Published in final edited form as:

Clin Pract (Lond). 2014 ; 11(5): 537–547. doi:10.2217/CPR.14.47.

Externalizing Disorders and Environmental Risk: Mechanisms of Gene-Environment Interplay and Strategies for Intervention

Diana R. Samek^a and Brian M. Hicks^b

^aDepartment of Psychology, University of Minnesota

^bDepartment of Psychiatry, University of Michigan

Summary

Though heritable, externalizing disorders have a number of robust associations with several environmental risk factors, including family, school, and peer contexts. To account for these associations, we integrate a behavioral genetic perspective with principles of a developmental cascade theory of antisocial behavior. The major environmental contexts associated with child externalizing problems are reviewed, as are the processes of gene-environment interplay underlying these associations. Throughout, we discuss implications for prevention and intervention. Three major approaches designed to reduce child externalizing behavior are reviewed. Prevention and intervention programs appear to be most successful when they target individuals or communities most at risk for developing externalizing disorders, rather than applied universally. We end by commenting on areas in need of additional research concerning environmental influences on persistent externalizing behaviors.

Keywords

Antisocial Behavior; Behavior Genetics; Developmental Theory; Environmental Risk; Externalizing Disorders; Gene-Environment Interplay

Introduction

Externalizing disorders are characterized by problematic behavior related to poor impulse-control, including rule breaking, aggression, impulsivity, and inattention. Specific child and adolescent externalizing disorders include conduct disorder (CD), oppositional defiant disorder (ODD), and attention-deficit-hyperactivity disorder (ADHD). Child and adolescent externalizing disorders are relatively common, with estimates ranging from 7–10% [1, 2], and with higher prevalent rates in males relative to females [2, 3]. Child externalizing disorders are highly predictive of impulse control disorders in adulthood including substance use disorders and antisocial personality disorder [4–7]. We review the links between externalizing disorders and environmental risk factors, framing the discussion within a

Correspondence: Address correspondence to Diana R. Samek, N218 Elliott Hall, 75 E River Road, Minneapolis, MN 55455, or to Brian M. Hicks, Department of Psychiatry, University of Michigan, 4250 Plymouth Rd, Ann Arbor, MI 48109. di.samek@gmail.com or brianhic@umich.edu.

The authors report no conflicts of interest.

developmental cascade model, wherein various person-situation transactions between dispositional characteristics and environmental contexts accumulate over time [8] and result in externalizing problems. We also review three major prevention and intervention programs aimed at reducing adolescent externalizing behavior and discuss lessons learned from the application of basic research to public policy.

A Developmental Cascade Model of Antisocial Behavior

Patterson and colleagues [8, 9] have articulated an influential theory of antisocial behavior that is a model of the behavioral contingencies between parents and children in which each “trains” the other to respond in ways that will increase and maintain a child’s aggressive and oppositional behavior while simultaneously decreasing parents’ control over such behaviors. Typically, such interactions are the result of children with a difficult or disinhibited temperament (difficult to soothe, high emotional reactivity, disinhibited behavior, and irregular in routines such as sleeping and eating)[10, 11] and parents who employ ineffective parenting practices, such as inconsistent discipline that ultimately reinforces aggressive behaviors. A common pattern is that a child acts out or demands something, parents deny the request and instead demand compliance, child protests and becomes aggressive, parent makes threats or also becomes aggressive to coerce compliance, the child escalates his or her aggression and demands, parent fails to follow through on threats and ultimately gives in to the child’s demands or fails to ensure the child terminates his or her disruptive behavior. As a result of these coercive cycles, a child learns that aggression is an effective strategy to meet his or her goals.

The behavioral strategy of aggression and defiance does not typically operate as effectively in other contexts such as in school and among peers. Teachers are less likely to tolerate defiance or acquiesce to aggressive demands. This often elicits additional aggressive, oppositional, and defiant behavior that marginalizes these children in daycare and classroom settings. Same aged-peers similarly have negative reactions to such behaviors that then result in rejection by prosocial peers. Such children also often have mild to moderate neuropsychological challenges such as lower than average IQ, deficits in working memory and executive function, learning disabilities, and problems with attention [12]. When coupled with disruptive behavior problems, these neuropsychological deficits result in academic difficulties [13]. Teachers also tend to be less motivated to work with students who exhibit disruptive or antisocial behavior, and parents are often poorly equipped to implement effective remediation.

For the aggressive child, this leads to frustration and embarrassment and disengagement from academic contexts. Failures to adapt or find a “place” in school and within a prosocial peer network often leads to depression, withdrawal, and anger in early to middle childhood. The accumulated losses resulting from these negative relationships and disengagement from the socializing agents of parents, school, and prosocial peers then pushes these children to affiliate with deviant peers in later childhood and early adolescence [14]. Such deviant peer groups establish a culture that values and reinforces norm violations [15, 16] and contributes to a dramatic escalation in antisocial behavior in adolescence including delinquency, early substance use, and precocious sexual behavior, further weakening the bonds to the

socializing influences and support networks of family, school, and prosocial peers. The accumulating interplay of environmental and person-level risk then begins to limit the available contexts for antisocial youth; a dynamic process referred to as “cascading constraints”[9]. In turn, these constraints then greatly increase the probability of poor outcomes such as school expulsion and educational disengagement, unemployment, criminal activity, and arrest.

Heritability and Gene-Environment Interplay in Externalizing Disorders

Before discussing the links and underlying mechanisms between environmental risk factors and externalizing, it is important to first note that the different externalizing disorders have a high rate of co-occurrence [2, 17] and tend to run in families. Twin and family studies have shown that parent-child similarity for externalizing is accounted for by the transmission of a general liability that increases risk for multiple disorders, rather than the transmission of disorder-specific liabilities [18, 19]. The familial resemblance (i.e., similarity among relatives) for this general externalizing liability has been estimated to be over 0.80, higher than any specific externalizing phenotype. However, the relative mix of genetic and shared environmental influences (i.e., environmental influences that contribute to similarity among relatives, in contrast to nonshared environmental influences that contribute to differences among relatives) underlying this familiarity changes over the course of development. In childhood and early adolescence, there is a substantial contribution of shared environmental influences in addition to heritable influences [1, 18], but by late adolescence and young adulthood, familial resemblance on the general externalizing liability is almost entirely due to genetic influences [20, 21]. Given the large and persistent influence of genetic factors on the development of externalizing behaviors, it is important to understand the processes that account for exposure to high risk environments, as well as how such environments may moderate inherited risk for externalizing.

