Social and Biological Changes During Adolescence That Precipitate the Onset of Antisocial Behavior

Lisa M. Gatzke-Kopp, David DuPuis, and Robert L. Nix

Adolescence is a period of development anecdotally synonymous with rebellion, defiance, and a vpropensity to engage in behaviors that call into question the existence of a human instinct to survive. Although these behaviors evolve from a normative motivation to establish independence and develop a personal and sexual identity, adolescence is a vulnerable time period for the development of pathological behavior. Criminological data indicate a nearly tenfold increase in crime during the adolescent period, a rate that substantially declines over the course of adulthood.

The observed rise in antisocial behavior during adolescence led to the proposal of classes of antisocial individuals that were considered to be taxonomically distinct (Moffitt, 1993). It was originally proposed that the behaviors associated with delinquency were relatively normative in adolescence, and thus carried less risk when onset occurred in adolescence versus in childhood. Onset of antisocial behavior during early childhood was significantly more predictive of chronicity of symptoms, whereas onset in adolescence appeared to be a more transient form of antisocial behavior, often remitting in adulthood in conjunction with the increase in responsibility associated with independence (Moffitt, 1993). Reflecting the differential prognostic value between the two categories, the terms "life course persistent" and "adolescent limited" were coined as labels for the two subgroups.

In the past two decades, however, evidence has amassed that these labels oversimplify the categorization of antisocial individuals. Specifically, onset in adolescence does not appear to be entirely normative, nor is it a relatively innocuous or necessarily temporary condition (Moffitt & Caspi, 2005; Roisman, Aguilar, & Egeland, 2004). Regardless of age of onset, individuals with elevated levels of antisocial behavior in adolescence are distinguishable from non-antisocial peers both in terms of their early risk factors (Roisman et al., 2010) and their adult outcomes (Woodward, Fergusson, & Horwood, 2002). Furthermore, antisocial individuals evidence common individual risk factors, such as poor fear conditioning, regardless of age of onset (Fairchild, Van Goozen, Stollery, & Goodyer, 2008). Therefore onset of antisocial behavior in adolescence appears to be along the continuum of risk associated with onset in childhood, and

L.M. Gatzke-Kopp, Ph.D. (✉)
Department of Human Development and Family Studies and Neuroscience, The Pennsylvania State University, 315 HHD East, University Park, PA 16802, USA
e-mail: lisakopp@psu.edu

D. DuPuis
Department of Human Development and Family Studies, The Pennsylvania State University, 315 HHD East, University Park, PA 16802, USA
e-mail: dud164@psu.edu; lisakopp@psu.edu

R.L. Nix, Ph.D.
The Prevention Research Center, The Pennsylvania State University, 316A Biobehavioral Health Building, University Park, PA 16802, USA
e-mail: rnix@psu.edu

W.T. O'Donohue et al. (eds.), *Handbook of Adolescent Health Psychology*,
DOI 10.1007/978-1-4614-6633-8_28,

the risky behaviors associated with this disorder can result in lifelong consequences to the individual, diverting them into an adverse developmental trajectory (Molero, Hodgins, Larsson, Larm, & Tengstrom, 2010; Odgers et al., 2008; Roisman et al., 2010).

This chapter will focus on the social and biological changes associated with adolescence that heighten the risk for the onset of antisocial behaviors during this period. Consistent with the notion of a continuum, increases in the severity, chronicity, or accumulation of contextual and individual risk factors decrease the latency to symptom onset. For individuals in the moderate range of risk exposure, symptom onset may not be evident until later in development, when the underlying vulnerability is amplified by developmentally normative changes in neural structure and function that occur across the adolescent period. Metaphorically, for some individuals, the flux of social and biological changes during this period represent the gasoline on a previously contained campfire.

Definitions

It is important to first address the different permutations and definitions of the broad category of antisocial behavior. Following from the American Psychiatric Association's diagnostic manual (American Psychiatric Association, 2000), the two most pertinent disorders to this discussion are oppositional defiant disorder (ODD) and conduct disorder (CD). ODD consists of a pattern of easy loss of temper, arguing (particularly with adults or authority figures), failing to abide by rules, deliberate attempts to annoy others, being angry or spiteful, and displacing blame for one's actions onto others. In an effort to avoid over-pathologizing developmentally normative behavior associated with toddlerhood and adolescence, diagnosis is reserved for individuals with four or more symptoms causing significant interpersonal impairment for a period of 6 months or longer (DSM-IV-TR, 2000). Symptoms classified in conjunction with CD tend to represent more severe behaviors along this continuum, including destruction of property, such as fire-setting; physically violent or cruel actions; theft or fraud; and statutory crimes, like regularly skipping school. Again, to avoid diagnosing sporadic or situation-specific behaviors, symptoms must be present over the preceding 12-month period in order to meet diagnostic threshold.

Comorbidity between ODD and CD exceeds the predicted rates that would occur if each diagnosis were truly independent of the other. This has led some researchers to argue that the distinction between the two is artificial, and that the criteria simply represent antisocial behavior at different developmental stages (see Biederman, Newcorn, & Sprich, 1991). For instance, young children can easily engage in deliberately annoying behavior and defying adult instruction; however, a certain degree of physical maturity is required before they can successfully engage in crimes like armed robbery or sexual assault. Thus ODD is often considered a developmental precursor for CD. However, the majority of individuals who obtain an ODD diagnosis in childhood will not go on to meet the criteria for CD, suggesting value in retaining the two diagnoses as separate entities. The interrelated nature of ODD and CD has led the American Psychiatric Association to establish a hierarchical structure between these disorders. Because ODD is frequently considered to be a developmental antecedent of CD or a less severe manifestation of the same underlying pathology, ODD is not diagnosed in the presence of CD (DSM-IV-TR, 2000).

In addition to these formal diagnoses, many other terms are used to characterize pathological behavior of this nature. Developmentally, problem behaviors defined by their outward manifestation and impact on others are often referred to as "externalizing" behaviors. Researchers often examine correlates of symptoms in this domain without requiring that full diagnostic criteria be met. Additionally, some researchers focus more specifically on sociological—rather than psychological—constructs, such as the development of delinquent and/or criminal behavior as defined legally. Other researchers focus on the personality constructs that underlie complex psychiatric syndromes, such as impulsivity. Generally speaking, researchers across different disciplines

acknowledge the conceptual overlap between these various approaches, often reviewing literature in one domain to inform another. To this end, the term "antisocial behavior" is often used to subsume the various instantiations of pathological behavior.

Common Vulnerability

An inclusive approach to studying these related behavioral profiles is supported by behavioral genetics research, which indicates that demarcation between disorders such CD, ODD, and even attention deficit hyperactivity disorder (ADHD) may be artificial. These disorders appear to be influenced by similar physiological and environmental risk factors, leading to their frequently comorbid presentation (Burt, Krueger, McGue, & Iacono, 2001; Hofvander, Ossowski, Lundstrom, & Anckarsater, 2009). In a study of twins followed longitudinally, externalizing disorders measured across childhood (CD), adolescence (delinquency), and adulthood (antisocial behavior and substance dependence), as well as non-pathological constructs such as disinhibited personality, could be explained primarily by a common latent vulnerability that is highly heritable (Krueger et al., 2002). This kind of common vulnerability has important implications for research and treatment of antisocial behavior over time. Rather than consider these diagnoses as independent contributions to adverse outcomes that may or may not be present in any given individual, this model proposes that any individual with one externalizing disorder is inherently at risk for the others.

