenting. More specifically, overprotective parenting exerted an effect on child safety signal learning when

parental anxiety did not, suggesting that overprotective parenting may be a viable target for the prevention and treatment of anxiety-related disorders in children. Indeed, a number of parent-training techniques (see Wei & Kendall, 2014 for a review) have been implemented within family-based interventions for child anxiety and could be used specifically to address overprotective parenting. These strategies include 1) psychoeducation about the costs of overprotective parenting; 2) training in recognizing overprotective parenting and in granting autonomy (Bodden et al., 2008); 3) training in encouraging non-avoidant behavior and adaptive coping (Silverman et al., 1999); 4) cognitive restructuring in order to identify and modify thoughts underlying overprotective parenting behaviors (Bodden et al., 2008); and 5) anxiety management (Khanna & Kendall, 2009). Alternatively, these techniques could be taught within an exclusively parent-based intervention (e.g., Lebowitz, Omer, Hermes, & Scahill, 2014). Such efforts aimed at reducing the frequency of overprotective parenting behaviors could allow children to explore and seek out the corrective learning experiences needed in order to effectively distinguish between what is and is not safe. This could, in turn, either decrease children's vulnerability to developing later anxiety-related psychopathology or, for children who are already struggling with anxiety, increase the likelihood of them experiencing remission from their symptoms.

PARENTAL WORRY NOT STRONGLY RELATED TO CHILD SAFETY

SIGNAL LEARNING

Contrary to our predictions, children whose parents were higher in worry did not differ significantly from children whose parents were lower in worry on FPS during the critical transfer test. In other words, within a largely non-pathological sample, higher parental worry is not a strong predictor of greater child impairments in fear inhibition. This finding is inconsistent with

previous findings of impaired fear inhibition in adults with GAD (Lissek et al., 2014), though it is important to note that worry was not assessed in the children in this study but in their parents. Consequently, it is possible that we would have found a relationship between worry and child FPS had we examined worry in children. Alternatively, a stronger relationship may have emerged between parental worry and child FPS had we examined worry in a larger sample of parents engaging in more pathological levels of worry.

Given the internal nature of worry (Heimberg et al., 2004), it may be that parental worry is less visible to children than other symptoms of anxiety and thus, may have less of an effect on children's defensive responding. Stated another way, higher levels of worry in parents may not necessarily translate into higher levels of communication of this worry from parents to their children. It may therefore be that parental worry only impacts child safety signal learning to the extent that parents express their worries to their children.

Notably, whereas children whose parents were lower in worry had similar levels of FPS throughout acquisition, children whose parents were higher in worry trended toward having greater FPS to the conditioned stimuli during early acquisition followed by a decrease in FPS with learning. These results align with previous findings in psychiatrically healthy adults of an association between higher levels of worry and larger FPS to threat cues (Dunning & Hajcak 2015). Additionally, the finding of heightened fear responding during early acquisition when the experimental contingencies were likely most ambiguous is consistent with evidence linking pathological worry to higher intolerance of uncertainty (Dugas, Marchand, Ladouceur, 2005) and a higher likelihood of interpreting ambiguous stimuli as threatening (Aikins & Craske, 2001). Children whose parents were higher in worry may have been more distressed by the ambiguity of the early AX+/BX- trials and more likely to interpret the stimuli presented during these trials as

threatening, thus leading to higher initial FPS. Although not examined in the present study, this mechanistic interpretation could be a consequence of the cross-generational transfer of intolerance of uncertainty, possibly via modeling or information transfer (Fisak & Grills-Taquechel, 2007), from parents who are higher in worry to their children.

BLUNTED FEAR RESPONDING IN CHILDREN OF PARENTS WITH HIGHER SYMPTOMS OF DEPRESSION

Parental depression was, after the initial block of trials, only weakly associated with child FPS. However, during the first block of acquisition, parental depression was strongly related to reduced child FPS to both the danger and safety cue (Table 9), suggesting that having a depressed parent may be related to blunted child fear responding. These results are consistent with findings of reduced fear responding to threat in adults with depression (Lang & McTeague, 2009; Melzig, Weike, Zimmermann, & Hamm, 2007) and extend these effects to their children. In fact, given little evidence for learning during early acquisition within conditional discrimination paradigms (e.g., Jovanovic et al., 2014), the danger and safety cues may have been considered to be similarly threatening during this initial block of trials. Thus, higher parental symptoms of depression were essentially associated with lower initial child FPS to stimuli that were likely perceived to be threatening or aversive. One possible explanation for these findings is that children of parents who are higher in depression may utilize avoidant coping strategies aimed at reducing the aversiveness of unpleasant circumstances (Grillon & Baas, 2003), particularly when approaching novel situations.

Indeed, potential explanations for this attenuation in fear responding include evolutionary accounts of depression, which argue that reduced responding may serve an adaptive function of conserving energy in unpropitious situations (Nesse, 2000). Alternatively, the present findings

may reflect maladaptive brain plasticity and brain circuit dysfunction secondary to depression that manifest in the form of dampened defensive responding (Lang, McTeague, & Bradley, 2014). Regardless of the mechanism, the finding that higher parental symptoms of depression were related to lower child FPS in the present study is notable. These findings are consistent with the theory of Gotlib and colleagues, which proposes that alterations in stress reactivity may be one mechanism underlying the familial aggregation of depression (Gotlib, Joorman, & Folland-Ross, 2014). That said, whereas Gotlib and colleagues (2014) posit that it is the transmission of *hyper-reactivity* to negative stimuli that may partially explain this increased risk, the present findings provide evidence for the cross-generational transmission of *hypo-reactivity* in the face of threat. Critically, these findings are not mutually exclusive, with the former potentially supporting findings of the transmission of general internalizing symptoms within families (Starr et al., 2014) and the latter potentially better accounting for findings of depression-specific transmission (Hammen, Shih, & Brennan, 2004).

