Cognition, Emotion, and Neurobiological Development: Mediating the Relation Between Maltreatment and Aggression

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Child maltreatment has been consistently linked to aggression, yet there have been few attempts to conceptualize precisely how maltreatment influences the development of aggression. This review proposes that biases in cognitive, emotional, and neurobiological development mediate the relation between childhood maltreatment and the development of aggression. In addition, it is posited that physical abuse and neglect may have differential effects on development: Physical abuse may result in hypervigilance to threat and a hostile attributional bias, whereas neglect may result in difficulties with emotion regulation because of a lack of emotional interactions. These processes may be "hardwired" into neural networks via the overactivation of certain brain regions and dysfunctional cognitive processes. The theoretical and necessarily speculative nature of this article is intended to stimulate hypotheses for future research. Only when the adverse effects of maltreatment on brain and cognitive development are understood can scholars hope to develop more effective interventions to alter the developmental pathway to aggression.

Keywords: *maltreatment; aggression; physical abuse; neglect; cognition; emotion regulation*

There is an abundant and consistent literature that demonstrates that children who are victims of physical abuse are at greater risk of themselves becoming physically aggressive (e.g., Manly, Kim, Rogosch, & Cicchetti, 2001; Stouthamer-Loeber, Loeber, Homish, & Wei, 2001; Widom, 1989, 1998). Retrospectively, 75% of violent, sexual offending, and delinquent adults report histories of childhood trauma (Seifert, 2003). However, researchers have yet to determine exactly how maltreatment affects development to increase the likelihood of aggression. The research literature on the effects of child maltreatment has matured in the past few decades from examining correlates of maltreatment in general to studying in depth the mechanisms through which maltreatment influences negative outcomes.

The experience of a chronic traumatic experience of an interpersonal nature such as physical abuse or neglect by a child's caregiver in all likelihood will have a fundamental impact on that child's psychological and neurobiological development, particularly as complex abilities and neural connections continue to develop well into adolescence and even adulthood. Childhood and early adolescence are times of rapid change and extensive malleability: mentally, physically, and environmentally. Researchers have increasingly delineated the developmental stages of both neurobiological and cognitive growth. Different lines

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of research have focused on which brain regions mature at which time and at what ages cognitive abilities are acquired. Cognitive development can affect neurological development and vice versa (Diamond, 2002), and the importance of adaptive emotion regulation is being increasingly acknowledged.

Adolescence is a period when the effects of earlier childhood maltreatment will have more opportunities to manifest. Not only are adolescents developing and consolidating more abstract and complex modes of thinking, but they are also exposed to a greater variety of situations in which distortions in thought processes will become more apparent, such as more intimate peer and dating relationships, greater opportunities for delinquency, and more independence, in which they increasingly rely on themselves for decision making. The present review posits that maltreatment increases the risk of aggression via its impact on cognitive and emotional processing with persistent effects because of interactions with the child's developing neurobiology. It is also conceivable that although both physical abuse and neglect may result in later aggression, this process may occur via differential effects on neurobiological and cognitive development.

Maltreatment

The prevalence of child maltreatment cannot be understated. Recently, a national survey of victimization experiences of children and adolescents in the United States ages 2 to 17 years found that more than 1 in 8 had experienced a form of child maltreatment (defined here as physical, sexual, or emotional abuse; neglect; or family abduction or custodial interference; Finkelhor, Ormrod, Turner, & Hamby, 2005). If only a small fraction of these youth develop psychopathological or behavioral problems, the number of affected individuals suffering from emotional distress or themselves victimizing others will be in the tens or hundreds of thousands in a given year.

The main foci of this review are chronic physical abuse and neglect by caregivers. One caveat, however, is that researchers have frequently included various types of maltreatment (e.g., physical, sexual, emotional abuse; neglect; witnessing domestic violence) in their "maltreated" group; therefore, it is difficult at this time to examine the differential impacts of physical abuse versus neglect in this brief review. Findings for physical abuse and neglect are separately presented where available. For the purpose of this review, *maltreatment* is used in reference to more than one type of maltreatment or discussion of child maltreatment in general. Where relevant, studies are presented here that refer to the effects of trauma in general, including discrete events such as accidents and physical assaults, to integrate what has been discovered in different lines of research into a tentative formulation of how cognitive and emotion variables may generally mediate the relation between maltreatment and aggression.

The National Clearinghouse on Family Violence (2005) has stated that neglect occurs when a child's caregivers do not provide the requisite attention to his or her emotional, psychological, or physical development, but this is not a universally used definition. Various researchers apply different criteria to what constitutes "neglect," whereas others have failed to differentiate neglect from their omnibus construct of "maltreatment." Research is increasingly indicating that childhood neglect may have more of a negative impact on social, emotional, cognitive, and academic functioning than physical abuse (Erickson & Egeland, 2002), particularly throughout childhood and early adolescence (Hildyard & Wolfe, 2002). Childhood maltreatment outcomes often look similar for children who have been physically abused versus neglected, but it is conceivable that they may develop via different mechanisms. Although both physical maltreatment and neglect may be very traumatic for children, it is plausible that the negative effects of physical abuse may be partially offset by episodic positive emotional interactions with the maltreating caregiver, whereas the neglected child may not receive many emotional experiences at all, thus lacking opportunities to develop adequate emotion regulation skills.

Maltreatment and Aggression

Researchers have identified two major subtypes of aggression: reactive and proactive aggression. *Reactive* aggression refers to impulsive retaliatory aggression in response to a perceived threat or provocation (Dodge & Coie, 1987). In contrast, *proactive* aggression refers to aggressive acts in pursuit of a goal or desired outcome. Most relevant to the present discussion are the effects of maltreatment on reactive aggression, as this is posited to be the most closely linked to emotional dysregulation. Child maltreatment has been linked to a number of psychopathological outcomes, including aggression, particularly reactive aggression. It is difficult to assess the specific effects of different types of maltreatment on aggression because, as indicated above, researchers have typically included various forms of maltreatment in a single "maltreated" group. Similarly, aggression research is difficult to disentangle as definitions of "aggression" differ among studies, often including delinquent, antisocial, or conduct disorder behaviors as outcomes. This is important to keep in mind when comparing findings across studies.

Both physical abuse and neglect have been linked to a number of negative outcomes, with consistent links to aggression for both types of maltreatment. Manly and colleagues (2001) demonstrated that the impact of certain types of maltreatment on later aggression can vary by the age when it is experienced: The severity of *emotional* maltreatment in infancy and toddlerhood and *physical* abuse during the preschool period were strong predictors of later externalizing (i.e., "acting out") behaviors and aggression. However, this study assessed only children up to 11 years of age, making it difficult to draw conclusions on the long-term effects of maltreatment during these early periods on later adolescent or adult functioning. A nationally representative study of 2- to 17-year-olds in the United States found that child maltreatment made an independent contribution to anger and aggression and that even though other types of victimization experiences independently contributed as well, physical abuse and neglect had the strongest associations with anger, aggression, and depression in comparison to sexual victimization, witnessing family violence, and other major types of violence (Turner, Finkelhor, & Ormrod, 2006). Although socioeconomic factors have been found to influence both neglect and aggression (Schumacher, Slep, & Heyman, 2001), one study found that supervisory (i.e., parental monitoring) neglect mediated the effects of social disadvantage on the development of children's aggression (Knutson, DeGarmo, Koeppl, & Reid, 2005).