The realization of this risk appears to be predicated on environmental exposures. For instance, although an individual displaying age-inappropriate oppositionality and a willingness to violate rules and social norms is at high risk for developing substance dependence, doing so requires exposure to substances of abuse, a phenomenon correlated with environmental factors such as neighborhood, peer, and family factors that make substances of abuse accessible. Thus, an individual's propensity to develop a psychological disorder may not manifest until, or unless, the right environmental circumstances interact with individual risk. It follows logically then, that the increase in manifestation of antisocial behavior in adolescence reflects the expansion of environments that the individual is exposed to. The identification of the environmental processes that contribute to the progression from disinhibited personality to a specific form of psychopathology is invaluable in identifying appropriate targets for intervention.

Epidemiologically, boys are more likely to garner a CD diagnosis than girls (DSM-IV-TR, 2000). This has led to some controversy regarding approaches to research and diagnosis of these disorders with respect to sex differences. This is consistent with males' tendency to be more aggressive in most mammalian species (Archer, 2009). However, some researchers have argued that the diagnostic discrepancy is an artifact of the gender-stereotyped behaviors listed as symptoms for CD, which favor behavior more typical of boys. In response, researchers have argued that females engage in aggressive behavior toward peers, but are less likely to do so in a physical domain, instead focusing on the use of gossip and slander to manipulate social status as a mechanism of harming peers (Crick, Bigbee, & Howes, 1996). Although this profile of behavior, termed "relational aggression," is more prototypical of females, it is likely a construct unrelated to CD. Furthermore, although the criteria for CD may contain legitimately sexually dimorphic symptoms, such as aggression (see Archer, 2009), females are quite capable of meeting the existing criteria for CD. However, they tend to do so by exhibiting different profiles of behavior than males. Females with CD are less likely to engage in physical fighting, but rather to express their impulsivity and proclivity for law breaking through behaviors such as truancy, lying, stealing (without confronting the victim), using drugs, and engaging in promiscuous sex, possibly for money (Vera, Ezpeleta, Granero, & de la Osa, 2010). Indeed, much of the research regarding risk factors for antisocial behavior does not support a sexually divergent model (Fontaine, Carbonneau, Vitaro, Barker, & Tremblay, 2009).

Family Processes

Parents represent the first socializing influence on children and, as such, play a fundamental role in the development of behavioral and emotional regulation. However, as children grow into adolescence, both parents and children struggle with the changing need for autonomy. Although parents might welcome adolescents' increased self-sufficiency, they often worry about what they do not know of their adolescents' independent lives. Parents continue to play an important role during this transitional period, even if an unwelcome one on the part of the child.

Research on general parenting styles reveals an association between an authoritative approach to child-rearing and adolescent adjustment (Steinberg, 2001; Steinberg, Mounts, Lamborn, & Dornbusch, 1991). Parents who are authoritative display high levels of sensitivity and responsiveness to their adolescents' needs but also expectations for respectful and responsible behavior. These parents tend to rely on more democratic means of control and encourage their adolescents to express themselves. In contrast, factors consistently associated with antisocial outcomes and early substance experimentation in youths include a lack of parental supervision, harsh punishment practices, and lack of emotional support (Murray & Farrington, 2010).

In addition to broad parenting domains, research has also revealed specific coercive cycles of exchanges that can take place between parents and young children or adolescents that contribute to the escalation of antisocial behavior (Granic & Patterson, 2006; Patterson, 1982). For instance, a single cycle might begin with a mother asking her son to clean his room. The child might resist by passively ignoring the instruction, actively defying it, or engaging in aversive behaviors such as whining. If the parent is feeling tired or stressed, she might decide not to follow through on the request. Because the child's behavior was aversive, the parent's decision to escape the argument is negatively reinforced and thus more likely to occur again. Likewise, the parent's withdrawal of her request negatively reinforces the child's aversive response. Thus both parent and child have reinforced each other in the exchange, which has failed to result in a satisfying and appropriate resolution. In future episodes, the parent is more likely to be exacerbated by the child's defiance and escalate her demand with yelling and threats of punishment. Over time, the child learns that anger, threats, and dominance are an effective means to accomplish goals.

This kind of coercive cycle of exchanges may be especially likely to occur in adolescence, when the newly developed need for autonomy leads most youths to experiment with more defiant responses to parental control. Parents may be inclined to avoid unpleasant arguments with irritable adolescents and retreat from a more active role in their adolescents' daily lives. Coercive exchanges that have undermined positive relationships and induced parents to withdraw and adolescents to withhold information may be particularly detrimental for high-risk adolescents, who often use unsupervised time to engage with deviant peer groups (Dishion, Nelson, & Bullock, 2004). As such, parenting practices and family interactions likely contribute to the confluence of risk factors that promote a rise in antisocial behavior during adolescence.

Peer Processes and the Moderating Influences of Parents

Friendship is a normative and fundamental aspect of adolescent development that exposes the individual to a world of social behaviors and expectations beyond that of the family context. However, the increasing influence of peers during adolescent development can introduce new contexts for psychopathological risk.

Association with deviant peers has been shown to have a facilitatory effect on individual delinquency (Brendgen, Vitaro, & Bukowski, 2000; Lacourse, Nagin, Tremblay, Vitaro, & Claes, 2003). This is likely a product of the tendency for antisocial individuals to associate with other deviant peers (Cairns, Leung, Buchanan, & Cairns, 1996), forming relationships in which peers mutually exacerbate each other's antisocial

behavior (Dishion, Bullock, & Granic, 2002). This phenomenon has been observed across developmental time and irrespective of age of onset of delinquency (Lacourse et al., 2003). Process-oriented research has determined that friendship interactions can increase antisocial behavior through mutually reinforcing responses to deviant talk (Dishion, Nelson, Winter, & Bullock, 2004). In fact, evidence from clinical interventions conducted in a group format has identified adverse outcomes of increased delinquency due to the introduction of deviant peers to each other (Dishion, Bullock, et al., 2002; Dishion, McCord, & Poulin, 1999; Poulin, Dishion, & Burraston, 2001). These findings support the notion of a causal role for peer influence in the development and exacerbation of delinquency, and indicate the powerful effect of social influences on deviance, even in a seemingly therapeutic context.

Despite the significant effect peers have on exacerbating antisocial tendencies, exertion of parental influence over adolescent friendships appears to have protective effects. There is some evidence that parenting practices, such as encouragement of achievement, influence adolescent adjustment, such as school grade point average, which in turn affects the peers adolescents tend to spend time with (Brown, Mounts, Lamborn, & Steinberg, 1993). Intervention research in which participants were randomly assigned to therapeutic resources during the middle school period indicates that strengthening parenting skills and parental monitoring of friendships resulted in a significant decline in deviant peer associations and delinquent behavior (Dishion, Bullock, et al., 2002).