PARENTAL PTSD NOT STRONGLY RELATED TO CHILD SAFETY

SIGNAL LEARNING

Similar to the findings for parental worry, parental PTSD was also not strongly linked with child FPS to either the danger or safety cues. This finding stands in contrast to a large body of research in adults linking PTSD with exaggerated responding to both threat (e.g., Blechert et al., 2007; Glover et al., 2011; Griffin, 2008; Norrholm et al., 2011; Pole et al., 2009; Wessa & Flor, 2007) and safety (Jovanovic et al., 2009; 2010) cues. Additionally, the findings from the current study are inconsistent with the theory presented in the introduction that parents with PTSD may fail to model appropriate safety signal responding for their children, thus resulting in child impairments in safety signal learning. However, though it is possible that parental PTSD

has little influence on how children learn to distinguish between threat and safety cues and how they learn to inhibit fear to the latter, caution must be taken in interpreting the present findings given that the sample had very few individuals with trauma exposure or related symptoms of PTSD.

It is possible that had we examined the effects of parental PTSD in a larger, clinical sample that we would have found a stronger effect of parental PTSD on child fear responding and inhibition. Given that maternal PTSD symptoms are uniquely related to offspring symptoms of PTSD (Starr et al., 2014), it is likely that parents and children with PTSD share one or more biomarkers, such as impaired safety signal learning, in common. It is also likely that the effects of parental PTSD on child safety signal learning vary depending on the degree of PTSD-related impairment, as well as the specific symptoms that the parent is experiencing. More specifically, although it is possible for an individual with the minimum number of six PTSD symptoms once per month to meet DSM-5 criteria for PTSD, that person would look very different clinically from an individual experiencing all twenty of the DSM-5 symptoms of PTSD many times per week. In one study examining safety signal learning in adults with PTSD, those who reported higher PTSD symptoms had greater impairments in safety signal learning than those who reported lower PTSD symptoms (Jovanovic et al., 2009). By contrast, other findings have shown reduced FPS to idiographic trauma scripts in individuals with more extensive trauma histories and more severe PTSD symptoms (Lang & McTeague, 2009). Accordingly, the extent to which parental PTSD influences child fear responding may vary depending on parents' symptom profiles and trauma histories. Future studies examining the relationship between parental PTSD and child safety signal learning should ideally be designed to detect these varied effects, and should thus include parents with a wide range of PTSD symptoms and both discrete and

extensive trauma histories.

COGNITIVE AND PHYSIOLOGICAL INDICATORS OF CHILD SAFETY SIGNAL LEARNING

In contrast to prior studies that found that children as young as eight can learn the contingencies associated with the stimuli in conditional discrimination paradigms (Glenn et al., 2011; Jovanovic et al., 2014), the retrospective expectancy ratings in the present study revealed that children were not fully aware of the experimental contingencies. Specifically, children were not significantly more likely to predict that the air puff had been paired with the danger cue than they were to predict that the air puff had been paired with the safety and safety transfer cues. Further, a similar pattern emerged when examining the effect of higher parental worry and overprotective parenting on expectancy ratings, consistent with previous studies showing that contingency awareness is not impacted by anxiety-related symptoms (Jovanovic et al., 2009, 2010, 2014).

On the one hand, these findings may help explain why overall FPS was not significantly greater to the danger cue than to the safety cue in the present study, especially in light of findings suggesting that explicit knowledge of the contingencies associated with threat and safety cues might be a prerequisite for appropriate fear inhibition (Jovanovic et al., 2006). Although prior work suggests that a developmental shift may occur in the ability to process safety signals around age 10 (Glenn et al., 2011; Jovanovic et al., 2014), the findings of poor cognitive and physiological discrimination between threat and safety cues in this study may indicate that the developmental honing of discrimination between danger and safety signals had yet to take place in the study's sample. Alternatively, it is possible that the study did not include enough acquisition trials for children to learn the contingencies associated with the conditioned stimuli.

It is also possible that the finding of poor safety signal learning at both the cognitive and physiological levels is an artifact of the current study's small sample size, as the general pattern of findings was in the expected direction. Despite not being statistically significant, children generally responded with greater FPS to the danger cue than to the safety cue and tended to rate the danger cue as more likely to be paired with the air puff than the safety cue.

LACK OF A STRONG INDIRECT EFFECT OF OVERPROTECTIVE PARENTING

Given that overprotective parenting has been proposed as a contributing factor in the link between parent and child anxiety-related psychopathology (e.g., Edwards et al., 2010), the present finding of a lack of a strong indirect effect of overprotective parenting on the relationship between parental worry and child FPS to the safety transfer cue was unexpected. In addition, this finding was inconsistent with the findings of Borelli and colleagues (2015), who found an indirect effect of the related construct of parental overcontrol on the relationship between maternal and child anxiety assessed two and a half years later. Notably, however, this discrepancy in findings may be explained by differences in the constructs that were investigated, as the present study specifically examined the indirect effect of overprotective parenting on the relationship between parental worry and child safety signal learning as opposed to the association between parent and child anxiety. In other words, it may be that overprotective parenting is more likely to influence the relationship between parent and child anxiety than the relationship between parental worry and child FPS. More specifically, given that worry is largely an internal process (Heimberg et al., 2004) that may, at non-pathological levels, regulate physiological arousal associated with anxiety (e.g., Ottaviani et al., 2014), higher parental worry within a psychiatrically healthy sample could translate into more mindful parenting and greater

consideration of the impact that certain behaviors might have on one's children. Accordingly, sub-clinical worry may curb the anxiety that might otherwise fuel overprotective parenting behaviors, thus reducing the influence of overprotective parenting on the relationship between parental worry and child FPS.

