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**GENETIC AND ENVIRONMENTAL INFLUENCES ON TACTILE REACTIVITY
FROM INFANCY TO TODDLERHOOD**

A Dissertation in

Psychology

by

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ABSTRACT

Touch is the main modality through which infants learn from and interact with their environments and caregivers, and infants who have prolonged difficulty tolerating or responding to tactile input may be at risk for atypical development and social-emotional problems. Using a prospective adoption design, the current study examined the development of tactile reactivity between 9 and 18 months, in relation to genetic and environmental influences. This study measured children's ($N = 561$; 57% male) tactile reactivity at 9 and 18 months during a novel tactile task in which adoptive parents painted children's hands and feet and pressed them to paper to make a picture. Adoptive parents were rated, using the physical involvement subscale from the Home Observation for Measurement of the Environment inventory, on the amount of support they provided children for engaging in their physical environments. Birth parents reported their level of negative affect on the Adult Temperament Questionnaire – Short Form, and birth mothers reported on prenatal environment. Overall, children showed a significant decrease in tactile reactivity from 9 to 18 months; however, children whose birth mothers reported higher levels of negative affect showed significantly less change in tactile reactivity. Findings suggest that 9 to 18 months is a time period characterized by significant change, namely a normative decrease, in tactile reactivity, but some children show more persistent tactile reactivity due to genetic influences, which may have an impact on their cognitive, motor, and social-emotional development.

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INTRODUCTION

Infants depend on sensory experiences to communicate and bond with caregivers, be successfully soothed, and learn about the world. Indeed, a large body of research has shown that early sensory experiences scaffold the development of higher-order cognitive, motor, and regulatory processes (Cascio, 2010; Feldman, Eidelman, Sirota, & Weller, 2002; Feldman, Singer, & Zagoory, 2010; Feldman, Weller, Sirota, & Eidelman, 2003). There is also evidence that infants show individual differences in behavioral responses to sensory stimuli (i.e., sensory processing patterns; Baranek, David, Poe, Stone & Watson, 2006; Ben-Sasson et al., 2007; Dunn, 2001; Dunn & Daniels, 2002; Miller, Anzalone, Lane, Cermak, & Osten, 2007; Miller, Reisman, McIntosh, & Simon, 2001). Whereas most infants are able to use early sensory experiences to engage in and learn from their environments, infants with atypical sensory responses (e.g., hypo- or hyper-responsiveness) have difficulty tolerating or responding to sensory input, which may have implications for later development. For instance, atypical sensory responses assessed as early as 8 months have been associated with developmental disabilities, poor motor skills, and difficulties regulating attention, emotion, and behavior (Ben-Sasson et al., 2007; DeGangi, Porges, Sickel, & Greenspan, 1993; Eeles et al., 2013; Mammen et al., 2015).

In particular, infants' responses to touch may have a far-reaching impact on child development because, as the first sensory system to develop, it is the main modality through which infants learn from and interact with their environments and caregivers (Cascio, 2010). Studies of humans and animals have shown that the exchange of tactile stimulation plays a major role in parent-infant interaction (e.g., tickling, rocking, cuddling; Hernandez-Reif, Diego, & Field, 2007) and in child regulatory development (Feldman et al., 2010; Ferber & Makhoul, 2004; Polan & Hofer, 1999). Indeed, higher levels of touch between parent and infant (e.g., skin-

to-skin contact, affectionate touch, tactile game play) have been linked to secure attachment relationships, more positive social responses in infants, and positive reciprocal parent-infant interactions (Feldman et al., 2002; Ferber et al., 2008; Hertenstein, Verkamp, Kerestes, & Holmes, 2006; Stack & Muir, 1992). As a result, when infants have problems tolerating or responding positively to touch, the parent and child may have difficulties developing a relationship that will support the development of age-appropriate social and self-regulatory behaviors (Field, 2010). These studies, along with evidence that tactile reactivity is related to, but distinct from other developmental processes and outcomes (e.g., psychopathology, effortful control; Hopkins, Lavigne, Gouze, LeBailly, & Bryant, 2013; Van Hulle, Schmidt, & Goldsmith, 2012) suggest the need to examine, specifically, the development of tactile reactivity across infancy and toddlerhood.

Although there is evidence that reactivity to sensory stimulation is influenced by genetic (Goldsmith, Van Hulle, Arneson, Schreiber, & Gernsbacher, 2006; Van Hulle, Schmidt, & Goldsmith, 2012), prenatal (e.g., prenatal stressors, maternal weight gain; Crepeau-Hobson, 2009), and environmental factors (e.g., the parent-child relationship; DeGangi, Sickel, Kaplan, & Wiener, 1997; Epstein, Saltzman-Benaiah, O'Hare, Goll, & Tuck, 2008; Miller, 2006; Sameroff & Fiese, 2000), little is known about how the interaction among these various factors influences the development of reactivity to sensory stimulation across time and context. To our knowledge, there have been no longitudinal, genetically informed studies of reactivity to touch. Indeed, most studies of child sensory processing have examined biologically related parents and children, which does not allow for the disentanglement of genetic and environmental influences on the development of child sensory processing patterns. As a result, we have limited understanding of how reactivity to sensory stimuli develops and is influenced by genetic and environmental

factors across time. Such research could inform current conceptualizations of normal and abnormal development because studies have shown that sensory processing patterns, particularly difficulties accepting tactile stimulation (Dunn & Bennett, 2002), are related to psychopathologies across the lifespan (e.g., Autism Spectrum Disorder, anxiety disorders, mood disorders, conduct problems, Attention Deficit Hyperactivity Disorder, schizophrenia; Brown, Cromwell, Filion, Dunn, & Tollefson, 2002; Cheung & Siu, 2009; Engel-Yeger & Dunn, 2011; Ermer & Dunn, 1998; Hoehn & Baumeister, 1994; Lane, Reynolds, & Thacker, 2010; Parham & Mailloux, 2001; Rieke & Anderson, 2009). There is evidence suggesting sensory processing difficulties in early childhood play a causal role in the development of later mental health problems (Bron, Van Rijen, Van Abeelenm, & Lambregtse-Van Den Berg, 2012; Ornitz, 1983). For instance, Gerrard and Rugg (2009) have proposed that abnormal responses to sensory stimuli hamper the development of skills necessary for language and social interaction, leading to symptoms of autism. Still, not all young children who have difficulties responding to touch go on to develop psychopathology, and research examining the impact of genetic and environmental factors on the development of tactile reactivity may increase understanding of different developmental pathways leading from early difficulties with tactile reactivity.

In addition to increasing understanding of atypical development, a longitudinal investigation of genetic and environmental influences on reactivity to touch could reveal developmental time-points when interventions may be most effective. Indeed, some children may be highly reactive to touch because of genetic influences, which could have an impact on the responses they evoke from parents and could also have implications for how children respond to different rearing environments. However, because prior research has not examined interplay between genetic and environmental influences on child reactivity to sensory stimulation across

time and context, it is unclear how sensory reactivity develops or how and when to provide effective interventions. The proposed study aims to examine reactivity to tactile stimulation: 1) in a naturalistic, social context, 2) between 9 and 18 months, a period of development when sensory responses appear to be most malleable (Dunn & Daniels, 2002), and 3) in relation to genetic and environmental influences using a prospective adoption design.

Sensory Processing

Theory and Measurement. Ayres (1979) originally proposed that the way in which the brain processes sensory stimuli results in behavioral responses to the environment and the development of adaptive behaviors. More specifically, the processes of sensitization, or the capacity to register a new sensory stimulus, and habituation, or the capacity to adapt to an ongoing or familiar sensory stimulus, support the individual's ability to organize a behavioral response that is appropriate for the characteristics of a given sensory stimulus. Individual differences in how the brain processes sensory stimuli are believed to result in distinct patterns of responding to sensory input (sensory processing patterns). Atypical sensory processing occurs when individuals show hyper- or hypo-responsiveness or inconsistent responding to sensory stimuli (Miller et al., 2007).

Researchers have built on Ayres' work by identifying specific patterns of responding to sensory stimulation from infancy to adulthood, in typically-developing and clinical samples (e.g., Baranek et al., 2006; Ben-Sasson et al., 2007; Dunn, 2001; Miller, Reisman, McIntosh, & Simon, 2001). Relevant to the current study, researchers have identified a pattern of Sensory Overresponsivity, which is characterized by responses to sensory stimulation that are quicker, more intense, and prolonged, compared to other children's responses. Infants who are overresponsive to sensory stimuli may show a variety of behavioral responses, including

resistance, avoidance, and distress. Additionally, Sensory Overresponsivity has been linked to heightened sympathetic nervous system reactivity (Miller et al., 1999). There is also evidence for a pattern of Sensory Underreactivity; however, this pattern is more prevalent in individuals with developmental disabilities and less likely to be found in normative samples (Dunn & Daniels, 2002), and as a result, the focus of the current study will be on heightened reactivity to tactile stimulation.

Typically, sensory processing patterns have been examined using caregiver report of children's emotional and behavioral reactions to a variety of sensory stimuli (e.g., Ben-Sasson, Carter, & Briggs-Gowan, 2009; Dunn & Daniels, 2002; Schoen, Miller, & Green, 2008). Less frequently, sensory processing has also been measured by clinical observations of young children's behavioral responses during systematic exposure to different types and intensities of sensory stimuli (DeGangi & Greenspan, 1989), or responses to multi-sensory toys during play-based assessment (Baranek, 1999).

