

REVIEW ARTICLE

Recent Research Findings on Aggressive and Violent Behavior in Youth: Implications for Clinical Assessment and Intervention

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Abstract: Assessing children and adolescents for potential violent behavior requires an organized approach that draws on clinical knowledge, a thorough diagnostic interview, and familiarity with relevant risk and protective factors. This article reviews empirical evidence on risk factors, the impact of peers, developmental pathways, physiological markers, subtyping of aggression, and differences in patterns of risk behaviors between sexes. We explore these determinants of violence in children and adolescents with attention to the underlying motivations and etiology of violence to delineate the complexity, unanswered questions, and clinical relevance of the current research. Interventions, including cognitive behavioral therapy, psychopharmacological treatment, and psychosocial treatment, are reviewed with acute recognition of the need to use multiple modalities with, and to expand research to define optimal treatment for, potentially violent children and adolescents. The information considered for this review focuses on violence as defined as physical aggression toward other individuals. Other studies are included with wider definitions of violence because of their relevance to assessing the potential for violent behavior. © Society for Adolescent Medicine, 2004

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Although arrest rates for serious violent crimes and juvenile homicides have fallen from an all-time high in the mid-1990s, many adolescents and children remain involved in aggressive delinquent and violent behaviors such as physical fighting, bullying, using weapons, verbal threats of harm to others, and chronic impulsive aggression [1]. In 1999, juveniles accounted for 16% of all violent crime arrests, and homicides committed by youth under 18 accounted for 10.1% of all homicides [1,2]. Although this homicide rate is lower than in previous years, the overall prevalence of other violent behaviors among youth remains high. These figures are the culmination of a tragic trajectory of violence that has an alarming impact on the physical safety and emotional well-being of our nation's youth.

Youth violence often emanates from multiple risk factors: biologic vulnerability [3–5]; inconsistent, overly permissive, or harsh discipline [6,7]; community deprivation [8–10]; easy access to guns [11]; and exposure to violence [12,13]. Violent behavior rarely appears spontaneously; it typically has a long developmental pathway [14–16]. In certain instances, aggression may be a response to stress that occurs during a vulnerable period, and an individual may not respond in the same volatile way at a different time in their life [17]. However, there is usually a strong continuity in violence between childhood, adolescence, and adult life. Aggressive behavior, conduct problems, and antisocial behaviors generate

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one-third to one-half of all child and adolescent psychiatric clinic referrals, and clinicians are frequently asked to provide evaluation and treatment recommendations for these patients [18]. In the context of disruptive disorders, extensive reviews examine the primary risk factors and developmental pathways while also recognizing that there is still a level of complexity that warrants further research to enhance our understanding of aggression and to inform effective interventions [19].

Even though many clinicians specializing in adolescent medicine may not have the expertise to conduct this type of psychiatric diagnostic assessment and to choose treatment modalities, it is helpful to be exposed to the relevant research about aggressive youth and to appreciate the practical limitations of our knowledge and possible areas of intervention. The role of the evaluating mental health clinician is critical in providing a diagnostic assessment that is based on a sophisticated clinical formulation. The initial steps are to carefully identify and understand the cumulative effects of risk and protective factors on the patient; assess acute safety considerations; evaluate the onset, severity, and course of the violent behavior; identify comorbidity; and determine the motivation for change and self-reflection. Currently, no validated screening instruments or protocols exist for the prediction of juvenile aggression. Although several assessment instruments appear promising, no single screening instrument has been established or generally accepted for predicting youth aggression.

The success in predicting treatment outcomes and violence for these high-risk patients is variable, and it is useful for clinicians to continue to assess these patients and to look for opportunities for preventive interventions. Offering the perspective of a community practitioner rather than that of an individual practitioner is crucial because these aggressive children usually need coordinated efforts drawing on resources from their family, medical, and mental health care providers, educators and other community members. These assessments may occur in emergency rooms, court clinics, schools, outpatient psychiatric clinics, or inpatient psychiatric units. In this review, we will present the salient information relevant to clinicians who may be asked to identify and/or assess violent children and adolescents, and to determine the capacity for intervention. Because there is extensive recent research on youth violence, particular attention is therefore focused on topics that have special relevance to clinicians. Most important are studies that provide information critical to

the evaluation of youth violence. These research findings are grouped into the areas of individual factors (gender, physiological markers, and social cognitive risk factors), social and environmental factors (family, peer and environmental factors), followed by sections addressing conceptual models (cumulative risk factors and aggression subtypes), considerations in risk assessment, and prevention/intervention approaches (cognitive behavioral therapy, psychopharmacological treatment and psychosocial treatment). Special emphasis is devoted to reports from areas that have not received consideration in previous general reviews but expand our clinical awareness and provide a better framework for understanding youth violence, such as aggression in girls and physiological markers.

Methodology of Search

Research literature on youth aggressive and violent behavior was reviewed after a systematic search of PsycInfo and Medline. Also, manual review of articles' reference lists identified additional pertinent studies. The review focuses on important findings in youth violence and topics that have not been covered in previous general reviews, including gender differences, conduct disorder, subtypes of aggression and risk factors, with emphasis on areas of current research.

Individual Factors

Gender

Most of the research on youth violence focuses on men and boys with relatively little attention given to aggressive females, primarily because a much larger percentage of males, as compared with females, commit violent acts [20]. Typically, gender differences were difficult to discern, as many studies (particularly those examining conduct disorder) included only male participants [21]. In the past, to understand the characteristics, history, and symptoms of girls with illegal or aggressive behavior, the most frequently implemented design relied on uncontrolled follow-up and cross-sectional studies with predominantly white samples [22–24]. However, in the last 10 years, researchers have generated more empirical studies of girls' aggression in several different disciplines (developmental psychology, child psychiatry, and criminology), with more attention to prospective longitudinal studies and more diverse participants [25–28]. However, there is still a long

way to go until the research on female youth violence and aggression provides the same depth of work as on boys, particularly with respect to longitudinal studies.

Most epidemiological studies have identified conduct disorder as one of the most severe mental disorders in adolescent girls, with prevalence rates varying from 4% to 9% [29,30]. Criminal statistics and diagnostic criteria of conduct disorder can be viewed as identifying adolescent females with the same underlying disruptive behaviors of concern. The Office of Juvenile Justice showed in national statistics on adolescent female violent crime arrests an increase of 23% as compared with an 11% increase in the arrests of male juveniles [31]. It is unclear if this marked increase in female arrests is owing to increased detection of females by the juvenile justice system and previous reluctance to arrest girls. The severity of adolescent female crime has also increased [31].

Girls may have different ways than boys of expressing aggression that are affected by biological, dispositional, and contextual factors. The challenge is to unravel the interaction of causal factors, the heterogeneity of risk factors, and the identification of different developmental trajectories to determine precise mechanisms of variable outcomes of female aggression. There is recognition that girls are often exposed to the same biological insults (e.g., prenatal maternal cigarette smoking) as boys, but that this exposure has a minimal effect on girls' relative risk (RR) of conduct disorder [32]. In contrast, there is an association of prenatal smoking with psychiatric morbidity specific to antisocial behavior in males [32]. These outcome measures have some methodological limitations owing to a reliance on cross-sectional studies and because there is difficulty measuring prenatal exposure with precision and separating risk factors that may have confounded the results. However, this study highlights the increased vulnerability of males to peri and postnatal stresses [32]. It would be clinically useful to delineate why females are less vulnerable to prenatal nicotine exposure and subsequent associated severe antisocial behavior.

The majority of developmental studies do not differentiate physical aggression and verbal aggression [33], and the studies tend to examine the externalizing observable behaviors that are more consistent with male aggression, such as openly confrontational verbal threats and physical assaults [34]. Existing classification methods of girls with conduct disorder may overlook behavior that may

subsequently evolve into serious psychopathology but does not necessarily reflect overt patterns of aggression. In a longitudinal study of 2251 girls entering kindergarten, who were examined over a period of 7 years with a 3-year follow-up, the DSM-IV diagnostic criteria of conduct disorder failed to identify the most impaired, persistently antisocial girls [29]. They suggested that the criteria for girls might need to be different from those used for boys, whether in reducing the number or type of symptoms. Crick expanded the criteria of female aggression from an emphasis on physical and overt aggression to verbal, indirect, and relational aggression [35]. Relational aggression refers to gaining control through manipulative behavior that affects peer status and that is recognized by girls as motivated by intent to harm and "meanness." Later studies demonstrated that relational aggression in females predicts concurrent psychosocial adjustment problems [36].

Separate criteria for identifying conduct disorder in females and males have not been developed. This issue was considered during the development of the DSM-IV but was not pursued because there was insufficient information available to support gender-specific criteria for identifying conduct disorder [37]. By developing accurate and useful criteria that examines a broad range of behavior for assessing female aggression, it may be ascertained that there is an unrecognized continuity between persistent troubling behavior (not the same type of disruptive behavior that is seen in males) that increases the probability of developing life-long impairment in females [26]. There may be gender-specific levels and types of behavior that identify girls as disruptive that are at low risk according to males' standards but predict subsequent impairment in girls. This identification may be useful in developing reliable clinical tools to provide early detection and support to those young girls who are at risk of developing late onset of dysfunction in multiple areas. Several longitudinal studies show that adolescent girls with conduct disorder predictably suffered in multiple adult outcomes after adolescence. Their dysfunction unfolded over time and included poor physical health [38], increased mortality rates, increased criminality rates, high rates of psychiatric comorbidity, and participation in violent relationships [24].

Antisocial adolescent females are often more vulnerable to family dysfunction and have a later onset of aggressive behavior than males [39]. Some preliminary evidence connects girls' depression and family discord to later antisocial behavior [40]. Expanding

the analysis of behavior linked to aggression is reflected in one of the first studies of an ethnically diverse group of adolescent female offenders that showed a link between trauma, psychopathology, and violence [41]. An examination of 96 incarcerated adolescent girls found that they were 50% more likely to show symptoms of posttraumatic stress disorder (PTSD) than male juvenile delinquents [41]. The difficulty with the study was that the sample was small and the researchers did not consider other comorbid pathology. Causality was not established because cross-sectional data were collected. The recognition of PTSD and subsequent aggression in incarcerated females may lead to focusing on this often unidentified association between PTSD and aggression. Such research also highlights the importance of screening and early intensive intervention with traumatized children.