IMPAIRED DISCRIMINATIVE CONDITIONING RELATED TO CHILD ANXIETY BUT NOT AGE

Although not a central focus of this study, higher child anxiety symptoms were associated with lower FPS to AX+ and higher FPS to BX- during the second block of acquisition, suggesting slower learning of the contingencies associated with the conditioned stimuli. Anxiety may be more strongly associated with impaired fear inhibition in the presence of safety cues than with heightened fear responding in the context of danger (Duits et al., 2015). Whereas learning to be fearful of dangerous situations has adaptive evolutionary implications, failing to inhibit fear in non-threatening contexts may interfere with effective functioning and corrective learning. Indeed, elevated startle responding to safe, but not dangerous, cues predicts child anxiety symptoms both cross-sectionally (Jovanovic et al., 2014) and longitudinally (Craske et al., 2012). It is worth noting, however, Jovanovic and colleagues (2014) found enhanced discriminative conditioning (i.e., elevated responding to threat cues relative to safety cues) in 10-13 year-olds, but not in 8-9 year-olds, who reported higher symptoms of anxiety, which the present study did not. Instead, the present study found an association between higher anxiety and poorer discriminative conditioning, and also found that child age was not a strong predictor of child FPS to either the danger or safety cues. One possible explanation for this discrepancy is that Jovanovic and colleagues (2014) recruited their sample of children from a low-income, urban, highly trauma-exposed population whereas the present study recruited a

younger sample of children who were from a more affluent population with a relatively low rate of trauma exposure. It is therefore possible that when examining safety signal learning across development, the ability to distinguish between threat and safety cues varies with both age and trauma exposure.

LIMITATIONS AND FUTURE DIRECTIONS

As noted above, this study has a small sample size, which rendered the study underpowered. A second, related limitation is that the study consisted of a largely psychiatrically healthy, community sample, which likely restricted the range of parental symptoms of worry, PTSD, and depression, thus limiting generalizability of the findings. Future studies should include separate groups of parents diagnosed with depression and anxiety-related disorders in addition to a psychiatrically healthy control group. Alternatively, future studies could take a more dimensional approach, consistent with the NIMH Research Domain Criteria (RDoC) initiative (Kozak & Cuthbert, 2016), and examine larger samples of children with intact to grossly impaired safety signal learning in order to identify the parent and child correlates of child responding to threatening and non-threatening cues.

Given these sample limitations and our interest in parental worry and overprotective parenting specifically, we created high and low groups for parental worry and overprotective parenting using median splits, thus allowing us to maximize variability. Although median splits are generally not recommended in part due to concerns that creating arbitrary groups may give way to misleading, oversimplified interpretations (MacCallum et al., 2002), median splits may be able to reveal important differences that otherwise might be lost and the risks of type I and II errors associated with their use are not as high or problematic as previously thought (Iacobucci et al., 2015).

Another limitation with the sample itself is that it only included one parent from each family, with the majority of included parents being female. Thus, this study was not capable of determining the unique or additive effects of maternal and paternal parenting style and psychopathology on child safety signal learning. Although prior longitudinal work has shown that child anxiety changes similarly regardless of whether children have one or two overprotective parents (Borelli, Margolin, & Rasmussen, 2015), the same study also found a stronger indirect effect of maternal overcontrol than paternal overcontrol on the relationship between parent and child anxiety. Further, studies examining the link between parent and child anxiety specifically have found an increased risk of child anxiety when both parents suffer from anxiety-related psychopathology (Li, Sundquist, & Sundquist, 2008; Steinhausen, Foldager, Perto, & Munk-Jorgensen, 2009). Moreover, it is theorized that the strength of these relationships likely varies throughout development, potentially intensifying during periods of increasing autonomy seeking (Borelli, Margolin, & Rasmussen, 2015). Thus, the use of a cross-sectional design does not allow for an examination of how child safety signal learning changes over the course of development, nor does it enable causal inferences to be made. It remains unclear whether higher overprotective parenting results in child safety signal learning impairments or whether parents engage in overprotective parenting behaviors in response to observing their children struggle to differentiate safe from unsafe cues in the environment. It is possible that the relationship between these variables is actually transactional, such that overprotective parenting encourages increased child fear in objectively safe contexts, which in turn, begets greater parent involvement and overprotection. Moreover, in two parent homes, the parenting style of the second parent may impact the relationship between these variables, thus further obscuring the direction of effects. Longitudinal designs that include multiple parents are

needed in order to better ascertain causality and potential iterative effects.