Measures of sensory processing have focused on reactivity specifically to the sensory features of one's environment. However, in the literature, sensory processing has been conceptualized both as part of broader measures of reactivity to one's environment, such as temperamental negative affect (Rothbart, 1981) and as a related but distinct construct (Dunn, 2001). Studies of the relation between sensory reactivity and broader affective reactivity (i.e., distress to novelty, blocked goals, being confined) have yielded mixed findings. For instance, Desantis and colleagues found evidence for a dimension of negative reactivity that spanned infants' responses to both sensory and non-sensory features of the environment (Desantis, Harkins, Tronick, Kaplan, & Beeghly, 2011), whereas work by Gouze and colleagues suggests that young children's reactivity to sensory stimuli is distinct from their general distress responses

to a variety of environmental situations (i.e., sensory *and* non-sensory features; Gouze, Lavigne, Hopkins, Bryant, & Lebailly, 2012).

There is some evidence that sensory reactivity and negative affect show distinct relations with child outcomes (Hopkins et al., 2013; Mammen et al., 2015) and have unique developmental trajectories, with sensory reactivity showing limited change and negative affect showing increases during the first two years of life (Ben-Sasson, Carter, & Briggs-Gowan, 2010; Brooker, Buss, Lemery-Chalfant, Aksan, Davidson, & Goldsmith, 2013; Wood, 2011). This suggests that sensory reactivity is a fundamental process that precedes negative affectivity or is perhaps a more neurobiological aspect of the broader distress response. Indeed, there is evidence that sensory reactivity is predictive of negative affect (e.g., fear, sadness, anxiety) in studies of children and adults (Engel-Yeger et al., 2016; Engel-Yeger, Palgy-Levin, & Lev-Wiesel, 2015; Green, Ben-Sasson, Soto, & Carter, 2012; Kagan & Snidman, 1991), and researchers have suggested early sensory reactivity as a marker of negative affect and/or a trait that leads to negative affect in adulthood (Engel-Yeger et al., 2016; Levit-Binnun, Szepeswol, Stern-Ellran, & Engel-Yeger, 2014). On the other hand, several studies have found significant correlations between sensory reactivity and negative affect in infancy, childhood, and adulthood (Case-Smith, Butcher, & Reed, 1998; Daniels, 2004; Engel-Yeger & Dunn, 2011; Goldsmith et al., 2006; Klein et al., 2008), and there is some overlap in genetic underpinnings of sensory reactivity and negative affect (Keuler et al., 2011). However, correlations between sensory reactivity and negative affect (and their genetic underpinnings) do not necessarily mean they are the same construct. Taken together, current studies suggest that sensory reactivity and negative affect share some genetic underpinnings, and there is a part of sensory reactivity that overlaps with negative affect and may even lead to negative affect in childhood and adulthood. However, there

is a need for more longitudinal studies of the relation between sensory reactivity and negative affect; for instance, it is possible that early sensory reactivity interacts with environmental experiences, thus affecting the manifestation of negative affect.

Stability and Change. Studies have shown that early deprivation or exposure to particular types of stimuli has lasting effects on processing centers in the central nervous system in human and animal samples, which provides evidence for critical periods in the development of sensory systems (Bavelier et al., 2001; Zhang, Bao, & Merzenich, 2001); however, little research has focused on the development of sensory processing patterns across time. There is evidence that behavioral responses to sensory stimuli develop across infancy, generally stabilizing by 7 months of age, and the development of sensory systems corresponds with other developmental changes (e.g., motor development and locomotion; DeGangi & Greenspan, 1988, 1989). However, Dunn (1999) found no differences in sensory processing *patterns* in cross-sectional studies across middle childhood, adolescence, and adulthood. Developmental differences in sensory processing patterns have been found only across infancy and toddlerhood (Dunn & Daniels, 2002). By comparing parent- and self-reports of sensory processing across different age groups, Dunn and Daniels (2002) found that, compared to infants, toddlers showed more sensitivity to different types of oral stimuli; the results of that study also indicated that infants and toddlers showed higher responsivity to tactile stimulation than do children, adolescents, and adults. This evidence suggests that sensory processing patterns may be more malleable during infancy and toddlerhood and that children become less sensitive to tactile stimulation across development.

To date, only two studies have examined developmental trajectories of sensory reactivity during early childhood. Ben-Sasson and colleagues showed that, on average, sensory reactivity

shows little change across the first three years of life (particularly after age 2); however, there was also significant individual variation in sensory reactivity over time, indicating that some children did change significantly over this time period (Ben-Sasson, Carter, & Briggs-Gowan, 2010). By examining sensory reactivity at ages 2 and 4, Van Hulle and colleagues identified several distinct developmental trajectories, including a large number of children who did not show elevated sensory reactivity at either time point, relatively smaller groups of children who either remitted or had late-onset symptoms of sensory reactivity, and a very small group of children who showed elevated sensory reactivity at ages 2 and 4 (Van Hulle, Lemery-Chalfant, & Goldsmith, 2015). Their findings also suggested overall modest stability in sensory reactivity from age 2 to age 4; while a large number of children showed elevated sensory reactivity at either age 2 or age 4, only 2.5% of their sample showed chronic sensory reactivity, and this group of children was also more likely to be less soothable, more fearful, and born prematurely (Van Hulle et al., 2015). Together, these two studies suggest the need to understand individual differences in change and stability in sensory reactivity during early childhood, as well as genetic and environmental factors that relate to these individual differences.

Stability in tactile reactivity across infancy and toddlerhood appears to be related to other early risk factors (e.g., premature birth) and could be predictive of future difficulties. Research on factors that influence change and stability of sensory processing patterns could inform current understanding of developmental pathways to mental health problems. For instance, it is possible that infants who do not show a normative decrease in touch reactivity have difficulty developing higher-order processes and are at risk for poor developmental outcomes. As a result, the proposed study will examine change and stability in children's responses to tactile stimulation

across infancy and toddlerhood in relation to genetic and environmental factors that have been found to be associated with sensory development.

Genetic and Environmental Influences on Sensory Processing

Evidence for Genetic and Environmental Influences. To our knowledge, no study of humans has examined the association between one generation's sensory responses and another's. However, research with non-human mammals suggests that sensory responses related to parenting behaviors are transmitted across generations. For instance, rodent research has shown that mothers with sensory deficits (e.g., anosmia) show fewer stimulatory behaviors toward pups (e.g., licking; Fleming, O'Day, & Cramer, 1999), and rat pups that receive less stimulation from mothers provide lower levels of stimulation to their own pups (Francis, Diorio, Liu, & Meaney, 1999). There is evidence from several studies of humans and animals showing genetic influences on sensory systems (e.g., neurological processing of sensory information; neurological sensitivity and responsiveness; neurological threshold; Fajardo, Meseguer, Belmonte, & Viana, 2008; Fiorio et al., 2007; Oertel et al., 2009; Weisbrod, Hill, Niethammer, & Sauer, 1999; Yokogawa, Hannan, & Burgess, 2012). In particular, there is evidence for significant genetic influences on sensory gating (neurological sensitivity to sensory stimuli; Anokhin, Vedeniapin, Heath, Korzyukov, & Boutros, 2007; Myles-Worsley et al., 1996) and for the interaction between genetic and environmental influences on sensory gating (Quednow et al., 2012). There is also evidence for the effects of pre- and peri-natal environment on infant sensory responses (e.g., maternal stress during pregnancy, birth weight, gestational age; Crepeau-Hobson, 2009; Keuler, Schmidt, Van Hulle, Lemery-Chalfant, & Goldsmith 2011; Van Hulle et al., 2015). The few studies that have used genetically informed designs to examine genetic and

environmental influences on children's sensory processing provide evidence for both genetic and environmental contributions.

To date, only findings from the Wisconsin Twin Project (WTP) have used a genetically informed design to study genetic and environmental influences on specific sensory processing patterns in children. Using a twin design, the WTP examined genetic, shared environmental (non-genetic influences resulting in similarities between family members), and nonshared environmental (non-genetic influences resulting in differences between family members) effects on sensory defensiveness (also described as over-responsivity to sensory stimulation) in toddlers (Goldsmith et al., 2006; Keuler et al., 2011). Findings revealed evidence for significant genetic, shared environmental, and non-shared environmental influences on tactile defensiveness, in particular, in the WTP sample (Keuler et al., 2011); only genetic and non-shared environmental influences accounted for significant variance in children with extreme tactile defensiveness (top 5% of sample; Goldsmith et al., 2006). Genetic influences accounted for most of the variance in extreme reactivity to tactile stimulation, which suggests that genetic effects are larger for elevated, and perhaps clinically significant, tactile defensiveness. In a more recent investigation, Van Hulle and colleagues investigated genetic and environmental influences on sensory defensiveness in middle childhood by conducting a follow-up study with the twins who had participated in the WTP (Van Hulle, Schmidt, & Goldsmith, 2012). Genetic influences accounted for most of the variance in sensory defensiveness (.65-.70), and the remaining variation was explained mainly by nonshared environmental influences. Taken together, the results of the two studies suggest that genetic effects on reactivity to sensory stimuli may increase from toddlerhood to middle childhood, whereas the effects of shared environment may decrease.

These studies have provided groundbreaking evidence for genetic and environmental influences on sensory processing patterns in toddlerhood and middle childhood; specifically, findings have shown that individual differences in heightened reactivity to sensory stimuli are heritable. By investigating specific genetic and environmental influences on child reactivity to tactile stimulation across time, the current study will increase understanding of the development of tactile reactivity and could reveal developmental time-points when environmental interventions may be most effective. In the following section, influences on the development of sensory processing within the parenting environment are discussed.