Two mechanisms of gene-environment (G-E) interplay are essential to understand the ways in which environmental influences correlate and interact with genetic risk in the development of externalizing: gene-environment correlation (rGE) and gene x environment (G x E) interaction. rGE refers to the fact that exposure to environmental risk is not random, but rather is partly a function of genetically influenced traits that help shape family contexts as well as the decisions and actions people take in selecting and shaping their environments [22, 23]. Thus, while a large literature has demonstrated an association between parenting behaviors and child adjustment [24–28], a confounding factor in this theorized causal relationship is the extent to which parents and children share a genetic liability that contributes to both ineffective parenting practices and the child’s disruptive behavior. This is known as *passive* rGE [29]. *Evocative* rGE can also occur, and refers to how children evoke parental response as a consequence of their own genetically influenced traits. Thus, a child with a genetically influenced disinhibited temperament style may evoke negative reactions from parents and other adults, such that harsh discipline and ineffective parenting may be as much a consequence as cause of a child’s externalizing behavior. Finally, *active* rGE refers to a situation in which heritable characteristics contribute to the selection of environments, typically environments that are consistent with and accentuate the selection traits. This might include affiliating with peers who have similar externalizing tendencies or a desire to

go to parties where drugs and alcohol are available, providing additional contexts for further antisocial behavior and increasing conflict in the parent-child relationship.

Family Influences: Parents and Siblings

Consistent with a rGE model, behavioral genetic research has shown that the association between negativity in the parent-child relationship and the child's antisocial behavior is largely accounted for by shared genetic influences [30–32]. Thus, it appears that parenting behaviors are less a causal environmental influence on the development of externalizing problems, but rather are, in part, a result of parent's reaction towards a child's behaviors or their own genetically influenced impulse control problems that are genetically transmitted to their offspring. These findings do not suggest that parenting interventions are futile; in fact, if shared genes are linking parenting behavior and child externalizing problems, it may be even more important to intervene on those that are the most susceptible to that genetic risk, rather than universally.

In addition to rGE processes linking parenting and child externalizing, several studies have also found that G x E interactions underlie this association [33–35]. G x E interaction refers to the situation wherein the heritability or effect of genes on a trait (externalizing) is dependent on some condition of the environment (parenting). Multiple studies have now demonstrated that the genetic variance of externalizing increases in the context of greater parent-child conflict, negativity, punitive discipline, and lower parental warmth [28, 34, 35]. In contrast, warm and supportive parenting practices seem to suppress genetic risk for externalizing [36]. These findings support proposals for the widespread use of parent-training interventions to reduce externalizing disorders in childhood and adolescence [37]. Such interventions reduce the mean or absolute level of disruptive behaviors, and likely do so by altering the environment such that the context is no longer conducive to the expression of genetic risk factors. Teaching parents effective parenting skills may be particularly effective for a high-risk population (e.g., children with parents with a current or past externalizing disorder; children with a disinhibited temperament), rather than universal parental training interventions. Indeed, community-based preventions have been shown to be more effective for high-risk versus low-risk children [38, 39], a point we will return to later.

Besides the parent-child relationship, a large body of research has shown that aspects of the sibling relationship contribute to the development of externalizing disorders in childhood and adolescence [40–44]. Ineffective parenting and sibling conflict are highly correlated, and both independently predict subsequent peer difficulties and antisocial behavior [40]. Additionally, some have argued that sibling relationship difficulties and conflict in early- and middle-childhood may be an early indicator of problems likely to occur in social relationships outside the family [45–47].

There is also evidence that siblings often act as “partners in crime” and engage in antisocial activity together [41, 48], particularly when they are same-sex, of similar age, and report close relationships and mutual friends [43, 49]. Indeed, twin, adoption, and other genetically informative designs have shown that relationship factors (social closeness, contact, and

mutual friendships) account for a substantial proportion of sibling similarity in externalizing—particularly for substance use—via environmental mechanisms [50, 51]. Thus, while parental transmission of externalizing disorders operates through a common genetic liability [18, 19, 52], sibling relationship influences on child and adolescent externalizing appear to be environmental in nature. To date, there has been limited research that has investigated sibling influences in prevention and intervention of externalizing in childhood or adolescence [45], although sibling facilitation of substance use (helping younger siblings get alcohol and drugs) appears to be a key source of sibling influence on adolescent substance use [53].

Academic Difficulties and Peer Factors

In addition to the family environment, there is ample evidence that academic difficulties and rejection by prosocial peers are associated with child and adolescent externalizing problems [13, 54–57]. Longitudinal studies have shown that externalizing problems in childhood predict subsequent academic difficulties in childhood [58] and adolescence [59], over and above prior academic competence. Further, Moilanen et al. [58] demonstrated that lower academic competence in early childhood was associated with greater internalizing (anxiety and depression) problems in middle childhood, and more externalizing problems in adolescence. In addition to a main effect on externalizing behaviors, school environments also seem to have a moderating influence on genetic risk for externalizing similar to that of family contexts. Specifically, genetic influences on externalizing tend to be lower among children with higher grade point averages (GPAs) and among those who report greater academic engagement [60]. Though a number of selection factors (e.g., IQ, parental involvement) contribute to academic competence, if such contexts can be established and maintained, they can help to suppress existing genetic risk for externalizing problems. Involvement in extracurricular activities also seems to reduce risk for persistent externalizing problems [61, 62], both by providing structure and an alternative to antisocial activities, and by fostering ties to school contexts. There is limited longitudinal research on this topic; however, and none that we know of that has employed a genetically informative design.

