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Intergenerational Transmission of Abuse: Implications for Parenting Interventions From a Neuropsychological Perspective

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Abstract

Neuroimaging provides robust evidence to demonstrate the functional and structural deficits in childhood and adult survivors of child maltreatment. These deficits mirror the neurocircuitry involved in parenting. Despite this, research into the mechanisms behind the intergenerational transmission of abuse fails to acknowledge the contribution of brain development on future parenting ability. A discussion of the cognitive, social, and emotional deficits of child and adult survivors of childhood maltreatment is presented. This is followed by a critical overview of how current parenting interventions fail to take into account the neuropsychological mechanisms behind the intergenerational transmission of abuse. A conceptual model of the neuropsychological transmission of childhood maltreatment is presented. This model will allow child welfare practitioners to gain a greater understanding of the specific deficits of individuals who have experienced childhood maltreatment and how parenting interventions may be enhanced for this population. This represents one step forward in breaking the cycle of the intergenerational transmission of childhood maltreatment.

Keywords

child maltreatment, intergenerational transmission of abuse, neuroimaging, neuropsychology, parenting

Background: Intergenerational Transmission of Abuse

Abuse and maltreatment in childhood is one of the most destructive developmental experiences (Schore, 2001). Childhood abuse has profound impact upon many aspects of an individual's life, including parenting. Initial research on the parenting abilities of individuals who had experienced childhood abuse argued that these individuals were at increased risk of becoming perpetrators of childhood abuse (Egeland, 1993). However, although some parents who have experienced childhood abuse or maltreatment do become parents who maltreat their children, the rates are far from 100% and are currently estimated at approximately 30% (Oliver, 1993). Subsequently, models have emerged that target the intergenerational transmission of abuse, why and how individuals who experience childhood maltreatment do or do not become perpetrators of childhood abuse themselves. The explanations for this are wide-ranging including: social learning theory (Simons, Whitbeck, Conger, & Wu, 1991), parental attitudes and values (Hanson & Mullis, 1986), parenting styles (Van Ijzendoorn, 1992), and genetics (Muller, Hunter, & Stollak, 1995). However, even more recent summaries of the knowledge of the mechanisms of intergenerational abuse lack a neuropsychological perspective (Tomison, 1996). This is a dangerous oversight on the part of child protection researchers. Neuroimaging research is beginning to emerge that demonstrates how individuals can become neurodevelopmentally compromised as a result of childhood trauma and how this impairment can lead to deficits in future social relationships, attachment, and parenting.

The present paper aims to extend what we know about the intergenerational transmission of abuse by presenting research on the impairments exhibited by children and adults who have experienced childhood maltreatment. The impact of these impairments in the structure and function of the social brain will be discussed. It will be argued that the components of the social brain are analogous to the neurocircuitry of maternal social behavior. This research will be summarized in a neuropsychological attachment model of the intergenerational transmission of childhood maltreatment.

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Child Maltreatment and Brain Development

Evidence from animal studies first highlighted the crucial link between childhood maltreatment and brain development. This research has consistently demonstrated the impact of neglect on brain development (e.g., Eastman & Mason, 1974), showing that deprivation during a critical period reduces brain size and functionality. Evidence from animal studies also indicates a direct relationship between maternal care and hippocampal development. Higher levels of maternal care in rats (pup licking and grooming) have been found to lead to increased neuron density and cell volume in the hippocampus (Liu et al., 2000). There is an extensive body of literature on the consequences of early maltreatment in animals but discussion is beyond the scope of the current review.

