Peer effects and molecular genetics in adolescent antisocial behavior

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Adolescent antisocial behavior is detrimental to individual development and poses a huge burden to society. Historically, first nature, later nurture were held responsible for the occurrence of antisocial behavior (Laub, 2004) but contemporary research embraces genetic and social factors and focuses particularly on their interplay in trying to understand individual variation in engaging in aggression, delinquency, and substance use. What does this new orientation teach us with regard to the peer environment? Aiming to elucidate how molecular genetics and peer environment function together in increasing the risk for antisocial behavior, the current chapter summarizes findings on antisocial and risk behaviors and discusses challenges for future studies.

Most past research into antecedents of antisocial behavior has attributed at least some influence to deviant peer affiliation which makes sense given that youths spend increasingly more time interacting with peers (Larson & Richards, 1991) and are more vulnerable to peer influence than children or adults (Brechwald & Prinstein, 2011; Steinberg & Morris, 2001). Studies provide ample evidence that deviant peer affiliation increases own antisocial behavior, even after accounting for peer-related confounders such as peer rejection and family-related factors (for reviews see Dishion & Tipsord, 2011; Monahan, Steinberg, & Cauffman, 2009). In fact, one of the strongest predictors of antisocial behavior in adolescence is peer behavior (Van Lier et al., 2007). Not only are adolescents influenced by their environment, they also shape their environment and actively decide whom they select as friends. Such selection effects are often driven by behavioral similarity so that antisocial adolescents will "flock together". Although studies have more often examined socialization effects, both processes are acknowledged in this chapter.

Importantly, adolescents are not uniformly vulnerable to deviant peer influence and individual factors also affect selection of friends (Caspi & Moffitt, 2006; Magnusson, 1985; Rutter, 1997). Prior research on this person-peer environment interplay has focused on

biological and psychological factors such as pubertal stage and temperament, but more recent studies reflect a growing interest in the role of specific genes. Building upon twin studies that suggested already for quite some time that heritability is important in antisocial behavior (Ferguson, 2010; Moffitt, 2005; Rhee & Waldman, 2002), scientific and technical advances now allow us to examine whether so-called candidate genes are linked to antisocial behavior. Candidate gene studies do not necessarily try to establish a main association between a specific gene and an outcome but assume gene-environment correlations (rGE) and gene-environment interactions (GxE).

The term gene-environment correlation subsumes three different mechanisms in which genetic factors are associated with exposure to particular environmental conditions (Knafo & Jaffee, 2013). With regard to the peer environment, evocative rGE and active rGE are likely (Figure 1). In evocative rGE, individuals' genetically influenced traits evoke particular responses from the environment. For instance, a sensation-seeking child will encounter different reactions from the peer group than an anxious child. Active rGE describes that individuals' selection of environments is to some extent genetically driven. Here, the sensation-seeking child will probably select other sensation-seekers as friends whereas the anxious child is more likely to affiliate with peers who prefer quieter spare time activities.

Gene-environment interactions describe that genotype elevates or buffers the effect of environmental exposure on the outcome, or that a direct association between a candidate gene and an outcome is qualified by exposure to a particular environment (Figure 2). For instance, if peer drinking is associated with adolescents' own alcohol consumption only for carriers of a specific variant of a gene, this gene would moderate the association between environmental exposure and outcome. If, in contrast, the association between a particular gene and aggressive behavior is only present if the adolescent affiliates with aggressive peers,

environmental exposure has moderating function. Theoretical assumptions about pathways should guide assignment of main predictor and moderator variables.

Since Caspi and colleagues' landmark study showed how carriers of specific variants of the monoamine-oxidase-A (*MAOA*) gene were more antisocial in adulthood after being maltreated as children than maltreated individuals who did not carry this variant (Caspi et al., 2002), studies on externalizing behavior, delinquency, aggression, and substance use have progressively employed GxE designs (Bakermans-Kranenburg & van IJzendoorn, 2006; Jaffee et al., 2005; McCrory, De Brito, & Viding, 2012; Propper, Willoughby, Halpern, Carbone, & Cox, 2007). Curiously, the majority of these studies remained within the family environment, that is, examined how genetic factors and negative family conditions interact in raising the risk of offspring maladjustment. While parents are crucially important for adolescent behavioral development, the significance of peers with regard to antisocial behavior is uncontested. But how can specific genes affect an adolescent's likelihood to pick up deviant behaviors from peers? Or vice versa, can peers really buffer genetic effects on antisocial behavior? The steadily growing number of studies on the interplay between peer environment and molecular genetic information appears to suggest this. Following a brief introduction to molecular genetics, we focus on particular neurotransmitter systems and candidate genes relevant to them as these are implicated as biological pathways in most candidate gene studies.

Peers, molecular genetics, and antisocial behavior: Where do we stand?