Any antisocial behavior (including violence) in girls should alert clinicians to the possibility of comorbid psychiatric disorders because girls with antisocial behaviors are at much greater risk than boys for suffering from a wide range of psychiatric illnesses [42]. In a recent study examining violence exposure, violent behaviors, psychological trauma, and suicide risk in a community sample of dangerously violent adolescents, one in five females was at a high risk for suicide compared with significantly lower percentages in all other comparison groups [43]. The distinctive vulnerabilities of violent females and their pattern of clinical presentation remain to be delineated.

Clinicians must be vigilant about screening for aggressive behavior in females, particularly between the female and intimate partners and/or family members. Practitioners must also consider that an assaultive adolescent girl may have had some underlying trauma and may need further counseling. If the clinician sees an aggressive adolescent female for a recent injury or routine examination, it is particularly relevant to screen for suicide risk, as they are at a greater risk [43,44].

Physiological Markers

Recently, researchers have attempted to identify biological markers that may be relevant to the further subtyping of aggression. Environmental stressors can affect hormone production, and experiences can affect physiological states that can, in turn, affect behavior. Aggressive behavior in both children and adults is associated with abnormalities in peripheral responses to stress.

One peripheral measure, salivary cortisol concentration, may reflect alterations in the hypothalamic-pituitary-adrenal axis. In a longitudinal study of 38 clinic-referred school-age boys, low salivary cortisol levels were associated with persistent and early onset of aggression [45]. Boys with low cortisol concentrations (measured at Year Two and Four in the study) had three times the number of aggressive symptoms than did boys with higher cortisol levels. Continually restricted (low) cortisol levels may be more relevant to predicting continuous aggression than an isolated low concentration of cortisol at a single point in time. This finding was correlated to the subtype of aggressive children [45].

Boys who bully often have low anxiety and show low cortisol levels [45]. In contrast, affective aggressive boys with high arousal show high cortisol levels. This study was limited by a relatively small sample consisting only of males and by the failure to control for time of the day in measuring cortisol, because salivary cortisol levels show diurnal/circadian variability [45]. The mechanism linking persistent aggression and low cortisol concentration is not yet elucidated. Yehuda et al examined the alteration in cortisol levels (lowered) in patients with posttraumatic stress disorder (PTSD) [46]. There may be some overlap with aggressive patients that have lowered cortisol levels. The brain plasticity of the developing child suggests that prenatal and early developmental stress (maternal prenatal smoking, abuse, and neglect) can change the hypothalamic-pituitary-adrenal axis permanently [47]. Another hypothesis postulates that attachment behaviors regulate arousal activity in the hypothalamic-pituitary-adrenal axis. Disorganized attachment relationship in infants is correlated with elevated cortisol levels [48]. The later correlates of disorganized attachment strategies can manifest in preschool years as disturbed and aggressive interactions with parents and teachers [49,50]. However, clinically, these physiological markers cannot be used as predictors of violence, as many children with disorganized attachment histories and elevated salivary cortisol levels do not become aggressive. Some studies have shown that it is not merely the basal level of cortisol that is key to understanding disruptive and aggressive behavior but rather the hypothalamic axis response to stressful stimuli [51]. Consequently, further studies are needed to fully understand these interactions.

Researchers have postulated that the inhibitory neurotransmitter serotonin (precursor 5-HT) may modulate aggressive behavior in youths. Several methods of measuring indirect serotonin activity in

the brain are employed, as serotonin cannot be directly, economically, or easily quantified: metabolites in the cerebrospinal fluid and platelet receptors indirectly demonstrate the neuronal functioning as do measurements of whole blood serum [27]. The hypothesized relationship between lowered CSF serotonin precursors and higher levels of aggression is supported by two longitudinal studies; however, there is not a simple inverse relationship [52,53]. Challenge studies use drugs such as dl-fenfluramine as a way to indirectly assess the CNS serotonin levels. These challenge studies of prepubertal boys suggest that there may be developmental changes in serotonin function. Prepubertal aggressive boys initially may have increased serotonin functioning as compared with nonaggressive boys [54]. This enhanced serotonin may decrease with the onset of adolescence [55]. If this hypothesis is substantiated in future studies, it could have direct clinical implications in terms of avoiding selective serotonin reuptake inhibitors in aggressive prepubertal boys [55]. It is a more complex picture with youths, possibly because developmental fluctuations with serotonin confound the results. Further research needs to delineate the relationship of the development of neurobiological systems and specific vulnerabilities in response to stressful environmental events [54].

Gender differences in the rates of aggressive behaviors have naturally focused on the potential role of androgens, especially testosterone, in the development of violence. Numerous studies have found a correlation between higher levels of testosterone and physical aggression in boys [56–58]. Most of the studies describing this relationship are with boys after the onset of puberty, suggesting that the activating effect depends on physical maturation [59–62]. There is also some evidence to suggest that testosterone is specifically related to provoked aggression, but not unprovoked aggression, in adolescent and young males [58,59].

There are no definitive mechanisms delineated to understand the hypothesized association between aggression in youth and fluctuations in testosterone, cortisol, or neurotransmitters. This is the new frontier as researchers attempt to further elucidate how neurobiology and hormones play out differently in aggressive versus nonaggressive individuals while still acknowledging the impact of environmental stressful events. Whereas selective serotonin reuptake inhibitors are used in the adult population to dampen aggression by increasing serotonin [63], preliminary findings in prepubertal boys suggest

that treatment for adults cannot be indiscriminately transferred to youth [54].

Social Cognitive Risk Factors

Social cognitive research has identified differences in the way that aggressive children process information [64–66]. Lochman et al and Dodge examined social cognitive variables in aggressive and nonaggressive boys at preadolescent and early adolescent developmental points [66,67]. They found that aggressive children often misread interpersonal cues and interpret ambiguous or prosocial communication as hostile and react aggressively. The children also often have heightened sensitivity to rejection derived from early experiences of physical abuse or emotional neglect that then triggers anxiety or angry states [68,69]. This tendency to identify affect arousal as anger can also lead to overlooking verbal solutions in favor of frequent and intense aggressive behavior.

Trauma-related emotions can trigger severe aggression in response to minor or trivial disappointments. Slaby and Guerra elaborated on the cognitive profile of these aggressive adolescents who believe that there are limited consequences for aggression, that aggression has concrete benefits, and that it is a legitimate response [70]. These findings are exceedingly important for clinicians working with aggressive children and their parents. Understanding the impact of impaired social communication can assist families in understanding violent outbursts and serve as the basis for developing potential interventions. This insight can also assist clinicians in recognizing how distorted social cognition in patients and their families impedes their efforts for intervention.

Social and Environmental Factors

Family Factors

The family environment is the intimate system wherein development is shaped. There is ample empirical evidence (longitudinal designs, randomized controlled clinical trials, and cross-sectional studies) demonstrating the pivotal role of consistent parental discipline in preventing early patterns of aggressive behavior [6,7,71,72].

Dishion et al and Patterson et al developed a model of coercion that starts with family practices beginning in early childhood [73,74]. In this typical scenario, when an oppositional child is aggressive, the parents fail to intervene early and to set reasonable standards for behavior. Instead, parents may

respond inconsistently by withdrawing, giving a neutral response, or overreacting with excessively harsh punishment or exaggerated negative affect. A reciprocal escalation of behavior may ensue with increasingly coercive parent-child interactions. The child learns that aggressive reactions to parental requests often lead to parental abdication and withdrawal. Thus, the child uses aggressive behavior to effectively terminate parental aversive requests, and in turn, the aggressive behavior is reinforced (escape conditioning). Often, the same parents may overlook or respond inappropriately to the prosocial behavior their children may occasionally demonstrate. The insights on family interaction reinforce the importance of clinician attention to parent-child interactions in dealing with aggressive behavior. Parents are often frustrated in their attempts to manage aggressive behavior in their offspring and may be unaware of how their responses may unwittingly sustain or even exacerbate behavior. This explanation does not mean that responsibility for violent acts by youth should be incorrectly placed on the parents, but rather points to the need for families to find more effective means to resolve the issues that contribute to aggressive behavior. In terms of assisting parents, ready information about how parents can use appropriate discipline methods, attend to positive reinforcement, and encourage conflict resolution is useful. Consistent parental discipline, increased positive parental involvement, and increased monitoring of the child's activities were accompanied by significant reductions in a child's antisocial behavior.

Peers and Gangs

As with the development of other social behaviors, peers have an impact on aggression and violence in adolescence. Studies with different age groups indicate that the influence of deviant peer behavior on the development of aggression is most pronounced during adolescence. Associating with delinquent peers was predictive of self-reported adolescent violence in several studies [75,76]. In addition, associating with peers who disapprove of antisocial behavior appears to reduce the likelihood of later violent acts [76]. Unfortunately, in mixed groups of children, nonaggressive children are more likely to become aggressive than are aggressive children to become nonaggressive [77]. Despite the contribution of deviant peers to the onset of adolescent aggression, the mechanism of the causal influence of peer networks is not delineated.

Gangs may be a special case in peer relationships and violence. Numerous studies report an association between gang involvement and increased violence and delinquency [78–82]. The result of Thornberry's analysis of gang members supports a facilitation model where the norms and group processes of the gang exacerbate the behavior patterns of the individual gang members [83]. Interestingly, before and after gang membership, these individuals do not have significantly different risk factors or profiles than nongang members. Also, gang members are disproportionately responsible for delinquent crime, particularly serious and violent offenses [78,84].

The Seattle Social Development Project also found that the influence of gangs was greater than just associating with deviant peers [79]. Parents can modify the effect of deviant peers, with a positive parent-child relationship providing protection for adolescents [85,86]. Another important peer influence on the development of aggression may be social ostracism, as seen in recent school shootings. In early childhood, both peer group rejection and victimization are associated with increased risk for aggressive behavior [87,88]. It is not clear whether this rejection and victimization are prompted by early aggressive behaviors or by some other individual risk factor, such as impulsivity. Certainly, social ostracism results in youth having fewer opportunities to learn and practice socially acceptable behaviors through positive peer relationships. To curtail bullying by aggressive children, Olweus designed systemic interventions in schools to increase monitoring and establish consequences for bullying [89]. Twemlow et al examined how coercive power dynamics in school are critical to understanding how bullying can be sustained in school settings [90]. By analyzing the school climate, the power dynamic can be rebalanced so as to decrease the potential for violence [90]. Whereas the negative effect of antisocial peers is a risk factor for aggressive behavior in youth, clinicians should recognize the heightened impact of gangs and their recent spread throughout American communities. It is important to learn not only about the patient's peer group, but also if there is gang presence and involvement.