However, as noted above, parent-child relationships are likely to become strained through the transition to adolescence as parental authority is met with adolescent autonomy needs. Research highlights the importance of warm, supportive family interactions that make adolescents comfortable disclosing what is happening in their lives (Kerr & Stattin, 2000; Stattin & Kerr, 2000) and allow parents to provide guidance regarding peer relationships (Mounts, 2000). Although supervision of adolescent's peer group is important, parents' approach to this responsibility cannot be overlooked (Steinberg, 2001). Authoritarian prohibition of a deviant friend can have the effect of increasing adolescent defiance of the parent, eroding the parent-child relationship, and strengthening the deviant peer friendship. Alternatively, parental prohibition of a specific friend that is delivered in an autonomy granting way, supporting the adolescent's perspective and providing a clear rationale, promoted acceptance of the parent's prohibition, and reduced the association with that deviant friend (Soenens, Vansteenkiste, & Niemiec, 2009).

Neural Correlates of Pathological Antisocial Behavior

Most research on antisocial behavior has focused on family and peer influences because of the desire to understand better those processes that might be amenable to intervention. However, in recent decades, more research has focused on individual variability in vulnerability to those influences. Although no specific brain region can be implicated in a complex behavioral syndrome, individual differences in neural function underlie traits such as emotional arousal, fear conditioning, and the ability to regulate behavior in the service of long-term goals. These systems are implicated in symptoms of conduct disorder, such as extreme anger and arousal that lead to fighting and aggressive behavior. Deficits in fear conditioning have also been implicated in covert delinquent behaviors, such as lying and cheating, and overt cruel behaviors, such as bullying. Variation in these systems interacts with environmental factors to establish patterns of behavior that become self-sustaining over time. Understanding the source of individual vulnerability may help refine intervention programs to more directly address the needs of individual adolescents.

Perhaps the most consistent findings associated with antisocial behavior are deficits in the structure and/or function of the prefrontal cortex (Raine, 2008; Yang & Raine, 2009). The prefrontal cortex in humans represents a large and functionally diverse region which is attributed the higher order cognitive functions that make humans unique.

Among these functions are the abilities to resolve competition between goals with conflicting timescales, to retain appropriate information in active memory while executing a behavior, and to update behavioral goals based on feedback from the environment. These actions often require the suppression of lower order brain regions that subserve emotions and otherwise would result in impulsive and reactive behavior. The evolutionarily older limbic system, including the nucleus accumbens, amygdala, and anterior cingulate cortex, is responsible for generating basic emotional drives such as fear, anger, and reward seeking, and has also been implicated in pathological behavior.

Several researchers have proposed a dynamic model of neural processing in which the prefrontal cortex and limbic nuclei each contribute to the execution of behavior, but do so adaptively under different circumstances (Lewis & Todd, 2007; Metcalfe & Mischel, 1999; Seeley et al., 2007). Under normal conditions, the prefrontal cortex is an invaluable resource in processing the complexities of judgment in social situations. To do this, the brain must integrate information from past experiences to generate a range of possible actions or responses and make predictions about their probable consequences; it then must select the action that has the highest likelihood of being effective. Although these computations take place quickly, they are far slower and more metabolically costly than the impulsive decisions executed by the limbic system. Under conditions of perceived threat, it is more advantageous to react immediately without concern for consequences. For instance, if one were to come across something that looks like a snake during a hike, it would be more reasonable to jump back immediately than to pause and notice upon closer inspection that the stimulus is in fact a twisted branch. Thus, under conditions of threat, emotional impulse overrides deliberate and possibly rational behavior. Metcalfe and Mischel (1999) coined the terms "cold" and "hot" cognitive processing to distinguish between the slow and methodical responses generated in the prefrontal cortex and the rapid and potentially irrational responses of the limbic nuclei. The balance between these systems is thus dynamic and contextually determined.

Neuroimaging research indicates reduced connectivity between the prefrontal and limbic regions in adolescent boys with CD (Shannon, Sauder, Beauchaine, & Gatzke-Kopp, 2009). This suggests that antisocial individuals may have difficulty modulating the relative balance between these two systems. This is consistent with findings revealing pathological disruptions in neurotransmitter systems that facilitate inhibitory control of limbic regions (Siever, 2008). Developmental research indicates that disruptions in neurotransmission in these regions is not sufficient to predispose an individual to aggressive behavior; however, such disruptions render an individual especially sensitive to environmental adversity that more readily programs hostility and aggression in response to provocation (see Buckholtz & Meyer-Lindenberg, 2008).

The confluence of these structural and functional deficiencies results in the individual's experiencing social-emotional cues differently than developmentally typical adolescents. It has long been observed that highly aggressive individuals show an increased tendency to infer hostile intent in others, especially when social situations are ambiguous (Dodge, 1986). By adolescence, most students who are bumped in a crowded hallway would dismiss the incident without much regard, presuming the contact to have been accidental, and noting only a minor physical annoyance. However, individuals characterized by psychopathology are more likely to suppose a purposeful and aggressive intention on the part of the bumping student. This attribution of hostility essentially engages the limbic system in the brain, eliciting a rapid, emotional, and defensive act of self-protection. This overidentification of threat results in a defensive override of appropriate judgment in the heat of the moment. This phenomenon explains why children and adolescents can often identify appropriate social responses to minor social threat in a hypothetical context, but frequently have difficulty enacting them in the real world, when the youths are more likely to be emotionally aroused.

Individuals with antisocial behavior may be more likely to attribute hostile intent in ambiguous or benign situations due to abnormalities in

processing emotional cues from facial expression. Adolescents with a history of CD show greater limbic activation in response to pictures with negative emotional content than age-matched controls, suggesting an over-reactive emotion response (Herpertz et al., 2008). However, eye tracking technology reveals that aggressive early adolescents do not focus on hostile social cues preferentially, but rather appear to be regulated by a preexisting social schema that construes social cues so they are consistent with expectations (Horsley, de Castro, & Van der Schoot, 2010). Thus, over-activation of emotion-processing regions of the brain appears to represent a functionally appropriate response to distorted cognitive interpretations, suggesting the importance of targeting these interpretive processes in intervention (Dodge, 2011).

Antisocial adolescents also appear to have deficits in processing reward-related information, even outside of the social context. This has important implications for understanding antisocial behavior, as reward and punishment contingencies are the driving forces behind behavior modification. Similar to individuals with acquired brain damage, adolescents and adults with antisocial behavior or substance abuse disorders have been documented to engage in poor decision-making in monetary tasks (Ernst et al., 2003; Stadler et al., 2007). Recently, researchers have examined the neural correlates of risky decision making in antisocial adolescents and found a consistent pattern of under-activation in the prefrontal and limbic regions during the decision-making process, suggesting a deficient recognition of the relative risk between decisions (Crowley et al., 2010). Furthermore, antisocial adolescents showed less activation during monetary wins than normally developing controls, and more activation during monetary loss (Crowley et al., 2010). These findings suggest that antisocial adolescents respond atypically to cues of reward and punishment; this difference likely contributes to the challenges of appropriately shaping these adolescents' prosocial behavior. Some researchers have suggested that externalizing disorders are characterized by deficient processing of reward cues, requiring larger, and more immediate, reinforcement to effectively shape behavior (Beauchaine, Gatzke-Kopp, & Mead, 2007; Sagvolden, Johansen, Aase, & Russell, 2005).