Maltreatment and aggression have been consistently linked, but what remains to be delineated is *how*. Cognitive, emotional, and neurobiological development are complex processes that continue into adulthood. The experience of physical abuse and/or neglect conceivably has implications for these developmental processes. It is proposed in this article that (a) maltreatment may increase the risk of aggression by overactivating certain cognitive processes, resulting in distortions or biases; (b) these distortions will be stronger relative to other, later-developing, higher-order processes and may in fact interfere with their acquisition; (c) emotion development, particularly regulation, will be compromised because of negative experiences and distortions in cognitive processing; and (d) these biases are consolidated in neural wiring as the brain continues to refine its neural connections and further develop the region responsible for higher-order processing, the prefrontal cortex (PFC).

It is hoped that in discussing how maltreatment can affect development, hypotheses can be formulated on how physical abuse and neglect can differentially affect subsequent development. Specifically, it is proposed that physical abuse may lead to aggression via cognitive biases for perceived threat, that neglect may lead to aggression via deficits in emotion regulation, and that both of these processes are strengthened by changes in neural "hardwiring" during development. Delineating the processes that lead from childhood maltreatment to adolescent aggression can indicate future research directions and eventually result in the design of more specific interventions earlier in childhood before the deleterious effects of maltreatment become more permanent and manifest as aggression.

COGNITION

A number of cognitive processes may be affected by maltreatment and/or have been associated with aggression. One difficulty is that researchers in different fields have not integrated their findings and appear to refer to some of the same processes with different terms. A discussion of executive functioning, or higher-order abstract processing, is presented, followed by a brief summary of higher-order processes that continue to develop from childhood into adulthood. In explicating the many processes involved in interpretation and decision making, it will become apparent that distorted development at any one level will have implications for processes that build on that level. Following that is a discussion of deficits in social information processing (SIP), which have been strongly associated with aggression.

Executive Functions (EFs)

EFs generally refers to the higher-order processes of self-regulation of thought, action, and emotion, all of which depend on the neural functioning systems involving the PFC (Séguin & Zelazo, 2005). The development of EFs appears to be more strongly and specifically involved in physical aggression than is general cognitive development, particularly in adolescence (Séguin & Zelazo, 2005). Components of EFs that are often measured in research include (a) vigilance and distractibility, (b) planning and organization, (c) response inhibition, (d) set shifting and categorization, (e) selective attention, (f) visual scanning, and (g) verbal learning (Biederman et al., 2004). Inhibition may be especially key to the development of executive functioning (Brocki & Bohlin, 2004). Developmentally, the ability to flexibly switch between two different perspectives on a situation appears at around 5 years (Séguin & Zelazo, 2005), whereas marked improvements are found on many cognitive tasks requiring the holding of information in the mind in addition to inhibition of behavior between

the ages of 3 and 7 (Diamond, 2002). Abstract reasoning and attentional set shifting steadily improve throughout adolescence, whereas response inhibition tends to level off by early adolescence (Rosso, Young, Femia, & Yurgelun-Todd, 2004). Although adult-level performance on many EF tests is attained at approximately 12 years of age, the development of more complex EF constructs continues to develop into adulthood (Séguin & Zelazo, 2005).

Although a number of basic cognitive functions are in place at an early age, the fact that executive functioning becomes more complex and continues to be expanded on well into adolescence highlights the risk for executive *dys*functions. Distortions or delays in the acquisition of normative functions at any level of this developmental process will likely hinder the appropriate development of metacognitive functions (e.g., planning). As one example, it is plausible that a selective attention bias for signs of threat would interfere with the abilities to sustain attention to another person or goal in an interpersonal interaction and to consider the perspective of another person when one is constantly distracted or overwhelmed by a potential perceived danger.

Maltreatment and executive functioning. Maltreated children of preschool and early school age have been shown to display heightened vigilance and attention patterns in response to aggressive stimuli as compared to nonmaltreated children of the same age (Rieder & Cicchetti, 1989). A study of previously neglected Romanian adoptees found deficits on executive functioning tasks assessing attentional and social deficits (Chugani et al., 2001). Children ages 5 to 12 years with a history of neglect have shown a decreased verbal learning capacity, likely stemming from deficits in registration and retrieval, relative to comparison children (Turgeon & Nolin, 2004). Interestingly, Pears and Fisher (2005) found differential effects of neglect versus physical abuse in preschoolers in foster care. Although neglect was negatively associated with executive functioning and physical development, physical abuse was found to be positively associated with these outcomes.

EF deficits and aggression. Irregularities have been reported in executive functioning in youth with aggressive tendencies (Brocki & Bohlin, 2004; Séguin, Nagin, Assaad, & Tremblay, 2004) that have been found to predict aggressive behavior 2 years later in older children and early adolescents (Giancola, 2000). Early evidence of EF weaknesses has been associated with externalizing behavior problems 2 years later in elementary school children (Nigg, Quamma, Greenberg, & Kusche, 1999), and with delinquent behavior in adolescence (Moffitt & Henry, 1989), independently of intelligence, reading ability, sex effects, or earlier behavior problems. Both verbal and nonverbal working memory deficits have also been associated with physical aggression even after controlling for IQ and general memory (Séguin et al., 2004). Although children and adolescents with disruptive behavior problems often present with comorbid attention deficit/hyperactivity disorder (ADHD) symptoms, a number of studies have found that EF difficulties continue to be related to physical aggression, even after controlling for ADHD (Giancola, Mezzich, & Tarter, 1998; Séguin, Boulerice, Harden, Tremblay, & Pihl, 1999; Toupin, Déry, Pauzé, Mercier, & Fortin, 2000). High levels of perseveration have been found in children with externalizing behaviors (Matson & Fisher, 1991) and in adolescents with a history of physical aggression (Séguin, Areseneault, Boulerice, Harden, & Tremblay, 2002), suggesting that youth who physically misbehave may have difficulty controlling their responding or considering alternative perspectives to situations.

A study with university students found that individuals who reported impulsive aggression (i.e., emotionally charged aggression out of proportion to the situation) performed worse on tasks requiring planning and organization than did their nonaggressive peers, although they did not differ on simple verbal measures (Villemarette-Pittman, Stanford, & Greve, 2003). Villemarette-Pittman and colleagues (2003) suggested that it was not language ability per se that was the main impairment in the impulsive aggressive group; rather, it was inefficient executive functioning that accounted for their poorer performance on complex verbal tasks. They concluded that EF deficits were a major contributing factor in the development and persistence of the hostile, aggressive, and antisocial behaviors exhibited by people with impulsive aggressive behaviors. One mechanism they proposed for this association was that poor expressive skills interfere with the verbal mediation of behavior, particularly in emotionally charged situations (Mungas, 1988). They hypothesized that impulsively aggressive individuals cannot "talk" themselves through alternative solutions or responses or adequately use verbal negotiation in a conflict situation (Villemarette-Pittman et al., 2003).