Gangs and Females

The finding that male gang involvement is associated with a disproportionate amount of serious and violent crime holds true with girl gangs as well [84]. Surveys have demonstrated that female gang mem-

bers are more likely to be violent than non-gang-involved boys [91]. Although female gangs represent a small proportion of gang members, the numbers on females in gangs vary widely depending on whether data are drawn from official law enforcement sources or self-report surveys. The law enforcement data may underestimate the presence of girls because of the law enforcement's limited capacity to get accurate internal information from the gangs and because of the extensive confusion around how to define a gang [92]. National surveys of law enforcement agencies over two decades, covering 61 police departments, show a total of 992 female gang members comprising approximately 4% of the gang population [91]. In a multisite, multistate cross-sectional survey of a public school sample of eighth grade students (not a random sample), 237 girls out of 623 gang members in an ethnically diverse group of 6000 students identified themselves as gang members (38%) [93].

The re-examination of the role of female gang members has redefined the earlier bias by male researchers who relied on interviews with male members [91,92]. Female gang members were initially seen as playing an auxiliary role in the gang and primarily acting as weapon bearers, sexually exploited members, or girlfriends [92,94]. The trajectory of female gang involvement may be different and more complex than originally posited. Ethnographic fieldwork has highlighted that the adolescent girls' participation in gangs may reflect frustration about a harsh, constricted future [84,95]. Females were more likely to look to the gang as a refuge than males and they often came from more troubled families than the male gang members [84].

Environmental and Situational Factors

Studies of communities and individuals confirm the popular impression that youth violence is more common in urban and impoverished neighborhoods [96,97]. Certainly the impact of poverty on the family system contributes to the risk for violence and aggression, but the analysis of neighborhood characteristics offers a more complex understanding. Collective efficacy (assessed by cross-sectional surveys of 8782 Chicago adult residents) shows that active engagement by adults to supervise and maintain order, neighborhood residential stability, and concentrated affluence decreases the likelihood of violence in a community [8,9]. Additionally, adults sharing relevant information and providing supervision for informal social control, known as intergen-

erational support, were more often identified in close proximity to other stable neighborhoods [10]. Another factor that adds to the vulnerability of the neighborhood occurs when youth are exposed to violence, as this exposure increases the risk for aggressive behavior in youth [12].

Access to a potentially lethal weapon, usually a firearm, increases the likelihood that a lethal event will result from an aggressive or violent altercation [98]. The relatively easy access to firearms for youth increases the risk of youth violence [11]. Weapon-carrying for some adolescents is relatively common, as identified in a 2001 Center for Disease Control and Prevention study, Youth Risk Behavior Surveillance System [99]. In that national study of high school students, 17.4% of adolescent boys carried a weapon (a knife, gun, or club) at some point during the month before the survey [99]. The rate was higher in some areas (e.g. one survey that was conducted in inner-city middle schools found that 25% of male students and 11% of female students reported carrying a gun with gun-carrying strongly linked to aggressive delinquency rather than to self-protection) [100]. Boys most likely to carry handguns were those with the most aggressive behaviors (i.e., initiating fights), who believed that shooting someone is justifiable under certain circumstances and who perceived their peers as accepting violence [101].

Pittel used clinical evaluations to describe some of the beliefs of students carrying weapons and categorized them as "deniers," "innocents," "fearfuls," and "defenders" [102,103]. For example, deniers claim ignorance of how the weapon came into their possession. They insist that they did not knowingly carry the weapon into school and claim an unknown culprit planted it in their book bag or locker. Innocents admit to possessing a weapon but claim they were holding it for someone else or found it. It is important to further elucidate the reasons that adolescents carry weapons, as it will inform clinical interventions.

A moderate relationship exists among illicit drug use, alcohol, and violence [104]. Alcohol can stir aggression by reducing threat-related inhibition and increasing arousability. Alcohol also decreases higher-order cognitive functioning by altering the adolescent's ability to communicate and judge the degree of threat in a social situation [104]. A study on youth violence in schools demonstrated that 40% of the students who drank alcohol at school reported carrying a weapon to school, as compared with 4% of youth who did not drink alcohol at school [105]. Aggression predicts substance use and substance use

predicts aggression [106]. An extended longitudinal study found that aggressive behavior in childhood is predictive of substance use in adolescence [107]. This research also indicated that the relationship appears to be influenced by the presence of associated symptoms of depression and impulsivity. Other factors that may affect the association between aggression and substance use in youth include family history of alcoholism and drug abuse and involvement with peers or gangs using drugs [107]. Clinicians must be aware of the vicious cycle that exists between substance use and violence in youth, as with adults.

These findings on specific environmental factors contributing to youth violence enable clinicians to assess the individual patient's potential risk, as well as current behavior patterns, in greater detail. Such understanding can provide the basis for a more tailored and individualized approach to developing prevention and intervention plans. Public health efforts can also be directed to address these defined risks within the broader community to reduce and hopefully prevent youth violence.

Conceptual Models

Cumulative Risk Factors

Numerous factors contribute to the relative risk for the development of violence and no single factor is associated with all aggression or provides absolute prediction. Studies utilizing multiple factors provide stronger prediction of violence and demonstrate the interaction and increased cumulative risk of these influences [108]. Evidence indicates that the impact of risk factors depends upon their presence during specific stages of development [96].

Specific models describing distinct pathways in the development and progression of aggressive behavior that incorporate multiple risk factors have been proposed based on longitudinal research [14,15]. As part of an overall model of the development of antisocial behaviors, Loeber et al describe a specific course of development of aggressive and violent acts. Minor fights and bullying characterize the early stage, progressing to the later stages of more serious assaults, weapon use, rape, and robbery [15]. Although many children will exhibit entry level behaviors, fewer progress to each successive stage of antisocial acts. The further a youth progresses in development of aggressive behaviors, the more likely that other antisocial behaviors will also appear. Therefore, youth with the most severe behaviors will often exhibit the widest variety of antisocial acts [16].

The central design of effective preventive efforts is twofold: (a) the examination of risk and protective factors at critical developmental periods, and (b) the understanding of the mechanisms through which these risk factors impair youth behavior. In the context of assessing violent/aggressive children, the principal questions are whether children are "hard-wired" and genetically primed to be aggressive, whether the environment is shaping the vulnerable child, or both. Raine's research showed substantial empirical evidence to support the interaction between biological and environmental variables to specifically explain violent behavior [3,4]. Raine drew comparisons from a large birth cohort (4269 male children in Denmark) and classified the children according to two variables. If children had birth complications or neurological impairment, they had about the same chance of becoming criminally violent 18 years later as those children with no risk factors. The group of children with both early childhood rejection and birth complications (4.5% of population) accounted for 18% of all violent crimes committed by the collective sample of 4269 subjects. Raine's study defines early childhood rejection as maternal rejection of the infant (unwanted pregnancy and attempt to abort the fetus) and disruption of the mother-infant bond (public institutional care of the infant). Significantly, the interaction effect was found to be specific to violent offending and did not generalize to nonviolent crimes or recidivism, per se. A different example of the critical interaction between genetic risk and environmental influence was provided from the Dunedin longitudinal study [5]. Physically abused boys with a variant of the monoamine oxidase A (MAOA) gene were twice as likely to develop aggressive behaviors and three times as likely to be convicted of a violent offense as an adult in comparison with abused boys without the MAOA variant. In the absence of a history of abuse, boys with the variant MAOA gene were at no greater risk for later aggressive behaviors than other nonabused boys.

This research provides specific information about some of the very early risk factors for violent behavior and has major policy implications and clinical relevance supporting intensive early intervention. Effective early interventions with nurse visitation in the home environment for high-risk families (average of 30 visits spanning from prenatal to the child's 2nd birthday and focusing on maternal functioning) have shown a significant reduction in adolescent antisocial behavior including arrests and convictions, in comparison to a control group [109]. This type of

intervention can compensate for negative birth complications and promote positive parenting, thereby preventing the more serious forms of antisocial behavior leading to arrests and convictions [109].

Aggression Subtypes

From a clinical perspective, research on subtypes of aggression may be helpful in understanding and treating aggression. Clinical observation, experimental paradigms in laboratories, and cluster/factor analytic studies show subtypes of aggression that may have implications for the management and treatment of aggressive patients [64,110,111]. These qualitatively distinct forms of aggression in youth may affect more tailored prevention and intervention approaches to help predict treatment response.

One subtype of antisocial behavior is classified according to time of onset: childhood-onset (prepubertal) or adolescent-onset [1,112–114]. The investigations primarily examined longitudinal groups of males at different intervals utilizing direct observation, peer nomination (wherein peers identify the most aggressive peers), or teacher/parent ratings of disruptive behavior. The results are usually presented in terms of variance (percentage) or stability coefficient (correlating individuals from one time to another time with certain behaviors present). Childhood-onset antisocial behavior is rarer than adolescent-onset, typically 5–6% in the general population of young males, but it is associated with more seriously persistent violent behavior and worse outcomes [7]. Childhood-onset antisocial behavior is more likely associated with neuropsychological deficits (e.g., impaired language and intellectual functioning, attention deficit hyperactivity disorder [ADHD]) and inconsistent discipline by parents when the child is young [113].