Consistent with the sibling deviance literature, there is also overwhelming evidence that deviant peer affiliation is associated with antisocial behavior and substance use [63–66]. Moreover, deviant peer affiliation has been shown to amplify the association between harsh parental discipline and externalizing behavior [67], indicating an interaction between peer and family influences on externalizing problems in adolescence. Given the notable impact of the peers, a logical target of intervention or prevention for externalizing would be peer groups of high-risk youth. Notably, Dishion et al. [68] have shown that such interventions often reinforce problematic externalizing behaviors, in part because many high-risk youth lack adequate social skills and involvement in more prosocial activities. As such, the conversations and social activities among friends who are both antisocial tend to be focused on the past and future commission of antisocial behaviors. However, a high degree of emotional and behavioral self-regulation may buffer against deviant peer influences [69], and cognitive emotional self-regulation is likely an important component to early

externalizing interventions [70], particularly those that aim to offset risk imposed by deviant peer affiliation.

Twin and other genetically informative designs have demonstrated genetic influences on deviant peer affiliation [71, 72], which is interpreted as the consequence of active rGE processes [29]. That is, to some extent, people select friends based on their similarity on genetically influenced traits and interests, including attitudes towards norm violation such as delinquent behavior and early substance use. As a consequence, measures of deviant peer affiliation exhibit heritable variance, and the importance of genetic influences on peer selection increases from childhood into adulthood as people gain greater autonomy in selecting their social environments [71]. G x E interactions also underlie the association between peer deviance and externalizing problems, such that the genetic risk for externalizing problems increases in the context of more deviant peer groups [35, 73, 74]. While some children with externalizing problems may begin to affiliate with each other in childhood, the greater autonomy afforded adolescents results in a dramatic increase in the formation of deviant peer groups that then fuels a dramatic increase in antisocial behavior in mid-to-late adolescence [75]. Preventing or disrupting the influence of deviant peers is crucial to deflect the trajectory of persistent antisocial behavior, but accomplishing such a goal is exceedingly difficult in practice [68]. Thus, successful interventions might focus more on strengthening the parent-child or family bond, improving emotional self-regulation, and fostering school bonding earlier in development, which should help to mitigate the influence of deviant peers in adolescence.

Hicks et al. [35] recently conducted a relatively comprehensive analysis that examined G-E interplay between externalizing at age 17 and six environmental risk factors including parent-relationship problems, deviant peer affiliation, academic disengagement, and stressful life events. For each environmental variable, the genetic variance of externalizing increased in the context of greater environmental adversity, indicative of a general mechanism of G-E interplay for externalizing regardless of the specific contextual risk. The aggregate finding is demonstrated in Figure 1. Further, the environmental risk factors were all correlated, and other research has shown that people typically experience not just one but several exposures to risk factors [76]. The non-independence and heritable nature of externalizing and environmental risk then suggests that the most severe and persistent antisocial behavior is exhibited by a small group of people that carry an especially high loading of both genetic and environmental risk [75]. The likely mechanisms for this are rGE processes that lead to greater exposure to environmental risk that is compounded by G x E interactions, such that the experience of greater environmental adversity leads to the expression of more genetic vulnerabilities.

A Review of Three Major Prevention and Interventions Programs

Given the high societal cost of adult externalizing behaviors, there is a societal imperative to intervene and ideally prevent such problem behaviors before their severity rises to the level of criminality, as such behaviors have potentially life altering consequences for both perpetrators and victims. As the “cascading constraints” of persistent antisocial narrow an individual’s potential developmental landscape [9, 75], the earlier an intervention is

implemented, the greater the likelihood for success in deflecting the trajectory toward persistent antisocial behavior. Another conclusion to draw from findings reviewed here and elsewhere is that interventions and preventions for externalizing problems will be most successful if they selectively target those children at greatest risk. Key characteristics that distinguish such children include a difficult or disinhibited temperament, family history of externalizing disorders, and behavioral and academic problems in school. Key components of such interventions include parent training and support, fostering prosocial skills (e.g., practice emotional self-regulation, reduce sibling conflict and deviancy training, improve prosocial peer affiliation), and promoting school engagement and bonding (e.g., extracurricular involvement, additional instruction for those not succeeding academically). Here, we review three major programs attempting to reduce externalizing problems (for additional program reviews and long-term follow-up results, see Henggeler & Sheidow [77], Fagan [78], Patterson et al. [79]). Effect sizes for these three prevention and intervention programs have been generally small to moderate [38, 80, 81].

Multisystemic therapy (MST) [82] is based on a social ecology framework [83], and aims to intervene across multiple systemic influences at the level of the individual, family, and community, to reduce the likelihood of chronic and violent juvenile offenses. MST targets 12–17 year olds who have engaged in serious antisocial behavior (criminal felonies if prosecuted as an adult), and aims to foster a greater degree of parent-child and family bonding, mitigate the effects of deviant peers through parental supervision or involvement in prosocial peer activities, and increase engagement in school and extra-curricular activities. While the goal is to reduce risk factors and strengthen protective factors, it is done at the individual rather than universal level, as specific factors may be more relevant to some youth compared to others [84]. In general, MST has been shown to be quite effective in reducing serious juvenile offenses and substance use [80, 85], even continuing into adulthood (up to 21 years later [86, 87]). Notably, MST is a time and labor-intensive intervention administered by a team of professionals that includes a therapist, social worker, and other specialists. A therapist is on duty 24 hours a day and 7 days a week to aid in crises, performs treatments in the adolescents' home at nights and on weekends, and the treatment typically lasts 4 months. MST has been widely implemented, with MST programs operating throughout 30+ states in the United States, and some internationally [84].

Communities that Care (CTC) is a prevention program that has been shown to be successful at reducing substance use, delinquency, and violent behavior [81, 88]. However, CTC is implemented universally at schools and communities deemed at high-risk (e.g., public schools in high-crime areas [88]), rather than towards individuals. CTC is less time-intensive compared to MST, and focuses on a broader population than those with serious juvenile offenses. Several program interventions are utilized, including teacher training (5 days), child social and emotional skill development (4 hours via teachers), and parental training (7-week session). Trainings began with children in the 1st through 3rd grade, and intervention effects have been demonstrated through age 18 [88], such that students in the intervention were less likely to report violent delinquent acts, heavy drinking, risky sexual behavior than those in the control condition.