Additional research indicates that adolescents with CD have deficits in processing changes in reward contingencies. During a monetary incentive task, adolescents with CD and ADHD were compared to developmentally normative peers (Gatzke-Kopp et al., 2009). In this task, participants were asked to complete a simple game while undergoing a functional imaging scan. During some blocks of this game, correct answers resulted in the administration of a monetary reward, which accumulated across trials in the center of the screen for the participant to watch. During other blocks, the monetary reward was reset to zero, with no reward for correct responses although the participant was instructed to continue responding. The simplicity of the task ensured equivalent performance across groups, allowing for the examination of how adolescents with and without externalizing disorders reacted to the same levels of reward incentives. Individuals in both the CD and developmentally normative groups showed a robust activation in the caudate nucleus during blocks in which correct answers were rewarded, consistent with the neural networks associated with behavioral responding for reward. However, when participants engaged in blocks in which the same performance was no longer accompanied by reward, the two groups differed significantly in the regional brain activation they demonstrated. Developmentally normative adolescents evidenced a shift of activation from the caudate to the anterior cingulate cortex. However, adolescents with a diagnosis of CD and/or ADHD did not show this typical shift in processing the contingency change. In fact, these adolescents continued to activate the caudate nucleus, with no significant difference between the reward and non-reward conditions (Gatzke-Kopp et al., 2009).

The failure of adolescents with CD to recognize the experimentally induced change in feedback suggests that they may be especially insensitive to cues of behavioral ineffectiveness. In normally developing adolescents, behavior that may have been successful in the past but now

consistently fails to yield desired results will extinguish naturally. It has been proposed that failure in this process to use feedback to make adjustments to behavior is due to deficits in dopamine (Sagvolden et al., 2005). Support for this hypothesis has also been reported with electroencephalographic techniques, which measure brain response to error feedback in real time. Individuals with externalizing symptoms showed reduced brain activation in response to error commission, indicating a reduction in monitoring behavioral success (Hall, Bernat, & Patrick, 2007). Thus individuals with antisocial behavior disorders may appear to perseverate in behaviors despite their obvious lack of success in achieving goals.

In addition to deficits in reinforcement and extinction that mediate behavioral change in normally developing individuals, antisocial individuals have also been shown to have deficient response to punishment. Antisocial behavior has frequently been associated with deficits in fear conditioning, which is thought to contribute to the relative ineffectiveness of punishment threat in deterring antisocial behavior. Research has consistently demonstrated low levels of physiological arousal in antisocial individuals (Gatzke-Kopp, Raine, Loeber, Stouthamer-Loeber, & Steinhauer, 2002). Physiological arousal has been proposed to play a fundamental role in the development of conscience and internalization of rule structures that guide behavior. Researchers propose that the experience of punishment or negative performance feedback elicits a physiological response (typically measured through skin conductance) that provides an internal marker or cue of the negative experience. Over time this results in a conditioned response whereby the individual is able to use situational cues to avoid punishing consequence (Bechara, Damasio, & Damasio, 2000). For normally developing children, a simple scolding of an unwanted behavior, such as touching an electrical outlet, establishes a physiological code—coupled with the emotional experience of the mildly punishing parental admonishment—that the behavior is inappropriate. In time the child learns to avoid the cueing stimulus (e.g., the electrical outlet), thus internalizing the situational rule and regulating his or her own behavior outside of parent supervision. Children with antisocial behavior regularly fail to internalize cues of punishment and continually engage in behaviors that result in negative consequences. Adolescents with CD, regardless of the age of onset, show deficits in fear conditioning, indicating an inability to pair negative experience in a manner contingent with environmental cues (Fairchild et al., 2008).

No research has yet identified a single necessary or sufficient neuropathology that underlies antisocial behavior. It seems most likely that individuals meeting criteria for CD represent a heterogeneous class (Jones & Westen, 2010). However, evidence has amassed to implicate profiles of psychological dysfunction that likely contribute to some individuals developing antisocial symptoms. Specifically, heightened sensitivity to emotional cues in social contexts appears to lead some children to experience emotional arousal disproportionate to the situation, which may be difficult for them to regulate appropriately and which may result in inappropriate and unnecessary behavioral retaliation. In addition, some antisocial individuals appear to be characterized by an increased drive to seek reward and excitement, accompanied by a tendency not to respond to punishment. Because punishment is not as effective in preemptively deterring behavior and because extinction learning is not as effective in eliminating unsuccessful behavior, it can be especially challenging to successfully change the behavior of adolescents with CD.

Normative Brain Changes in Adolescence

Although a significant amount of research indicates pathological neural processing in adolescents with antisocial behavior, normative developmental changes in adolescence may also serve as a catalyst for vulnerable individuals who experience onset of antisocial behavior during this time. Just as adolescence is defined by the robust physical changes in body shape, size, and reproductive function, dramatic changes occur in neural development during this time as well. Increasing

research on adolescent brain development indicates that, in some domains, adolescents exist midway along a continuum from childhood to adulthood, with a steady linear increase in function and ability. In other domains, however, adolescence represents a unique stage of development, deviating both from the previous state of childhood as well as the future state of adulthood. This likely contributes to the dramatic rise in antisocial behavior during adolescence, when moderate levels of environmental risk are met with developmental changes in impulsivity, culminating in antisocial behaviors not previously seen.

Poor judgment in adolescence has often been attributed to the prolonged maturation of the prefrontal cortex. This region, responsible for higher order processing of information, decision-making, and regulatory control over other brain regions, is among the last region of the brain to reach full maturity (Colby, Van Horn, & Sowell, 2011; Sowell et al., 2003). This is reflected in the linear increase from childhood to adulthood observed for performance on a number of cognitive tasks (Levin et al., 1991; Swanson, 1999; Williams, Ponesse, Schachar, Logan, & Tannock, 1999). However, risky decision making does not demonstrate a parallel linear decrease across this developmental span, suggesting that the increase of these types of behaviors in adolescence is not solely attributable to delayed prefrontal maturation. Recently, researchers have focused on the relative balance between the prefrontal and limbic regions to understand the nonlinear development in reward processing and decision-making. Unlike the prefrontal cortex, limbic regions show adultlike activation patterns by adolescence (Galvan et al., 2006). This has led to the proposal that during childhood, both the prefrontal and limbic systems are relatively immature, and during adulthood, both have reached appropriate maturation. It is during adolescence, when the maturity of these two systems is out of balance, that there is a spike in impulsive, emotionally charged, reward-focused behaviors that originate in the relatively more developed limbic system and that are not mollified by the relatively less mature prefrontal cortex (Ernst & Fudge, 2009; Somerville & Casey, 2010).