Investigations about aggression and conduct disorder-like behavior demonstrate aggression as a relatively stable trait, often compared with intelligence [17]. Olweus carefully reviewed 16 longitudinal studies of subjects 2 to 18 years of age and showed high stability coefficients (.81 in males). Subsequent studies, with varying methods of assessment, also demonstrated high rates of stability of aggression in clinically referred samples and community samples with a range from 32% to 81% of children continued with their disruptive, aggressive behavior in adolescence [17]. Although these studies emphasized high stability of aggression over time, it is critical to enhance the understanding about the significant proportion of aggressive youth that do not maintain

aggressive behavior over time, and to recognize that a small portion of adult violent offenders had short-term escalation of aggression at late onset [115]. It is critical that clinicians not interpret the relative stability of aggression as equivalent to aggression being relatively intractable as a fixed and predetermined behavior. Although there is a consistent finding in the stability of aggression, this finding has not translated into an understanding of patterns of aggressive behavior within individuals. Nor has this categorization generated an understanding about the large individual differences in the stability of aggression; which individuals may replace aggression with better adaptive behavior, which individuals are at greater risk for persistent aggression, and which youth are intermittently aggressive.

There are several limitations with childhood-onset and adolescent-onset subtyping. The problem with the term “childhood-onset” is that it implies a fixed, determined behavior, and does not seem to reflect the ongoing exposure to risk factors and cumulative insults that shape and reinforce persistent aggression. The variability in aggression or antisocial behavior suggests that different ways of measuring aggression may result in different indices of stability or discontinuity [116]. This type of measurement does not capture the periodicity of aggression, and high correlation does not demonstrate the change in severity level of aggression with age. The inadequacy of the categories was further illustrated when Tolan and Thomas’ examined early- and late-onset offenders and showed that both populations looked surprisingly similar in their cumulative risk factors [117]. In creating onset curves in a longitudinal sample of 500 males from the Pittsburgh Youth Group Study, the age of onset of aggression gradually increased for each level of aggression and there was no bimodal distribution that would support early versus late onset [116].

The most empirical research analyzing distinct patterns of aggressive antisocial and delinquent behaviors relates to the trajectory of overt and covert behaviors [15,111,116]. These underlying dimensions of aggression were developed almost exclusively on males, and non-Anglos were underrepresented.

Despite the limitation, a temporal sequence of escalating aggressive behaviors was proposed by examining the Pittsburgh Youth Study of 1500 males in three cohorts, ranging from ages 7 to 13 years at the first sampling time, with 6-month intervals between assessments followed over 10 years [96]. The cohorts were chosen so as to cover the age-range of development under investigation (7 years to young

adulthood), but the three cohorts do not represent separate pathways, just separate age groups. In the "overt" pathway, males start by annoying and bullying others, followed by physical fighting, then by assaultive behavior and forced sex. The "covert" pathway entails sneaky acts such as stealing and lying, followed by property damage, vandalism, and fire setting; culminating in fraud, burglary, and serious theft. The third proposed developmental pathway involved those males with "authority conflict." This research highlights how identifying common clusters of aggression and sequences of behaviors may improve early identification. When this theoretical framework was applied to the National Youth Data of a nationally representative sample, a larger proportion of serious and violent youth offenders followed the overt developmental sequence than the general population [118]. The initial step of detailing the developmental patterns of aggression over time and identifying the probable trajectory of serious and violent offenders may allow a clinician to identify patients at risk when they have a progression of behavior and not by the presence of a specific behavior. Although the cumulative acts of aggression are detailed, the mechanism of how individuals begin with minor aggressions, progress to more severe forms of violence, and how individuals with similar behavior will follow these predictable trajectories, remain to be elucidated. Winnicott's essays on deprivation and delinquency [119] or Aichhorn's observations on Wayward Youth still provide insight about the inner experience and psychic turmoil [120]. These authors illuminate the meaning of the outward manifestations of behavior through insightful interviews of individual patients, often overlooked in the population-based studies.

Another subtype of aggression emanates from multicultural studies that assessed proactive aggression and reactive aggression [64,121]. Children initiate proactive aggression to obtain specific rewards and establish social dominance. Proactive aggression involves a minimal level of physiological arousal and relates to predatory aggression. Conversely, reactive aggression or affective aggression involves the defensive use of force against a perceived threat or provocation. This defensive stance is triggered by activation of the fight-or-flight response, with a high level of physiologic arousal.

Different neuroanatomical chemical pathways underlie these forms of aggression. Affective/reactive aggression is characterized by impulsive/explosive anger and decreased levels of serotonin metabolites in cerebrospinal fluid [122,123]. The autonomic acti-

vation is fear-induced and leads to irritability and hyperarousability [124]. In animal models, stimulation of the ventromedial hypothalamus reproduces (simulates) an affective type of aggression [125]. Predatory aggression involves minimal levels of autonomic activation and the information processing is different [124,126].

In a small clinical sample, Vitiello et al provided preliminary evidence of the clinical validity of subtypes of aggression [127]. A scale was constructed with items that demonstrated good internal consistency, reliability, and stability for identifying predatory and affective aspects of aggression. The instrument was used to differentiate the types of aggression of 73 aggressive boys and girls aged 10 through 18 years who were inpatients or enrolled in a partial hospitalization program. Most of the patients had either predominantly affective or mixed predatory-affective scores. Vitiello's findings suggest that those children who are purely proactive/predatory aggressors are not as frequently treated or admitted to psychiatric hospitals. Patients with a high affective aggression score had a higher incidence of psychotic symptoms and a higher likelihood of receiving lithium or neuroleptics.

Distinguishing whether adolescents' aggression is primarily reactive or proactive may suggest the therapeutic direction of prevention and treatment, as well as prognosis [128]. If adolescents have reactive aggression, they most likely have impaired social cognitive processing that misinterprets information and can be responsive to cognitive behavioral therapy that provides an alternative approach to fearful stimuli than reacting aggressively [128-130]. These types of patients may also benefit from medications that alter their hyperaroused state. Proactive aggressive youth are more likely to progress to externalizing behaviors and subsequent criminal behavior than males assessed as having reactive aggression in adolescence and followed into adulthood [131]. Proactive boys have the expectation of positive outcomes from aggressive behavior and thus the emphasis is on systematic interventions, increased monitoring, and consistent consequences [90].

Considerations in Risk Assessment

Assessing children and adolescents for potential violence requires an organized approach that draws on clinical knowledge, a thorough diagnostic interview, and familiarity with relevant risk and protective factors. Even with guidelines and checklists for iden-

Table 1. Assessment Guidelines for Clinicians

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1. What are the capabilities and skills of the parents?
 2. Is there any evidence of disorganized attachment to the primary caregiver?
 3. Are there any other medical problems that suggest abnormalities with regulation of behavior or affect?
 4. Does the patient's aggression fall into predatory aggression or affective aggression?
 5. What is the range, severity and frequency of the aggressive behavior?
 6. Is there a clear precipitant to the aggression, (predictable triggers or situations)?
 7. Has the patient been traumatized, and could that lead to hypervigilance and hostile attributions?
 8. Is there a past history of violent episodes?
 9. What are the parents' attitudes towards violence?
-

tifying risk factors, there is the possibility of errors: false positives, false negatives, or both. False positives are children and adolescents who may have risk factors but do not act violently, whereas false negatives are youth who are overlooked but who subsequently act violently. In the absence of validated and reliable screening instruments or effective protocols, we propose a rational approach to the clinical interview, conducted by a mental health practitioner, that will help in evaluating individual children or adolescents for potential violence [132]. Unfortunately, there are practical barriers regarding some adolescents that practitioners would ideally like to refer, such as time lag, financial limitations, and family or patient distrust of practitioners. If the patient makes explicit verbal threats or appears to have prominent symptoms suggestive of a comorbid state (exacerbating his/her aggression), the treating clinician is advised to make a referral.

The starting point of an evaluation is a general diagnostic psychiatric interview to determine if the young patient has a major mental illness, medical disorder, or substance abuse that could be contributing to his or her aggressive behavior. A clinician should cover specific areas of information in an organized fashion using a format similar to the one illustrated in Table 1.

The questioning can then move on to facts about the immediate context of the aggression. It is important to obtain collateral information from parents, teachers, court records, or security guards, because minimization of responsibility for actions and denial are to be expected. It is critical to carefully assess the patient's attitudes toward carrying a weapon, access to a weapon, and the risk of using a weapon in a fight. It also is important to identify which adults support this young patient, including other clini-

cians, and to get details of past treatment attempts. These clinicians may note what has already been done for the patient. When a clinician has enough information to make a preliminary formulation, it is useful to explain to the patient the clinician's current understanding in addition to exploring the patient's insight and motivation.

Essential to the diagnostic interview is for the clinician to clarify whether the child or adolescent wants to change and is willing to work to change his/her assumptions, behavior patterns, denial of responsibility, and lack of trust. It is important to identify whether the child or adolescent who enjoys hitting or hurting the victim has any empathy or understanding of the distress inflicted on another person.

If the patient expresses no motivation to change and does not have any desire to control aggression or homicidal ideation, the assessment has reached a critical juncture. At this point, it is the clinician's responsibility to provide feedback to the adults (e.g., parents, court personnel, school staff) who have initiated the assessment. If the patient poses a very high violence risk, preventive action needs to be initiated [133].

Coercive measures such as hospitalization and the question of warning potential victims also need to be addressed. Although risk factors can indicate the potential for violence, it is still difficult to determine why some children are on a chronic trajectory of aggressive behavior and others manage to compensate despite exposure to many of the cumulative risk factors that lead to violence. Violence is rarely random, yet the dynamic and situational variables can change so quickly that an assessment is extremely time-sensitive. Developing a rational strategy for evaluating adolescents and children at risk for violence leads to the development of a treatment plan/program to contain and reduce the risk.

Prevention/Interventions

Cognitive-Behavioral Therapy

Cognitive-behavioral therapy (CBT) seeks to change social cognitive deficits and distortions in aggressive children and adolescents. It focuses on defining the problem, generating alternative solutions, anticipating consequences and introducing behavioral monitoring, and prioritizing responses. Interventions usually involve role-playing, practicing, homework assignments, and specific skill-building to change cognitive distortions and responses. Cognitive-be-

havioral problem-solving skills training (PSST), totaling 20 sessions for preadolescent children evaluated in inpatient and outpatient support settings, supports the efficacy of the treatment compared with therapeutic changes of relationship therapy (RT) and attention placebo control conditions [129,130]. The effects were demonstrated in a 1-year follow-up assessment in school and at home with changes in behavior at home and at school [134]. A more detailed review of CBT outcome research showed improvements in social competency and lessened aggressive behavior [130]. Nevertheless, further research is required to examine child and treatment characteristics that predict outcome and demonstrate clinically meaningful improvement.