A subsequent randomized trial of 24 small towns in 7 states implemented CTC beginning in 5th grade (again, targeting schools in high crime and low income areas), and has shown that delinquent behavior and alcohol and cigarette use were lower in CTC relative to control communities at the 10th grade [81]. By the 12th grade, CTC communities were more likely to abstain from substance use, but among people that had used substances, there were no differences in the intervention arm for past-month or past-year substance use, nor were there differences in past-year delinquency or violence [89]. Results have also been partially replicated in Pennsylvania, where CTC had a significant effect on reduced delinquency and better academic performance, but had no significant effect on adolescent substance use through the 12th grade.

A final systemic prevention program we discuss is PROMoting School-community-university Partnerships to Enhance Resilience (PROSPER) [38, 90–92]. The purpose of PROSPER is to link university-based prevention researchers to elementary and secondary school personnel (counselors, curriculum directors, principals) and community providers of youth services, to implement the most efficacious prevention strategy given the school's needs. In consultation with researchers, school personnel select from a menu of prevention programs that fit best with their overall goals and populations including school-based and parenting interventions. Thus, unlike CTC and MST, neither high-risk individuals nor whole schools are selected for interventions; rather, interventions are implemented based on the specific needs of the school or community. PROSPER, however, has been shown to be more effective for high-risk versus low-risk children [38]. Other studies have also found that similar family and school interventions are more effective at reducing alcohol use among high-risk adolescents [39].

Future Perspective

While we have focused on research on the developmental cascade of antisocial behavior through adolescence, less research has evaluated the processes that contribute to the persistence versus desistence of antisocial behavior in adulthood. For example, much of the research on G-E interplay has been limited to cross-sectional findings in childhood and adolescence [1, 34, 35]. Thus, it remains unclear if such demonstrated gene-environment influences are relatively proximal in effect or have long-lasting effects through young adulthood. It will be important for future research to address the long-term impact of family, peer, and school or work environments on adult psychosocial outcomes, including how these environmental risk variables correlate and interact with person-level variables to predict persistent externalizing problems in adulthood.

Additionally, while there has been extensive work done to incorporate parent training into prevention and intervention programs aimed at reducing externalizing disorders or substance use in childhood and adolescence, there has been limited research that has incorporated siblings. Given the substantial sibling influences in child and adolescent externalizing behaviors, an important avenue of future research will be to address how sibling factors can be incorporated into prevention and intervention efforts [42, 43]. A recent program titled *Siblings are Special* (SAS)[45] has the long-term goal of reducing siblings' risk for maladjustment and substance use. SAS is implemented in a 12-week, after-school session,

teaching children emotion regulation and problem-solving skills. It will be important to evaluate the efficacy of the program as data becomes available.

Finally, there has been relatively little research that has systematically evaluated the efficacy of the timing of prevention and interventions in childhood and adolescence. While earlier is generally better, how early is necessary versus optimal? Additionally, interventions or preventions may be most effective if they occur just prior to or at the beginning of crucial developmental transitions (e.g., pubertal onset, transitioning from elementary to middle to high school). It would be useful to compare the results of a specific program (MST, CTC, PROSPER) that intervened at various age groups (e.g., age 5, 10, 11, 12, 13...) and evaluate the current and long-term outcomes of that specific program through young adulthood. We are not aware of any programs such as MST, CTC, or PROSPER that have tested and compared age-specific targeted interventions and preventions in this manner, and testing age-specific prevention and intervention programs is a likely candidate for future research.

Conclusion

Substantial evidence indicates that externalizing problems are embedded within a larger context of interacting individual and environmental influences. A largely inherited disposition towards behavioral disinhibition correlates and interacts with well-established environmental risk factors (parenting, peer, and school contexts) to influence the developmental trajectory of externalizing problems. Prevention and intervention research aimed at reducing problematic antisocial behavior has been shown to be successful, particularly programs that target high-risk individuals and populations early in development, and that focus on strengthening bonds to the socializing agents of family and school. To date, little research has incorporated siblings into prevention and intervention programs. Given the research demonstrating sibling similarity in adolescent substance use and externalizing problems, this area seems ripe for future prevention research to incorporate. Additionally, more research is needed that addresses the long-term impact of child and adolescent environmental contexts on adult outcomes, as well as the long-term influence of child and adolescent prevention and intervention programs on adult outcomes.

Acknowledgments

This work was supported by United States Public Health Service grants R01 DA034606 and K01 DA025868 to Brian M. Hicks from the National Institute on Drug Abuse.

References

1. Burt SA, Krueger RF, McGue M, Iacono WG. Sources of covariation among attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder: The importance of shared environment. *Journal of Abnormal Psychology*. 2001; 110(4):516–525. [PubMed: 11727941]
2. Kessler RC, Berglund P, Demler O, Jin R, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*. 2005; 62(6):593–602. [PubMed: 15939837]
3. King SM, Iacono WG, McGue M. Childhood externalizing and internalizing psychopathology in the prediction of early substance use. *Addiction*. 2004; 99(12):1548–1559. [PubMed: 15585046]