The utilization of neuroimaging techniques in the field of childhood maltreatment has established a link between child maltreatment and comprised neurodevelopment. This has included children who have been raised in orphanages who were subject to substantial periods of neglect. Neglect leads to reduced frontal-occipital circumference, decreased metabolic activity in the orbitofrontal gyrus, infralimbic prefrontal cortex, amygdala, and hippocampus (Chugani et al., 2001). This has consequences for the social and emotional well-being of the child, with increased incidences of psychopathology and social difficulties being found in maltreated children (Molnar, Buka, & Kessler, 2001). Social, emotional, and cognitive difficulties often present in children who have been abused and are wide-ranging and pervasive, for example deficits in declarative memory (Cheatham, 2010), everyday memory (Moradi, Doost, Taghavi, Yule, & Dalgleish, 1999), language comprehension (Katz, 1992), executive functioning, and attention (Beers & De Bellis, 2002). These social, emotional, and behavioral deficits have profound implications for the ability of children to thrive and develop into resilient secure adults with a stable base through which to tackle the many challenges and adversities of life.

These social, emotional, and cognitive difficulties can be traced directly to brain development. More recent neuroimaging research in children who have been maltreated has established a link between the neuropsychological profiles of maltreated children and subsequent socioemotional and cognitive deficits. Structural and functional deficits in maltreated children span a range of different types of maltreatment. Maltreated children in psychiatric care have a significantly smaller corpus callosum; this relationship is stronger for children who have been neglected (Teicher et al., 2004). Reduced sized corpus callosum has also been found in children with posttraumatic stress disorder secondary to sexual or physical abuse (De Bellis et al., 2002). In addition to the corpus callosum, the size of the orbitofrontal cortex (OFC) is also reduced in children who have experienced physical abuse (Hanson et al., 2010). Differences between the responses to emotional cues have also been found in maltreated children, with greater neural responses to angry faces found in maltreated than nonmaltreated children (Cicchetti & Curtis, 2005). A maltreated child responding differently to negative affect has important implications for their social functioning. Overall, neuroimaging research on maltreated children presents a strong argument for the link between childhood maltreatment, brain development, and subsequent functioning. However, the specific neuropsychological profiles, socioemotional, and cognitive deficits of children who have been subjected to differing types and lengths of maltreatment are yet to be determined.

To further elucidate the link between child maltreatment and brain development, research on this area has also focused on retrospective research with adults who have been maltreated. This area of research focuses on retrospective self-report of child abuse in adults, as well as on the clinical and neuropsychological profiles of adults with borderline personality disorder (e.g., Schmahl, Vermetten, Elzinga, & Bremner, 2003). This area of research will be considered after a discussion of the mechanisms that lead to alterations in the structure and functioning of the social brain in maltreated children.

Mechanisms of Comprised Social Brain Functioning: Neurodevelopment and Stress

As a result of childhood maltreatment, the social brain can be significantly altered both structurally and functionally. Neurodevelopmental theorists propose a link between the environmental experience of a child, social brain development, and behavior—viewed as occurring through disruption to neurodevelopmental processes (Perry, 1995, 2001, 2002) or through sensitization of the stress systems (Heim & Nemeroff, 2001; Teicher, 2002). Maltreatment and abuse during childhood will disrupt neurodevelopment, for example neurogenesis, synaptogenesis, and myelination (Perry, 2002). The brain develops in a "use it or lose it" fashion—it requires a specific set of sensory stimulation from the caregiver as these are translated into neural activations that later form the basis of the neural representations of socioemotional bonds (Perry, 2002). Without these adequate inputs (as occurs in childhood maltreatment), necessary neural representations in the social brain will not develop and later effects will be seen in the social relationships. Human relationships including the caregiver relationship represent the "active ingredient" of the environment on child brain development (Shonkof, 2003, p. 72). Maturation of the "social brain" areas, for example amygdala, anterior cingulate, and limbic structures are thought to be particularly sensitive to the stress caused by early childhood maltreatment and trauma (Schore, 2001). Altered neurodevelopmental processes represent one possible way that the social brain and

therefore subsequent social behavior may be affected by early environmental influences, including the traumatic experience of childhood abuse.