Research on the relation between sensory processing and parenting has focused on parenting behaviors that support the development of sensory systems, the influence of child sensory processing patterns on parenting behaviors, and the potential for parenting to mitigate sensory processing difficulties. In general, considerable evidence from animal and human research has shown the importance of the exchange of tactile stimulation between parent and child for the development of attentional, emotional, and behavioral regulation (Polan & Hofer, 1999; Feldman & Eidelman, 2003a; Ferber & Makhoul, 2004). The exchange of tactile stimulation has been found to support the regulation of biological and behavioral processes, including sensory systems (Hofer, 1995; Hrdy, 1999), to decrease infant stress reactivity (Hernandez-Reif, Diego, & Field, 2007), and to promote more exploration of the environment (Feldman et al., 2003). Further, the exchange of tactile stimulation during infancy is predictive of more positive parenting behaviors (e.g., lower intrusiveness, higher sensitivity), as well as closer proximity and more synchrony between parent and child behaviors (Feldman et al., 2003).

Several environmental factors have been found to relate to sensory processing difficulties, including maternal psychopathology, family conflict, parent hostility, and low socioeconomic

status (Hopkins et al., 2013). Studies of internationally adopted children who have had early, prolonged stays in institutional care, which typically lacks social and sensory stimulation, go on to have difficulties responding to sensory input (Lin, Cermak, Coster & Miller, 2005; Wilbarger, Gunnar, Schneider & Pollak, 2010). Taken together, the results of prior studies suggest that rearing environments that interfere with the parent's support of child positive engagement in sensory stimulation may lead to later sensory processing difficulties. Indeed, certain parenting styles and responses to child reactivity to sensory stimuli may have a negative impact on children's development of sensory processing patterns over time. Studies have shown that child sensory processing difficulties are related to higher parenting stress and parental overprotection (Epstein, Saltzman-Benaiah, O'Hare, Goll, & Tuck, 2008; Gourley, Wind, Henninger & Chinitz, 2013), although direction of effects is unclear. Further, parents of children with sensory processing difficulties often experience confusion in response to their children's reactions to sensory stimuli and have a tendency to interpret their children's behavior in a negative light, which can lead to parent insensitivity to children's sensory processing patterns and sensory needs, as well as difficult parent-child interactions (DeGangi, 2000; Dunn, 2004; Miller, 2006; Sameroff & Fiese, 2000). DeGangi et al. (1997) compared the quality of mother-child interactions during tactile play between dyads in which the child had regulatory and sensory processing difficulties and dyads in which the child did not show difficulties. Results indicated that in mother-child dyads in which the child had regulatory and sensory processing difficulties, mothers showed less movement and symbolic play, infants showed less exploration and more aggression, and the dyads showed less synchrony and more flat affect. Such findings are indicative of the disconnection that can occur between parents and their children who have abnormal sensory responses; findings also suggest the distress and frustration a parent may

experience and the negative patterns that can develop when even the most basic parent-child interactions, such as feeding, cuddling, and dressing, are disrupted by a child's difficulty responding to the sensory stimuli involved in the activity. It is possible that parents who are better able to manage such frustration and support their children's positive engagement with sensory stimuli can more effectively promote their children's development of healthy sensory processing patterns.

Dunn (2004) suggested intervention strategies aimed at improving parent-child interactions for children experiencing sensory processing difficulties. Dunn called for parent-infant interventions that focus on facilitating caregivers' understanding of and sensitivity to child sensory processing patterns, which could promote caregiver anticipation of the child's needs. By attending to cues related to child reactivity to sensory stimuli, parents can structure daily environments to facilitate children's development of adaptive responses to and management of sensory stimulation, which is likely to improve children's interactions with others and their environments (e.g., providing structured patterns of sensory stimulation so that highly reactive children can attend to tasks with fewer distractions caused by intruding stimuli). In sum, Dunn (2004) proposed that increasing parents' understanding of a child's behavior in relation to his or her sensory processing pattern and training parents to match their responses to their children's sensory and regulatory needs can improve the quality of parent-child interactions, which is believed to protect against negative effects of sensory processing difficulties on children's development. Jaegermann and Klein (2010) showed that a parenting intervention designed to increase parent awareness of and responsiveness to cues related to child sensory processing patterns resulted in more positive parenting behaviors and parent-child interactions than a parenting intervention designed to increase overall parenting sensitivity. There is evidence that

interventions aimed at increasing parents' awareness of their children's sensory needs can ameliorate sensory processing difficulties (Schaaf & Anzalone, 2001; Williams & Shellenberger, 1996). Further, the provision of a variety of sensory stimuli and activities and supporting children's engagement in sensory stimulation can decrease sensory processing difficulties and associated problem behaviors (Hall & Case-Smith, 2007; Miller, Coll, & Schoen, 2007).

In sum, there is evidence for associations among child sensory processing, parenting behaviors, and the quality of parent-child relationships, and these findings can guide research on specific environmental influences on sensory processing. However, because studies in this area of research have examined biologically related parents and children, it is impossible to disentangle genetic and environmental influences on the development of child sensory processing patterns. Gene-environment correlations, wherein a child's heritable characteristics evoke or are reflected in their rearing environments (Plomin, DeFries, & Loehlin, 1977), and gene-environment interactions, defined as moderation of environmental influences on outcomes by genetic influences or environmental moderation of effects of genotype on outcomes (Reiss, Leve, & Neiderhiser, 2013), cannot be examined in such studies. Indeed, prior research has shed little light on how sensory processing may be transmitted across generations. In the following section, examples are presented on how correlations and interactions between genetic and environmental influences could influence the development of sensory processing.

Gene-environment Interplay. Studies of gene-environment interplay during infancy and toddlerhood comprise a relatively new area of research, which, prior to the proposed study, has not examined child sensory processing (aside from studies that have examined main effects of genetic and environmental influences; Goldsmith et al., 2006; Van Hulle et al., 2012). However, there is evidence for several different types of interplay between genetic and environmental

influences that affect socio-emotional processes during toddlerhood (e.g., fussiness, externalizing problems; Natsuaki et al., 2010; Pemberton et al., 2010; Rutter, Moffitt, & Caspi, 2006). For instance, in passive gene-environment correlation, a parent and child share genes and environment, and a child's genetic propensities are correlated with the family environment inherited from parents (Kendler, 1996); as a result, a child's inherited reactivity to sensory stimuli may be correlated with a family environment characterized by limited sensory stimuli (e.g., few opportunities for sensory stimuli in home environment; few exchanges of sensory stimulation). In evocative gene-environment correlation, a child's heritable traits evoke specific responses from others (McGue, Elkins, Walden, & Iacono, 2005); for example, a child's inherited tendency to react negatively to sensory stimuli may evoke harsh or insensitive parenting responses. With regard to gene-environment interaction, there is evidence that children show differential responses to parenting styles and interventions, based on genetic risk for psychopathology (Leve et al., 2009), and studies have shown that interventions for child behavior problems are most effective for children at high genetic risk for psychopathology (Shaw, Dishion, Supplee, Gardner, & Arnds, 2006). In the case of children's sensory processing patterns and difficulties, it is possible that parenting behaviors that support children's positive engagement with sensory stimulation may benefit primarily children with genetic propensity for heightened reactivity to sensory stimuli. However, studies of gene-environment interplay have yet to examine sensory processing patterns. As a first step to understanding how the interplay of genetic and environmental influences relates to sensory processing patterns during infancy and toddlerhood, the current study used a genetically informed adoption design to examine genetic and environmental factors and interactions in relation to children's tactile reactivity across 9 and 18 months.

The Current Study

The current study used a prospective adoption design to examine: 1) the development of child tactile reactivity from 9 to 18 months, 2) genetic and environmental influences on the development of child tactile reactivity, and 3) interplay between genetic and environmental influences on the development of child tactile reactivity. The adoption design can be used to identify interplay between genetic and environmental influences on child development (Cadoret et al., 1996; Leve et al., 2009; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998; O'Connor et al., 2003). Because adopted children are reared by adoptive parents (who are not genetically related to the child), associations between adopted child characteristics and adoptive parent characteristics are presumed to result from environmental influences. Similarly, because birth parents and the adopted child do not share an environment, associations between the adopted child and birth parents are presumed to result from genetic influences or effects of prenatal environment. Therefore, the prospective adoption design effectively controls for passive gene-environment correlation. Further, the current study measured prenatal influences, which allows for the detection of genetic influences that could be confounded with prenatal environment and of interplay between genetic influences (as measured by birth parent characteristics) and the rearing environment (e.g., adoptive parent characteristics).

To assess the development of child tactile reactivity across 9 and 18 months, a structured task that required parents to deliver tactile stimulation to their infants by painting the infants' feet and hands and pressing them on paper to make a picture was used (Flower Print Task).

Consistent with prior measures of sensory reactivity, which have used children's observed or parent-reported resistance behaviors to assess infant overresponsivity to sensory stimuli (e.g., Ben-Sasson, Carter, & Briggs-Gowan, 2009; DeGangi & Greenspan, 1989; Dunn & Daniels,

2002; Schoen et al., 2008; Goldsmith, 1996), we coded infants' resistance (e.g., struggling, pushing or pulling away from stimulus) during tactile stimulation. Prior research has shown that individual differences in these behavioral responses to sensory stimulation can be reliably measured by 9 months of age (DeGangi & Greenspan, 1989; Fairhurst, Löken, & Grossman, 2014). Further, prior research from the current sample has shown that infants' patterns of responding to tactile stimulation during the Flower Print Task are consistent with those identified by parent-report (Mammen et al., 2016).