Additionally, limitations with the assessment strategy used in the present study must also be noted. First, the reliance on only self-report questionnaires to measure parental worry, depression, and overprotective parenting may have biased the findings. This may be particularly the case for overprotective parenting as prior work shows discordance between self-reported and observed overprotective parenting behaviors in mothers (Clarke et al., 2013) and similarly suggests that mothers' reports of their own parenting style, including overprotection, tend to be more favorable than child and partner reports of maternal parenting (Bögels, & van Melick, 2004). Furthermore, this study did not assess more general symptoms of parental anxiety such as avoidance and fear of anxiety-related sensations, nor did it examine safety signal learning in parents. This latter limitation prevented us from investigating the hypothesis that impairments in fear inhibition may be transmitted from parents to their children. Relatedly, it is possible that the included parents were not impaired in safety signal learning, thus reducing the likelihood of us finding impairments in their children. Alternatively, it is possible that overprotective parenting predicts child safety signal learning impairments over and above parents' own impairments in fear inhibition, or that the combination of overprotective parenting and impaired fear inhibition in parents predicts even greater child impairments in safety signal learning. In addition, this study did not include a clinical interview of child anxiety, PTSD, and depression. This would have provided valuable information particularly for child PTSD symptoms given that there was some discrepancy in parent and child reports of child trauma exposure, with some children reporting trauma exposure that was not endorsed in their parents' reports of child trauma exposure. Consequently, it is possible that at least some children endorsed symptoms of PTSD that were not related to a specific, DSM-5 Criterion A traumatic event. That said, although

conducting clinical interviews of child anxiety, PTSD, and depression would have provided useful information about specific diagnoses, structured assessments in a largely non-pathological sample would have provide limited, dichotomous information and would have greatly increased assessment burden for children.

An additional caveat is that the overprotective parenting and parent-reported child depression and internalizing symptoms of dyads that were not included in the FPS analyses ($n = 15$) were higher than those of dyads that were included in the FPS analyses. Although reasons for exclusion from these analyses varied, these included child distress during the psychophysiological task ($n = 4$) and artifacts or related problems ($n = 5$), which included excessive movement. Thus, it is possible that the AX+/BX- paradigm was too demanding or aversive for the children from dyads that were higher on baseline measures of parenting and child symptoms. Conversely, other dyads were excluded from the FPS analyses because their responses to the experimental stimuli were indistinguishable from their baseline EMG activity, suggesting that the psychophysiological task was unlikely to be too difficult for these children, and that they may have even experienced it as being boring.

Further, it is possible that our use of the noise alone (NA) trials from the same acquisition block as an index from which to calculate FPS to each CS may have obscured our findings, as some children may have exhibited heightened startle responses to the NA trials within the acquisition and testing phase due to carryover effects from the danger trial. Using the mean of the values from the NA trials during the habituation phase, prior to acquisition, may have served as a more useful anchor point from which to calculate FPS.

A final limitation and important area for future work concerns developing a better understanding of the phenomenon of impaired safety signal learning, including the biological

and neurological underpinnings of these impairments. Conceptually, inappropriate fear responding to safety cues may reflect deficient fear inhibition, an overgeneralization of fear from danger to safety cues, or carryover effects from the danger cues to the safety cues. Whereas impaired safety signal learning is most likely related to weakened prefrontal control of the amygdala, overgeneralization of fear and carryover effects from the danger to the safety cues may be most closely linked to hyperactivity of the amygdala (Jovanovic & Ressler, 2010). Of note, although this study was not designed to rule out these mechanistic accounts, it is unlikely that stimulus generalization could explain the safety signal learning impairments found given the very distinct shapes that were used in this study. Studies examining the overgeneralization of fear typically include stimuli such as circles that vary only subtly in size (e.g., Lissek et al., 2010; 2014), and thus that are much more similar, and likely, to result in overgeneralization, than the stimuli used in the current study. That said future studies that examine fear responding and inhibition to even more disparate conditioned stimuli, such as a combination of auditory and visual cues, could deepen knowledge of the factors that are directly implicated in impaired safety signal learning.

STRENGTHS AND CONCLUSIONS

The present study is the first to examine child safety signal learning in the context of parental overprotection and psychopathology. This is an important area of inquiry given evidence suggesting that child impairments in safety signal learning may be a risk factor for the development of later anxiety-related psychopathology (e.g., Craske et al, 2012) and given accumulating evidence implicating parenting in the etiology of both child anxiety (Lebowitz et al., 2016) and depression (Goodman, 2007). Children whose parents were higher in overprotective parenting had impaired transfer of fear inhibition from the safety cue to the safety

transfer cue during the critical test of safety signal learning, indicating that overprotective parenting may be a viable target for prevention and intervention efforts aimed at reducing the incidence and course of child anxiety-related disorders.