Second, adverse effects on the social behavior of maltreated children may occur as a result of the brain becoming sensitized to and developmentally altered by stress. Inconsistent and infrequent caregiver responses that occur as part of the sequelae of childhood abuse and maltreatment are perceived as chronically stressful for the child (Bowlby, 1988). This chronic stress in infants is thought to lead to long-term hyperarousal (Heim & Nemeroff, 2001; Perry, 1995) that can be persistent, lifelong (Heim et al., 2001), and ultimately "trait" like (Perry, 2002). "The trauma of abuse induces a cascade of effects, including changes in hormones and neurotransmitters that mediate development of vulnerable brain regions" (Teicher, 2002, p. 4). These brain regions are thought to include areas that support social functioning through emotional responses, for example the hippocampus (Bremner, 1999). This view is supported by evidence that shows individuals who have experienced child abuse or maltreatment experience chronic hyperarousal to stress (Heim & Nemeroff, 2001) and that these same individuals experience comprised brain development (Cohen et al., 2006). The chronic hyperarousal that occurs in a sensitized brain is thought to affect the later social behavior of maltreated children, predominantly due to their difficulties with regulating affect (van der Kolk, 2005). These stress-induced difficulties with self-regulation lead to difficulties in social settings as the child (or adult) may be unable to differentiate, manage or express emotions, and control impulses (van der Kolk & Fisler, 2004). Cognition may also become more primitive (i.e., less able to focus upon consequences) when they are exposed to later stress (Perry, 2002). The view that early childhood abuse and maltreatment can have consequences for the social brain structure and functioning may have important implications for the way practitioners respond to both children and adults who have been maltreated.

"Social Brain Deficits": Adult Sequelae of Childhood Maltreatment

Social Brain Deficits Explained

Evidence on the relationship between specific brain structures (e.g., OFC, anterior cingulate cortex, limbic system, and the amygdala) and social and emotional functioning represents a possible explanation as to why individuals who experience childhood maltreatment may exhibit specific difficulties with social relationships (including the parent–child relationship). The OFC has been widely implicated in social behavior. Lesions of the OFC lead to a lack of affect (Rolls, 2006), impaired interpersonal behavior (Beer, John, Scabini, & Knight, 2006), altered fear behaviors (Babineau et al., 2011), and impulsivity and aggression (Anderson et al., 1999). Moreover, the OFC is also associated with representing the positive aspects of touch (Francis et al., 1999). Thus, individuals with impairments to this area may have difficulties in experiencing the positive emotions associated with their infant and responding to their infant in a social appropriate manner. Activity in the OFC is also affected by the level of cognitive demand inherent in a situation. Cognitive demand reduces neural responses in the amygdala and the OFC to emotional pictures (Kellermann et al., 2011). This can have worrying implications for the care of infants, as caring for an infant can have many competing demands, which inherently increases cognitive load. For example, the cognitive load associated with making a bottle for their infant while remaining emotionally responsive to the needs of the infant. The reduced activation of the OFC caused by this cognitive demand could be particularly more harmful for individuals who already have childhood trauma-induced compromised functioning in this area.

The anterior cingulate cortex (ACC) is another area of the social brain implicated in the social precursors of parenting. Activation in the ACC is associated with increased emotional awareness and the ability to detect emotional signals (Lane et al., 1998). Lesions of the ACC lead to emotional instability and apathy (Kennard, 1954). The ACC is part of a wide region of areas with reciprocal connections that are related to emotional regulation. The OFC, the ventromedial prefrontal cortex (VMPFC), the dorsolateral prefrontal cortex (DLPFC), the amygdala, and the ACC are key structures underlying emotional regulation and are shaped by early social influences (Davidson, Putnam, & Larson, 2000).