In addition, hormonal changes that accompany the onset of puberty have been implicated in exacerbating vulnerability in some individuals, leading to an increase in antisocial behavior (Susman et al., 2010). In particular, the timing of pubertal onset relative to peers appears to play a role in increasing antisocial behaviors. Girls with early-onset menarche are at a higher risk for CD than their peers. Early-onset menarche has been shown to be more common in girls with high levels of familial stress and, in particular, the experience of sexual abuse (Zabin, Emerson, & Rowland, 2005). Although the presence of these risk factors likely contributes independently to the risk for antisocial behaviors, research suggests that early menarche also may increase risk for girls by altering aspects of their social development. Girls with visible bodily changes associated with puberty well in advance of their peers often feel isolated from age-comparable peers and are more likely to receive the attention of older boys. Furthermore, those boys who are less successful in cultivating romantic relationships with girls their own age may be more likely to seek out younger girls. Thus, early-onset puberty appears to become a mechanism by which young girls begin an association with deviant peer groups, thus exacerbating their own risk (Burt, McGue, DeMarte, Krueger, & Iacono, 2006). In homes with low parental supervision, these emerging social relationships are likely to significantly contribute to female delinquency and other risk behaviors, illustrating the synergistic effects of parental, peer, and biological mechanisms in precipitating risk.

Interestingly, similar phenomena have been observed for boys. Pubertal development significantly out of sync with age-matched peers appears to exacerbate risk in vulnerable boys. However, pubertal onset was not a sufficient predictor of risk; rather it interacted with biological measures of trait stress reactivity to predict risk. Only boys with late-onset pubertal development who were characterized by low levels of salivary alpha amylase (thought to reflect low levels of reactive arousal) demonstrated an increased propensity toward antisocial behavior (Susman et al., 2010). Alternatively, boys with early-onset pubertal

development were at risk only if additionally characterized by high levels of cortisol reactivity (thought to reflect stress; Susman et al., 2010). Each of these biological predispositions may reflect vulnerability for antisocial behavior through mechanisms such as poor fear conditioning or hypersensitivity to threat. It may be that the resultant social stresses involved in being physically different from peers during this sensitive period of development interact with these predisposing traits to increase risk for antisocial behavior.

Prevention and Intervention

Regardless of the etiology or age of onset of antisocial behavior, engaging in illegal and dangerous acts carries substantial risk for consequences that may permanently alter an adolescent's developmental trajectory. For instance, experimentation with drugs of abuse is common in this time period, with 46 % of high school seniors reporting having used an illicit drug at least once and 56 % reporting having been drunk at some point (Johnston, O'Malley, Bachman, & Schulenberg, 2009). Although research in substance dependence suggests that not all individuals are equally vulnerable to the transition between initiation of use and development of addiction, even nonaddictive substance experimentation can carry substantial risks. Overdose of a single drug, lethal combinations of multiple drugs, accidental death, and victimization are among the life-altering consequences of even experimental use. Relatively minor illegal acts committed for the first time can go awry and result in incarceration, and risky decisions related to sexual behavior can have life-altering consequences. The severity of these potential consequences suggests that prevention and intervention efforts are important, regardless of the severity of the symptoms expressed.

Prevention programs may alleviate some of the social risk factors that contribute to antisocial behavior, and successfully redirect development along a more positive trajectory. Several of the most effective antisocial behavior prevention programs deliver services to adolescents and their families. The Strengthening Families Program is a universal prevention program designed to address issues parents and children face in the transition to adolescence (Spoth, Redmond, & Shin, 2001). The program is administered in seven weekly sessions with concurrent parent and child components, followed by a structured family interaction time. The program focuses on providing parents with strategies to improve monitoring of child behavior, support open communication and bonding, and implement effective discipline strategies. Children are also guided in communicating with parents and managing challenges in peer relationships.

Randomized clinical trials demonstrated that the Strengthening Families Program significantly reduced the onset of adolescent substance use through high school, suggesting that effects are maintained over the course of several years (Spoth, Guyll, & Shin, 2009; Spoth et al., 2001). Moreover, it appears that this reduction in use is due to limited exposure. Thus, parental supervision, communication, and support appear to guide adolescents into friendships with peers who are less likely to seek out drugs and alcohol (Spoth et al., 2001, 2009). This is an excellent example of how preventing exposure to substances during this sensitive period of brain development may divert otherwise at-risk youth from potential addiction and the adverse consequences of experimental use.

Often antisocial behavior involves criminal activity that requires legal intervention. Although it is usually adolescents with life-course-persistent antisocial behavior who engage in more serious criminal activity, this is not always the case. Behavior of this sort is often met with punitive rather than therapeutic responses. However, as described above, punitive responses, whether in school or through the justice system, have little impact in deterring or redirecting antisocial behavior. Equally important, punitive responses may further alienate individuals from prosocial influences, increase their exposure to antisocial peers, and reduce the academic and skill development that would support a positive outcome.

One program that appears effective in reducing the recidivism of adolescents engaged in criminal activity is Functional Family Therapy (Sexton &

Alexander, 1999). Initially developed for first-time status offenders from mostly middle-income families, Functional Family Therapy is a short-term intervention that integrates social-learning and systems theories to motivate family members to change by identifying and building upon unique strengths and offering families specific ways to improve. Like many family interventions, Functional Family Therapy helps enhance parenting skills, promote supportive communication, and reduce negative exchanges, including blaming.

Functional Family Therapy has been replicated independently in several clinical trials with diverse samples, including adolescents who have committed multiple serious offenses. These trials indicate that Functional Family Therapy reduces dramatically the likelihood of being convicted of another crime compared to normal practices in the criminal justice system, such as probation or placement in residential treatment, or other forms of therapy. In one follow-up study, Functional Family Therapy, compared to probation, accounted for an 83 % reduction in recidivism during the remainder of adolescence (11.1 % versus 66.7 % across 2.5 years) and a 79 % reduction during early adulthood (8.7 % versus 40.9 % across 3 years; Gordon, Graves, & Arbuthnot, 1995). There is even some intriguing evidence that Functional Family Therapy can reduce the likelihood that younger siblings in the family will become involved in criminal activity in the first place (Klein, Alexander, & Parsons, 1977).

Adolescents who are involved in the criminal justice system are often mired in an array of risk-exacerbating factors, including low academic achievement (possibly due to low IQ or unaddressed learning disabilities); an antisocial peer network; potential use or dependence on illicit substances; a harsh, hostile, or abusive home environment; and parental antisocial behavior or substance dependence. Multisystemic Therapy (MST) was designed to address the compounding effect of multiple risk factors by focusing on adolescents and the family, peer, school, and community systems within which they are embedded (Henggeler, Cunningham, Schoenwald, Borduin, & Rowland, 2009). MST provides short-term, but intensive, in-home treatment that is problem-focused and highly individualized, based on the resources individual adolescents have access to and the particular challenges they face.