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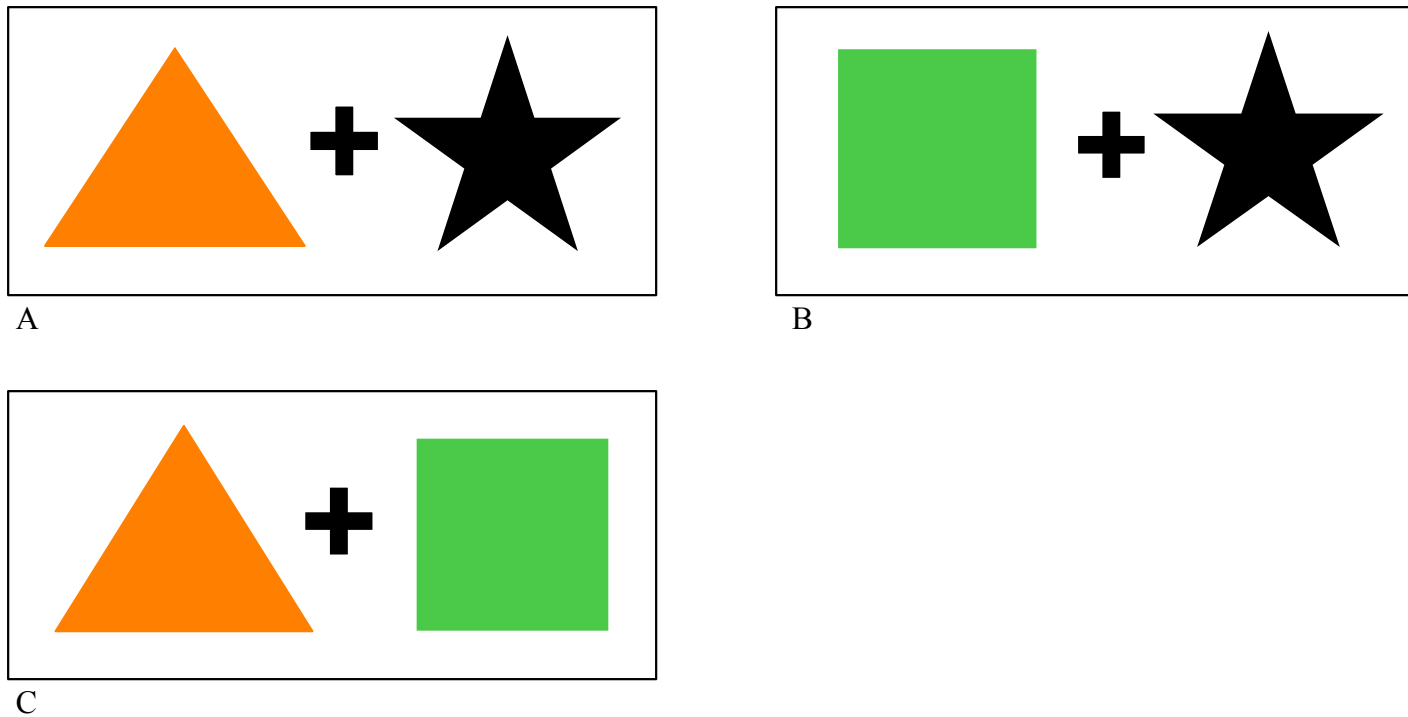


Figure 1. *Examples of the CSs presented on the computer monitor during fear acquisition and the transfer test. (A) AX+, the reinforced stimulus; (B) BX-, the non-reinforced stimulus; and (C) AB, the fear inhibition test stimulus.*

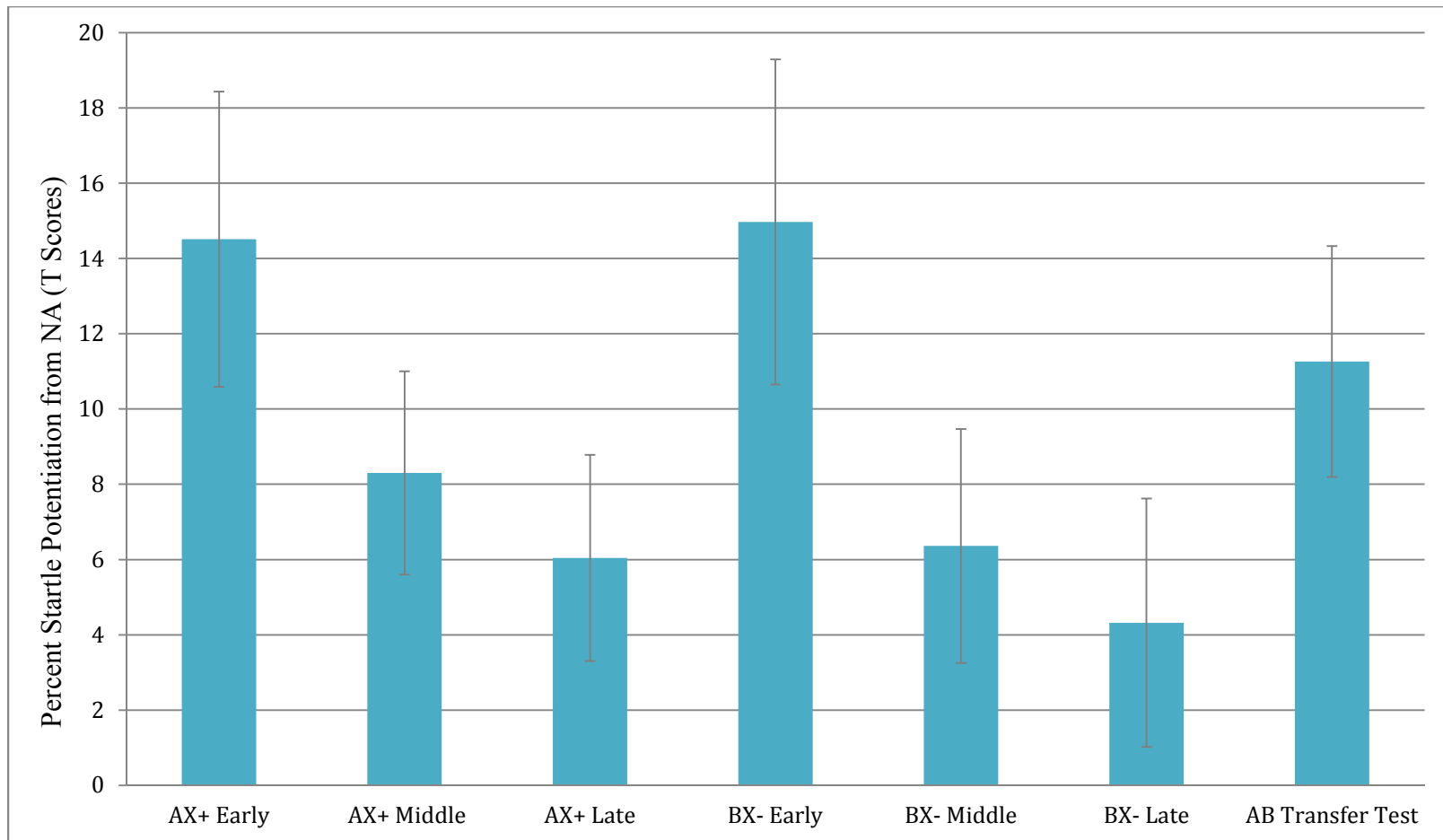


Figure 2. *Startle Potentiation to Each CS During Acquisition and the Transfer Test*