To assess genetic and environmental influences on child tactile reactivity (and their interplay), we measured specific adoptive parent characteristics (environmental influences) and birth parent characteristics (genetic influences) relevant to child sensory reactivity. To our knowledge, it is currently unclear which birth parent characteristic would be the best index for genetic influence on child tactile reactivity. Although it would likely be ideal to use a measure of sensory reactivity developed for adults to assess genetic influence on child tactile reactivity (e.g., Adolescent/Adult Sensory Profile; Brown & Dunn, 2002), this was not available in the current study. Moreover, there is no extant research suggesting adult sensory reactivity is a genetically transmitted characteristic that increases the likelihood of offspring showing sensory reactivity in childhood. However, there is evidence that sensory reactivity and temperamental negative affect are related across development and share genetic underpinnings (Case-Smith et al., 1998; Daniels, 2004; Engel-Yeger & Dunn, 2011; Goldsmith et al., 2006; Keuler et al., 2011; Klein et al., 2008). Studies have also shown that early sensory reactivity predicts later negative affect (Engel-Yeger et al., 2016; Engel-Yeger et al., 2015; Green et al., 2012; Kagan & Snidman, 1991; Levit-Binnun et al., 2014), and it is possible that sensory reactivity in childhood develops into or becomes subsumed in adult temperamental negative affect.

Based on this evidence, birth parent negative affect is likely a measure of broad genetic risk for child development, including child sensory reactivity. As a result, to measure genetic influence on child tactile reactivity, birth parents completed the negative affect scale on the Adult Temperament Questionnaire (Rothbart, Ahadi, & Evans, 2000). With regard to environmental influences relevant to child tactile reactivity, the HOME physical involvement subscale (Caldwell & Bradley, 1984) was used to measure how adoptive parents supported and managed their 9-month-old infant's engagement with stimuli in the home environment (e.g., proximity to child, structuring of activities, supporting child's exploration of toys), and this was based on evidence that providing regular support for children's engagement in a variety of stimuli and activities can decrease sensory processing difficulties and associated problem behaviors (Hall & Case-Smith, 2007; Miller, Coll, & Schoen, 2007).

Several hypotheses were examined. With regard to Aim 1 of the study (i.e., to examine the development of tactile reactivity from 9 to 18 months), it was expected that children would show modest stability and also a normative decrease in reactivity to tactile stimulation between 9 and 18 months. This prediction was consistent with prior research suggesting that children show modest stability in sensory reactivity during early childhood (Van Hulle et al., 2015) and that children become less sensitive to tactile stimulation, specifically, across development (Dunn & Daniels, 2002). With regard to Aim 2 (i.e., to examine effects of genetic and environmental factors on the development of tactile reactivity), it was hypothesized that children with genetic propensity for heightened tactile reactivity (i.e., birth parents had higher negative affect scores) would be less likely to show a normative decrease from 9 to 18 months, rather, they would show a lesser degree of change in tactile reactivity over time. This prediction was supported by evidence from studies of genetic influence on broader temperamental reactivity and regulation in

early childhood (Plomin et al., 1993; Plomin, 1986; Saudino, Plomin, & DeFries, 1996), finding that, in general, genetic influences account for stability and environmental influences account for change in children's reactivity and behavioral regulation in response to environmental stimuli (which may or may not include sensory stimuli) across time. It was also expected that higher levels of adoptive parents' support of their children's engagement in their physical environments (i.e., higher adoptive parent HOME physical involvement scores) would predict greater decreases in child tactile reactivity. This prediction was supported by prior research suggesting that parents' capacity to support their children's engagement with stimuli can promote more adaptive responses to sensory stimuli (Hall & Case-Smith, 2007; Jaegermann & Klein, 2010; Miller, et al., 2007).

With regard to Aim 3, a specific gene-environment interaction ($G \times E$) was hypothesized to influence the development of children's reactivity to tactile stimulation. For $G \times E$, in the context of an expected normative decrease in tactile reactivity between 9 and 18 months, it was expected that adoptive parent support of child engagement with the physical environment would predict greater decreases in child tactile reactivity when children have a genetic propensity for heightened tactile reactivity (gene-environment interaction; see Figure 1). That is, the effects of parenting on the development of child reactivity to tactile stimulation would vary based on genetic influences. This hypothesis was supported by prior studies showing that environmental supports are most effective for children at risk due to genetic influences (Shaw, Dishion, Supplee, Gardner, & Arnds, 2006).

With regard to gene-environment correlation (rGE), there is limited research on which to base a specific hypothesis for how genetic influences on children's reactivity to tactile stimulation contribute to their postnatal environments by evoking particular responses from

adoptive parents (evocative gene-environment correlation). Prior research has shown that children's sensory processing difficulties (e.g., extremely high or low reactivity to sensory stimuli) are associated with more negative parenting responses and parent-child interactions in biologically-related parents and children (DeGangi et al., 1997; Epstein et al., 2008). This may suggest that children with a genetic propensity for heightened tactile reactivity evoke less support for engaging in their physical environments from adoptive parents based on their genetic propensity for heightened reactivity to tactile stimuli (see Figure 2). However, it is also possible that children with a genetic propensity for heightened tactile reactivity evoke greater support from adoptive parents based on their genetic propensity for heightened tactile reactivity. As a result, no specific hypothesis was made for *r*GE and, instead, the associations between birth parent negative affect (genetic propensity for heightened tactile reactivity) and adoptive parent support of child engagement with stimuli in the physical environment were explored.

METHOD

Participants

This study included participants ($N = 561$; two cohorts collected 2 years apart; Cohort 1 $n = 361$ and Cohort 2 $n = 200$) from the ongoing Early Growth and Development Study (EGDS), a prospective, longitudinal study of adoptive families in the Pacific Northwest, West/Southwest, Mid-West, and Mid-Atlantic regions of the United States. Each adoption triad consisted of the adopted child, adoptive parents (APs), and birth mother (BM). Additionally, data from the birth father (BF) were collected for a subset of families ($n = 208$).

The current investigation included data collected from the adoptive parents and children when children were 9 ($M = 9.36$, $SD = .56$, range = 8.25 - 11.76) and 18 ($M = 17.96$, $SD = .86$, range = 15.64 - 20.47) months of age (T1 and T2, respectively), birth mother reports of prenatal experiences at 4 months postpartum, and birth mother and birth father self reports on temperament (T2).

The median child age at adoption placement was 2 days ($SD = 12$ days, range = 0 - 91), and 57.2 percent of the children were male. Over half the children were White (55.6%), 19.3% were multiracial, 13% were African American, 10.9% were Latino, less than one percent were Asian, less than 1 percent were American Indian, and less than 1 percent were of unknown/unreported ethnicity. In general, adoptive parents were older (T1 Adoptive Mother Age: $M = 38.2$, $SD = 5.6$, range = 24.5 - 55.8 years; T1 Adoptive Father Age: $M = 39.2$, $SD = 5.9$, range = 25.1 - 60.6 years), college-educated, and middle class (T1 Household Income: $Median = 110,000.00$, $SD = 104,959.43$, range = 7,000 - 150,000). Adoptive mothers were primarily White (91.8%, 3.9% African American, 2.0% Latino, 0.9% Multiracial, 1.4% Other), as were adoptive fathers (91.8%, 4.9% African American, 1.6% Latino, 1.1% Multiracial, 2.0%

Other). Birth parents (BPs) were generally younger (T1 Birth Mother Age: $M = 24.8$, $SD = 6.0$, range = 14.3 - 44.1 years; T1 Birth Father Age: $M = 26.9$, $SD = 7.9$, range = 16.0 - 59.6 years) and more ethnically and racially diverse (Birth Mother Race/Ethnicity: 70.1% White, 13.3% African American, 6.7% Latino, 4.9% Multiracial, 5.0% Other; Birth Father Race/Ethnicity: 69.9% White, 11.5% African American, 9.1% Latino, 4.8% Multiracial, 4.7% Other). The majority of birth parents had completed high school or trade school and most reported household incomes below \$25,000. See Leve, Neiderhiser, Shaw, Ganiban, Natsuaki, and Reiss (2013) for detailed information on recruitment and assessment procedures and detailed sample information.

Procedure

The current investigation included data provided by the adoptive parents and birth parents, as well as observations of the adoptive parents and adopted children at 9 and 18 months. At both times, a 2.5 hour birth parent assessment was completed in the home or some other convenient site, and the 2.5-3.5 hour adoptive parent assessment took place in the home. Interviewers asked birth and adoptive parents to complete computer-assisted and paper-pencil interview questions, which all participants completed independently. Adoptive parents were observed in several tasks, separately, with their adopted child except for the Flower Print Task, which parents completed together with their child. The Flower Print Task is the focus of the current study. All participants received monetary compensation for volunteering their time to participate in the study. Separate teams of trained interviewers completed birth and adoptive family assessments to preclude any sharing of data across birth and adoptive families. Detailed information on the EGDS procedures and assessments can be found in Leve et al. (2013).