Although it is critical to continue the development and evaluation of CBT, several limitations exist. First, there is the high attrition rate of severely stressed families that are hindered by the associated costs, scheduling difficulties, inconvenience, and reluctance to participate in a treatment intervention [135]. This attrition, which can be as high as 50% to 75% of children referred for treatment, can result in over-inflated support for using CBT to reduce problem behavior because the most difficult families don't participate [136]. Although the attrition rate may not be the exclusive problem to this modality, it points to the need for further improvements in the implementation of this approach.

Similarly, as in any therapy, children in CBT require motivation to change; obtaining this motivation can be challenging when aggressive behavior is egosyntonic. Garbarino, a psychologist who has worked with extremely violent boys in juvenile detention systems, cautions: "Some of the boys have memorized the list of techniques and concepts but can do no more than parrot what is in the textbook. Others say that they can not imagine being able to apply these techniques in the situations that they face in the world" [137]. Another aspect to consider is the cognitive development of the child, as it has been demonstrated that preschool and early school-age children who are preoperational in their thinking do not respond to CBT as well as older children (ages 11–15 years) who are more cognitively sophisticated [134]. Another dilemma is that the most vulnerable aggressive children often have language expressive deficits, executive functioning difficulties, and impulse control problems. These limitations make it especially difficult for children to put their emotions into words rather than actions, and they may have difficulty understanding and internalizing the cognitive scripts.

Long-term CBT follow-up usually consists of a 1-year follow-up and frequently does not include direct observation of the child's behavior or assessment of exact skills that may diminish behavior, such as aggression. Critical indices of treatment efficacy still need to be developed with the caveat that it may be more prudent to conceptualize aggression conduct disorder as a "chronic disease model." Optimizing treatment of aggression occurs if experienced clinicians are used, which is not always true outside of the research setting. Also, it is important to note that incremental gains are achieved with longer treatment (up to 50 or 60 sessions) including periodic booster sessions [130].

Psychopharmacological Interventions

Medications should be considered for violent aggressive children only in the context of a careful diagnostic assessment that reviews multiple risk factors and generates a complex formulation. Managing violent children and adolescents with solely pharmacological methods is not recommended. Failure to consider and initiate an active comprehensive treatment plan sets up the treating clinician for dangerous liability. For a treatment plan to be effective in modifying aggression, it needs to be comprehensive and address family competency, relational capabilities, and educational progress.

It is common clinical practice to identify target symptoms in an aggressive/violent child, such as irritability, impulsiveness, or affective liability. Only then are medication trials conducted that try to ameliorate the symptoms. However, this approach is tenuous because there is minimal research demonstrating its efficacy. Frequently, the research on aggression in adults is extrapolated to provide pharmacotherapy treatment suggestions for adolescents and children. The concern is that the findings on adults are not applicable to adolescents and children. There are no specific antiaggressive drugs currently available; rather there are some drugs, including atypical antipsychotics, anticonvulsants, mood stabilizers, anxiolytics, beta-blockers, and alpha-agonists that are used for their capacity to indirectly decrease aggression. There is a growing body of research on the indications and efficacy of medication in the treatment of aggression in youth. Most of the reports are of open trials rather than randomized controlled studies and among all these investigations, the reported duration of treatment is seldom longer than 2 months [138]. One striking example of the importance of rigorous research is a report that found,

among youth admitted for inpatient treatment for severe aggression, in a double-blind study, almost 50% responded to placebo [139]. Most of these randomized clinical studies use a relatively small sample of aggressive adolescents, do not identify comorbid disorders, and do not consider the impact of other treatment modalities.

Clinicians need to identify the specific conditions that may contribute to the patient's aggressive behavior and to use this information as a guide in the selection of potential medications. To determine efficacy, empirical trials of agents should be sufficiently long. Clinicians should rely on studies that use double-blind and placebo design in medication trials. Additionally, aggressive and violent behaviors should be assessed with standardized ratings [140]. A further complication is that frequently, aggressive patients may have simultaneous multiple medication trials, making it difficult to determine the pharmacodynamic effect of the combination of medications and the contribution of single agents. Connor and Steingard [141], and more recently, Frazier [142], reviewed many of the controlled studies that look at each category of psychiatric conditions that may be responsive to medication and may lead to reduction in aggressive behavior. The critical clinical recommendation is that if a comorbid condition exists, then treating it with indicated medications might reduce the aggressive behavior as well.

A guiding principal in the evaluation of violent and aggressive children is that they often have a wide range of psychopathology, including ADHD, mood disorders [143], learning and communication disorders, obsessive-compulsive disorder with associated anxiety, PTSD, substance use and abuse, and even rare cases of psychotic disorder with paranoid ideation [142]. Puig-Antich studied a subset of depressed boys with aggressive behavior and showed that if their depression improved, the antisocial behaviors also improved, whether the improvement was spontaneous or the result of treatment for depression [144]. Aggression in ADHD children is reduced if young patients are treated with stimulants [145]. Some clinicians suggest that clonidine (Catapres) treatment can be useful for ADHD children who display overaroused behavior, excessive hyperactivity, and extreme aggression [146]. Furthermore, lithium and divalproex (Depakote) have been found useful in double-blind, placebo-controlled studies for children and adolescents with disruptive disorders characterized by explosive temper and mood lability or bipolar disorder and comorbid conduct disorder [147–149]. Lastly, some clinicians suggest that a trial

with selective serotonin reuptake inhibitors may alleviate symptoms in irritable, depressed children [150,151]. Nevertheless, the best guideline is to use the least toxic and safest intervention first.

Patients with conduct disorder and associated aggressive behavior pose a particular challenge. They are difficult to build an alliance with because they often oppose adult authority and have concurrent substance use. Although there is no medication with labeling approved by the U.S. Food and Drug Administration for conduct disorder, clinicians may feel pressured to address the explosive impulsive aggression with medications. The comorbid condition of conduct disorder is critical to determine. One recent study that carefully examined 50 youths (aged 11 to 17 years) in a juvenile detention center found that 84% of the sample met criteria for conduct disorder (CD) or oppositional defiant disorder (ODD) (60% CD, 24% ODD), 20% had major depression, and 15% met criteria for ADHD [152]. Lithium has had equivocal results in trials of patients with conduct disorder [153]. Findling et al demonstrated that the use of risperidone was reported as superior to a placebo in short-term use with a small number of outpatient children and adolescents with conduct disorder, although it is difficult to determine the efficacy because of the small sample size [154]. Van Bellinghen and De Troch found that risperidone was significantly more effective than placebo in reducing aggression in a sample of children between the ages of 6 and 14 years at doses ranging from 0.03 to 0.06 mg/kg/day [155]. Risperidone's use is best limited to cases where the aggressive behavior severely affects functioning. Further systematic prospective treatment trials are needed to fully determine the effective medications for aggression in conduct disorder and comorbid conditions.

Psychosocial Treatment

A careful assessment of the developmental stage of the child or adolescent will define the therapeutic approach. The therapist tries to promote the development of new skills and encourage adopting new ways of coping. Although there are a variety of techniques that the therapist may employ, adolescents demand an inordinate amount of flexibility. The focus usually is on the adolescent's current functioning and his current relationships with an emphasis on renegotiating the adolescent-parent relationship and exploring the role of peers. The therapist usually sees the adolescent alone first, whereas with a child, the parent may be interviewed first.

Adolescents often do not recognize their need for help and may project their difficulties as derived from unrealistic responses of teachers or parents. If clinicians are making a referral to a therapist they can help to anticipate with the adolescent that it is a normal reaction to balk at this type of treatment initially. Children are usually more receptive to building a trusting relationship with a therapist than adolescents. Therapists often rely on role playing and engaging game activities with children that help model how children can control their impulses [156].

In Parent Management Training (PMT), the focus is on parents acquiring concrete skills that concentrate on teaching prosocial behavior [130]. Parents learn to observe antecedents to their child's behaviors and to modify the consequences. There also is an emphasis on active role-playing, practice, and feedback. Outcome studies have shown gains that have been maintained 1 to 3 years after this form of treatment [157]. However, most PMT studies focus on children 3 to 10 years of age [130,158].

Multisystemic Treatment (MST), a family-based intensive therapeutic approach, has been demonstrated to be effective with adolescent juvenile offenders [159]. MST is tailored to the needs of each family with the goal of improving the communication skills and management of the family's problem behavior. Borduin also showed that juvenile offenders (they averaged 4.2 previous arrests) who received the MST intervention were less likely to be arrested for violent crimes than were youths who had received individual therapy [159]. The long-term effects of MST have promising outcomes [160].

Discussion and Summary

Aggression and violence in youth have grave implications for the progression of psychiatric impairment, school difficulties, and legal involvement. As clinicians, it is useful to develop insight about how to conceptualize and organize biopsychosocial information to better guide patients and incorporate new information about treatment. Clinicians are well positioned to identify those individuals that are exposed to multiple risk factors, such as poor social attachments, comorbid psychiatric disorders, coercive family discipline, and access to fire arms, and can ideally suggest interventions before the aggressive behavior is chronic, frequent, pervasive, and severe. Although there are substantial data defining subtypes of aggression (covert/overt aggression, reactive/proactive aggression), further refinement of

these subtypes is needed to develop better screening instruments to identify particular behavior. In turn, this information may inform how clinicians prioritize interventions. Researchers still need to develop and confirm different models that explain the progression or deterrence of adolescents engaging in these troubling behaviors. More investigation is warranted to discern certain correlates of aggression in both community populations and clinically referred patients so that tailored prevention, early interventions, and evidence-based treatment can be mobilized. As treatment interventions are more rigorously tested and meaningful algorithms are generated, clinicians may come to see the aggressive teenager as challenging, and yet also know how to build on the adolescents' strengths and help to substantially modify their aggression. The pattern of violence will perpetuate or not, depending on how clinical understanding deepens regarding the causes of aggression and how this understanding is turned into prevention, intervention, and treatment. The insight and practical suggestions that are generated will allow our children and adolescents to make meaningful alternative choices.