Interference with verbal abilities may also be in part because of a selective development of *nonverbal* cognitive capacities when under chronic stress; in other words, the youth may be constantly hypervigilant for danger cues and may overinterpret nonverbal cues at the expense of verbal cues (Perry, 2001). Because of their traumatic experiences, these youth

may have learned that picking up on nonverbal cues (e.g., facial expressions) is more important than verbal information; thus, chronically traumatized children can be less efficient at processing and storing information (Perry, 2001). Because of this overreliance on nonverbal cues, cognition may become dominated by the more "emotional" neural regions (e.g., limbic areas; Perry, 2001; see Neurobiological Development section below).

There is preliminary evidence for cognitive deficits in maltreated children and accumulating evidence that EF deficits are associated with aggressive behaviors. It appears likely that EF deficits mediate, at least in part, the relation between maltreatment and aggression via such biases and deficits as selective attention to threatening cues, misperceiving and misinterpreting others' intentions, and difficulties in response inhibition, to name just a few possibilities. No models, however, have yet been proposed by EF researchers explaining how EF deficits may arise from trauma and affect later aggression.

Higher-Order Cognitive Development

As they develop, older children and adolescents increasingly acquire the abilities needed to think abstractly and logically, they begin to consider the future more, they increasingly take on the perspective of the third person, and they begin to place greater importance on their roles in life (Zubenko, 2002). Although early adolescents are still somewhat egocentric, adolescence is typically the period when the ability to think abstractly emerges (Garbarino, Eckenrode, & Powers, 1997). Thus, adolescents are still acquiring the skills needed to abstractly think about their own, others', and society's problems in general. More than ever before, adolescents continue to develop alternative points of view and the ability to independently evaluate the motives and behaviors of others (Garbarino et al., 1997). This also means that they are better able than a few years prior to infer the motives behind others' actions (e.g., maltreatment), although they may still not be able to fully comprehend the reasons.

Decision-making processes and the development of a metacognitive orientation, or the ability to reflect on and evaluate one's own knowledge or that of another person (Moshman, 1998), continue to be elaborated on in adolescence (Byrnes, 2003). By late adolescence or early adulthood, there is increasing insight into the notion that there are evidence- or reasoning-based techniques for evaluating the accuracy and validity of multiple perspectives (Kitchener, King, Wood, & Davidson, 1989; Schommer, Calvert, Garigleietti, & Bajaj, 1997).

The full impact of childhood maltreatment may not be appreciated until the child further develops and engages in more mature relationships and responsibilities and develops more sophisticated cognitive capabilities (Lubit, Rovine, DeFrancisci, & Eth, 2003). Consequently, the impact of earlier childhood trauma may not manifest itself until the adolescent has matured enough to fully comprehend the experience and has avenues in which to express the effects (e.g., delinquency, substance abuse).

Theory of mind (ToM). This refers to the understanding of human action in terms of mental states, such as intentions, beliefs, and desires (Zelazo, Qu, & Müller, 2005). The development of ToM begins in infancy and develops across the lifespan (Zelazo et al., 2005). There are at least four steps involved in successfully inferring another person's mental state and using that inference to predict behavior (Flavell, Miller, & Miller, 2002): (a) appreciate that one may have a different perspective from another in a particular situation, (b) formulate a hierarchy of inferences for determining the other person's mental states in relation to one's own, (c) keep track of changes in the environment and in the other person's behavior so as to make appropriate adjustments to one's ideas about the person's mental states, and (d) deduce likely behavior based on these inferred mental states.

What may appear to be a relatively simple process is dependent on the successful development of a number of prior cognitive skills. Distortions in any one of these steps may lead to an inaccurate perception of what the other individual is thinking and planning. It becomes clear when considering all the more basic skills that provide the foundation for higher-order skills that any deviations or dysfunctions in prior skills will create difficulties in attaining more abstract and logical capacities. It may, therefore, become even more difficult to overcome biases in determining what others' intentions are. Delays in the development of ToM have been found in maltreated children, thereby indexing their deficits in the ability to attribute beliefs, desires, emotions, and intentions to others (Cicchetti, Rogosch, Maughan, Toth, & Bruce, 2003).

The successful attainment of these higher-order abstract skills seem to be dependent on the development of the some of the more basic executive functioning "building blocks," such as attending to relevant information needed to infer another person's state of mind and being able to shift from one frame of reference to consider alternative perspectives. Paralleling the developmental psychology literature, researchers in cognition and neuropsychology

have examined similar higher-order cognitive processes in the form of EFs. Although there is a more specific focus on planning, regulation, and reasoning than in social cognition, there is arguably a great deal of overlap between the social development and neuropsychology arenas, with findings that are relevant for maltreatment and aggression in one field having implications for research in the other.

Social Information Processing

Deficits in SIP mechanisms in youth with aggressive behaviors have been extensively studied, particularly in children. Dodge and his colleagues (Crick & Dodge, 1996; Dodge, 1993; Dodge & Schwartz, 1997) have described distortions in encoding, retrieving, and recalling information and in response selection and have proposed that aggressive children hold a *hostile attributional bias* wherein ambiguous actions by other people are interpreted as having hostile intent.

Five main information-processing steps have been identified with respect to situations of aggression. Dysfunctions in the first two steps (i.e., encoding, mental representation) have particularly been associated with reactive aggression (Crick & Dodge, 1996; Dodge & Coie, 1987; Schwartz et al., 1998), whereas the last three steps (i.e., accessing responses, evaluating responses, response selection) have been implicated in proactive aggression (Crick & Dodge, 1996; Dodge & Coie, 1987). Relevant empirical findings for the first two information-processing steps are presented below.

Encoding involves attending to and encoding relevant cues into the working memory (Dodge & Pettit, 2003). For example, children prone to perceiving threatening cues may notice only perceived threats to the exclusion of other information that may disconfirm that perception. Children with aggressive behavior problems have been found to selectively attend to hostile cues (Dodge, Pettit, Bates, & Valente, 1995; Dodge, Pettit, McClaskey, & Brown, 1986), selectively recall hostile cues (Milich & Dodge, 1984), and neglect to take in relevant nonhostile cues (Dodge, Bates, & Pettit, 1990).

Mental representation involves the representation and interpretation of encoded cues in a meaningful way (Dodge & Pettit, 2003). The hostile attributional bias is implicated at this stage when the other person's intent is ambiguous (Dodge & Frame, 1982) or in the form of interpretational errors when the intent is clear (Waldman, 1996). Early adolescents with aggression problems have readily accessible hostile social constructs (Graham & Hudley, 1994) and tend to make hostile attributions rather than benign interpretations (Dodge, Price, Bachorowski, & Newman, 1990). Youth with aggressive behaviors may not seek out additional facts that disprove their interpretation (Slaby & Guerra, 1988). This involves interpretation, which could include such steps as perspective taking, perceptions of facial expressions, and ToM.