Birth Parent Negative Affect

Birth parent negative affectivity was used as an index of genetic influences on child reactivity to tactile stimulation, with a correlation between birth parents' negative affectivity and child tactile reactivity indicating genetic influence. At T2, birth parents completed the negative affect scale from the Adult Temperament Questionnaire – Short Form (ATQ; Rothbart, Ahadi, & Evans, 2000), a self-report measure of temperament, which was adapted from Derryberry and Rothbart's Physiological Reactions Questionnaire (1988). The negative affect scale of the ATQ short form is comprised of the fear (7 items; e.g., *Loud noises sometimes scare me*), sadness (7 items; e.g., *Sometimes minor events cause me to feel intense sadness*), discomfort (6 items; e.g., *Colorful flashing lights bother me*), and frustration subscales (6 items; e.g., *I find it very annoying when a store does not stock an item that I wish to buy*; Evans & Rothbart, 2007). Negative affect scores were computed by averaging the 26 items. Birth parent negative affect scale scores offer information on the extent to which they show general negative affective reactions to environmental stimuli. The inter-item alpha for the negative affect scale in the EGDS sample was .82 for birth mothers and .79 for birth fathers.

Reactivity to Tactile Stimulation in Parent-Child Interaction

Flower Print Task. The Flower Print Task was originally designed for the EGDS to assess co-parenting as it required both parents to work together to complete a fun but somewhat challenging task with their child. Both adoptive parents and their adopted child participated in this task at 9 and 18 months, although the task was discontinued at 18 months for Cohort I because adoptive parents complained that the task was difficult to complete when children became mobile (see Missing data description below). The task required adoptive parents to paint the child's hands and feet and then to press them to a piece of paper to create a flower design.

The adoptive parents used step-by-step instructions, paper, paintbrushes, several bottles of paint, a bib, and wet wipes and worked together to complete the task. Typically, one parent held and moved the infant while the other parent painted and pressed each of the infant's hands and feet to the paper. Although initially designed to assess the manner in which parents worked together, this task was ideal for assessing infants' responses to tactile stimulation within a social context because it structured parents' delivery of tactile inputs.

Child behaviors were microcoded second-by-second using coding software (Mangold International, INTERACT, Germany) at T1 and T2. Since the current study aimed to examine the development of children's responses during the exchange of tactile stimulation, child behaviors were coded only during the delivery of stimulation, that is, only when the child's hands and feet were being painted, pressed to the paper, and cleaned off with the wet wipes. The coding scheme was informed by existing coding systems that have been used reliably to code touch and response to touch in parent-child interactions, as well as reactivity to novel stimuli (Feldman et al., 2002; Feldman & Eidelman, 2003b; Feldman, Keren, Gross-Rozval, & Tyano, 2004). Existing coding systems were adapted for the ages of the children in the current study and to capture children's affective (e.g., neutral, positive, negative emotion) and behavioral (e.g., gaze to and away from tactile stimuli, exploration of tactile stimuli, body response to stimuli, self-soothing, physical resistance) responses specifically to tactile stimulation from parents. To assess reactivity to tactile stimuli, the proportion of time children showed physical resistance behaviors in response to tactile input (e.g., arching the back, squirming or struggling, shaking head, banging head against parent, pushing, kicking, pulling hand or foot out of reach) was computed. The decision to code these behaviors was supported by empirical evidence that physical resistance is a response indicative of overresponsivity to sensory stimuli and is

consistent with parent-report measures of heightened reactivity to tactile stimulation (Dunn & Daniels, 2002; Mammen et al., 2016). Further, research with the current sample (Mammen et al., 2016) found that behaviors coded during this task yielded sensory processing patterns consistent with prior research (Miller, Anzalone, Lane, Cermak, & Osten, 2007), and that children observed to have the most difficulty engaging in tactile stimuli (e.g., low levels of gazing at and long latency to explore tactile stimuli) during the Flower Print Task also showed the highest levels of physical resistance during the task (Mammen et al., 2016).

A team of undergraduate research assistants was trained to code child behaviors during the delivery of tactile stimulation. Seventeen percent of the interactions were double coded at T1 ($n = 87$) and T2 ($n = 50$) to establish inter-rater reliability. Coefficient kappas of .80 at T1 and .66 at T2 for physical resistance coding demonstrated adequate inter-rater reliability.

Missing data. At T1, data were missing due to: only one parent available to participate because of scheduling constraints ($n = 17$) or single-parent family ($n = 10$); technical problems with video/sound equipment ($n = 14$); and data unavailable because the family had not yet joined the study or did not participate in the T1 adoptive family assessment ($n = 23$). At T2, the Flower Print Task was conducted in the full Cohort 2 sample but only for a subset of the Cohort 1 sample. Researchers began Cohort 1 data collection using the Flower Print Task but stopped administering the task because adoptive parents reported difficulties completing it when children were 18 months old due to the children's mobility. In addition, data were missing due to: only one parent available to participate because of scheduling constraints ($n = 13$) or single-parent family ($n = 14$); the family did not participate in the T2 adoptive family assessment ($n = 23$); the child was too distressed ($n = 3$); and technical problems with equipment ($n = 2$). As a result, data from the task were available for 497 families at T1 and 295 at T2.

Physical Involvement in Home Environment

Following the T1 adoptive parent assessments, interviewers assessed each adoptive parent's physical involvement (e.g., proximity to child, structuring of activities, supporting child's exploration of toys) with his or her child in the home environment using the physical involvement subscale from the Home Observation for Measurement of the Environment inventory for children ages 0-3 (HOME; Caldwell & Bradley, 1984). The HOME has been used to assess the amount and quality of social-emotional and cognitive stimulation and support available in the rearing environment. Specific to the proposed study, the HOME physical involvement subscale was used to measure how parents support and manage their child's engagement with physical stimuli in the home environment (6 items; e.g., *Mother/father provides toys that challenge child to develop new skills*). Interviewers responded to the yes-no format of the HOME to complete the subscale, and adoptive mothers and fathers were rated separately for physical involvement with the child ($\alpha = .69$ and $.75$ for adoptive mother and adoptive father, respectively). Adoptive mother and adoptive father HOME physical involvement scores were standardized and averaged.

Covariates

Adoption Openness. It is possible that contact between birth parents and the adoptive parents or the adopted child has an influence on adopted child development. The degree of openness in an adoption can confound the separation of environmental and genetic influences on child development by increasing gene-environment correlation if, for instance, the rearing environment provided by adoptive parents is influenced by their interactions with birth parents. To control for adoption openness, at T1, birth and adoptive parents were asked to report on their perceptions of the openness of the adoption, the amount of information birth and adoptive

parents knew about one another, as well as the level of contact birth and adoptive parents had with one another. These items were used to create a continuous variable indicating the level of openness in the adoption. See Ge et al. (2008) for more details on the creation of the adoption openness variable.

Obstetric Complications. Prior research suggests that the EGDS sample evidenced somewhat elevated levels of pre- and perinatal risks (Marceau et al., 2013), which have been found to be associated with abnormal sensory responses (Crepeau-Hobson, 2009). Further, effects of pre- and perinatal experiences may confound the estimation of genetic influences (Pemberton et al., 2010) because they could contribute to a correlation between birth parent and child attributes, which, in an adoption design, indicates genetic influence. Birth mothers reported on pre- and perinatal experiences across five categories: exposure to drugs and alcohol, exposure to toxins, maternal psychopathology during pregnancy, pregnancy complications, and neonatal complications. A perinatal risk index was created using a scoring procedure similar to the McNeil-Sjöström System, which has been used to quantify pre- and perinatal risk exposure (Marceau et al., 2013; McNeil, 1995; McNeil & Sjöström, 1995). A measure of overall obstetric complications was created by compositing scores across the five categories.

Data Analytic Plan

Linear Mixed Models were conducted using maximum likelihood estimation in SPSS Version 24 to examine: change in child tactile reactivity from 9 to 18 months (Aim 1), effects of genetic and environmental factors on change in child tactile reactivity (Aim 2), and effects of gene-environment interactions on change in child tactile reactivity (Aim 3). Child tactile reactivity was entered as the dependent variable, and a repeated effects variable, *Time*, indicated the point at which child tactile reactivity was assessed (9 or 18 months). Because data were

collected from a subset of birth fathers ($N = 208$), separate models were conducted for birth mothers and birth fathers (i.e., for BM and BF negative affect). Adoption openness and obstetric complications were included as covariates in both BM and BF analyses, since prenatal environment and postnatal contact between birth parents and adoptive parents may confound the estimation of genetic influences.

To test the hypothesis that child tactile reactivity would show a normative decrease across time, the effect of *Time* (9 or 18 months) on child tactile reactivity was examined in the Linear Mixed Models (Aim 1). A significant negative parameter estimate for the repeated factor *Time* would indicate a significant decrease in tactile reactivity. To examine the hypothesis that children with genetic propensity for heightened tactile reactivity (i.e., birth parents show higher negative affect) would show a lesser degree of change in tactile reactivity, and that adoptive parents' support of their children's engagement in the physical environment (i.e., higher levels of AP physical involvement scores) would predict greater decreases in child tactile reactivity (Aim 2), BP negative affect scores and AP physical involvement scores were each specified as a fixed interaction effect (with *Time*) in the Linear Mixed Models. A significant positive parameter estimate for the BP negative affect and *Time* interaction would suggest genetic propensity was related to increases in tactile reactivity over time, and a significant negative parameter estimate for the AP physical involvement and *Time* interaction would suggest AP physical involvement was related to decreases in tactile reactivity. To test the hypothesis that children with a genetic propensity for heightened tactile reactivity (i.e., birth parents had higher levels of negative affect) would show greater decreases in tactile reactivity as a function of AP physical involvement ($G \times E$; see Figure 2), a 3-way fixed interaction effect (*Time* x BP negative affect x AP physical involvement) was specified in the Linear Mixed Models (Aim 3). To examine significant

interaction effects, children's tactile reactivity trajectories from 9 to 18 months were plotted at 1 standard deviation below and 1 standard deviation above the mean of each moderator variable (Preacher, Curran, & Bauer, 2006).