Note. Early = early acquisition; Middle = middle acquisition; Late = late acquisition. Error bars represent standard errors.

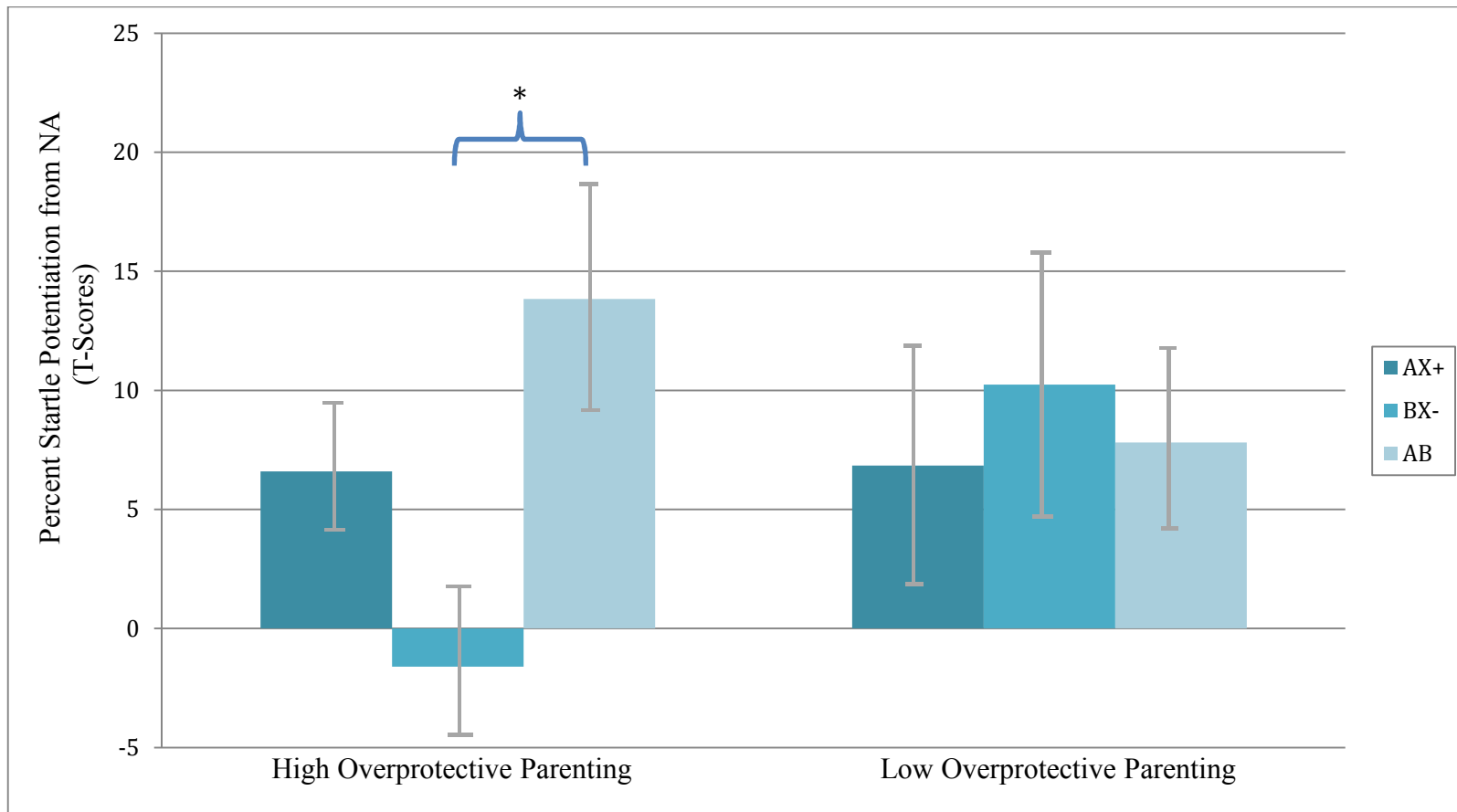


Figure 3. *The Effect of Overprotective Parenting on Startle Potentiation During the Transfer Test and the Last Block of Acquisition*

Note. * = $p < .05$

Table 1

Inclusion and Exclusion Criteria for Study

	Inclusion Criteria	Exclusion Criteria
Children	<ol style="list-style-type: none"> 1. Between the ages of 8-11. 2. Able to read, write, and speak English. 3. Normal hearing and normal or corrected-to-normal vision. 	<ol style="list-style-type: none"> 1. A parent-reported cognitive disability (not including ADHD).
Parents	<ol style="list-style-type: none"> 1. Between the ages of 18-65. 2. Able to read, write, and speak English. 3. Biological parent and primary caregiver of the eligible child. 	<ol style="list-style-type: none"> 1. Current diagnosis of substance dependence, schizophrenia, bipolar disorder, or other disorder with delusional content. 2. Depression severe enough to require immediate psychiatric treatment (i.e., active suicidality). 3. If trauma-exposed, no clear memory or an index trauma that occurred before the age of three years.