A hostile attributional bias appears to be particularly relevant to instances of reactive aggression rather than proactive aggression. Children and adolescents who have experienced maltreatment may develop a tendency toward a hypervigilance to perceived threatening cues that emerged as an adaptive response to actual threats in the past (Dodge, 2003). Yet the ability to process information regarding other people's intentions will vary with level of cognitive development; for example, reactive aggression in response to misinterpretations of peers' intentions has only been found around age 11 and may not be present at ages 8 to 9 (Crick & Dodge, 1996).

SIP and aggression. More than 100 studies have reported significant correlations between SIP and measures of aggressive behavior problems (Dodge, 2003). A meta-analysis of 41 studies found robust, significant associations between a hostile attribution of intent to peers and aggressive behavior (de Castro, Veerman, Koops, Bosch, & Monshouwer, 2002). Larger effect sizes were found when examining more severe aggressive behavior. This included studies examining 8- to 12-year-olds versus younger children, suggesting that hostile attributional biases require the development of more sophisticated cognitive abilities. Although it has been suggested that children's behavioral problems cause processing biases and deficits, Dodge (2003) has demonstrated through a review of longitudinal studies that it is likely that SIP mechanisms leads to later aggression. It is also quite probable that aggressive behavior and its social and interpersonal consequences help to maintain and/or solidify these processing biases. Dodge and his colleagues reported that cognitive processes, including selective attention and hostile attributional biases, partially mediated the relationship between physical maltreatment experienced prior to kindergarten and later externalizing behaviors in second grade (Weiss, Dodge, Bates, & Pettit, 1992), in fifth grade (Dodge et al., 1995), and in adolescence (Dodge, Crozier, & Lansford, 2001), even when controlling for initial levels of aggression.

Integration of EFs and SIP. Although research on executive functioning and SIP has rarely been integrated, these constructs have overlapping meanings and findings that are integrable in a model of the effects of maltreatment on aggression in developing

children and adolescents. It appears that these two literatures from different psychological disciplines have overlapping constructs and often refer to similar types of variables. For example, it has been posited that adults with low levels of executive functioning may demonstrate poor SIP skills and an inability to cope with overwhelming response options; thus, these individuals may fail to access socially appropriate alternatives and automatically access their default aggressive responses to perceived provocative situations (Hoaken, Shaughnessy, & Pihl, 2003). Vigilance, selective and sustained attention, and the inability to process multiple relations simultaneously could conceivably relate to hypervigilance and how social information is encoded and interpreted. Difficulties with set shifting, categorization, spontaneous organization, and the ability to consider another person's perspective could contribute to a hostile attributional bias in that aggressive individuals may have difficulty shifting criteria for what they consider to be "hostile" signs and to consider alternative intentions on the part of the other person. It appears quite likely that there are overlapping constructs, and thus findings from one discipline could likely be applied to the other in terms of future aggression outcomes.

Although emotions have been acknowledged by SIP researchers as the energy driving information processing, they have not been explicitly addressed in SIP models. The two fields have been relatively separately studied (de Castro, Merk, Koops, Veerman, & Bosch, 2005). Recently, however, de Castro and his colleagues (2005) examined emotion regulation and SIP variables, including hostile intent attribution, response generation, and response evaluation, finding emotion regulation to be especially important to the SIP model. In subsequent regression analyses, associations between aggression and other SIP variables became nonsignificant after hostile intent attribution was entered into the equation. Thus, de Castro and his colleagues suggested that aggressive behavior is a function of hostile intent attribution and maladaptive emotion regulation.

EMOTION

Cognition and emotion have not typically been integrated in studies of cognitive processing and of emotion regulation; however, it is highly probable that cognitions and emotions strongly interact with one another. It is difficult to differentiate the effects of each; moreover, one cannot completely understand cognitive functions without an appreciation for the social and emotional context surrounding them (Phelps, 2006). Because of the intricate interweaving of neural networks, particularly between the regions responsible for executive functioning (i.e., PFC) and emotion regulation (i.e., amygdala; see Neurobiological Development section below), the activation of one type of network will inevitably affect the others. Bell and Wolfe (2004) have argued that emotion regulation research must necessarily include cognition as it is likely that the neural mechanisms are the same as those underlying cognitive processes. Emotions will also determine to which stimuli the mind should allocate attention (Siegel, 1999).

Disruptions in the development of emotion regulation have been put forward as a factor in the development of aggressive behavior. Emotion-regulation difficulties are associated with disruptive behaviors (Eisenberg & Fabes, 1999; Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002). Aggressive children have been found to exhibit more intense and frequent expressions of anger in emotionally arousing situations (Bohnert, Cmic, & Lim, 2003), although the monitoring and regulation of emotions may reduce aggressiveness (de Castro, Bosch, Veerman, & Koops, 2003). Although this type of research has been studied in children and adults, adolescents have rarely been the focus.

Emotional Development

Adolescents have to master a variety of emotion regulation skills for adaptive development at a time of exposure to more stressors, negative emotional experiences, and interpersonal relationships. Developmental achievements in adolescence do not include simply the development of more abstract cognitive capacities but also the emotional adjustment to new ways of perceiving the world. Some of the skills adolescents need to develop include regulating intense emotions; modulating rapidly vacillating emotions; achieving awareness of, and successfully attending to, one's own emotions without becoming overwhelmed by them; distinguishing emotions from facts to avoid emotional reasoning; and negotiating and maintaining interpersonal relationships in the presence of strong emotion (Rosenblum & Lewis, 2003). If these skills are deficient or never appropriately develop, the individual's capacity to regulate affect in emotion-arousing situations may be compromised. With the emergence of abstract thought in adolescence, emotions may come to be triggered by abstract ideas, anticipated future events, or recalled past events (Rosenblum & Lewis, 2003). It appears that the development of more abstract cognitive skills brings with it the improved ability to infer others' emotions and to cope with conflicting emotions. Thus, youth who have

developed maladaptive modes of cognitive processing because of maltreatment would likely show impaired ability to cope with the increased complexities and intensity of interpersonal interactions in adolescence.

Emotion Regulation

Emotion dysregulation has been found to mediate the relations between maltreatment and reactive aggression (Shields & Cicchetti, 1998); maltreatment, bullying, and victimization (Shields & Cicchetti, 2001); and harsh parenting by mothers and child aggression (Chang, Schwartz, Dodge, & McBride-Chang, 2003). Differential effects on emotion processing have been found for physical abuse versus neglect. For example, a history of physical abuse has been linked to a response bias to perceptions of *angry* facial expressions, whereas a history of neglect has been found to relate more strongly to difficulty in *discriminating* emotional expressions and a bias toward sad faces (Pollak, Cicchetti, Hornung, & Reed, 2000). Considering their maltreatment experiences, this makes sense. For children who have experienced physical maltreatment, displays of anger in their environment are their strongest predictors of threat; thus, a selective attention to threat-related (i.e., anger-related) signals at the expense of attention to other emotional cues would be adaptive (Pollak et al., 2000). In contrast, neglect is typically associated with an emotionally impoverished environment, with few opportunities for meaningful social interactions. If children are deprived of interactive emotional experiences with others, their capacity to tolerate intense emotion states, including positive emotions, may be underdeveloped (Siegel, 1999).