Additionally, to explore gene-environment correlations (Aim 3), hierarchical linear regressions were used to assess associations between birth parents' negative affect (separate models for birth mothers and birth fathers) and adoptive parents' physical involvement, controlling for obstetric complications and adoption openness.

Missing Data Handling. The number of values present for each measure is reported in Table 1. Multiple imputation was used to address missing data in the current sample. Prior research suggests multiple imputation of missing data leads to less biased results, compared to listwise deletion and full information maximum likelihood estimation, in cases where data are not missing at random (Ji, Chow, Schermerhorn, Jacobson, & Cummings, 2018; Mustillo & Kwon, 2014). Missing values were imputed for all study variables with missing data using SPSS 24 multiple imputation with linear regression method. To assist in the estimation of missing child tactile reactivity values, three auxiliary variables were included along with study variables in the imputation model: Adoptive Mother-reported distress to limitations subscale from the Infant Behavior Questionnaire (IBQ collected at T1; Rothbart, 1981), Adoptive Mother reports on the Infant Characteristics Questionnaire (ICQ; an 11-item abridged version included in the EGDS at T2; Bates, Freeland, & Lounesbury, 1979), and a factor indicating Cohort (1 or 2). The distress to limitations and ICQ measures were chosen because they assess broad reactivity to environmental stimuli, which is conceptually distinct from tactile reactivity (Dunn, 2001) but has shown modest to large associations with tactile reactivity during infancy and toddlerhood (Case-Smith, Butcher, & Reed, 1998; Daniels, 2004; Goldsmith et al., 2006). Cohort was selected

because this variable was associated with missingness in child tactile reactivity (see below). To assist in the estimation of missing BF negative affect scores, BF reports on the harm avoidance scale of the Temperament Character Inventory, which measures temperamental negativity and fearfulness (collected at T1; Cloninger, Svaric, & Pryzbeck, 1993), were included in the imputation model. Twenty imputations were created and findings for main analyses were pooled from the 20 imputed datasets. Following imputation, BP negative affect scores and AP physical involvement scores were standardized to assist in interpretation of interaction effects.

RESULTS

Preliminary Analyses

Means and standard deviations for study variables can be found in Table 1. Because the 18-month measure of child tactile reactivity showed high skewness and kurtosis, the 9- and 18-month measures of child tactile reactivity were log-transformed; however, because the results using log-transformed variables were similar to results using original data, findings are reported from analyses conducted with the untransformed variables. Correlations were computed across all study variables, pooling results from the imputed datasets (Table 2). A small, positive correlation was found between obstetric complications and BM negative affect.

ANOVA revealed no sex differences on child tactile reactivity during the Flower Print Task at 9 or 18 months. Given the large amount of data missing at the 18-month time point, ANOVAs were used to examine potential differences between children who participated only in the 9-month assessment and children with data at both 9 and 18 months. ANOVA revealed that children with data at both time-points showed significantly higher tactile reactivity at 9 months compared to children with data at only the 9-month time-point ($F(1, 495) = 9.09, p < .01$). ANOVAs revealed no other differences on study variables between children with data only at 9 months and children who participated at both time points. To follow up on the difference in tactile reactivity between children with data from both time points and children with data from only the 9-month time point, differences in study variables based on cohort were examined, since the majority of children missing 18-month data were in Cohort 1 due to the decision by researchers to stop using the task for Cohort 1. ANOVA revealed that children in Cohort 1 showed significantly higher tactile reactivity at the 9-month time point than children in Cohort 2 ($F(1, 495) = 26.60, p < .001$) and that adoptive parents in Cohort 2 had significantly higher

physical involvement scores than adoptive parents in Cohort 1 ($F(1, 491) = 13.04, p < .001$).

There was a marginally significant difference between BMs in Cohort 1 and Cohort 2 on negative affect, such that BMs in Cohort 1 had higher negative affect scores ($F(1, 466) = 3.87, p = .05$). Because of these differences between Cohorts 1 and 2, *Cohort* was included as a covariate in main analyses.

Tactile Reactivity across Early Childhood

Correlations examining stability in child tactile reactivity from 9 to 18 months and associations between genetic factors and postnatal environment (gene-environment correlations) are reported in Table 2. Full results from the Linear Mixed Models (separate BM and BF models) with BP negative affect and AP physical involvement, and their interaction, predicting change in child tactile reactivity are reported in Table 3 (for BM) and Table 4 (for BF). Results pooled from analyses using multiple imputation to handle missingness are reported. Results of analyses conducted with imputed datasets were very similar to analyses conducted using only cases with full data. Study findings were also consistent when Linear Mixed Models were conducted with and without the three covariates.

Aim 1: Stability and Change in Tactile Reactivity. A small but significant correlation was found between child tactile reactivity at 9 months and at 18 months ($r = .19, p < .05$), suggesting modest stability in child tactile reactivity from infancy to toddlerhood.

Linear Mixed Models revealed a significant effect of *Time* (i.e., change in child tactile reactivity from 9 to 18 months), such that children's tactile reactivity was significantly lower at 18 months, compared to 9 months ($b = -.07, SE = .02, t = -4.44, p < .001$), as predicted.

Aim 2: Main Effects of Genetic and Environmental Factors on Change in Tactile Reactivity. In the BM analysis, the interaction between BM negative affect and *Time* was significant ($b = .01, SE = .00, t = 2.48, p < .05$). Follow-up Linear Mixed Models, examining the effect of *Time* at high (1 SD above the mean) and low (1 SD below the mean) levels of BM negative affect showed that children whose BMs had low negative affect showed a greater decrease in tactile reactivity from 9 to 18 months ($b = -.06, SE = .01, t = -5.41, p < .001$) compared to children whose BMs had high negative affect ($b = -.02, SE = .01, t = -2.07, p < .05$; see Figure 3). The BM analysis revealed no effect of AP physical involvement on change in child tactile reactivity.

The BF model revealed no significant effects of BF negative affect or AP physical involvement on change in child tactile reactivity.

Aim 3: Gene-Environment Interplay. To test for gene-environment interaction, the effect of the 3-way interaction *Time* x BP negative affect x AP physical involvement on change in child tactile reactivity was examined. Neither the BM nor the BF Linear Mixed Model revealed a significant effect of the 3-way interaction.

To test for evocative *r*GE, hierarchical linear regressions were used to examine associations between birth parents' negative affect and adoptive parents' physical involvement, controlling for covariates (obstetric complications, adoption openness, and cohort). There was no significant association between BP negative affect (for BM or BF) and AP physical involvement (see full results for hierarchical linear regressions in Table 5).

Additional Findings. In the BM Linear Mixed Model, there was a significant negative effect of BM negative affect ($b = -.02, SE = .01, t = -2.42, p < .05$) on initial level of tactile reactivity (9-month tactile reactivity), indicating that children with higher genetic propensity had

lower initial levels of tactile reactivity. A follow up Linear Mixed Model examining the effect of BM negative affect on final level of tactile reactivity (18-month tactile reactivity) was not significant ($b = .01, SE = .00, t = 1.55, p = .13$).

DISCUSSION

Infants rely on tactile experiences to engage with caregivers and their physical environments (Cascio, 2010; Feldman et al., 2002; Feldman et al., 2010; Feldman et al., 2003). Infants who have difficulties tolerating tactile input may be at risk for cognitive, motor, and regulatory difficulties (Ben-Sasson et al., 2007; DeGangi et al., 1993; Eeles et al., 2013; Mammen et al., 2015). Not only does infant reactivity to touch appear to have a meaningful and far-reaching impact on child development, but prior research also suggests that sensory reactivity is more malleable during the transition from infancy to toddlerhood (Ben-Sasson et al., 2010; Dunn & Daniels, 2002). As a result, the current study focused on the development of infant tactile reactivity from infancy to toddlerhood and on how genetic and environmental factors influence change over time.

Findings from the current study suggest that, on average, children show a decrease in tactile reactivity from infancy to toddlerhood. This is consistent with prior cross-sectional work by Dunn and Daniels (2002), which found that infants and toddlers showed higher responsivity to tactile stimulation than did children, adolescents, and adults. To date, developmental differences in sensory reactivity have only been found across infancy and toddlerhood (Dunn & Daniels, 2002), and prior research suggests limited change in sensory reactivity after age 2 (Ben-Sasson et al., 2010). It is possible that, given the developmental milestones children reach in, for instance, motor skills (e.g., independent movement) and communication (e.g., beginning to use words to communicate), between 9 and 18 months, they may begin to rely on other sensory modalities (e.g., for bonding, communication, exploration, comfort-seeking, regulation) and, thus, show overall less sensitivity to tactile stimuli by 18 months. Touch is also believed to play an important role in the development of the attachment relationship across the first year of life

(Duhn, 2010), and it is possible that infants' sensitivity to touch tapers off following this critical period for developing a secure connection to primary caregivers. On the other hand, children who do not show a normative decrease in tactile sensitivity (which may be due to genetic or environmental factors) may become overwhelmed as their environments become more stimulating and as they are expected to use a greater variety of sensory modalities to engage in social and physical activities.