4. Babinski LM, Hartsough CS, Lambert NM. Childhood conduct problems, hyperactivity-impulsivity, and inattention as predictors of adult criminal activity. *Journal of Child Psychology and Psychiatry and Allied Disciplines*. 1999; 40(3):347–355.
5. Biederman J, Petty CR, Dolan C, et al. The long-term longitudinal course of oppositional defiant disorder and conduct disorder in ADHD boys: findings from a controlled 10-year prospective longitudinal follow-up study. *Psychological Medicine*. 2008; 38(7):1027–1036. [PubMed: 18205967]
6. Elkins IJ, McGue M, Iacono WG. Prospective effects of attention-deficit/hyperactivity disorder, conduct disorder, and sex on adolescent substance use and abuse. *Archives of General Psychiatry*. 2007; 64(10):1145–1152. [PubMed: 17909126]
7. Fergusson DM, Boden JM, Horwood LJ. The developmental antecedents of illicit drug use: Evidence from a 25-year longitudinal study. *Drug and Alcohol Dependence*. 2008; 96(1–2):165–177. [PubMed: 18423900]
- **8. Patterson GR, DeBaryshe BD, Ramsey E. A Developmental Perspective on Antisocial-Behavior. *American Psychologist*. 1989; 44(2):329–335. Patterson et al. is a landmark paper that proposed a developmental model on antisocial behavior based on coercion theory; Granic and Patterson is a review and elaboration on this line of research that integrates dynamics systems theory. This work was used to guide the current review. Ineffective parenting is proposed to lead to parent-child aggressive interactions, subsequent peer and school problems, and subsequent deviant peer affiliation. [PubMed: 2653143]
- **9. Granic I, Patterson GR. Toward a comprehensive model of antisocial development: A dynamic systems approach. *Psychological Review*. 2006; 113(1):101–131. Patterson et al. is a landmark paper that proposed a developmental model on antisocial behavior based on coercion theory; Granic and Patterson is a review and elaboration on this line of research that integrates dynamics systems theory. This work was used to guide the current review. Ineffective parenting is proposed to lead to parent-child aggressive interactions, subsequent peer and school problems, and subsequent deviant peer affiliation. [PubMed: 16478303]
10. Thomas, A.; Chess, S. The New York Longitudinal Study: From infancy to early adult life. In: Plomin, RJD., editor. *The study of temperament: Changes, continuities, and challenges*. Erlbaum; Hillsdale, NJ: 1986. p. 39-52.
11. Caspi A, Silva PA. TEMPERAMENTAL QUALITIES AT AGE-3 PREDICT PERSONALITY-TRAITS IN YOUNG ADULTHOOD - LONGITUDINAL EVIDENCE FROM A BIRTH COHORT. *Child Development*. 1995; 66(2):486–498. [PubMed: 7750379]
12. Morgan AB, Lilienfeld SO. A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*. 2000; 20(1): 113–136. [PubMed: 10660831]
13. Hinshaw SP. Externalizing Behavior Problems and Academic Underachievement in Childhood and Adolescence - Causal Relationships and Underlying Mechanisms. *Psychological Bulletin*. 1992; 111(1):127–155. [PubMed: 1539086]
14. Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin*. 1992; 112(1):64–105. [PubMed: 1529040]
15. Dishion TJ, Spracklen KM, Andrews DW, Patterson GR. Deviancy training in male adolescent friendships. *Behavior Therapy*. 1996; 27(3):373–390.
16. Dishion TJ, Andrews DW, Crosby L. ANTISOCIAL BOYS AND THEIR FRIENDS IN EARLY ADOLESCENCE - RELATIONSHIP CHARACTERISTICS, QUALITY, AND INTERACTIONAL PROCESS. *Child Development*. 1995; 66(1):139–151. [PubMed: 7497821]
17. Kadesjo B, Gillberg C. The comorbidity of ADHD in the general population of Swedish school-age children. *Journal of Child Psychology and Psychiatry and Allied Disciplines*. 2001; 42(4):487–492.
18. Bornovalova MA, Hicks BM, Iacono WG, McGue M. Familial transmission and heritability of childhood disruptive disorders. *Am J Psychiatry*. 2010; 167(9):1066–1074. [PubMed: 20634367]
19. Hicks BM, Krueger RF, Iacono WG, McGue M, Patrick CJ. Family transmission and heritability of externalizing disorders - A twin-family study. *Archives of General Psychiatry*. 2004; 61(9):922–928. [PubMed: 15351771]

20. Young SE, Rhee SH, Stallings MC, Corley RP, Hewitt JK. Genetic and environmental vulnerabilities underlying adolescent substance use and problem use: General or specific? *Behav Genet.* 2006; 36(4):603–615. [PubMed: 16619135]
21. Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology.* 2002; 111(3):411–424. [PubMed: 12150417]
22. Dick DM. Gene-Environment Interaction in Psychological Traits and Disorders. *Annual Review of Clinical Psychology.* 2011; 7:383–409.
23. Moffitt TE. The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. *Psychological Bulletin.* 2005; 131(4):533–554. [PubMed: 16060801]
24. Buehler C, Gerard JM. Marital conflict, ineffective parenting, and children's and adolescents' maladjustment. *Journal of Marriage and Family.* 2002; 64(1):78–92.
25. Kaczynski KJ, Lindahl KM, Malik NM, Laurenceau JP. Marital conflict, maternal and paternal parenting, and child adjustment: A test of mediation and moderation. *Journal of Family Psychology.* 2006; 20(2):199–208. [PubMed: 16756395]
26. Pettit GS, Bates JE, Dodge KA. Supportive parenting, ecological context, and children's adjustment: A seven-year longitudinal study. *Child Development.* 1997; 68(5):908–923.
27. Steinberg L. We know some things: Parent-adolescent relationships in retrospect and prospect. *Journal of Research on Adolescence.* 2001; 11(1):1–19.
28. Snyder J, Cramer A, Afsar J, Patterson GR. The contributions of ineffective discipline and parental hostile attributions of child misbehavior to the development of conduct problems at home and school. *Developmental Psychology.* 2005; 41(1):30–41. [PubMed: 15656735]
29. Scarr S, McCartney K. How People Make Their Own Environments - a Theory of Genotype-Environment Effects. *Child Development.* 1983; 54(2):424–435. [PubMed: 6683622]
30. Burt SA, Krueger RF, McGue M, Iacono W. Parent-child conflict and the comorbidity among childhood externalizing disorders. *Archives of General Psychiatry.* 2003; 60(5):505–513. [PubMed: 12742872]
31. Neiderhiser JM, Reiss D, Hetherington EM, Plomin R. Relationships between parenting and adolescent over time: Genetic and environmental contributions. *Developmental Psychology.* 1999; 35(3):680–692. [PubMed: 10380859]
32. Pike A, McGuire S, Hetherington EM, Reiss D, Plomin R. Family environment and adolescent depressive symptoms and antisocial behavior: A multivariate genetic analysis. *Developmental Psychology.* 1996; 32(4):590–603.
33. Button TMM, Lau JYF, Maughan B, Eley TC. Parental punitive discipline, negative life events and gene-environment interplay in the development of externalizing behavior. *Psychological Medicine.* 2008; 38(1):29–39. [PubMed: 17803832]
34. Feinberg ME, Button TMM, Neiderhiser JM, Reiss D, Hetherington EM. Parenting and adolescent antisocial behavior and depression - Evidence of genotype x parenting environment interaction. *Archives of General Psychiatry.* 2007; 64(4):457–465. [PubMed: 17404122]
- **35. Hicks BM, South SC, Dirago AC, Iacono WG, McGue M. Environmental Adversity and Increasing Genetic Risk for Externalizing Disorders. *Archives of General Psychiatry.* 2009; 66(6):640–648. This comprehensive paper evaluated relationships between adolescent externalizing disorders and several measures of environmental risk and found overwhelming evidence for gene-environment interaction, such that genetic risk for externalizing was greater under more adverse conditions of environmental context than less adverse conditions (see Figure 1). [PubMed: 19487629]
36. Johnson W. Genetic and environmental influences on behavior: capturing all the interplay. *Psychol Rev.* 2007; 114(2):423–440. [PubMed: 17500633]
37. Forgatch MS, Patterson GR, Gewirtz AH. Looking Forward: The Promise of Widespread Implementation of Parent Training Programs. *Perspectives on Psychological Science.* 2013; 8(6): 682–694. [PubMed: 24443650]
- *38. Spoth R, Redmond C, Shin C, Greenberg M, Clair S, Feinberg M. Substance-use outcomes at 18 months past baseline - The PROSPER community-university partnership trial. *American Journal*