Not only do interactive experiences enable children to experience high levels of emotionally engaged arousal, but they also may entrain neural systems to be able to manage such states (Schore, 1996). Without the experience of meaningful interactions with caregivers, children may be at a disadvantage later in life when increasingly coming into contact with interpersonal relationships outside of the home. Goldsmith and Davidson (2004) have questioned why affective states are so adaptable in childhood yet resistant during psychopathological states in adulthood and have posited that this may be because of developmental changes in neural circuitry. Hence, the importance of intervening as early as possible during the development of emotion regulation processes becomes apparent.

Empathy

It is likely that the development of empathy will prominently factor in some aspects of aggressive behaviors. Empathy is the product of the coordinated operation of both emotional and cognitive processes (Hoffman, 1984). The levels of role-taking skills developed are predictive of levels of empathy (Roberts & Strayer, 1996). Rosenblum and Lewis (2003) have theorized that although the basic skills for empathic awareness are in place by adolescence, they are not enough to promote empathic *responding*; additional emotion regulation skills are needed for the experience and expression of empathy. To develop the ability to empathically respond, one must be able to tolerate the affect generated by an empathic connection (Eisenberg et al., 1994). Otherwise, in the presence of negative affect, individuals who have poor emotion regulation skills may experience high levels of negative arousal and personal distress (Eisenberg, 2000).

It has therefore been argued that if emotional regulation is associated with empathic responding, then the skills the child or adolescent has developed or will develop for coping with negative affect will fundamentally affect that individual's capacity to empathize with others throughout his or her life (Eisenberg, Fabes, Schaller, Carlo, & Miller, 1991). If these skills are deficient or never appropriately develop, the individual's capacity to regulate affect in emotion-arousing situations may be compromised. For example, adolescents with conduct disorder exhibit more personal distress, their emotional experiences become more self-focused and aversive, and they are less able to match the affective states of another when confronted with scenarios designed to evoke empathic responding (D. Cohen & Strayer, 1996).

Integration of Cognition and Emotion

As discussed, the relations among the following constructs are fairly well established: (a) biases in SIP, especially toward perceiving threat and attributing hostile intent to others, are related to displays of aggressive behaviors; (b) maltreatment has been associated with deficits in executive functioning skills (e.g., inhibition, selective attention, set shifting), which in turn are associated with increased rates of aggression; and (c) maltreatment is often associated with emotion dysregulation, which in turn has been related to aggression. Theorizing and preliminary evidence for impairments in ToM and empathy also imply roles in the relation between maltreatment and aggression. Thus, it appears that deficiencies and biases at various stages of cognitive processing and emotion regulation have a strong impact on the development of aggressive behaviors and are likely implicated in maltreatment, although delineating the latter relationship requires further investigation.

Although these lines of research have developed relatively independently of one another, there seem to be many similarities among them. Future research needs to find a way to integrate the results into a coherent picture of the effects of maltreatment on cognitive and emotional development and the subsequent effects on aggression.

As the brain continues to develop throughout childhood and adolescence, it is extremely difficult to attain a complete understanding of how maltreatment influences aggression without considering its impact on neurobiological, cognitive, and emotional development and the interactions among these variables. The following section provides a brief summary of relevant neurobiological development and proposes that repeated activation of certain cognitive and emotional processes (e.g., selective attention, hypervigilance to threat) may hinder the acquisition of more abstract abilities (e.g., perspective taking), partially through the overactivation of corresponding neural networks.

NEUROBIOLOGICAL DEVELOPMENT

The human brain continues to develop throughout childhood, adolescence, and beyond (Sowell et al., 1999). One implication is that children acquire increasingly complex cognitive abilities as they get older. This is not a genetically predestined course, however. Experiences during development may affect current and subsequent development, both physically and cognitively. Although the effects on neurobiological development are numerous, the following sections outline research most relevant to executive functioning, emotion regulation, and aggression, beginning with the development of the neural regions governing higher-order cognitive processing (e.g., PFC) and emotional processing (e.g., limbic system), a description of the physiological stress response and the effects of continued stress on the brain, followed by findings with maltreated and aggressive populations.

Neural Development

Although the majority of brain development occurs within the first 5 years of life, brain structures and synaptic "pruning" away of relatively inactive neurons continue to develop throughout middle childhood and adolescence. Much of the brain's higher cognitive processing occurs in unmyelinated portions of neurons (i.e., grey matter), which dramatically grow during the first 2 years of life and in preadolescence (Giedd et al., 1999) to early adolescence (Kanemura, Aihara, Aoki, Araki, & Nakazawa, 2003). Myelination refers to the white fatty substance covering the axons of some neurons; it greatly increases the speed and efficiency of information transmission between neurons. The number of myelinated axons (i.e., white matter) increases in a roughly linear pattern throughout childhood and adolescence (Giedd, 2004). It is thought that structural changes in grey and white matter improve the efficiency of frontal lobe functioning by selectively pruning away unused neural synapses and by improving the conductance of electrical signals among active neurons (Giedd et al., 1999; Spear, 2000), the most intense period of which occurs between the ages of 7 to 16 years (Pihl & Benkelfat, 2005). This results in use-dependent patterns of connectivity; hence, experience plays a major role in the development of the PFC (Pihl & Benkelfat, 2005). If certain neural regions are overactivated relative to others, it is conceivable that more neuron pruning will occur in the underutilized area. One implication for maltreated youth if they are chronically hypervigilant for signs of threat rather than seeking out alternative explanations is that it would physically become that much harder for them to use other more adaptive cognitive processes that are governed by relatively underdeveloped neural regions with fewer synaptic connections.

Prefrontal Cortex

Although executive functioning is not synonymous with the functioning of the PFC, the construct of executive functioning was originally derived from examinations of the consequences of damage to the PFC (Zelazo et al., 2005). The PFC is part of a neural circuit that plays a central role in fear conditioning and stress responsivity (Ishikawa & Raine, 2003) that continues to develop into early adulthood (Kostovic, Skavic, & Strinovic, 1988; Sowell et al., 1999) and the fourth decade of life (Bartzokis, Beckson, Neuechterlein, Edwards, & Mintz, 2001), making it particularly vulnerable to disruptions during development. The majority of PFC development rapidly occurs in the first 11 years of life, reaching adult levels of neuronal myelination in adolescence, which dramatically increases the transmission speed of information between neurons (Diamond, 2002). Improvements in cognitive processing that occur after the age of 7 years appear to be tied to the PFC, particularly in the dorsolateral PFC (DLPFC; Diamond, 2002). The PFC regions most relevant to the present discussion of cognition and emotion are the DLPFC and the orbitofrontal cortex (OFC).