Children in the current study also showed modest stability in tactile reactivity over time, which is consistent with Van Hulle and colleagues' (2015) finding of limited stability in children's sensory reactivity from age 2 to age 4. Although there seems to be some stability in children's reactivity to tactile stimuli from infancy to toddlerhood, the current results suggest that this time period is characterized by change, specifically decreasing reactivity to tactile stimuli. It may be that children's tactile response patterns are more malleable during the first year of life, perhaps making this an optimal time for interventions aimed at increasing children's positive responses to touch. However, there is also evidence that young children show distinct developmental trajectories in sensory reactivity (Ben-Sasson et al., 2020; Van Hulle et al., 2015) and some children show chronic sensory reactivity across early childhood (Van Hulle et al., 2015), which may be influenced by genetic and environmental factors.

In the current study, as predicted, children showed individual differences in levels of change in tactile reactivity from 9 to 18 months as a function of birth parent characteristics. Even though BM negative affect was associated with lower initial levels of child tactile reactivity, children with genetic propensity for heightened tactile reactivity (i.e., children whose BMs reported higher negative affect) showed a significantly smaller decrease in tactile reactivity from 9 to 18 months. Therefore, the current study showed that, genetic factors may contribute to

continued tactile reactivity across early childhood, which adds to prior findings that prenatal environment (e.g., lower birthweight, shorter gestation; Van Hulle et al., 2015) and emotional responses (e.g., less soothability, higher fear; Van Hulle et al., 2015) lead to chronic sensory reactivity in early childhood. Indeed, because Van Hulle and colleagues did not examine genetic influences on trajectories of sensory reactivity, it is possible that their findings for the influence of prenatal environment and emotionality on sensory reactivity actually reflected genetic influence. The inclusion of both genetic and prenatal factors is a strength of the current study. Children with genetic propensity for heightened tactile reactivity appear to be less amenable to change, which may cause them to show more persistent reactivity to tactile stimuli in their environments, perhaps influencing their cognitive, motor, and social-emotional development (Ben-Sasson et al., 2007; DeGangi et al., 1993; Eeles et al., 2013; Mammen et al., 2015). That there is a genetic factor related to a lesser decrease in tactile reactivity so early in development is important, particularly since genetic influences tend to become stronger across early childhood (Davis, Finkel, Turkheimer, & Dickens, 2015; Saudino, 2005). Ongoing tactile reactivity in early childhood may profoundly shape a young child's environment, particularly as children begin to take on a greater role in evoking and self-selecting environments based genetic propensity.

Moreover, contrary to the prediction that children with higher genetic propensity would benefit more from adoptive parent support for engaging in their physical environments ($G \times E$; see Figure 1), AP physical involvement did not affect children's change in tactile reactivity, and this did not vary based on genetic propensity for heightened tactile reactivity. Not only did children with genetic propensity for heightened tactile reactivity show a lesser decrease in tactile reactivity over time, the level of support they received from their adoptive parents did not modify this pattern. Further, negligible associations found between BP negative affect and AP physical

involvement did not indicate that children with higher genetic propensity received different levels of support from adoptive parents. That is, children with a genetic propensity for heightened tactile reactivity did not evoke more or less support for engaging in their physical environments due to genetic influences (rGE).

Overall, the current study found some support for genetic influences on patterns of change in tactile reactivity from infancy to toddlerhood, though only from birth mothers, not birth fathers, which may have been due to the smaller sample of birth fathers in the study. Current findings did not provide evidence that environmental support of children's engagement in their physical environments or gene-environment interplay affect the development of child tactile reactivity across infancy and toddlerhood. Children with genetic propensity for heightened tactile reactivity showed a distinct developmental trajectory in tactile reactivity, namely a significantly smaller developmental decrease. However, genetic effects on change in tactile reactivity were not altered by supportive parenting, nor was there a main effect of parenting behavior, as measured by parents' observed support of their children's engagement with their physical environments, on the development of child tactile reactivity. Although prior studies have found that, following short-term interventions supporting children's engagement with sensory stimuli, children showed more adaptive responses to sensory stimuli (Hall & Case-Smith, 2007; Miller et al., 2007), it is possible that the naturalistic supportive parenting behaviors measured in the current study (e.g., proximity to child, structuring of activities, supporting child's exploration of toys) were not sufficiently specific to promoting children's engagement in tactile stimuli. Future research should consider other parenting measures, which may have a stronger effect on change in tactile reactivity and may modify genetic effects on tactile reactivity.

Limitations and Future Directions

Although the current study has important implications for understanding the early development of tactile reactivity in relation to genetic and environmental influences, there are limitations to consider. Prior research with this sample provides evidence for the validity of our measure of tactile reactivity (Mammen et al., 2016), however, it is important to acknowledge that children's responses to sensory stimulation have traditionally been assessed using caregiver reports or by systematically examining infants' behavioral responses during exposure to specific types of sensory stimulation by a trained examiner (e.g., DeGangi & Greenspan, 1989; Dunn & Daniels, 2002). Although there are advantages to measuring children's responses to tactile stimulation within a naturalistic, parent-child interaction, there was limited control over specific types of touch provided by parents (e.g., light touch versus firm touch; amount of touch used to reassure or soothe child), and it is possible that children responded differently to specific types of tactile stimulation. Further, the semi-structured nature of the Flower Print Task limited the variability and frequency of parenting behaviors that could be observed, as children's tactile reactivity was measured only when parents were painting the child's hands and feet and pressing them to paper. Thus, AP behavior during the Flower Print Task was not included in this study. Future research including both a parent-report measure and an interaction task that allows for the observation of a variety of parenting behaviors during the exchange of touch could further increase understanding of how tactile reactivity develops over time.

Additionally, the current study used a broad measure of AP support of children's engagement in their physical environments, and child tactile reactivity may decrease over time in response to parenting behaviors that are more specific to supporting children's approach of tactile stimuli. Future research should examine other environmental variables (e.g., parents'

insight into child sensory needs, child's daily levels and variety of sensory stimulation), which may have a stronger impact on change in tactile reactivity and may alter genetic influences on tactile reactivity in early childhood.

The decision to use BP negative affect as an index for genetic influence on child tactile reactivity was guided by prior research showing that early sensory reactivity predicts later negative affect (Engel-Yeger et al., 2016; Engel-Yeger et al., 2015; Green et al., 2012; Kagan & Snidman, 1991; Levit-Binnun et al., 2014). The current finding of BM negative affect predicting lesser change in child tactile reactivity provides initial support for using birth parent negative affect as an index of genetic influence on child tactile reactivity and should be replicated in other samples. Ideally, future research should examine the transmission of tactile reactivity across generations, using a measure of birth parent sensory reactivity, to determine whether the genetic effect found using BP negative affect is also found using a more specific measure of adult sensory reactivity. Future studies can shed light on the genetic variables that influence child tactile reactivity across development, as well as how these genetic influences impact children's environments and are altered by environmental supports.

Finally, the current study was limited by having only two ages for examining the development of child tactile reactivity and also by missing data at the second time, which was related to how much tactile reactivity children showed at the first time point (i.e., children with higher levels of tactile reactivity at 9 months were less likely to participate at 18 months). Although the missing data were handled appropriately using multiple imputation, the current findings may be more conservative, given that children with data at both ages were likely less reactive to tactile stimuli overall. Further, the missing data from potentially more reactive children may give an incomplete understanding of how tactile reactivity develops across early

childhood. Future research should examine the development of tactile reactivity both in typical and high-risk samples, ideally using three or more time points in order to examine fluctuations in tactile reactivity across time.

Conclusions

As the first study of genetic and environmental influences on the development of tactile reactivity from infancy to toddlerhood, the current investigation makes an important contribution to research on early sensory responses. Prior research has shown that while many children show difficulty tolerating sensory input during childhood, very few continue to show ongoing difficulties engaging in sensory stimuli (Van Hulle et al., 2015), which have deleterious effects on later development (Ben-Sasson et al., 2007; DeGangi et al., 1993; Eeles et al., 2013; Mammen et al., 2015). The current findings suggest that 9 to 18 months is a time period characterized by significant change, namely a normative decrease, in tactile reactivity, which may be due to developmental milestones children reach during this stage (e.g., increased mobility, verbal communication). Children may begin to rely more on other sensory modalities (e.g., for bonding, communication, exploration, comfort-seeking, regulation) and, thus, show overall less sensitivity to tactile stimuli by 18 months. However, some children show more persistent tactile reactivity due to genetic influences, and this may have an impact on their cognitive, motor, and social-emotional development. Future research should take into account how genetic influences relate to change in children's sensory responses, examine how chronic tactile reactivity affects children's development, and focus on identifying environmental supports that promote more positive responses to tactile stimuli.

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Appendix A. Tables.

Table 1

Descriptive Statistics for Variables.

Variable	<i>N</i>	Range	Mean	<i>SD</i>	<i>Skewness/Kurtosis</i>
ATQ					
BM Negative Affect	468	1.62-6.19	3.98	.74	-.11/.07
BF Negative Affect	157	1.92-5.42	3.53	.68	.01/-.13
Flower Print Task					
Proportion Child Resist 9 Mos	497	.00-.35	.07	.06	1.32/1.89
Proportion Child Resist 18 Mos	295	.00-.30	.03	.05	3.04/10.51
HOME					
AM Physical Involvement 9 Mos	492	.00-6.00	4.99	1.36	-1.20/.33
AF Physical Involvement 9 Mos	459	.00-6.00	4.54	1.63	-.92/-.31
Covariates					
Obstetric Complications	561	.00-39.00	9.49	6.57	1.06/1.48
Adoption Openness	561	-2.23-1.86	.04	.93	-.15/-.52

Table 2

Correlations Among Study Variables.