- of Preventive Medicine. 2007; 32(5):395–402. This paper demonstrated that the PROSPER prevention program was associated with lower levels of substance-use 18 months after completion of the program, particularly for the high-risk students. [PubMed: 17478265]
39. Verdurmen JE, Koning IM, Vollebergh WA, Van Den Eijnden RJ, Engels RC. Risk moderation of a parent and student preventive alcohol intervention by adolescent and family factors: A cluster randomized trial. *Prev Med*. 2013
 40. Bank L, Burraston B, Snyder J. Sibling conflict and ineffective parenting as predictors of adolescent boys' antisocial behavior and peer difficulties: Additive and interactional effects. *Journal of Research on Adolescence*. 2004; 14(1):99–125.
 - *41. Bullock BM, Dishion TJ. Sibling collusion and problem behavior in early adolescence: Toward a process model for family mutuality. *Journal of Abnormal Child Psychology*. 2002; 30(2):143–153. This paper showed that sibling collusion, a process in which siblings form a coalition that undermines parenting and promotes deviance, is strongly related to adolescent antisocial behavior, above and beyond the influence of peer deviance. [PubMed: 12002395]
 42. Defoe IN, Keijsers L, Hawk ST, et al. Siblings versus parents and friends: longitudinal linkages to adolescent externalizing problems. *Journal of Child Psychology and Psychiatry*. 2013; 54(8):881–889. [PubMed: 23398022]
 43. Rowe DC, Gulley BL. Sibling Effects on Substance Use and Delinquency. *Criminology*. 1992; 30(2):217–233.
 44. Snyder J, Bank L, Burraston B. The consequences of antisocial behavior in older male siblings for younger brothers and sisters. *Journal of Family Psychology*. 2005; 19(4):643–653. [PubMed: 16402880]
 45. Feinberg ME, Solmeyer AR, Mchale SM. The Third Rail of Family Systems: Sibling Relationships, Mental and Behavioral Health, and Preventive Intervention in Childhood and Adolescence. *Clinical Child and Family Psychology Review*. 2012; 15(1):43–57. [PubMed: 22105663]
 46. Lewin LM, Hops H, Davis B, Dishion TJ. Multimethod Comparison of Similarity in School Adjustment of Siblings and Unrelated Children. *Developmental Psychology*. 1993; 29(6):963–969.
 47. Stormshak EA, Bellanti CJ, Bierman KL. The quality of sibling relationships and the development of social competence and behavioral control in aggressive children. *Developmental Psychology*. 1996; 32(1):79–89.
 48. Slomkowski C, Rende R, Conger KJ, Simons RL, Conger RD. Sisters, brothers, and delinquency: Evaluating social influence during early and middle adolescence. *Child Development*. 2001; 72(1):271–283. [PubMed: 11280484]
 49. Whiteman SD, Jensen AC, Maggs JL. Similarities in Adolescent Siblings' Substance Use: Testing Competing Pathways of Influence. *Journal of Studies on Alcohol and Drugs*. 2013; 74(1):104–113. [PubMed: 23200155]
 50. Rende R, Slomkowski C, Lloyd-Richardson E, Niaura R. Sibling effects on substance use in adolescence: Social contagion and genetic relatedness. *Journal of Family Psychology*. 2005; 19(4):611–618. [PubMed: 16402876]
 51. Slomkowski C, Rende R, Novak S, Lloyd-Richardson E, Niaura R. Sibling effects on smoking in adolescence: evidence for social influence from a genetically informative design. *Addiction*. 2005; 100(4):430–438. [PubMed: 15784052]
 52. Hicks BM, Foster KT, Iacono WG, McGue M. Genetic and Environmental Influences on the Familial Transmission of Externalizing Disorders in Adoptive and Twin Offspring. *Jama Psychiatry*. 2013; 70(10):1076–1083. [PubMed: 23965950]
 53. McGue, M.; Iacono, WG. Siblings and the socialization of adolescent deviance: An adoption study approach. In: McCartney, K.; Weinberg, R., editors. *Experience and Development: A festschrift to honor Sandra W Scarr*. Taylor & Francis; London, UK: 2009. p. 179-202.
 54. Arnold DH. Co-occurrence of externalizing behavior problems and emergent academic difficulties in young high-risk boys: A preliminary evaluation of patterns and mechanisms. *Journal of Applied Developmental Psychology*. 1997; 18(3):317–330.