Despite the fact that MST tends to serve adolescents exhibiting serious antisocial behavior, one randomized study showed that—compared to treatment as usual in the Department of Youth Services—it reduced subsequent arrests by 43 % (0.87 versus 1.52), reduced incarceration by 10.4 weeks on average, and improved family cohesion (Henggeler, Melton, & Smith, 1992). In an independent replication, MST, compared to individual therapy, reduced the likelihood of recidivism across 4 years by 63 % (26.1 % versus 71.4 %).

With any intervention program, participation is a critical factor in success. Unfortunately, the investment in terms of both time and mental commitment are high, often serving as a barrier to success for many families. Even when available at no cost, attendance in weekly sessions can be a low priority for busy families. This might be especially true for programs that provide preventive services before serious problems have developed. In the Strengthening Families Program, as many as one-half of the participants assigned to the intervention did not participate in the weekly sessions, indicating that intensive therapeutic programs do not serve the needs of a large number of families (Spoth et al., 2001). Not surprisingly, those families who fail to participate or who drop out of therapeutic programs are often the families with the most risk factors and the greatest need for intervention (Kazdin, Mazurick, & Bass, 1993).

Addressing the diversity of parental needs and motivating drives requires a flexible approach to delivering intervention. In order to accommodate individual and cultural variation in acceptance of mental health intervention, an adaptive program called the Family Check-Up (FCU) was developed (Dishion & Kavanagh, 2003). The FCU model addresses the intervention process in stages, working with each family to deliver the intervention service that best matches expectations and best meets individual needs. After a comprehensive assessment, feedback is delivered to the family and potential goals for treatment are identified and discussed. The clinician then

describes a range of empirically supported treatment options that target the family's most salient concerns, and the family selects to participate in specific services from available resources. Not all families select the same level of intervention, and some families forego the intervention most likely to succeed. However, by involving families in the decision-making process, participation is enhanced and some level of intervention is delivered for families who may have otherwise refused treatment. In this way, the dynamic and client-oriented approach to therapy maximizes client buy-in and appears to result in treatment effects on the reduction of substance use that are as large or larger than other prevention programs (Dishion, Kavanagh, Schneiger, Nelson, & Kaufman, 2002).

Despite the effectiveness in increasing intervention participation that the FCU approach has had, biases against seeking mental health support as well as cost, time, and the availability of appropriately trained clinicians still represent significant barriers to access for many families. To address this, researchers have begun to explore alternative dissemination strategies for reaching parents on a global scale. By documenting families engaged in a therapeutic program in a six-part reality TV series, researchers were able to effectively disseminate information related to normative and nonnormative child development, destigmatize and model the value of seeking mental health assistance to deal with problem behaviors, and provide information about where to locate those additional targeted services. Findings suggest that such an approach can result in significant improvements in child behavior and parental reports of their own anger and depression (Calam, Sanders, Miler, Sadhnani, & Carmont, 2008). Web-based programming to supplement the television program was assigned to half of the intervention group, although treatment effects were evident even at the viewing-only level (Sanders, Calam, Durand, Liversidge, & Carmont, 2008; Sanders & Prinz, 2008). Capitalizing on technological advances and integrating educational missions within entertainment media may prove to diversify access to therapeutic strategies and educational information about parenting.

Summary and Conclusions

Although adolescence is a time of significant experimentation and risky decision making, it is not a pathological condition. However, individuals with certain patterns of emotional reactivity and/or deficits in social reinforcement learning may be especially vulnerable during this period. If family relationships are characterized by ongoing and escalating coercive exchanges, adolescents might not have the close relationships necessary for parents to positively influence behavior and provide appropriate guidance. Moreover, this breakdown in family relationships occurs as adolescents spend more unsupervised time, often with similar adolescents who share a propensity for antisocial behavior. Although the limbic system appears to be more advanced than the prefrontal cortex in all adolescents, these more vulnerable adolescents may be especially predisposed to exhibit impulsive, sensation-seeking, reward-focused actions that are less modulated by deliberative consideration of long-term consequences.

There are clear differences between individuals who first display serious antisocial behavior during early childhood and those who first display antisocial behavior during adolescence. However, those differences appear to be ones of degree, not kind. Individuals who first display serious antisocial behavior during early childhood experience a well-documented array of risk factors. Individuals who display antisocial behavior during adolescence may not experience as many of these risk factors at such intense levels. For these vulnerable individuals, however, the changes in family and peer relationships and cognitive and physical maturation that are defining features of normal adolescence might also change dynamics just enough to precipitate the onset of antisocial behavior.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (Revised 4th ed.). Washington, DC: American Psychiatric Association.

Archer, J. (2009). Does sexual selection explain human sex differences in aggression? *The Behavioral and Brain Sciences, 32*, 249–311.

Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. (2007). Polyvagal theory and developmental psychopathology. Emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology, 74*, 174–184.

Bechara, A., Damasio, H., & Damasio, A. R. (2000). Emotion, decision making and the orbitofrontal cortex. *Cerebral Cortex, 10*, 295–307.

Biederman, J., Newcorn, J., & Sprich, S. (1991). Comorbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety, and other disorders. *The American Journal of Psychiatry, 148*, 564–577.

Brendgen, M., Vitaro, F., & Bukowski, W. M. (2000). Stability and variability of adolescents' affiliation with delinquent friends: Predictors and consequences. *Social Development, 9*, 205–225.

Brown, B. B., Mounts, N., Lamborn, S. D., & Steinberg, L. (1993). Parenting practices and peer group affiliation in adolescence. *Child Development, 64*, 467–482.

Buckholtz, J. W., & Meyer-Lindenberg, A. (2008). MAOA and the neurogenetic architecture of human aggression. *Trends in Neuroscience, 31*, 120–129.

Burt, S. A., Krueger, R. F., McGue, M., & Iacono, W. G. (2001). Sources of covariation among attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder: The importance of shared environment. *Journal of Abnormal Psychology, 110*, 516–525.

Burt, S. A., McGue, M., DeMarte, J. A., Krueger, R. F., & Iacono, W. G. (2006). Timing of menarche and the origins of conduct disorder. *Archives of General Psychiatry, 63*, 890–896.

Cairns, R. B., Leung, M., Buchanan, L., & Cairns, B. (1996). Friendships and social networks in childhood and adolescence: Fluidity, reliability, and interrelations. *Social Development, 66*, 1330–1345.

Calam, R., Sanders, M. R., Miler, C., Sadhnani, V., & Carmont, S.-A. (2008). Can technology and the media help reduce dysfunctional parenting and increase engagement with preventative parenting interventions? *Child Maltreatment, 13*, 347–361.

Colby, J. B., Van Horn, J. D., & Sowell, E. R. (2011). Quantitative in vivo evidence for broad regional gradients in the timing of white matter maturation during adolescence. *NeuroImage, 54*, 25–31.

Crick, N. R., Bigbee, M. A., & Howes, C. (1996). Gender differences in children's normative beliefs about aggression: How do I hurt thee? Let me count the ways. *Child Development, 67*, 1003–1014.