The amygdala was first implicated in social behaviors with the discovery of Klüver-Bucy syndrome (Klüver & Bucy, 1937). Animals with lesions to the amygdala demonstrated odd behaviors and approached objects that were previously feared without any fear. The amygdala plays an important role in memory, specifically the formation of episodic memory via modulation of hippocampal consolidation and also the recollection of emotional memories. It is also involved in attention and perception. It is thought to facilitate attention, especially in situations with limited attentional resources and in recognizing emotion in social stimuli, specifically recognizing fear in faces (Phelps, 2006). The role of the amygdala has important implications for the ability of parents to attend to the display of fear in infants and respond appropriately. This impaired amygdala functioning in parents can precipitate early trauma in infants as parents may not be able to respond appropriately to the emotional needs of their infant.

Childhood Maltreatment and the Adult Social Brain

Neuroimaging research demonstrates that adults who have experienced early childhood maltreatment show significant deficits in "social brain" areas, the adult sequelae of

childhood maltreatment. In adults who have experienced childhood maltreatment, the size of the following "social brain" areas such as the hippocampus (Dannlowski et al., 2012), amygdala (Weniger, Lange, Sachsse, & Irle, 2008), and ACC (Thomaes et al., 2010) has been found to be significantly reduced. In addition, some studies have implicated all these regions in the adult sequelae of childhood maltreatment (Cohen et al., 2006). Moreover, there is a "dose–response" relationship between adverse childhood experience and likelihood of impairments across multiple domains of cognition, emotion, and behavior (Anda et al., 2006). Research into the adult sequelae of childhood maltreatment further strengthens the link between early child maltreatment and future social brain structure and functioning.

The relationship between early adverse events in childhood and negative psychological outcomes in adulthood has also been highlighted by the study of psychological disorders such as borderline personality disorder (BPD). Childhood maltreatment has been linked to the development of BPD (Skodol et al., 2002) and borderline patients have unresolved experiences of trauma and preoccupied attachments (Fonagy, 2000). BPD is characterized by deficits in affect (e.g., chronic feelings of emptiness, inappropriate displays of anger, affective instability), cognition (e.g., stress related paranoid ideation and identity disturbance, impulsive behavior, and suicidal ideation), and unstable and intense interpersonal relationships. Patients with BPD have been found to show smaller volumes of the hippocampus and amygdala (Schmahl et al., 2003). Evidence from research on BPD contributes to our knowledge of the neurodevelopmental and functional (cognitive, emotional, and behavioral) impacts of childhood maltreatment.

Research into the BPD model of childhood maltreatment extends beyond the generalized interpersonal and affective impairments of sufferers to include more specialized areas of social relationships. BPD has been linked to disturbed parenting by Newman, Harris, and Allen (2011) as core features of BPD such as affective dysregulation, impulsivity, and overactivity to emotional stimuli that have significant implications for capacity to respond normally to stimuli provided by the infant. Parents with BPD often have experienced adverse childhood circumstances (e.g., emotional trauma is related to neurobiological deficits that impact on parenting). They state that parents who experience early abuse develop core difficulties in recognizing and responding to their child's needs and are in turn at risk of repeating disturbed parenting behaviors. Synthesizing the neurobiological evidence on parents with BPD they propose a "transgenerational regulatory model of parent-infant interactional disturbance" (Newman et al., 2011, p. 110). Within this model they suggest the intergenerational transmission of early trauma, disorganized attachment, and subsequent neurodevelopment sequelae is mediated by increased stress and decreased capacity for regulation, representation of self, reflective capacity, affect recognition/representation, affective regulation, and interactional reward. These characteristics result in an infant's early trauma-based representation of disorganized attachment caused by the parent being unattuned to the infant's emotional states. The subsequent neurodevelopmental changes result in similar emotional and social deficits present in the infant that are concurrently in the parent (i.e., decreased capacity for regulation, decrease reflective capacity, etc.). The theory presented by Newman et al. provides the crucial link between childhood maltreatment and the "social brain" deficits of parenting.