55. Kupersmidt JB, Burchinal M, Patterson CJ. Developmental Patterns of Childhood Peer Relations as Predictors of Externalizing Behavior Problems. *Development and Psychopathology*. 1995; 7(4): 825–843.
56. Laird RD, Jordan KY, Dodge KA, Pettit GS, Bates JE. Peer rejection in childhood, involvement with antisocial peers in early adolescence, and the development of externalizing behavior problems. *Development and Psychopathology*. 2001; 13(2):337–354. [PubMed: 11393650]
57. Prinstein MJ, La Greca AM. Childhood peer rejection and aggression as predictors of adolescent girls' externalizing and health risk behaviors: A 6-year longitudinal study. *Journal of Consulting and Clinical Psychology*. 2004; 72(1):103–112. [PubMed: 14756619]
58. Moilanen KL, Shaw DS, Maxwell KL. Developmental cascades: Externalizing, internalizing, and academic competence from middle childhood to early adolescence. *Development and Psychopathology*. 2010; 22(3):635–653. [PubMed: 20576184]
59. Masten AS, Roisman GI, Long JD, et al. Developmental cascades: Linking academic achievement and externalizing and internalizing symptoms over 20 years. *Developmental Psychology*. 2005; 41(5):733–746. [PubMed: 16173871]
60. Johnson W, McGue M, Iacono WG. School Performance and Genetic and Environmental Variance in Antisocial Behavior at the Transition From Adolescence to Adulthood. *Developmental Psychology*. 2009; 45(4):973–987. [PubMed: 19586174]
61. Mahoney JL, Stattin H. Leisure activities and adolescent antisocial behavior: The role of structure and social context. *Journal of Adolescence*. 2000; 23(2):113–127. [PubMed: 10831137]
62. Harrison PA, Narayan G. Differences in behavior, psychological factors, and environmental factors associated with participation in school sports and other activities in adolescence. *Journal of School Health*. 2003; 73(3):113–120. [PubMed: 12677730]
63. Barnes GM, Hoffman JH, Welte JW, Farrell MP, Dintcheff BA. Effects of parental monitoring and peer deviance on substance use and delinquency. *Journal of Marriage and Family*. 2006; 68(4): 1084–1104.
64. Dekovic M, Wissink IB, Meijer AM. The role of family and peer relations in adolescent antisocial behaviour: comparison of four ethnic groups. *Journal of Adolescence*. 2004; 27(5):497–514. [PubMed: 15475043]
65. Kim JE, Hetherington EM, Reiss D. Associations among family relationships, antisocial peers, and adolescents' externalizing behaviors: Gender and family type differences. *Child Development*. 1999; 70(5):1209–1230. [PubMed: 10546341]
66. Stormshak EA, Comeau CA, Shepard SA. The relative contribution of sibling deviance and peer deviance in the prediction of substance use across middle childhood. *Journal of Abnormal Child Psychology*. 2004; 32(6):635–649. [PubMed: 15648530]
67. Lansford JE, Criss MM, Pettit GS, Dodge KA, Bates JE. Friendship quality, peer group affiliation, and peer antisocial behavior as moderators of the link between negative parenting and adolescent externalizing behavior. *Journal of Research on Adolescence*. 2003; 13(2):161–184. [PubMed: 20209019]
68. Dishion TJ, McCord J, Poulin F. When interventions harm - Peer groups and problem behavior. *American Psychologist*. 1999; 54(9):755–764. [PubMed: 10510665]
69. Gardner TW, Dishion TJ, Connell AM. Adolescent self-regulation as resilience: Resistance to antisocial behavior within the deviant peer context. *Journal of Abnormal Child Psychology*. 2008; 36(2):273–284. [PubMed: 17899361]
70. Fonagy P, Target M. Early intervention and the development of self-regulation. *Psychoanalytic Inquiry*. 2002; 22(3):307–335.
71. Kendler KS, Jacobson KC, Gardner CO, Gillespie N, Aggen SA, Prescott CA. Creating a social world - A developmental twin study of peer-group deviance. *Archives of General Psychiatry*. 2007; 64(8):958–965. [PubMed: 17679640]
72. Samek DR, Keyes MA, Iacono WG, McGue M. Peer Deviance, Alcohol Expectancies, and Adolescent Alcohol Use: Explaining Shared and Nonshared Environmental Effects Using an Adoptive Sibling Pair Design. *Behav Genet*. 2013; 43(4):286–296. [PubMed: 23644917]

73. Agrawal A, Balasubramanian S, Smith EK, et al. Peer substance involvement modifies genetic influences on regular substance involvement in young women. *Addiction*. 2010; 105(10):1844–1853. [PubMed: 20569232]
74. Fowler T, Shelton K, Lifford K, et al. Genetic and environmental influences on the relationship between peer alcohol use and own alcohol use in adolescents. *Addiction*. 2007; 102(6):894–903. [PubMed: 17523983]
75. Moffitt TE. Adolescence-limited and life course persistent antisocial behavior: A developmental taxonomy. *Psychological Review*. 1993; 100(4):674–701. [PubMed: 8255953]
76. Appleyard K, Egeland B, Van Dulmen MHM, Sroufe LA. When more is not better: the role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry*. 2005; 46(3):235–245. [PubMed: 15755300]
77. Henggeler SW, Sheidow AJ. Empirically supported family-based treatments for conduct disorder and delinquency in adolescents. *J Marital Fam Ther*. 2012; 38(1):30–58. [PubMed: 22283380]
78. Fagan AA. Family-Focused Interventions to Prevent Juvenile Delinquency: A Case Where Science and Policy Can Find Common Ground. *Criminology & Public Policy*. 2013; 12(4):1–34.
79. Patterson GR, Forgatch MS, Degarmo DS. Cascading effects following intervention. *Development and Psychopathology*. 2010; 22(4):949–970. [PubMed: 20883592]
- *80. Curtis N, Ronan KR, Borduin CM. Multisystemic treatment: A meta-analysis of outcome studies. *J Fam Psychol*. 2004; 18(3):411–419. This paper conducts a meta-analysis of Multisystemic treatment outcomes, reporting an average effect size of $d = .55$. [PubMed: 15382965]
- *81. Hawkins JD, Oesterle S, Brown EC, et al. Sustained Decreases in Risk Exposure and Youth Problem Behaviors After Installation of the Communities That Care Prevention System in a Randomized Trial. *Archives of Pediatrics & Adolescent Medicine*. 2012; 166(2):141–148. This paper demonstrates the Communities that Care prevention program (a randomized community trial) was associated with lower levels of substance use and antisocial behavior six years after the completion of the program (Grade 10). [PubMed: 21969362]
82. Henggeler, SW.; Schoenwald, SK.; Borduin, CM.; Rowland, MD.; Cunningham, PB. Multisystemic therapy for antisocial behavior in children and adolescents. The Guilford Press; New York, NY: 2009.
83. Brofenbrenner, U. The ecology of human development: Experiments by design and nature. Harvard University Press; Cambridge, MA: 1979.
84. Henggeler SW. Efficacy studies to large-scale transport: the development and validation of multisystemic therapy programs. *Annu Rev Clin Psychol*. 2011; 7:351–381. [PubMed: 21443449]
85. Henggeler SW. Efficacy studies to large-scale transport: The development and validation of multisystemic therapy programs. *Annu Rev Clin Psycho*. 2011; 7:351–381.
86. Sawyer AM, Borduin CM. Effects of multisystemic therapy through midlife: A 21.9 year follow-up to a randomized clinical trial with serious and violent juvenile offenders. *J Consult Clin Psych*. 2011; 79(5):643–652.
87. Schaeffer CM, Borduin CM. Long-term follow-up to a randomized clinical trial of multisystemic therapy with serious and violent juvenile offenders. *J Consult Clin Psych*. 2005; 73(3):445–453.
88. Hawkins JD, Catalano RF, Kosterman R, Abbott R, Hill KG. Preventing adolescent health-risk behaviors by strengthening protection during childhood. *Archives of Pediatrics & Adolescent Medicine*. 1999; 153(3):226–234. [PubMed: 10086398]
89. Hawkins JD, Oesterle S, Brown EC, Abbott RD, Catalano RF. Youth Problem Behaviors 8 Years After Implementing the Communities That Care Prevention System: A Community-Randomized Trial. *JAMA Pediatr*. 2013
90. Spoth R, Greenberg M, Bierman K, Redmond C. PROSPER community-university partnership model for public education systems: Capacity-building for evidence-based, competence-building prevention. *Prevention Science*. 2004; 5(1):31–39. [PubMed: 15058910]
91. Spoth R, Guyll M, Lillehoj CJ, Redmond C, Greenberg M. Prosper study of evidence-based intervention implementation quality by community-university partnerships. *Journal of Community Psychology*. 2007; 35(8):981–999. [PubMed: 20376336]
92. Spoth R, Guyll M, Redmond C, Greenberg M, Feinberg M. Six-Year Sustainability of Evidence-Based Intervention Implementation Quality by Community-University Partnerships: The