Crowley, T. J., Dalwani, M. S., Mikulich-Gilbertson, S. K., Du, Y. P., Lejuez, C. W., Raymond, K. M., et al. (2010). Risky decision and their consequences: Neural processing by boys with antisocial substance disorder. *PLoS One, 5*, e12835.

Dishion, T. J., Bullock, B. M., & Granic, I. (2002). Pragmatism in modeling peer influence: Dynamics, outcomes, and change processes. *Development and Psychopathology, 14*, 969–981.

Dishion, T. J., & Kavanagh, K. (2003). *Intervening in adolescent problem behavior: A family-centered approach*. New York: Guilford Press.

Dishion, T. J., Kavanagh, K., Schneiger, A., Nelson, S., & Kaufman, N. K. (2002). Preventing early adolescent substance use: A family-centered strategy for the public middle school. *Prevention Science, 3*, 191–201.

Dishion, T. J., McCord, J., & Poulin, F. (1999). When interventions harm: Peer groups and problem behavior. *American Psychologist, 54*, 755–764.

Dishion, T. J., Nelson, S. E., & Bullock, B. M. (2004). Premature adolescent autonomy: Parent disengagement and deviant peer process in the amplification of problem behavior. *Journal of Adolescence, 27*, 515–530.

Dishion, T. J., Nelson, S. E., Winter, C. E., & Bullock, B. M. (2004). Adolescent friendship as a dynamic system: Entropy and deviance in the etiology and course of male antisocial behavior. *Journal of Abnormal Child Psychology, 32*, 651–663.

Dodge, K. A. (1986). A social information processing model of social competence in children. In M. Perlmutter (Ed.), *Minnesota symposium in child psychology* (pp. 77–125). Hillsdale, NJ: Lawrence Erlbaum.

Dodge, K. A. (2011). Social information processing as mediators of the interaction between genetic factors and life experiences in the development of aggressive behavior. In P. R. Shaver & M. Mikulincer (Eds.), *Human aggression and violence: Causes, manifestations, and consequences* (pp. 165–185). Washington, DC: American Psychological Association.

Ernst, M., & Fudge, J. L. (2009). A developmental neurobiological model of motivated behavior: Anatomy, connectivity and ontogeny of the triadic nodes. *Neuroscience and Biobehavioral Reviews, 33*, 367–382.

Ernst, M., Grant, S. J., London, E. D., Contoreggi, C. S., Kimes, A. S., & Spurgeon, L. (2003). Decision making in adolescents with behavior disorders and adults with substance abuse. *The American Journal of Psychiatry, 160*, 33–40.

Fairchild, G., Van Goozen, S. H., Stollery, S. J., & Goodyer, I. M. (2008). Fear conditioning and affective modulation of the startle reflex in male adolescents with early-onset or adolescence-onset conduct disorder and healthy control subjects. *Biological Psychiatry, 63*, 279–285.

Fontaine, N., Carbonneau, R., Vitaro, F., Barker, E. D., & Tremblay, R. E. (2009). Research review: A critical review of studies on the developmental trajectories of antisocial behavior in females. *Journal of Child Psychology and Psychiatry, 50*, 363–385.

Galvan, A., Hare, T. A., Parra, C. E., Penn, J., Voss, H., Glover, G., et al. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience, 26*, 6885–6892.

Gatzke-Kopp, L. M., Beauchaine, T. P., Shannon, K. E., Chipman, J., Fleming, A. P., Crowell, S. E., et al. (2009). Neurological correlates of reward responding in adolescents with conduct disorder and/or attention-deficit/hyperactivity disorder. *Journal of Abnormal Psychology, 118*, 203–213.

Gatzke-Kopp, L. M., Raine, A., Loeber, S., Stouthamer-Loeber, M., & Steinhauer, S. R. (2002). Serious delinquent behavior, sensation-seeking and electrodermal arousal. *Journal of Abnormal Child Psychology, 30*(5), 477–486.

Gordon, D. A., Graves, K., & Arbuthnot, J. (1995). The effect of functional family therapy for delinquents on adult criminal behavior. *Criminal Justice and Behavior, 22*, 60–73.

Granic, I., & Patterson, G. R. (2006). Toward a comprehensive model of antisocial development: A dynamic systems approach. *Psychological Review, 113*, 101–131.

Hall, H. R., Bernat, E. M., & Patrick, C. J. (2007). Externalizing psychopathology and the error-related negativity. *Psychological Science, 18*, 326–333.

Henggeler, S. W., Cunningham, P. B., Schoenwald, S. K., Borduin, C. M., & Rowland, M. D. (2009). *Multisystemic therapy for antisocial behavior in children and adolescents* (2nd ed.). New York: Guilford Press.

Henggeler, S. W., Melton, G. B., & Smith, L. A. (1992). Family preservation using multisystemic therapy: An effective alternative to incarcerating serious juvenile offenders. *Journal of Consulting and Clinical Psychology, 60*, 953–961.

Herpertz, S. C., Huebner, T., Marx, I., Vloet, T. D., Fink, G. R., Stoecker, T., et al. (2008). Emotional processing in male adolescents with childhood-onset conduct disorder. *Journal of Child Psychology and Psychiatry, 49*, 781–791.

Hofvander, B., Ossowski, D., Lundstrom, S., & Anckarsater, H. (2009). Continuity of aggressive antisocial behavior from childhood to adulthood: The question of phenotype definition. *International Journal of Law and Psychiatry, 32*, 224–234.

Horsley, T. A., de Castro, B. O., & Van der Schoot, M. (2010). In the eye of the beholder: Eye-tracking assessment of social information processing in aggressive behavior. *Journal of Abnormal Child Psychology, 38*, 587–599.

Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2009). *Monitoring the future: National results on adolescent drug use*. Bethesda, MD: National Institute on Drug Abuse.

Jones, M., & Westen, D. (2010). Diagnosis and subtypes of adolescent antisocial personality disorder. *Journal of Personality Disorders, 24*, 217–243.

Kazdin, A. E., Mazurick, J. L., & Bass, D. (1993). Risk for attrition in treatment of antisocial children and families. *Journal of Clinical Child Psychology, 22*, 1–16.

Kerr, M., & Stattin, H. (2000). What parents know, how they know it, and several forms of adolescent adjustment: Further support for a reinterpretation of monitoring. *Developmental Psychology, 36*, 366–380.

Klein, N. C., Alexander, J. F., & Parsons, B. V. (1977). Impact of family systems intervention on recidivism and sibling delinquency: A model of primary prevention and program evaluation. *Journal of Consulting and Clinical Psychology, 45*, 469–474.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111*, 411–424.

Lacourse, E., Nagin, D., Tremblay, R. E., Vitaro, F., & Claes, M. (2003). Developmental trajectories of boys' delinquent group membership and facilitation of violent behaviors during adolescence. *Development and Psychopathology, 15*, 183–197.

Levin, H. S., Culhaneb, K. A., Hartmannb, J., Evankovichb, K., Mattsonb, A. J., Harward, H., et al. (1991). Developmental changes in performance on tests of purported frontal lobe functioning. *Developmental Neuropsychology, 7*, 377–395.