The Neuropsychology of Parenting: Crucial Role of the "Social Brain"

Although this research presents a solid foundation for the idea that comprised neurodevelopment due to childhood trauma would have consequences on parenting via impaired social deficits, it has not been until the last decade that research using neuroimaging paradigms (e.g., fMRI) has begun to emerge on the neural correlates of parenting. This research provides robust evidence that trauma compromised "social brain" regions are explicitly involved in the neurocircuitry of maternal behavior. The view that the neurocircuitry of maternal behavior mimics that of circuits responsible for other social attachments is also shared by other researchers (Swain, Lorberbaum, Kose, & Strathearn, 2007).

Areas involved in social reward are also implicated in the neurocircuitry of human maternal behavior. Lorberbaum et al. (2002) investigated the thalamocingulate circuitry of human maternal behavior. They compared the reaction of mothers to the cries of infants, with noise and a white noise plus rest condition. Mothers in their study reported an increased feeling of sadness in response to the cries of their own infants (which ensured they were measuring an attachment reaction). The anterior and posterior cingulate cortices, medial thalamic nuclei, and the bilateral mesial prefrontal cortex were activated in this condition compared to control noise. Their findings demonstrate that adaptive and positive maternal behaviors reflect general brain activity in response to appetitive (approach) stimuli. This is consistent with the view that frontal regions are involved in the representation of reward in social processes (Beer et al., 2006) and therefore that maternal behavior is experienced as rewarding in mothers with functional neuroanatomy in these areas. The experience of maternal care as rewarding acts as an important evolutionary mechanism to ensure security in the mother–infant dyad.

Nitschke et al. (2004) also improved on this study by incorporating unfamiliar infants as a comparison group. As most infant–mother relationships are positive, they predicted that that areas previously implicated in manifestations of positive emotion (i.e., OFC) would be activated. Consistent with this prediction, they found that mothers rated their mood as more pleasant after viewing a familiar,

as opposed to an unfamiliar, infant. The greatest bilateral OFC activation occurred in response to the mothers own infant rather than an unfamiliar infant. Ranote et al. (2004) also replicated this finding using more salient video stimuli. They compared mothers' responses to videos of their own infant, a neutral video, and an unfamiliar infant. Increased activation in bilateral OFC and medial prefrontal cortex was found for familiar infants as compared to the other conditions. The involvement of the OFC in the neurocircuitry of maternal behavior {suggests that}, for mothers who have neurodevelopmental impairments in this area the relationship with their infant will not be experienced as positive and therefore they may have difficulties providing consistent emotional warmth to their infant.

Other research on the neurocircuitry of maternal behavior indicates that the regions involved are extensive and closely mimic that of the social brain. Noriuchi, Kikuchi, and Senoo (2008) examined mothers' brain activity while her own infant (vs. other infant) displays a variety of attachment behaviors (smiling at her and crying for her). Videos presented were actual interactions between the mother and the infant. Mothers reported more positive emotions (e.g., happy, warmth, love) with their own infant versus other infants. Greater activation was shown in the medial frontal gyrus (MFG), superior frontal gyrus, angular gyrus, posterior gyrus, OFC, medial temporal gyrus (MTG), and dorsomedial prefrontal cortex (DMPFC) during the presentation of the video of play (smiling) in own infant versus other infants. During the presentation of the separation situation video they found increased activation in the OFC, MFG, MTG, superior temporal gyrus (STG), hypothalamus, and left putamen in own, as compared to other, infants. The wide-ranging regions involved in the neurocircuitry of maternal behavior indicates the importance of preventing and treating neurodevelopmental impairment in individuals who are, or likely to become, parents. As the neurodevelopmental impairments of childhood maltreatment span across the social brain, any impairment as a result of childhood maltreatment is likely to have an impact on parenting ability.