Table 2

Ranges, Means, and Standard Deviations for Parent Interview and Self-Report Measures

Measure	Range	<i>M</i>	<i>SD</i>
Parental Worry (PSWQ)	20-69	42.88	14.32
Parental PTSD (PSSI-5)	0-41	4.82	10.11
Parental PTSD (PDS-5)	0-64	5.1	13.79
Parental Depression (QIDS)	0-20	4.91	5.11
Overprotective Parenting (OP)	10-66	32.25	13.40

Note. PSWQ = Penn State Worry Questionnaire; PSSI-5 = Posttraumatic Symptom Scale Interview for DSM-5; PDS-5 = Posttraumatic Diagnostic Scale for DSM-5; QIDS = Quick Inventory of Depressive Symptomatology; OP = Overprotective Parenting Measure.

Table 3

Ranges, Means, and Standard Deviations for Parent- and Child-Report Measures

Measure	Range	<i>M</i>	<i>SD</i>
Child Anxiety (RCADS)	2-43	16.35	10.39
Child Anxiety, Parent Report (RCADS-P)	4-55	16.28	11.46
Child Depression (RCADS)	0-17	5.35	3.68
Child Depression, Parent Report (RCADS-P)	0-13	3.92	3.45
Child PTSD (PTSD-RI)	0-50	8.39	12.09
Child PTSD, Parent Report (PTSD-RI-P)	0-63	5.92	14.57
Internalizing Symptoms (CBCL)	33-71	49.92	9.96
Externalizing Symptoms (CBCL)	33-76	50.84	10.93
SUDs Pre-Task	0-50	15.53	15.17
Δ SUDs Pre- Minus Post-	-50-40	-.85	17.83

Note. RCADS = Revised Child Anxiety and Depression Scale; RCADS-P = Revised Child Anxiety and Depression Scale, Parent Version; PTSD-RI = PTSD Reaction Index; PTSD-RI-P = PTSD Reaction Index, Parent Version; CBCL = Child Behavior Checklist for Ages 6-18; SUDs = subjective units of distress; Δ SUDs = Pre- AX+/BX- Minus Post- AX+/BX-.

Table 4

Bivariate Correlations among the Parent Interview and Self-Report Measures

	1	2	3	4	5
1. Parental Worry (PSWQ)	--				
2. Parental PTSD (PSSI-5)	.51*	--			
3. Parental PTSD (PDS-5)	.51*	.90*	--		
4. Parental Depression (QIDS)	.59*	.85*	.78*	--	
5. Overprotective Parenting (OP)	.34	.14	.43	.34	--

Note. * = $p < .05$, adjusting for multiple comparisons; PSWQ = Penn State Worry Questionnaire; PSSI-5 = Posttraumatic Symptom Scale Interview for DSM-5; PDS-5 = Posttraumatic Diagnostic Scale for DSM-5; QIDS = Quick Inventory of Depressive Symptomatology; OP = Overprotective Parenting Measure.

Table 5

Bivariate Correlations among the Parent-Report and Child Self-Report Measures

	1	2	3	4	5	6	7	8
1. Child Anxiety (RCADS)	--							
2. Child Anxiety, Parent Report (RCADS-P)	.20	--						
3. Child Depression (RCADS)	.75*	.23	--					
4. Child Depression, Parent Report (RCADS-P)	.10	.81*	.11	--				
5. Child PTSD (PTSD-RI)	.15	.73*	.25	.70*	--			
6. Child PTSD, Parent Report (PTSD-RI-P)	.10	.88*	.26	.77*	.76*	--		
7. Internalizing Symptoms (CBCL)	.18	.75*	.25	.67*	.68*	.64*	--	
8. Externalizing Symptoms (CBCL)	.10	.55	.22	.48	.43	.34	.71*	--

Note. * = $p < .05$, adjusting for multiple comparisons; RCADS = Revised Child Anxiety and Depression Scale; RCADS-P = Revised Child Anxiety and Depression Scale, Parent Version; PTSD-RI = PTSD Reaction Index; PTSD-RI-P = PTSD Reaction Index, Parent Version; CBCL = Child Behavior Checklist for Ages 6-18.

Table 6

Bivariate Correlations among the Parent Interview, Parent Self-Report, and Parent-Report Measures

	Parental Worry (PSWQ)	Parental PTSD (PSS-I-5)	Parental PTSD (PDS-5)	Parental Depression (QIDS)	Overprotective Parenting (OP)
Child Anxiety (RCADS-P)	.53	.05	.70*	.46	.59*
Child Depression (RCADS-P)	.45	.17	.64*	.48	.37
Child PTSD (PTSD-RI-P)	.31	.31	.56	.30	.14
Internalizing Symptoms (CBCL)	.53	.18	.58	.36	.49
Externalizing Symptoms (CBCL)	.42	.22	.40	.34	.34

Note. * = $p < .05$, adjusting for multiple comparisons; PSWQ = Penn State Worry Questionnaire; PSS-I-5 = Posttraumatic Symptom Scale Interview for DSM-5; PDS-5 = Posttraumatic Diagnostic Scale for DSM-5; QIDS = Quick Inventory of Depressive Symptomatology; OP = Overprotective Parenting Measure; RCADS-P = Revised Child Anxiety and Depression Scale, Parent Version; PTSD-RI-P = PTSD Reaction Index, Version; CBCL = Child Behavior Checklist for Ages 6-18.