Lewis, M. D., & Todd, R. M. (2007). The self-regulating brain: Cortical-subcortical feedback and the development of intelligent action. *Cognitive Development, 22*, 406–430.

Metcalfe, J., & Mischel, W. (1999). A hot/cool systems analysis of delay of gratification: Dynamics of willpower. *Psychological Review, 106*, 3–19.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100*, 674–701.

Moffitt, T. E., & Caspi, A. (2005). Life-course persistent and adolescence-limited antisocial males: Longitudinal follow up to adulthood. In D. M. Stoff & E. J. Susman (Eds.), *Developmental psychobiology of aggression* (pp. 161–186). New York: Cambridge University Press.

Molero, S. Y., Hodgins, S., Larsson, A., Larm, P., & Tengstrom, A. (2010). Adolescent antisocial behavior as predictor of adverse outcomes to age 50: A follow up study of 1,974 individuals. *Criminal Justice and Behavior, 37*, 158–174.

Mounts, N. S. (2000). Parental management of adolescent peer relationships: What are its effects on friend selection. In K. A. Kerns, J. M. Contreras, & A. M. Neal-Barnett (Eds.), *Family and peers: Linking two social worlds* (pp. 169–193). Westport, CT: Praeger.

Murray, J., & Farrington, D. P. (2010). Risk factors for conduct disorder and delinquency: Key findings from longitudinal studies. *Canadian Journal of Psychiatry, 55*, 633–642.

Odgers, C. L., Moffitt, T. E., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., et al. (2008). Female and male antisocial trajectories: From childhood origins to adult outcomes. *Development and Psychopathology, 20*, 673–716.

Patterson, G. R. (1982). *Coercive family process*. Eugene, OR: Castalia.

Poulin, F., Dishion, T. J., & Burraston, B. (2001). 3-year iatrogenic effects associated with aggregating high-risk adolescents in cognitive-behavioral preventive interventions. *Applied Developmental Science, 5*, 214–224.

Raine, A. (2008). From genes to brain to antisocial behavior. *Current Directions in Psychological Science, 17*, 323–328.

Roisman, G. I., Aguilar, B., & Egeland, B. (2004). Antisocial behavior in the transition to adulthood: The independent and interactive roles of developmental history and emerging developmental tasks. *Development and Psychopathology, 21*, 417–439.

Roisman, G. I., Monahan, K. C., Campbell, S. B., Steinberg, L., Cauffman, E., & The National Institute of Child Health and Human Development Early Child Care Research Network. (2010). Is adolescence-onset antisocial behavior developmentally normative? *Development and Psychopathology, 22*, 295–311.

Sagvolden, T. A., Johansen, E. B., Aase, H., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *The Behavioral and Brain Sciences, 28*, 397–419.

Sanders, M. R., Calam, R., Durand, M., Liversidge, T., & Carmont, S. A. (2008). Does self-directed and web-based support for parents enhance the effects of viewing a reality television series based on the Tripe P-Positive Parenting Programme? *Journal of Child Psychology and Psychiatry, 49*, 924–932.

Sanders, M. R., & Prinz, R. J. (2008). Using the mass media as a population level strategy to strengthen parenting skills. *Journal of Clinical Child and Adolescent Psychology, 37*, 609–621.

Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., et al. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *Journal of Neuroscience, 27*, 2349–2356.

Sexton, T. L., & Alexander, J. F. (1999). *Functional family therapy: Principles of clinical intervention, assessment, and implementation*. Henderson, NV: RCH Enterprises.

Shannon, K. E., Sauder, C., Beauchaine, T. P., & Gatzke-Kopp, L. M. (2009). Disrupted effective connectivity between the medial frontal cortex and the caudate in adolescent boys with externalizing behavior disorders. *Criminal Justice and Behavior, 36*, 1141–1157.

Siever, L. J. (2008). Neurobiology of aggression and violence. *The American Journal of Psychiatry, 165*, 429–442.

Soenens, B., Vansteenkiste, M., & Niemiec, C. P. (2009). Should parental prohibition of adolescents' peer relationships be prohibited? *Personal Relationships, 16*, 507–530.

Somerville, J. H., & Casey, B. J. (2010). Development neurobiology of cognitive control and motivational systems. *Current Opinion in Neurobiology, 20*, 236–241.

Sowell, E. R., Peterson, B. S., Thompson, P. M., Welcome, S. E., Henkenius, A. L., & Toga, A. W. (2003). Mapping cortical change across the human life span. *Nature Neuroscience, 6*, 309–315.

Spoth, R., Guyll, M., & Shin, C. (2009). Universal intervention as a protective shield against exposure to substance use: Long-term outcomes and public health significance. *American Journal of Public Health, 99*, 2026–2033.

Spoth, R. L., Redmond, C., & Shin, C. (2001). Randomized trial of brief family interventions for general populations: Adolescent substance use outcomes 4 years following baseline. *Journal of Consulting and Clinical Psychology, 69*, 627–642.

Stadler, C., Sterzer, P., Schmeck, K., Krebs, A., Kleinschmidt, A., & Poustka, F. (2007). Reduced anterior cingulate activation in aggressive children and adolescents during affective stimulation: Association with temperament traits. *Journal of Psychiatric Research, 41*, 410–417.

Stattin, H., & Kerr, M. (2000). Parental monitoring: A reinterpretation. *Child Development, 71*, 1072–1085.

Steinberg, L. (2001). We know some things: Parent-adolescent relationships in retrospect and prospect. *Journal of Research on Adolescence, 11*, 1–19.

Steinberg, L., Mounts, N. S., Lamborn, S. D., & Dornbusch, S. M. (1991). Authoritative parenting and adolescent adjustment across varied ecological niches. *Journal of Research on Adolescence, 1*, 19–36.

Susman, E. J., Dockray, S., Granger, D. A., Blades, K. T., Randazzo, W., Heaton, J. A., et al. (2010). Cortisol and alpha amylase reactivity and timing of puberty: Vulnerabilities for antisocial behavior in young adolescents. *Psychoneuroendocrinology, 35*, 557–569.

Swanson, H. L. (1999). What develops in working memory? A life span perspective. *Developmental Psychology, 35*, 986–1000.

Vera, J., Ezpeleta, L., Granero, R., & de la Osa, N. (2010). Antisocial behavior, psychopathology and functional impairment: Association with sex and age in clinical children and adolescents. *Child Psychiatry and Human Behavior, 41*, 465–478.

Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the life span. *Developmental Psychology, 35*, 205–213.

Woodward, L. J., Fergusson, D. M., & Horwood, L. J. (2002). Romantic relationships of young people with childhood and adolescent onset antisocial behavior problems. *Journal of Abnormal Child Psychology, 30*, 231–243.

Yang, Y., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta analysis. *Psychiatry Research: Neuroimaging, 174*, 81–88.

Zabin, L. S., Emerson, M. R., & Rowland, D. L. (2005). Childhood sexual abuse and early menarche: The direction of their relationship and its implications. *Journal of Adolescent Health, 36*, 393–400.