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References

1. Fox JA, Zawitz MW. Homicide Trends in the United States. Washington, DC: Bureau of Justice Statistics, 2001.
2. Snyder HN. Juvenile Arrests in 1999. Washington, DC: Office of Juvenile Justice and Delinquent Behavior, 2000.
3. Raine A, Brennan P, Mednick SA. Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: Specificity to serious, early-onset violence. *Am J Psychiatry* 1997;154:1265-71.
4. Raine A, Brennan P, Mednick SA. Birth complications combined with early maternal rejection at age 1 year predispose to violent crime at age 18 years. *Arch Gen Psychiatry* 1994;51:984-8.
5. Caspi A, McClay J, Moffitt T, et al. Role of genotype in the cycle of violence in maltreated children. *Science* 2002;297:851-3.
6. Patterson GR, Southamster-Loeber M. The correlation of family management practices and delinquency. *Child Dev* 1984;55:1299-1307.
7. Tolan P. Socioeconomic, family, and social stress correlates of adolescent antisocial and delinquent behavior. *J Abnorm Child Psychol* 1988;16:317-31.
8. Brewer DD, Hawkins JD, Catalano RF, et al. Preventing serious, violent, and chronic juvenile offending: A review of evaluations of selected strategies in childhood, adolescence, and the community. In: Howell JC, Krisberg B, Hawkins JD, et al (eds). *Sourcebook on Serious, Violent, and Chronic*

- Juvenile Offenders. Thousand Oaks, CA: Sage Publications, 1995:271-6.
9. Peeples F, Loeber R. Do individual factors and neighborhood context explain ethnic differences in juvenile delinquency? *J Quant Criminol* 1994;10:141-58.
 10. Sampson RJ, Morenoff JD, Earls F. Beyond social capital: Spatial dynamics of collective efficacy for children. *Am Sociol Rev* 1999;64:633-60.
 11. Ash P, Kellermann AL, Fuqua-Whitley D, Johnson A. Gun acquisition and use by juvenile offenders. *JAMA* 1996;275:1754-8.
 12. Sampson RJ, Raudenbush SW, Earls F. Neighborhoods and violent crime: A multilevel study of collective efficacy. *Science* 1997;277:918-24.
 13. Schwab-Stone M, Chen C, Greenberger E, et al. No safe haven II: The effects of violence exposure on urban youth. *J Am Acad Child Adolesc Psychiatry* 1999;38:359-67.
 14. Loeber R, Wung P, Keenan K, et al. Developmental pathways in disruptive child behavior. *Dev Psychopathol* 1993;5:103-33.
 15. Loeber R, Green SM, Keenan K, et al. Which boys will fare worse? Early predictors of the onset of conduct disorder in a six-year longitudinal study. *J Am Acad Child Adolesc Psychiatry* 1995;34:499-509.
 16. Loeber R, Hay DF. Developmental approaches to aggression and conduct problems. In: Rutter M, Hay DF (eds). *Development Through Life: A Handbook for Clinicians*. Oxford, UK: Blackwell Scientific, 1994:488-515.
 17. Olweus D. Stability of aggressive reaction patterns in males: A review. *Psychol Bull* 1979;86:852-75.
 18. Kazdin AE, Esveltd-Dawson K, French NH, et al. Problem-solving skills training and relationship therapy in the treatment of antisocial child behavior. *J Consult Clin Psychol* 1987;55:76-85.
 19. Burke JD, Loeber R, Birmaher B. Oppositional defiant disorder and conduct disorder: A review of the past ten years, part II. *J Am Acad Child Adolesc Psychiatry* 2002;41:1275-93.
 20. Ellickson PL, McGuigan KA. Early predictors of adolescent violence. *Am J Public Health* 2000;90:566-72.
 21. Tremblay RE, Pihl RO, Vitaro F, et al. Predicting early onset of male antisocial behavior from preschool behavior. *Arch Gen Psychiatry* 1994;51:732-9.
 22. Lewis DO, Yeager CA, Cobham-Portorreal CS, et al. A follow-up of female delinquents. *J Am Acad Child Adolesc Psychiatry* 1991;30:197-201.
 23. Robins LN. The consequences of conduct disorder in girls. In: Olweus D, Block J, Radke-Yarrows M (eds). *Development of Antisocial and Prosocial Behavior: Research, Theories, and Issues*. Orlando, FL: Academic Press, 1986:385-414.
 24. Pajer KA. What happens to "bad" girls? A review of the adult outcomes of antisocial adolescent girls. *Am J Psychiatry* 1998;155:862-70.
 25. Serbin L, Cooperman JM, Peters PL, et al. Intergenerational transfer of psychosocial risk in women with childhood histories of aggression, withdrawal, or aggression and withdrawal. *Dev Psychol* 1998;34:1246-62.
 26. Wangby M, Bergman LR, Magnusson D. Development of adjustment problems in girls: What syndromes emerge? *Child Dev* 1999;70:678-99.
 27. Connor DF. *Aggression and Antisocial Behavior in Children and Adolescents: Research and Treatment*. New York, NY: Guilford, 2002.
 28. Deater-Deckard K, Dodge KA. Externalizing behavior problems and discipline revisited: Nonlinear effects and variation by culture, context and gender. *Psychol Inq* 1997;8:161-75.
 29. Zoccolillo M, Tremblay R, Vitaro F. DSM-III-R and DSM-III criteria for conduct disorder in preadolescent girls: Specific but insensitive. *J Am Acad Child Adolesc Psychiatry* 1996;35:461-70.
 30. Lahey BB, Miller TL, Gordon RA, Riley AW. Developmental epidemiology of the disruptive behavior disorders. In: Quay HC, Hogan AE (eds). *Handbook of Disruptive Behavior Disorders*. New York, NY: Plenum Press, 1999:23-48.
 31. Poe-Yamagata E, Butts JA. *Female Offenders in the Juvenile Justice System*. Washington, DC: Office of Juvenile Justice and Delinquency Prevention, 1996.
 32. Wakschlag L, Pickett K, Cook E, et al. Maternal smoking during pregnancy and severe antisocial behavior in offspring: Are they causally related? *Am J Public Health* 2002;92:966-74.
 33. Nagin DS, Tremblay RE. Trajectories of boys' physical aggression, opposition, and hyperactivity on the path to physically violent and nonviolent juvenile delinquency. *Child Dev* 1999;70:1181-96.
 34. Crick NR, Dodge KA. A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychol Bull* 1994;115:74-101.
 35. Crick NR, Grotpeter JK. Relational aggression, gender, and social-psychological adjustment. *Child Dev* 1995;66:710-22.
 36. Crick R, Werner NE. Response decision processes in relational and overt aggression. *Child Dev* 1998;69:1630-9.
 37. Shaffer D, Widiger TA, Pincus HA. DSM-IV child disorder, part II: final overview. In: Widiger TA, Frances AJ, Pincus HA, et al (eds). *DSM-IV Sourcebook, Volume 4*. Washington, DC: American Psychiatric Association, 1998:963-77.
 38. Bardone AM, Moffitt TE, Caspi A, et al. Adult physical health outcomes of adolescent girls with conduct disorder, depression, and anxiety. *J Am Acad Child Adolesc Psychiatry* 1996;37:594-601.
 39. Loeber R, Farrington DP, Tolan PH, et al. The implications of age of onset for delinquency risk: II. Longitudinal data. *J Abnorm Child Psychol* 1995;23:157-81.
 40. Obeidallah DA, Earls FJ. *Adolescent Girls: The Role of Depression in the Development of Delinquency*. National Institute of Justice Research Preview. Washington, DC: U.S. Department of Justice, 1999.
 41. Cauffman E, Feldman S, Waterman J, et al. Posttraumatic stress disorder among female juvenile offenders. *J Am Acad Child Adolesc Psychiatry* 1998;37:1209-16.
 42. Loeber R, Farrington DP. *Serious and Violent Juvenile Offenders: Risk Factors and Successful Interventions*. Thousand Oaks, CA: Sage Publications, 1998.
 43. Flannery DJ, Singer MI, Wester K. Violence exposure, psychological trauma, and suicide risk in a community sample of dangerously violent adolescents. *J Am Acad Child Adolesc Psychiatry* 2001;40:435-42.
 44. Kataoka SH, Zima BT, Dupre DA, et al. Mental health problems and service use among female juvenile offenders: Their relationship to criminal history. *J Am Acad Child Adolesc Psychiatry* 2001;40:549-55.
 45. McBurnett K, Lahey BB, Rathouz PJ, et al. Low salivary cortisol and persistent aggression in boys referred for disruptive behavior. *Arch Gen Psychiatry* 2000;57:38-43.
 46. Yehuda R, Boisoneau D, Lowy MT, et al. Dose-response changes in plasma cortisol and lymphocyte glucocorticoid receptors following dexamethasone administration in combat veterans with and without posttraumatic stress disorder. *Arch Gen Psychiatry* 1995;52:583-93.
 47. Perry BD, Pollard RA. Homeostasis, stress, trauma, and adaptation: A neurodevelopmental view of childhood trauma. *Child Adolesc Psychiatr Clin N Am* 1998;7:33-51.