Just as infant attachment style is related to adult attachment style, maternal care in childhood is related to the expression of maternal care in adulthood. Taken with consideration of research that shows poor maternal care impacts upon maturation of brain structures, the mechanisms for the intergenerational transmission of abuse begin to emerge. A recent study by Kim et al. (2010) linked the neurocircuitry of maternal behavior directly to attachment style of the mothers. However, this was not measured directly and relied on self-report. Mothers who reported higher maternal care in childhood showed greater activations in the middle frontal gyrus, superior temporal sulcus, and fusiform gyrus in response to infants cries. Moreover, the first few months of motherhood were accompanied by structural changes in areas related to maternal care. This has implications for the timing of assessment and intervention of mothers who have experienced traumatic events during childhood and experience subsequent neuropsychological deficits.

Although fMRI studies convincingly demonstrate the existence of neurocircuitry designed for the regulation of learning and reward responses towards the infants and the regulation of parental responses, this knowledge has been poorly accommodated for within parenting interventions. Given that unresolved and disorganized attachment in mothers is a stable trait (Benoit & Parker, 1994) and the strong link between early attachment style and neuropsychological development and social and emotional functioning, parenting interventions should be taking account of the impact of childhood trauma on neuropsychological functioning and the ability to parent.

Practice Implications for Parenting of a Neuropsychological Perspective to the Intergenerational Transmission of Abuse

One of the most widely used, recommended, and researched parenting intervention is currently the Webster-Stratton Incredible Years Programme (Webster-Stratton, 1998). However, much research on the efficacy of the programme does not take into account the characteristics of the parent but instead assesses the outcomes of interventions on the complex parent–child dyad through outcomes based on the child's behavior only (e.g., antisocial behavior; Scott et al., 2001). Moreover, although The Incredible Years Programme contains elements such as providing tactile stimulation to your baby, strengthening children's social skills, and emotional regulation, it does not explicitly contains elements around "fear behaviors," which are prevalent in mothers with unresolved attachment. Given this it is entirely possible that the impact and efficacy of parenting programs may be enhanced by considering the existence of neuropsychological deficits caused by childhood maltreatment. Particular elements of the programme, for example promoting emotional regulation in their child may pose difficulties for parents with unresolved attachment due to potential underlying neuropsychological deficits in social brain areas.

Research suggests that unresolved attachment in mothers can impact upon the effectiveness of attachment-based interventions. These interventions focus on promoting a strong and healthy attachment relationship through enhancing the responsiveness and sensitivity of the mother's interaction with her infant (Moran, Pederson, & Krupka, 2005). Moran et al. assessed the effectiveness of an attachment-based program and found that the intervention that was designed to enhance maternal sensitivity was not effective for mothers who were classified as "unresolved" (Moran et al., 2005). A meta-analysis by Bakermans-Kranenburg, Van Ijzendoorn, and Juffer (2005) highlighted the lack of studies which evaluated interventions that were specifically

designed to prevent disorganized attachment. This meta-analysis shows the crucial need for parenting interventions to be tailored to parents with unresolved attachment issues. This is particularly poignant as infants may be removed from their caregivers due to the caregiver not being able to complete therapy within the timescale of the infant.

The transmission of the intergenerational transmission of abuse has been discussed in depth, both in the current review and in the wider research field. However, it is apparent that there is a gap between what we know about the neurodevelopment sequelae of childhood trauma and the subsequent impact on parenting and integration into treatment (and eventually effectiveness). Research by Bailey, Moran, DeOliveira, Gleason, & Pederson (2006) echoes the research by Moran et al. that sensitivity-based interventions are not effective for mothers who are classified as unresolved. They suggest some specific reasons why attachment-based interventions may fail parents who are unresolved, focusing on transference as a mechanism. They theorize that the transference of themes present in unresolved mothers (rejection, disappointment, lack of flexibility) leads them to have similar expectations of the mother–infant relationship. Mothers interpret the infant's behaviors within this theme and have atypical perceptions of the infants' emotions. This limits the ability of unresolved mothers to benefit from a sensitivity-based parenting intervention. This is consistent what we know from neuropsychology about how childhood trauma can lead to deficits in the function of the amygdala, which in turn leads to deficits in processing of emotional cues.