PROSPER Study. American Journal of Community Psychology. 2011; 48(3–4):412–425.
[PubMed: 21394561]

Practice points

- Externalizing behaviors (rule breaking, aggression) and disorders (Conduct Disorder, Oppositional Defiant Disorder, and Attention-Deficit-Hyperactivity Disorder) are relatively common, more prevalent in boys than girls, and are highly predictive of impulse control disorders in adulthood.
- A combination of a difficult child temperament and ineffective parenting practices is theorized to lead children to learn to interact with parents in aggressive and defiant ways. This aggressive interaction strategy generalizes to those within and outside of the family, leading to increased sibling conflict, rejection by prosocial peers, and academic problems in early childhood. This developmental cascade continues by leading children to affiliate with deviant rather than prosocial peers, which reinforces antisocial attitudes and persistent antisocial behavior through adolescence.
- Externalizing disorders are substantially heritable, particularly by late adolescence.
- Gene-environment interaction and correlation is evident for child and adolescent externalizing disorders, such that genetic risk tends to increase in the context of greater environmental risk, and that children tend to select into environments (e.g., deviant peer groups) or are exposed to environments (e.g., ineffective parenting practices) that are correlated with the genetic influences on externalizing behaviors.
- Gene-environment correlation and interaction is evident in the relationship between ineffective parenting and child externalizing problems, suggesting common genetic influences on both ineffective parenting and child externalizing, and that greater environmental risk amplifies genetic risk for externalizing disorders. This suggests parental training interventions may be particularly important for high-risk children (e.g., children of parents with current or past externalizing disorders).
- Siblings are also highly influential to the development of child externalizing behavior. Sibling conflict may be an early indicator of social problems likely to occur outside the family. Siblings may act as “partners in crime” and simultaneously reinforce one another’s antisocial behavior. Behavioral genetic research has shown that these processes are highly influenced by environmental, rather than genetic factors, suggesting that siblings may be important socializing agents of antisocial behavior in childhood and adolescence.
- Longitudinal research shows that rejection by prosocial peers and experiences of academic difficulties is widely evident for children with externalizing disorders. There is some evidence that maintaining a high GPA during high-school may offset genetic risk for externalizing problems, and that involvement in extra-curricular activities may reduce risk for persistent externalizing problems, although this evidence is mostly cross-sectional in design.

- There is overwhelming evidence that deviant peer affiliation is associated with increased risk of externalizing problems via gene-environment correlation processes (i.e., children select friends based on common interests and traits). Gene-environment interaction is also evident in this relationship, suggesting an amplifying effect of deviant peer affiliation beyond selection effects.
- Three major programs are reviewed, including Multisystemic Therapy (MST), Communities that Care (CTC), and Promoting-School-Community-University-Partnerships to Enhance Resilience (PROSPER). In general, these programs have small to moderate effect sizes on reducing externalizing problems and substance use in adolescence, and seem to be particularly effective when targeting children or communities most at risk for developing externalizing disorders.
- In the next 5–10 years, we expect to see more research that extends the developmental cascade hypothesis past adolescence, as well as longitudinal research that aims to evaluate what may disrupt such a cascade. Additionally, we expect to see more prevention and intervention programs incorporate siblings, as well as evaluations of age-specific interventions.
- Substantial evidence supports a developmental cascade theory of antisocial behavior in childhood and adolescence. Behavior genetic research has shown complex processes of gene-environment interplay evident in the relationship between environmental risk factors and a liability towards externalizing. Longitudinal research is needed that evaluates the long-term influences of gene-environment interactions and child and adolescent prevention and intervention programs.

Figure 1. Gene-Environment Interaction: Genetic Variance in Adolescent Externalizing Disorders Varies as a Function of Environmental Risk

This figure represents the overall pattern of findings described in Hicks et al. [35]. A = additive genetic influence, C = shared environmental influences, E = nonshared environmental influences, SD = Standard Deviation. Environmental risk was evaluated in terms of mother- and father-child relationship problems, antisocial and prosocial peer affiliation, academic achievement and engagement, and a composite of stressful life events (e.g., parental divorce). Across all environmental risk measures, genetic variance of adolescent externalizing disorders was greater in the context of greater environmental risk, and lower in the context of more protective environments. This figure shows findings in the aggregate across environmental risk measures.