Table 7

Bivariate Correlations among the Parent Interview Measures, Parent Self-Report Measures, and Child-Report Measures

	Parental Worry (PSWQ)	Parental PTSD (PSS-I-5)	Parental PTSD (PDS-5)	Parental Depression (QIDS)	Overprotective Parenting (OP)
Child Anxiety (RCADS)	.06	-.05	-.17	-.17	.02
Child Depression (RCADS)	.14	.11	.15	.05	.01
Child PTSD (PTSD-RI)	.44	.09	.81*	.49	.37

Note. $\alpha = .05$, adjusting for multiple comparisons; PSWQ = Penn State Worry Questionnaire; PSS-I-5 = Posttraumatic Symptom Scale Interview for DSM-5; PDS-5 = Posttraumatic Diagnostic Scale for DSM-5; QIDS = Quick Inventory of Depressive Symptomatology; OP = Overprotective Parenting Measure; RCADS = Revised Child Anxiety and Depression Scale; PTSD-RI = PTSD Reaction Index.

Table 8

Means and Standard Deviations for Fear-Potentiated Startle to the Conditioned Stimuli Across Acquisition and the Transfer Test and for Retrospective Expectancy Ratings of the Air Puff to the Conditioned Stimuli

Conditioned Stimuli	<i>M</i>	<i>SD</i>
Danger Cue (AX+)	9.62	9.99
Early Acquisition	14.51	17.53
Middle Acquisition	8.3	12.08
Late Acquisition	6.04	12.27
Expectancy of the Air Puff	0.25	0.79
Safety Cue (BX-)	8.55	10.11
Early Acquisition	14.97	19.36
Middle Acquisition	6.36	13.91
Late Acquisition	4.32	14.75
Expectancy of the Air Puff	-0.25	0.85
Safety Transfer Cue (AB)	11.26	13.74

Expectancy of the US	-0.05	.89
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Note. Fear-potentiated startle is reported in standard T-score units and was calculated as percent potentiation from the noise alone trials.

Table 9

Bivariate Correlations among Fear-potentiated Startle and Parent Interview and Self-report Measures

	AX Early	BX Early	AX Middle	BX Middle	AX Late	BX Late	AB Transfer
Parental Worry (PSWQ)	.22	.33	-.08	.03	.13	-.01	.04
Parental PTSD (PSS-I-5)	.10	-.21	-.02	-.16	-.15	.04	-.01
Parental PTSD (PDS-5)	.34	-.32	-.03	-.06	-.04	-.02	-.07
Parental Depression (QIDS)	-.58*	-.68*	.03	.12	.04	-.11	-.06
Parental Overprotective Parenting (OP)	-.07	.02	-.23	-.002	-.05	-.30	.04

Note. * = $p < .05$; Early = early acquisition; Middle = middle acquisition; Late = late acquisition; Transfer = safety transfer test; PSWQ = Penn State Worry Questionnaire; PSS-I-5 = Posttraumatic Symptom Scale Interview for DSM-5; PDS-5 = Posttraumatic Diagnostic Scale for DSM-5; QIDS = Quick Inventory of Depressive Symptomatology; OP = Overprotective Parenting Measure.

Table 10

Bivariate Correlations among Fear-Potentiated Startle and Child Age, Self-Report Measures, and Subjective Units of Distress

	AX Early	BX Early	AX Middle	BX Middle	AX Late	BX Late	AB Transfer
Child Age	.00	.16	.05	-.33	.09	.28	.02
Child-Reported Anxiety (RCADS)	-.27	-.24	-.27	.47	.20	.03	.07
Child-Reported Depression (RCADS)	-.28	-.30	-.32	.41	.06	.02	-.11
Child-Reported PTSD (PTSD-RI)	-.22	.27	-.12	-.26	-.33	.10	.24
SUDs Pre-Task	.15	.08	-.39	.10	.02	.13	-.04
Δ SUDs Pre-to-Post	.19	.38	-.01	-.66	-.22	-.30	-.27

Note. $\alpha = .05$, adjusting for multiple comparisons; Early = early acquisition; Middle = middle acquisition; Late = late acquisition; Transfer = safety transfer test; RCADS = Revised Child Anxiety and Depression Scale; PTSD-RI = PTSD Reaction Index; SUDs = subjective units of distress; ; Δ SUDs = Pre- AX+/BX- Minus Post- AX+/BX-.

Table 11

Bivariate Correlations between Fear-Potentiated Startle and the Parent-report Measures

	AX Early	BX Early	AX Middle	BX Middle	AX Late	BX Late	AB Transfer
Parent-Reported Anxiety (RCADS-P)	-.28	-.13	-.56	.26	-.31	-.20	-.20
Parent-Reported Depression (RCADS-P)	-.01	-.35	-.36	.08	-.31	-.15	.03
Parent-Reported PTSD (PTSD-RI)	.16	.34	-.45	-.36	-.09	-.12	-.42
Internalizing Symptoms (CBCL)	.34	.11	-.28	.01	-.25	-.07	.05
Externalizing Symptoms (CBCL)	.23	.20	.04	.40	.11	.30	-.06

Note. $\alpha = .05$, adjusting for multiple comparisons; Early = early acquisition; Middle = middle acquisition; Late = late acquisition; Transfer = safety transfer test; RCADS-P = Revised Child Anxiety and Depression Scale, Parent Version; PTSD-RI = PTSD Reaction Index; CBCL = Child Behavior Checklist for Ages 6-18.

VITA

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