Newman (2010) takes one step in closing the gap between current neuropsychological knowledge and parenting interventions. They have designed a program for "high risk" parents called parenting with feeling (PWF). Newman defines "high risk" parenting as a style of parenting that includes a spectrum of behaviors likely to lead to disorganized attachment. The PWF program, which was developed from neuropsychological knowledge of mothers with BPD, is a group intervention targeted at high-risk parents and infants. The PWF differs from many other parenting programs as it aims to address trauma issues, increase parental reflective capacity, and to help individuals "parent in the face of adversity."

Parenting interventions should take into account the neuropsychological deficits parents have as a consequence of experiencing childhood trauma, and provide specific support to individuals to help them cope with the stresses and strains of parenting. Interventions will thus inevitably need to take into account neuropsychological assessment of individuals who have experienced childhood trauma. The neurodevelopmental consequences of childhood trauma are wide-ranging and subsequently the manner in which neuropsychological deficits are translated into parenting deficits in parents who have experienced trauma will thus also be wide-ranging. This highlights the need for comprehensive neuropsychological profiling of parenting who have experienced childhood trauma. This type of approach is captured by a new and exciting branch of applied neuropsychology, "Neurosequential Model of Therapeutics" (NMT; Perry, 2009). This approach aims to identify the key areas and systems in the brain that have been impacted as a consequence of adverse neurodevelopment and provide recommendations for enrichment and intervention to help restore functioning. A particular branch of NMT, known as the neurosequential model of caregiving (NMC), integrates the core principles of neurodevelopment and practically applies this model to inform parenting (Child Trauma Academy, n.d.) and is intended as compliment to NMT. Although a promising approach is becoming more widely used in clinical practice, use of NMC to tackle the childhood trauma has not been rigorously and systematically investigated. Research in this area will be a high priority to secure the future of the neuropsychological approach to tackling parenting deficits and increasing our understanding of how neuropsychology can contribute to tackling the intergenerational transmission of abuse.

Conclusion

This review has outlined the effect of childhood maltreatment on neurodevelopment, the cognitive, emotional, and social impairments that ensue as a result of childhood maltreatment. The neuropsychological sequelae can be severe in adulthood, with deficits in the "social brain" limiting an individual's success in their social world and ultimately their quality of life. When an individual becomes a parent these deficits affect the parent–child relationship as many of the "social brain" deficits are precursors for parenting. This article has outlined how new and promising neuropsychological evidence may be applied to parenting interventions and avenues for future research and specified future directions for this field. A number of caveats should be noted here. First, that despite the link between childhood trauma in parents, potential neuropsychological deficits and childhood behavioral problems that trauma during childhood does not prevent an individual from parenting per se but that it may represent one of many possible mechanisms through which abuse may be transmitted. As previously stated (Oscar, 1993), approximately one third of clients who have experienced abuse and neglect do not go onto abuse their child, one third do, and one third remain vulnerable. It is both the third that do and the third that remain vulnerable where this research may provide helpful suggestions for intervention. The research on neuropsychological consequences of childhood trauma provides a potential link in suggesting where we can intervene and break this link, where it does exist. The second caveat that should be emphasized is in the relation to the infancy of some of the therapeutic approaches grounded in neuropsychology. Both the NMT and the NMC proposed by the Child Trauma Academy have not been subject to wide scale efficacy studies. At this stage, there are no interventions that have been proven to assist with the effects of

neuropsychological deficits in the social brain on improving parenting. However, overall the research literature is highly suggestive that childhood trauma can have consequences on the functioning of adult social brain and subsequently can, in some individuals, affect parenting. Compromised neuropsychological functioning in children, who then become parents, is an area practitioners working with both parents and child in the child protection system should be aware. In future, with the evaluation and dissemination of promising therapeutic approaches grounded in neuropsychology, practitioners may have more in their toolkit with which to assist this vulnerable population.

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