48. Spangler G, Grossman KE. Biobehavioral organization in securely and insecurely attached infants. *Child Dev* 1993;64:1439–50.
49. Lyons-Ruth K. Attachment relationships among children with aggressive behavior problems: The role of disorganized early attachment patterns. *J Consult Clin Psychol* 1996;64:64–73.
50. Wartner U, Grossman K, Fremmer-Bombik E. Attachment patterns at age six in South Germany: Predictability from infancy and implications for preschool behavior. *Child Dev* 1994;65:1014–27.
51. Van Goozen SM, Matthys W, Cohen-Kettenis PT, et al. Hypothalamic-pituitary adrenal axis and autonomic nervous system activity in disruptive children and matched controls. *J Am Acad Child Adolesc Psychiatry* 2000;39:1438–45.
52. Kruesi MJP, Hibbs ED, Zahn TP, et al. A 2-year prospective follow-up study of children and adolescents with disruptive behavior disorders. *Arch Gen Psychiatry* 1992;49:429–35.
53. Clarke RA, Murphy DL, Constantino JN. Serotonin and externalizing behavior in young children. *Psychiatry Res* 1999;86:29–40.
54. Halperin JM, Newcorn JH, Kopstein I, et al. Serotonin, aggression, and parental psychopathology in children with attention-deficit disorder. *J Am Acad Child Adolesc Psychiatry* 1997;36:1391–8.
55. Halperin JM, Newcorn JH, Schwartz ST, et al. Age-related changes in the association between serotonergic function and aggression in boys with ADHD. *Biol Psychiatry* 1997;41:682–9.
56. Scerebo A, Kolko D. Salivary testosterone and cortisol in disruptive children: Relationship to aggressive, hyperactive, and internalizing behaviors. *J Am Acad Child Adolesc Psychiatry* 1994;33:1174–84.
57. Gerra G, Zaimovic A, Giucastro G, et al. Neurotransmitter-hormonal responses to psychological stress in peripubertal subjects: Relationship to aggressive behaviors. *Life Sci* 1998;62:617–25.
58. Brooks J, Reddon J. Serum testosterone in violent and non-violent young offenders. *J Clin Psychol* 1996;52:475–83.
59. Constantino J, Grosz D, Saenger P, et al. Testosterone and aggression in children. *J Am Acad Child Adolesc Psychiatry* 1993;32:1217–22.
60. Berenbaum S, Resnick S. Early androgen effects on aggression in children and adults with congenital adrenal hyperplasia. *Psychoneuroendocrinology* 1997;22:505–15.
61. Olweus D, Mattisson A, Schalling D, et al. Circulating testosterone levels and aggression in adolescent males: A causal analysis. *Psychosom Med* 1988;50:261–72.
62. Cohen D, Nisbett R, Bowdle B, et al. Insult, aggression, and the Southern culture of honor: An “experimental ethnography.” *J Pers Soc Psychol* 1996;70:945–60.
63. Fava M, Rosenbaum JF, Pava JA, et al. Anger attacks in unipolar depression with and without anger attacks. *Am J Psychiatry* 1993;150:1158–63.
64. Dodge KA, Coie JD. Social information processing factors in reactive and proactive aggression in children’s peer groups. *J Pers Soc Psychol* 1987;53:1146–58.
65. Lochman JE, Whidby JM, Fitzgerald D. Cognitive-behavioral assessment and treatment with aggressive children. In: Kendall PC (ed). *Child and Adolescent Therapy: Cognitive-Behavioral Procedures*. New York, NY: Guilford Press, 2000:31–83.
66. Lochman JE, Coie JD, Underwood M, et al. Effectiveness of a social relations intervention program for aggressive and non-aggressive, rejected children. *J Consult Clin Psychol* 1993;61:1053–8.
67. Dodge KA. Social cognition and children’s aggressive behavior. *Child Dev* 1980;51:162–70.
68. Dodge KA, Bates J, Pettit GS. Mechanisms in the cycle of violence. *Science* 1990;250:1678–83.
69. Feldman S, Downey G. Rejection sensitivity as a mediator of attachment behavior in young adults exposed to family violence. *Dev Psychopathol* 1994;6:231–47.
70. Slaby RG, Guerra NG. Cognitive mediators of aggression in adolescent offenders. *Dev Psychol* 1988;24:580–8.
71. Farrington DP. The family backgrounds of aggressive youths. In: Hersov LA, Berger M, Shaffer D (eds). *Aggression and Antisocial Behavior in Childhood and Adolescence*. Oxford, UK: Pergamon Press, 1978:73–93.
72. Loeber R, Dishion T. Boys who fight at home and school: Family conditions influencing cross-setting consistency. *J Consult Clin Psychol* 1983;52:759–68.
73. Dishion TJ, Patterson GR, Kavanagh KA. An experiential test of the coercion model: Linking theory, measurement, and intervention. In: McCord J, Tremblay RE (eds). *Preventing Antisocial Behavior: Interventions from Birth Through Adolescence*. New York, NY: Guilford Press, 1992:253–82.
74. Patterson GR, DeBaryshe BD, Ramsey E. A developmental perspective on antisocial behavior. *Am Psychol* 1989;44:329–35.
75. Farrington DP. Early predictors of adolescent aggression and adult violence. *Violence Vict* 1989;4:79–100.
76. Elliott DS. Serious violent offenders: Onset, developmental course, and termination—The American Society of Criminology 1993 presidential address. *Criminology* 1994;32:1–21.
77. Snyder H, Finnegan T, Stahl A, et al. *Easy Access to Juvenile Court Statistics: 1986–1995*. Pittsburgh, PA: National Center for Juvenile Justice, 1997.
78. Thomas C, Holzer C, Wall J. Serious delinquency and gang membership. *Adolesc Psychiatry* 2003;27:61–81.
79. Battin S, Hill K, Abbott R, et al. The contribution of gang membership to delinquency beyond delinquent friends. *Criminology* 1998;36:93–115.
80. Bjerregaard B, Lizotte AJ. Gun ownership and gang membership. *J Crim Law Criminol* 1995;86:37–59.
81. Elliott DS, Menard S. Delinquent friends and delinquent behavior: Temporal and developmental patterns. In: Hawkins JD (ed). *Delinquency and Crime: Current Theories*. New York, NY: Cambridge University Press, 1996:28–67.
82. Coie JD, Miller-Johnson S. Peer factors and interventions. In: Loeber R, Farrington DP (eds). *Serious and Violent Juvenile Offenders: Risk Factors and Successful Interventions*. Thousand Oaks, CA: Sage Publications, 2001:191–209.
83. Thornberry TP. Membership in youth gangs and involvement in serious violent offending. In: Loeber R, Farrington DP, (eds.), *Serious and Violent Juvenile Offenders: Risk Factors and Successful Interventions*. Thousand Oaks, CA: Sage Publications, 1998:147–66.
84. Fagin J. The social organization of drug use and drug dealing among urban gangs. *Criminology* 1990;27:633–69.
85. Poole R, Regoli R. Parental support, delinquent friends, and delinquency: A test of interaction effects. *J Crim Law Criminol* 1979;70:188–93.
86. Henggeler SW. *Delinquency in Adolescence*. Newbury Park, CA: Sage Publications, 1989.
87. Coie JD, Dodge KA, Kupersmidt JB. Peer group behavior and social status. In: Asher SR, Coie JD (eds). *Peer Rejection in*

- Childhood. Cambridge, UK: Cambridge University Press, 1990:17–59.
88. Schwartz D, McFadyen-Ketchum SA, Dodge KA, et al. Peer group victimization as a predictor of children's behavior problems at home and in school. *Dev Psychopathol* 1998;10:87–99.
 89. Olweus D. *Bullying at School: What We Know and What We Can Do*. Cambridge, MA: Blackwell, 1993.
 90. Twemlow SW, Fonagy P, Sacco FC. An innovative psychodynamically influenced approach to reduce school violence. *J Am Acad Child Adolesc Psychiatry* 2001;40:377–9.
 91. Curry GD. Female gang involvement. In: Miller J, Mason CL, Klein MW (eds). *The Modern Gang Reader*. Los Angeles, CA: Roxbury Publishing Company, 2001:121–33.
 92. Chesney-Lind M, Shelden RG. *Girls, Delinquency, and Juvenile Justice*. Belmont, CA: West/Wadsworth, 1998.
 93. Esbensen FA, Deschenes EP, Winfree LT. Differences between gang girls and gang boys results from a multi-site survey. *Youth Soc* 1999;31:27–53.
 94. Spergel IA. *The Youth Gang Problem*. New York, NY: Oxford University Press, 1995.
 95. Moore JW, Hagedorn J. What happens to the girls in the gang? In: Huff CR (ed). *Gangs in America*, 2nd edition. Thousand Oaks, CA: Sage Publications, 1996:205–18.
 96. Hawkins JD, Herronkohl T, Farrington DP, et al. A review of predictors of youth violence. In: Loeber R, Farrington DP (eds). *Serious and Violent Juvenile Offenders: Risk Factors and Successful Interventions*. Thousand Oaks, CA: Sage Publications, 1998:106–47.
 97. Elliott D, Huizinga D, Menard S. *Multiple Problem Youth: Delinquency, Substance Use and Mental Health Problems*. New York, NY: Springer-Verlag, 1989.
 98. Halliday-Boykins CA, Graham S. At both ends of the gun: Testing the relationship between community violence exposure and youth violent behavior. *J Abnorm Child Psychol* 2001;29:383–402.
 99. Centers for Disease Control and Prevention. *CDC Surveillance Summaries, Data for 2001: June 28, 2002*. *MMWR* 2002;51(No. SS-4).
 100. Callahan CM, Rivara F. Urban high school youth and handguns. A school-based survey. *JAMA* 1992;267:3038–42.
 101. Webster DW, Gainer PS, Champion HR. Weapon carrying among inner-city junior high school students: Defensive behavior versus aggressive delinquency. *Am J Public Health* 1993;83:1604–8.
 102. Pittel EM. How to take a weapons history: interviewing children at risk for violence at school. *J Am Acad Child Adolesc Psychiatry* 1998;37:1100–2.
 103. Pittel EM. Observation, interview, and mental status assessment (OIM): Violent. In: Noshpitz JD, Harrison S, Eth S (eds). *Handbook of Child and Adolescent Psychiatry: Vol. V, Clinical Assessment and Intervention Planning*. New York, NY: John Wiley & Sons, Inc., 1998:483–95.
 104. Volavka J. *Violence and Psychoactive Substance Abuse in Neurobiology of Violence*, 2nd edition. Washington, DC: APPI, 2002, 197–218.
 105. Bell C, Gamm S, Ballas P, et al. Strategies for the prevention of youth violence in Chicago public schools. In: Saffi M, Saffi SL (eds). *School Violence: Assessment, Management, Prevention*. Washington, DC: American Psychiatric Press, 2001:251–72.
 106. Brook JS, Whiteman M, Finch SJ, et al. Aggression, intrapsychic distress, and drug use: Antecedent and intervening processes. *J Am Acad Child Adolesc Psychiatry* 1995;34:1076–84.
 107. Lipsey MW, Derzon JH. Predictors of violent or serious delinquency in adolescence and early adulthood: A synthesis of longitudinal research. In: Loeber R, Farrington DP (eds). *Serious and Violent Juvenile Offenders: Risk Factors and Successful Interventions*. Thousand Oaks, CA: Sage Publications, 1998:86–105.
 108. Herrenkohl TI, Maguin E, Hill KG, et al. Developmental risk factors for youth violence. *J Adolesc Health* 2000;26:176–86.
 109. Olds D, Henderson CR Jr, Cole R, et al. Long-term effects of nurse home visitation on children's criminal and antisocial behavior: 15-year follow-up of a randomized controlled trial. *JAMA* 1998;280:1238–44.
 110. Atkins MS, Stoff DM. Instrumental and hostile aggression in childhood disruptive behavior disorders. *J Abnorm Child Psychol* 1993;21:165–78.
 111. Loeber R, Schmalzing KB. Empirical evidence for overt and covert patterns of antisocial conduct problems: A meta-analysis. *J Abnorm Child Psychol* 1985;13:337–53.
 112. Lahey BB, Loeber R, Hart EL, et al. Four-year longitudinal study of conduct disorders in boys: Patterns and predictors of persistence. *J Abnorm Psychol* 1995;104:83–93.
 113. Moffitt TE. Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychol Rev* 1993;100:674–701.
 114. Simons RL, Wu C, Conger RD, et al. Two routes to delinquency: differences between early and late starters in the impact of parenting and deviant peers. *Criminology* 1994;32:247–76.
 115. Farrington DP. Childhood, adolescent, and adult features of violent males. In: Huesmann LR (ed). *Aggressive Behavior: Current Perspectives*. New York, NY: Plenum Press, 1994:215–40.
 116. Loeber R, Hay DF. Key issues in the development of aggression and violence from childhood to early adulthood. *Annu Rev Psychol* 1997;48:371–410.
 117. Tolan PH, Thomas P. The implications of age of onset for delinquency risk: II. Longitudinal data. *J Abnorm Child Psychol* 1995;23:157–81.
 118. Tolan PH, Gorman-Smith D, Huesmann LR, et al. Assessment of family relationship characteristics: A measure to explain risk for antisocial behavior and depression in youth. *Psychol Assess* 1997;9:212–23.
 119. Winnicott DW. *Deprivation and Delinquency*. London, UK: Tavistock/Routledge, 1984.
 120. Aichhorn A. *Wayward Youth*. Vienna, Austria: Internationaler Psychoanalytischer Verlag, 1925.
 121. Dodge KA. The structure and function of reactive and proactive aggression. In: Pepler DJ, Rubin KH (eds). *The Development and Treatment of Childhood Aggression*. Hillsdale, NJ: Lawrence Erlbaum, 1991:210–8.
 122. Coccaro EF, Siever LJ, Klar HM, et al. Serotonergic studies in patients with affective and personality disorders: Correlates with suicidal and impulsive aggressive behavior. *Arch Gen Psychiatry* 1989;46:587–99.
 123. Kruesi MJP, Rapoport JL, Hamburger S, et al. Cerebrospinal fluid, monoamine metabolites, aggression, and impulsivity with disruptive behavior disorders of children and adolescents. *Arch Gen Psychiatry* 1990;47:419–26.
 124. Lewis DO. From abuse to violence: Psychophysiological consequences of maltreatment. *J Am Acad Child Adolesc Psychiatry* 1992;31:383–91.
 125. Siegel A, Schubert K, Shaikh MB. Neurochemical mechanisms underlying amygdaloid modulation of aggressive behavior in the cat. *Aggress Behav* 1995;21:49–62.