DLPFC. The DLPFC is hypothesized to be involved in emotion processing, necessary for the manipulation

and encoding of information in the working memory and important for the maintenance of information in the presence of distracting stimuli (Ishikawa & Raine, 2003). Dysfunction in the DLPFC may play a role in perseverative functioning (Diamond, 2002; Ishikawa & Raine, 2003) and response inhibition (Liddle, Kiehl, & Smith, 2001), which may cause difficulty in considering alternatives, even when faced with evidence that current behaviors are not working. The ability to "set-shift," or shift to alternative views of a situation, is especially vulnerable to damage to the DLPFC as compared to damage elsewhere in the brain (Stuss et al., 2000). The DLPFC is important for controlling impulses and is among the last of the brain regions to mature. It does not develop to adult dimensions until the individual reaches his or her early 20s (Giedd, 2004); thus, this may be especially vulnerable to disruptions in development.

OFC. The OFC helps to regulate negative affect and autonomic activity (Ishikawa & Raine, 2003). Along with the amygdala (discussed below), the OFC has a role in extracting emotional content from environmental stimuli (Ishikawa & Raine, 2003) and perceiving social signals that indicate anger (Bufkin & Luttrell, 2005). In terms of cognitive associations, the OFC has been linked to delayed gratification, intentional behavior, and the control of motivation states such as aggression (Paus, 2005). Damage to the OFC has been associated with poor impulse control, aggressive outbursts, and a lack of interpersonal sensitivity in adult criminal offenders and psychiatric patients (Bufkin & Luttrell, 2005). The mechanism underlying the suppression of negative emotion is thought to be through an inhibitory connection from regions of the PFC, likely the OFC, to the amygdala (Davidson, Jackson, & Kalin, 2000).

Because the OFC detects signals connoting anger and the DLPFC appears to be more involved in response inhibition and perseverative responding, could it be that chronic overactivation of the OFC contributes to the relative underdevelopment of the DLPFC? If so, that suggests that stronger and more numerous connections in the OFC developed via a history of physical abuse could lead an individual to be hypervigilant to anger signals along with a weaker ability to inhibit responses and shift perspectives. Davidson and colleagues (Davidson, Jackson, et al., 2000; Davidson, Putnam, & Larson, 2000) have suggested that dysfunctions in one or more prefrontal regions or the interconnections among them may be associated with impairments in the regulation of negative emotion and, thus, an increased propensity for impulsive aggression.

Limbic System

Major structures in the limbic system include the hippocampus, hypothalamus, and amygdala. The hippocampus has a role in memory formation and retrieval and is thought to act in response to signals from the amygdala (Amaral, Price, Pitkänen, & Carmichael, 1992), whereas the hypothalamus regulates the autonomic nervous system, and therefore stress response, through hormonal production and release. Both the hippocampus and hypothalamus play key roles in the physiological stress response by regulating cortisol, a stress hormone (Lovallo & Thomas, 2000; see below). Most pertinent to this discussion is the amygdala, which has a primary role in emotion processing and regulation. The amygdala is extensively and reciprocally connected to the OFC (Kaufer & Lewis, 1999). Although the OFC in the PFC is primed to detect angry affect, the amygdala is activated in response to environmental cues connoting threat, such as expressions of fear (Bufkin & Luttrell, 2005). Information about these cues is conveyed to the amygdala and integrated with projections from the OFC about the social context and then projected to other structures in the limbic system (Bufkin & Luttrell, 2005).

Higher levels of amygdala activation have been linked to excessive negative affect, whereas too little activation may lead to a decreased level of sensitivity to social cues that regulate emotion (Davidson, Putnam, et al., 2000). Activation of the limbic system during and after a traumatic experience could impede the development of information processing, specifically memory retrieval and recall, that is essential for discerning intentionality, personal responsibility, sense of control, and trust in others. This would restrict a child from developing cognitive schemas to deal with interpersonal intimacy, possibly resulting in aggression, avoidance, or both (Hartman & Burgess, 1993). Alternatively, perhaps the lack of soothing emotional experiences in childhood neglect leads to overactivation of the limbic system because the child does not experience adequate resolution of fearful experiences by caregivers.

Physiological Stress Response

The hypothalamic-pituitary-adrenal (HPA) axis (also known as the hypothalamic-pituitary-adrenocortical axis) is considered the body's stress response system and appears to specifically respond to situations of psychological stress involving negative emotion and perceptions of lack of control (Biondi & Picardi, 1999). The HPA axis, with its central nervous system (CNS) controls, is considered to be a complete

integration of hormonal outflow with thoughts and feelings (Lovallo & Thomas, 2000). When the HPA axis is activated by a stressor or perceived threat, the hypothalamus secretes corticotropin releasing factor, which stimulates the release of adrenocorticotropic hormone from the pituitary, finally stimulating the release of cortisol from the adrenal cortex. Cortisol, a key regulator of glucose metabolism, enters the blood–brain barrier, allowing it to reach all parts of the CNS (Lovallo & Thomas, 2000). Cortisol terminates the stress response through feedback at various levels of the HPA axis (Van Voorhees & Scarpa, 2004), particularly the hippocampus (Jacobson & Sapolsky, 1991). The effects of a dysregulated stress response will affect a variety of neural regions. In particular, cortisol reaches the limbic system (i.e., amygdala, hippocampus, and hypothalamus; Lovallo & Thomas, 2000); thus, it is assumed to be linked to both emotional and cognitive processes because of the intricate interconnections between the limbic and prefrontal regions (Wolkowitz et al., 1990).

The expression of a stress response is dependent on whether the situation is perceived as potentially threatening, the outcome is perceived to be important, and the individual expects to have resources necessary to manage the perceived threat (Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997). Stressors may lead to *hypercortisolism*, or chronic hyperactivity of the HPA system, which, over time, may have deleterious effects on the immune system and brain regions involved in memory and attention and may influence the expression of defensive behaviors (Gunnar et al., 1997), delay the development of myelination, produce abnormalities in synaptic pruning, and impair affective and cognitive ability (Sapolsky, 1992). The chronic activation of a stress response (e.g., via heightened cortisol levels) results in neuronal death (Sapolsky, 2000). Chronic stress could also lead to the converse, *hypocortisolism*, in which there is decreased adrenocortical secretion and reactivity (Gunnar & Vazquez, 2001), which may result in neuronal damage (Heim, Ehlert, & Hellhammer, 2000). Neural systems that are repeatedly activated through trauma or chronic stress may undergo permanent changes, whereas chronic activation of the HPA axis may result in damage to the limbic system with cortisol as a possible mediator of this process (Nelson & Carver, 1998; Sapolsky, 1996). Thus, chronic stressors such as maltreatment could be expected to influence the developing brain by impairing neuronal growth, particularly in the prefrontal and limbic regions.