Variable	1	2	3	4	5	6
1. BM NA	--					
2. BF NA	.03	--				
3. 9m TR	-.07	-.05	--			
4. 18m TR	.11	-.07	.19*	--		
5. AP Involvement	-.03	-.16 ⁺	-.01	.01	--	
6. Obstetric	.18**	.06	.04	-.01	.02	--
7. Openness	.09 ⁺	.01	-.03	-.02	.07	-.01

Note. ⁺ $p < .10$. * $p < .05$. ** $p < .01$. NA = Negative Affect, TR = Tactile Reactivity.

Table 3

BM Linear Mixed Model of Change in Tactile Reactivity from 9 to 18 Months.

Effect	<i>b</i>	<i>SE</i>	<i>t</i>
Intercept	.18	.02	7.76**
<i>Time</i>	-.07	.02	-4.44**
Negative Affect	-.02	.01	-2.42*
Physical Involvement	.00	.01	.38
NA x PI	.00	.01	-.48
Negative Affect x <i>Time</i>	.01	.00	2.48*
Physical Involvement x <i>Time</i>	.00	.00	-.21
NA x PI x <i>Time</i>	.00	.00	.50
Obstetric Complications	.00	.00	1.13
Adoption Openness	.00	.01	-.16
Cohort	-.06	.01	-4.53**
Obstetric x <i>Time</i>	.00	.00	-1.12
Openness x <i>Time</i>	.00	.00	-.06
Cohort x <i>Time</i>	.03	.01	3.25**

* $p < .05$. ** $p < .01$. NA = Negative Affect, PI = Physical Involvement.

Table 4

BF Linear Mixed Model of Change in Tactile Reactivity from 9 to 18 Months.

Effect	<i>b</i>	<i>SE</i>	<i>t</i>
Intercept	.18	.02	7.96**
<i>Time</i>	-.07	.02	-4.65**
Negative Affect	.00	.01	-.05
Physical Involvement	.00	.01	.38
NA x PI	.00	.01	.19
Negative Affect x <i>Time</i>	.00	.01	-.26
Physical Involvement x <i>Time</i>	.00	.00	-.30
NA x PI x <i>Time</i>	.00	.00	-.30
Obstetric Complications	.00	.00	.62
Adoption Openness	.00	.01	-.42
Cohort	-.06	.01	-4.41**
Obstetric x <i>Time</i>	.00	.00	-.56
Openness x <i>Time</i>	.00	.00	.22
Cohort x <i>Time</i>	.03	.01	3.18**

* $p < .05$. NA = Negative Affect, PI = Physical Involvement.

Table 5

Hierarchical Linear Regressions with BP Negative Affect Predicting AP Physical Involvement.

Variable	<i>B</i>	<i>SE B</i>	<i>t</i>
Final BM Model			
Obstetric Complications	.01	.01	.83
Adoption Openness	.08	.05	1.61
Cohort	.35	.09	3.82**
Negative Affect	-.03	.05	-.64
Final BF Model			
Obstetric Complications	.01	.01	.92
Adoption Openness	.08	.05	1.62
Cohort	.38	.09	4.14**
Negative Affect	-.18	.09	-1.92 ⁺

Note. ⁺ $p < .10$. * $p < .05$. ** $p < .01$.

Appendix B. Figures.

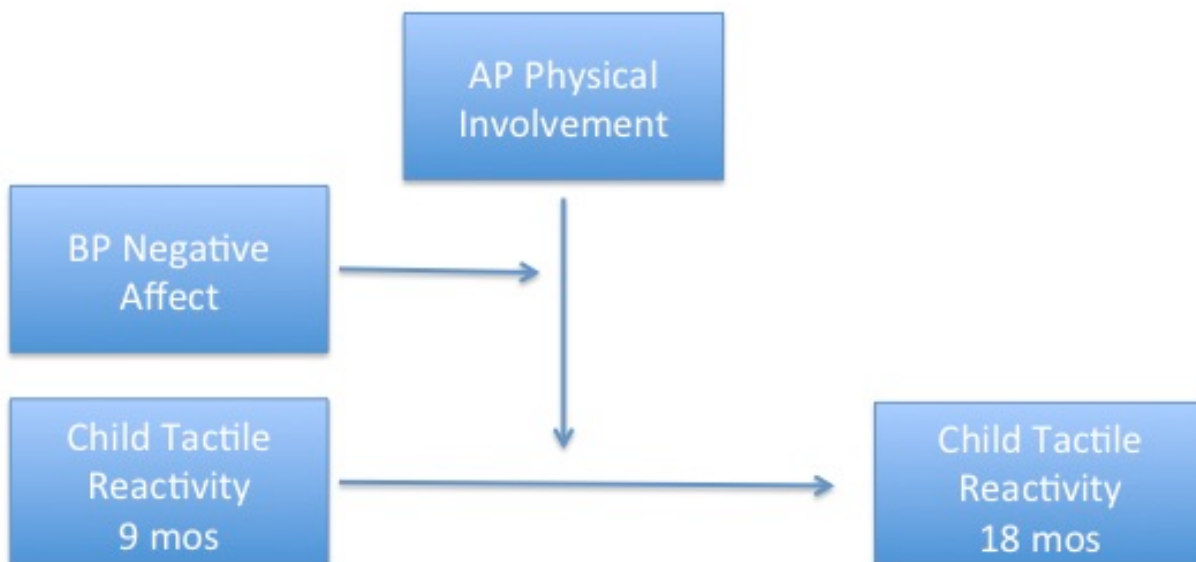


Figure 1. G x E: Children with Genetic Propensity for Tactile Reactivity Hypothesized to Show Greater Decreases in Tactile Reactivity from 9 to 18 months as a Function of Adoptive Parent Support of Child's Engagement in the Physical Environment.



Figure 2. rGE: Children with Genetic Propensity for Tactile Reactivity Evoke Adoptive Parent Support for Engagement in the Physical Environment.

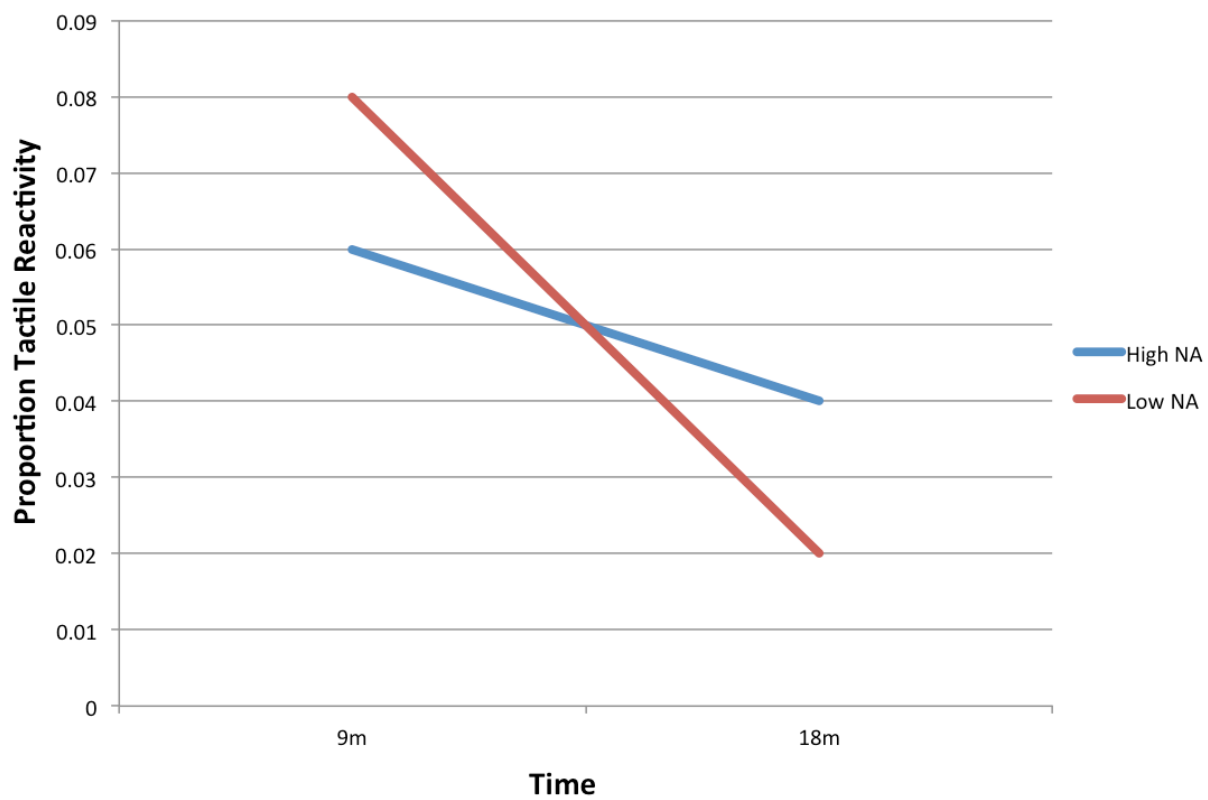


Figure 3. Mean Child Tactile Reactivity from 9 to 18 Months, at 1 SD above and 1 SD below Mean BM Negative Affect.

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