126. Eichelman B. Animal and evolutionary models of impulsive aggression. In: Hollander E, Stein DJ (eds). *Impulsivity and Aggression*. New York, NY: John Wiley & Sons, 1995:59–90.
127. Vitiello B, Behar D, Hunt J, et al. Subtyping aggression in children and adolescents. *J Neuropsychiatry Clin Neurosci* 1990;2:189–92.
128. Connor DF, Steingard RJ, Anderson JJ, et al. Gender differences in reactive and proactive aggression. *Child Psychiatry Hum Dev* 2003;33:279–94.
129. Kazdin AE. *Conduct Disorders in Childhood and Adolescence*, 2nd edition. Thousand Oaks, CA: Sage Publications, 1996.
130. Kazdin AE. Practitioner review: Psychosocial treatments for conduct disorder in children. *J Child Psychol Psychiatry* 1997;38:161–78.
131. Pulkkinen L. Proactive and reactive aggression in early adolescence as precursors to anti and prosocial behavior in young adults. *Aggress Behav* 1996;22:241–57.
132. Verlinden S, Hersen M, Thomas J. Risk factors in school shootings. *Clin Psychol Rev* 2000;20:3–56.
133. Fein RA, Vossekuil B. *Threat Assessment: An Approach to Prevent Targeted Violence*. Washington, DC: National Institute of Justice Research in Action, U. S. Department of Justice, 1995.
134. Kazdin A, Bass D, Siegel T, et al. Cognitive-behavioral therapy and relationship therapy in the treatment of children referred for antisocial behavior. *J Consult Clin Psychol* 1989; 57:522–35.
135. Kazdin AE, Wassell G. Therapeutic changes in children, parents, and families resulting from treatment of children with conduct problems. *J Am Acad Child Adolesc Psychiatry* 2000;39:414–20.
136. Webster-Stratton C, Hammond M. Treating children with early-onset conduct problems: A comparison of child and parent training interventions. *J Consult Clin Psychol* 1997;65: 93–109.
137. Garbarino J. *Lost Boys: Why Our Sons Turn Violent and How We Can Save Them*. New York, NY: Free Press, 1999.
138. Malone RP, Delancy MA, Luebbert JF. A double-blind placebo-controlled study of lithium in hospitalized aggressive children and adolescents with conduct disorder. *Arch Gen Psychiatry* 2000;57:649–54.
139. Malone RP, Luebbert JF, Delaney MA, et al. Nonpharmacological response in hospitalized children with conduct disorder. *J Am Acad Child Adolesc Psychiatry* 1997;36:242–7.
140. Lindenmayer JP, Kotsaftis A. Use of sodium valproate in violent and aggressive behaviors: A critical review. *J Clin Psychiatry* 2000;61:123–8.
141. Connor DF, Steingard RJ. A clinical approach to the pharmacotherapy of aggression in children and adolescents. *Ann NY Acad Sci* 1996;794:290–307.
142. Frazier JA. Agitation and aggression. In: Martin A, Scahill L, Charney DS, Leckman JF (eds). *Pediatric Psychopharmacology: Principles and Practice*. London: Oxford University Press, 2003:671–86.
143. Biederman J, Faraone SV, Chu MP, et al. Further evidence of a bidirectional overlap between juvenile mania and conduct disorder in children. *J Am Acad Child Adolesc Psychiatry* 1999;38:468–76.
144. Puig-Antich J. Major depression and conduct disorder in prepuberty. *J Am Acad Child Adolesc Psychiatry* 1982;21: 118–28.
145. Hinshaw SP. Stimulant medication and the treatment of aggression in children with attentional deficits. *J Clin Child Psychol* 1991;15:301–12.
146. Hunt RD, Minderaa RB, Cohen DJ. Clonidine benefits children with attention deficit disorder and hyperactivity: Report of a double-blind placebo-cross-over therapeutic trial. *J Am Acad Child Adolesc Psychiatry* 1985;24:617–29.
147. Donovan SJ, Stewart JW, Nunes EV, et al. Divalproex treatment of youth with explosive temper and mood lability: A double-blind, placebo-controlled crossover design. *Am J Psychiatry* 2000;157:818–20.
148. Kovacs M, Pollock M. Bipolar disorder and comorbid conduct disorder in childhood and adolescence. *J Am Acad Child Adolesc Psychiatry* 1995;34:715–23.
149. Malone RP, Bennett DS, Luebbert JF, et al. Aggression classification and treatment response. *Psychopharm Bull* 1998;34:41–5.
150. Ambrosini PJ, Wagner KD, Biederman J, et al. Multicenter open-label sertraline study in adolescent outpatients with major depression. *J Am Acad Child Adolesc Psychiatry* 1999;38:566–72.
151. Emslie GJ, Rush AJ, Weinberg WA, et al. A double-blind, randomized, placebo-controlled trial of fluoxetine in children and adolescents with depression. *Arch Gen Psychiatry* 1997; 54:1031–7.
152. Pliszka SR, Sherman JO, Barrow MV, et al. Affective disorder in juvenile offenders: A preliminary study. *Am J Psychiatry* 2000;157:130–2.
153. Campbell M, Adams PB, Small AM, et al. Lithium in hospitalized aggressive children with conduct disorder: A double-blind and placebo-controlled study. *J Am Acad Child Adolesc Psychiatry* 1995;34:445–53.
154. Findling RL, McNamara NK, Branicky LA, et al. A double-blind pilot study of risperidone in the treatment of conduct disorder. *J Am Acad Child Adolesc Psychiatry* 2000;39:509–16.
155. Van Bellinghen M, De Troch C. Risperidone in the treatment of behavioral disturbances in children and adolescents with borderline intellectual functioning: A double-blind, placebo controlled pilot trial. *J Child Adolesc Psychopharmacol* 2001; 11:5–13.
156. Meeks J, Bernet W. *The Fragile Alliance: An Orientation to Psychotherapy of the Adolescent*. Malabar, FL: Krieger Publishing, 2001.
157. Webster-Stratton C, Spitzer A. Parenting a young child with conduct problems: New insights using qualitative methods. In: Ollendick TH, Prinz RJ (eds). *Advances in Clinical Child Psychology*, Vol 18. New York, NY: Plenum, 1996:1–62.
158. Graziano AM, Diamant DM. Parent behavioral training. An examination of the paradigm. *Behav Modif* 1992;16:3–38.
159. Borduin C. Multisystemic treatment of criminality and violence in adolescents. *J Am Acad Child Adolesc Psychiatry* 1999;38:242–9.
160. Schoenwald S, Thomas C, Henggeler S. Treatment of serious antisocial behavior. In: Scruggs TE, Mastropieri M (eds). *Advances in Learning and Behavioral Disabilities*, Vol. 10 Part B. Greenwich, CT: JAI Press, 1996:1–23.