Maltreatment and Neurobiology

Maltreatment during early childhood may have an especially profound impact on neurological development when rapid neuronal growth and extensive neuroplasticity render the brain particularly sensitive to environmental input (Black, Jones, Nelson, & Greenough, 1998; Cicchetti & Tucker, 1994). As the PFC is continuing to develop throughout childhood, the potential for maltreatment to affect developing cognitive structures and processes becomes apparent. Exposure to even mild uncontrollable stress can impair the cognitive functioning of the PFC (Arnsten & Shansky, 2004). Adverse impacts in early childhood can have cumulative and cascading effects on future brain development (Repetti, Taylor, & Seeman, 2002). Subsequent alterations in cognition or social interaction may amplify the effects of the original traumatic experience or experiences. Although research is lacking of the effects of neglect on neuropsychological development in children and adolescents, de Bellis (2005) has hypothesized that neglect interferes with the effective development of prefrontal cortical regions and therefore executive functioning. Child neglect has been associated with delayed cognitive development and head growth in very young children (Strathearn, Gray, O'Callaghan, & Wood, 2001). Similarly, a study of previously institutionalized Romanian children adopted by American parents who experienced caregiver neglect early in life has revealed decreased metabolism in the brain structures involved in cognitive function and social intelligence, such as the orbital frontal gyrus, PFC, and amygdala, as compared with other children with chronic illness and healthy adults (Chugani et al., 2001).

Most interhemispheric connections between the frontal lobes traverse through the corpus callosum (Kaufer & Lewis, 1999). Children in psychiatric hospitals with a history of maltreatment have a corpus callosum that is, on average, 17% smaller than in psychiatric control children (Teicher et al., 2004), suggesting that there is less integration of information between the right and left sides of the frontal lobes. This study found that children who had been neglected had the largest reductions in corpus callosum size relative to children who had been physically or sexually abused, lending credence to the notion that the underutilization of neural connections in neglected children can result in permanent altered growth or neuronal death.

The developing brain is extremely sensitive to stress: Early exposure to unpredictable or chronic stress results in functional deficits and vulnerability to

future stressors (Perry, 2001). Chronic exposure to trauma can fundamentally and permanently alter brain and physiological development. Changes in brain wiring may have a fundamental impact on how the adolescent processes and interprets information. For example, Perry (2001) has elucidated three ways in which persistent fear states may affect brain and, hence, cognitive functioning. One, the development of the brain itself and its myriad of interconnecting networks may change in response to a traumatic experience in a use-dependent way. That is, brain systems that are normally relatively inactive may be chronically activated by trauma, and thus change how the brain develops. Two, chronic fear may alter how the brain internalizes and *stores* information experiences with an element of fear to them. And finally, trauma may change how the brain *retrieves* stored information.

Compared to a nontraumatized child living in a safe and calm environment, the traumatized child may have different parts of the brain regulating his or her cognitive functioning. The capacity to internalize new verbal cognitive information depends on having portions of the frontal and related cortical areas being activated, which itself depends on a state of attentive calm (Perry, 2001). Because of chronic experiences of danger, the traumatized youth may have a foreshortened sense of the future, placing too much emphasis on immediate reward (Perry, 2001) and thus impeding the normal cognitive development in adolescence of the effects of his or her actions on the future. When constantly in an alarm state, the internal regulating capabilities of cortex may malfunction, for example, the brainstem (which controls more of the basic emotional survival functioning) may reflexively, impulsively, and aggressively act to any *perceived* threat (Perry, 2001).

Aggression and Neurobiology

In 2004, a National Institutes of Health (NIH) conference, titled Preventing Violence and Related Health Risk-Taking, Social Behaviors in Adolescents, identified a concern that there has been relatively minimal incorporation of research on human brain development in the field of violence prevention (NIH, 2006). Yet if a child or adolescent has experienced intensely stressful experiences with the subsequent overactivation of certain neural regions (e.g., amygdala), perhaps connections with those areas are strengthened relative to other later-developing areas (e.g., DLPFC). One possible consequence of this dysfunction may be poor inhibition over aggressive behavior (Ishikawa & Raine, 2003) or an overactivation of the OFC, resulting in a bias toward detecting anger signals. In relation to emotion regulation, Paus (2005) has proposed that the development of such structures as the amygdala underlie the initial development of aggressive behavior earlier in life but that the relatively late development of cortical regions such as the PFC could enable adaptive regulation of aggression in the social context. It could be the case that intervention in late childhood or adolescence could help to overcome initial dysfunctions caused by maltreatment.

Findings on the relation between cortisol levels and aggression have been very mixed in children. Slightly more consistent findings have been found for adults: Higher levels of aggressive or antisocial behaviors are associated with lower levels of cortisol (Van Goozen, 2005). These findings have been replicated in children with externalizing and aggressive behaviors (Hart, Gunnar, & Cicchetti, 1996; McBurnett, Lahey, Rathouz, & Loeber, 2000). In contrast, some studies have found a positive relationship between aggression and cortisol response level in normal adolescents during experimentally induced aggression (Gerra et al., 1997) and during an emotion-arousing and painful procedure (Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos, 1997), whereas other studies have no relation between the two (Scerbo & Kolko, 1994; Schulz, Halperin, Newcorn, Sharma, & Gabriel, 1997; Stoff et al., 1992). Although the precise nature of the relation between cortisol levels and aggression remains unclear, it appears that abnormal cortisol levels are present in people displaying aggressive behaviors. It seems quite likely that a chronic traumatic experience such as physical abuse or neglect by a caregiver will interfere with the adaptive regulation of the HPA stress response; thus, if a youth has difficulty regulating his or her cortisol levels and therefore emotional responses, one effect may be a tendency toward overreacting to perceived threats with aggressive responses.

It is evident that maltreatment can have adverse impacts on the developing brain, which can also affect later development and the fine-tuning of neural networks. Environmental experiences may dramatically affect the development of the brain, its structures, and its interconnections. One way that neurological development may be affected is via chronic activation of hormonal stress responses. Another influence may be the over- or underactivation of certain cognitive processes, such as chronic hypervigilance to threat, which can lead to specific parts of the brain being overutilized and thus expanded on. Alternatively, certain structures of connections may be underutilized and undergo selective "pruning" of relatively inactive neuronal synaptic connections. For example, heightened and chronic stress levels will influence brain anatomy that affects

the processing of information and the perceptions of threatening stimuli. Increased activation of the amygdala in relation to the PFC may generate an excessive focus on emotions and disrupt the inhibition of negative affect. Disruptions to the PFC may also interfere with adaptive processing, memory, and interpretations of environmental, particularly social, stimuli. These dysfunctions in cognitive processing and emotion regulation may in turn affect further neuronal activation and pruning in the circuitry regulating the inhibition of emotions and behaviors.

CONCLUSION

Although it is widely acknowledged that child maltreatment increases the risk for aggression, the mechanisms through which this occurs remain unclear. A number of interventions have been proposed for victims of maltreatment and for aggressive youth, but no "gold standard" treatment has been identified for maltreated youth who may become prone to aggression. To devise such an intervention, one must identify which processes account for a large proportion of the relation between maltreatment and aggression. The impact of maltreatment on adolescents has been understudied, despite the fact that they are continuing to develop and are in the process of acquiring higher-order, abstract cognitive skills at the same time that their brains are maturing and perhaps being permanently altered via myelination and synaptic pruning.

One proposed mechanism through which maltreatment may influence aggression is via disruptions in cognitive development, particularly the development of higher-order executive functioning. The development of more complex, abstract processes continues throughout adolescence; this of course depends on the successful attainment of more basic cognitive processes in childhood, such as response inhibition, attention, working memory, emotion regulation, and recognizing emotions and intentions in other people. If there are fundamental dysfunctions in any of these lower-level processes, this could conceivably introduce bias and dysregulations to higher-order processes such as planning, reasoning, and perspective taking. SIP theory provides specific mechanisms through which cognitive errors may occur in the development of aggression and specifies at what stages in cognitive processing dysfunctions may occur. The integration of cognitive and developmental psychology research in the study of maltreatment and aggression would provide improved macro- and micro-level explanations for disruptions in cognitive development and which processes to specifically address in children and adolescents. The impact of major stressors in each of these stages of development may disrupt optimal development of specific cognitive functions, which can have cascading effects on the development of later higher cognitive functions. These stressors may have a direct impact on cognitive mechanisms, for example, through a bias for attending to threatening cues in the environment, or a more indirect impact, via the disrupted development of brain regions.

Neurobiological structures and functions continue to develop into adulthood, particularly those implicated in higher-order cognitive functioning, such as the PFC. As a result of this continued development, maltreatment could be expected to have a differential impact at various developmental stages, especially through interactions with cognitive and neuropsychological development. It is quite conceivable that parts of the brain that are chronically activated by stressors (e.g., limbic system by threat, PFC by anger) may have those connections strengthened at the expense of more adaptive but underutilized neural connections, which may be permanently pruned away (i.e., those underlying emotion regulation).

What can be concluded from this overview is that although there has been a great deal of research on the effects of maltreatment on aggression, the specific mechanisms through which this influence occurs remain unclear. Cognitive and neuropsychological irregularities conceivably could interact with each other to strengthen dysfunctional connections and processing. Not only is it expected that cognitive and neuropsychological dysfunctions would interact, but it is also likely that they have additive effects on outcomes because of "cementing" dysfunctional cognitive processes in neural wiring. It may also be the case that aggression may result from physical abuse and neglect, but through slightly different pathways. The need to examine the differential impact of physical abuse versus neglect is being increasingly recognized. The devastating consequences of childhood neglect on development are being acknowledged more and more, and while neglect is not typically viewed with the horror with which other types of maltreatment are viewed, it is the most common form of maltreatment (USDHHS, 2004) and may actually have more detrimental effects on the developing child (Hildyard & Wolfe, 2003). Furthermore, neglect may lead to more permanent consequences if neurobiological regions or cognitive and emotion capacities do not develop appropriately. It is entirely plausible that different *types* of maltreatment will have specific effects on development. If hypervigilance for signs of threat is adaptive in the environments of

physically abused children and an impoverished emotional environment is a result of neglect, it is conceivable that difficulties for physically abused children will include hypervigilance for threat and hostile attributional biases, whereas neglected children may primarily experience difficulties with emotion regulation. Although the outcome (i.e., aggression) may look the same, it may be via quite distinctive processes. If the primary cue for threat in the environment of a physically abused child is anger, it would be adaptive in that environment for the child to be vigilant to signs of anger. This may affect the development of neural regions associated with anger, such as the OFC, consequently "hard-wiring" the overactivity of this region. This selective attention, or hypervigilance for signs of threat, in addition to a cognitive bias to perceive hostile intent in others' actions, may reinforce one another and eventually manifest as a "default" tendency to misinterpret situations as threatening and thus aggressively react in retaliation or preemptive defense. Analogously, if a neglected child lacks emotional interactions and experiences of being soothed by a caregiver, this may result in irregularities in the neural regions guiding emotion regulation, primarily the limbic system. Thus, as the child enters adolescence, he or she may experience increasing difficulty regulating emotions as he or she is exposed to a greater variety of, and more intense, interpersonal and social situations. Of course, physical abuse and neglect often co-occur, but it will be of paramount importance to distinguish between the two as much as possible in future research.

The interaction between biological and environmental factors is important to consider, and the field of behavioral genetics has demonstrated great promise in recent years in illuminating developmental pathways involving genetics, maltreatment, and aggression. In particular, Moffitt (2005) has outlined methods to delineate this complex relationship beyond the traditional genotype versus phenotype approach. This was illustrated in a longitudinal study by Caspi and colleagues (2002) that examined the interaction between genes and environment in children with a history of maltreatment. These researchers found that maltreated children with high levels of a certain neurotransmitter-metabolizing enzyme were less likely than those with low levels of the same enzyme to develop antisocial problems, whereas maltreated youth with low levels of this enzyme were 3 times more likely than nonmaltreated youth to develop conduct disorder and 10 times more likely to be convicted of a violent crime. Future research into how biological and environmental factors interact to mediate the relation between maltreatment and aggression will provide meaningful insights into how to intervene with maltreated youth.

Presenters at the 2004 NIH conference on violence prevention in adolescents lamented the fact that scientific evidence is rarely considered when awarding public funding for violence prevention programs (Tuma, Loeber, & Lochman, 2006). Childhood maltreatment remains a prevalent societal issue, and concerns around youth aggression appear to be increasing in the mass media. A number of interventions have been found to be moderately effective for traumatized youth (e.g., trauma-focused cognitive behavioral therapy; J. A. Cohen, Mannarino, & Knudsen, 2005) and aggressive youth in terms of reducing behavioral problems. A recent meta-analysis of behavioral parent training and cognitive behavioral therapy for antisocial youth (including aggression) found medium effect sizes for both (McCart, Priester, Davies, & Azen, 2006). For maximal effect, it is necessary to address developmental issues and the effects on, and interaction of, cognitive and neuropsychological development. If aggression is manifested via different pathways in physically abused versus neglected children, optimal interventions will need to more specifically target these pathways. In addition, differences in neurocognitive-emotive processes may contribute to differential responses to intervention (Fishbein, Hyde, Coe, & Paschall, 2004).

Most concerning is the lack of research focusing on adolescents. Interventions typically consist of downward adaptations of adult treatments or upward extensions of child interventions (Weisz & Hawley, 2002). Yet adolescence is a time when the effects of maltreatment become more evident (e.g., deficient development of more abstract cognitive abilities such as empathy and perspective taking), and it provides more opportunities to aggress in terms of increasing independence from caregivers, greater deviant peer influence, and exposure to risky activities (e.g., substance use, sexual activity). Adolescents must learn how to cope with the intense emotions introduced by hormonal changes in puberty, more complex and involved peer interactions, and negotiating with dating partners. It is crucial to integrate the unique developmental needs of adolescents into the design of prevention and intervention. Research in different areas appears to be converging on similar results and needs to be integrated to elucidate a comprehensive model of the effects of childhood maltreatment on subsequent adolescent aggression, taking into account cognitive, emotional, and neurobiological development.

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