Human DNA consists of about 3 million pairings of adenine and thymine or guanine and cytosine that combine the double helix. Some stretches of DNA code for protein in that they are first transcribed into messenger RNA (mRNA) that then translates into amino acids, the building blocks for protein. Traditionally, those DNA stretches that are functional in coding for proteins are called genes. The remaining DNA transcribes into messenger RNA

but not further into amino acids. Current research is trying to identify the precise use of what was known as "junk DNA", suggesting that one of its functions is the regulation of protein-coding DNA.

Individual variation in some genes arises due to errors in DNA copying that result in base substitution or deletion. Although such variations in DNA are consequential (as we describe below), those of interest to social scientist are common and not eliminated by evolution, suggesting that they have some adaptive function as well. These characteristics differentiate genetic polymorphisms (as such genetic variations are called) from genetic mutations, which occur much less frequent and do not seem to serve an adaptive function. Different types of genetic polymorphisms are known: Apart from quantitative differences in efficiency as is the case in variable number tandem repeat polymorphisms (VNTR) where a DNA sequence varies in length, other genetic polymorphisms cause insertion of a different amino acid into a protein (single nucleotide polymorphisms, SNPs). Proteins are implicated in practically all processes in the human body thus also in those chemical processes that occur in the human brain. Here, proteins produce, process, and recognize neurotransmitters, chemicals that direct many brain processes such as dopamine, serotonin, and acetylcholine. Neurotransmitters are important because differences in their activity in the brain appear to be directly linked to behavior and emotion regulation (Raine, 2008) and to modulate how susceptible we are to environmental conditions, including those provided by peers.

Dopamine

Dopamine plays an important role in reward-related behaviors with brain dopamine levels increasing upon experience of reward. Many addictive substances like cocaine and amphetamines have the same effect. Candidate genes that are implicated in the dopaminergic system are the dopamine active transporter 1 gene (*DAT1*), as well as dopamine D2 and D4 receptor genes (*DRD2* and *DRD4*). Dopamine transporter cells affect reuptake speed of

dopamine, that is, the amount of time needed to clear dopamine from the synaptic cleft and essentially stop the dopamine signaling from one synapsis to the next. Dopamine receptor genes are responsible for receptor density and functionality in different brain regions.

Individual Variation in the *DAT1* gene occurs through a VNTR polymorphism. Most frequent variants include 9- and 10-repeat units but between 3- and 11-repeat versions have been observed. Highest expression levels were reported for the 10-repeat variant, thus this variant works most effectively in clearing dopamine from synapses. Increased duration of dopamine activity is associated with greater reward expectancy. The 9-repeat allele has been linked to greater self-reported impulsivity and increased activity in the ventral striatum, a brain region implicated in reward processing (Forbes et al., 2007), as well as to externalizing behavior in children (Young et al., 2002). However, research is not conclusive and some studies report more negative outcomes for carriers of the 10-repeate allele, e.g., with regard to attention-deficit/hyperactivity disorder (Cornish et al., 2005) and pathological criminal behavior in youth (Vaughn, Beaver, & DeLisi, 2009). Studies have yet to establish whether the *DAT1* gene moderates associations between peer characteristics and adolescent antisocial behavior. Beaver, Wright, and DeLisi (2008), however, showed that carriers of the 10-repeat allele who lived in high-risk neighborhoods were at greater risk to seek out delinquent peers, a prime example of active gene-environment correlation.

DRD2 and *DRD4* are dopamine receptor genes that regulate receptor density and functioning in various brain regions. Variation in the *DRD2* gene results from a SNP located on the neighboring *ANKK1* gene that gives rise to the *DRD2* A1 allele. This allele has been associated with psychopathic personality traits (Wu & Barnes, 2013), alcohol dependence (Wang, Simen, Arias, Lu, & Zhang, 2013), and cannabis addiction (Nacak et al., 2012). *DRD4* is located on chromosome 11 and contains a VNTR polymorphism which takes the form of a long (7-repeat) or short (4-repeat) allele with the short allele being much more

common than the long variant (Oak, Oldenhof, & Van Tol, 2000). Less frequent variants range from 2- to 10-repeats and research has shown that *DRD4* is more potent in binding dopamine in the brain in the presence of the short variant. The long allele has been linked to attention-deficit/hyperactivity disorder (Faraone, Doyle, Mick, & Biederman, 2001) and mood disorders (López León et al., 2005). Some studies have also linked the *DRD4* polymorphism to personality traits including novelty and thrill seeking (Dmitrieva, Chen, Greenberger, Ogunseitan, & Ding, 2011; Ray et al., 2009) but results remain inconclusive (Munafò, Yalcin, Willis-Owen, & Flint, 2008; Schinka, Letsch, & Crawford, 2002). Finally, associations between *DRD4* and substance use have been reported (Filbey et al., 2008; Hutchison, LaChance, Niaura, Bryan, & Smolen, 2002).

Important to note is a recent meta-analysis that did not support direct associations between any of the dopaminergic system polymorphisms discussed here and aggression (Vassos, Collier, & Fazel, 2013). Combined with some ambiguous associations (i.e., no clarity as to which variant increases risk, inconclusive results, replication failure), research that also takes into account environmental influences on antisocial outcomes seems more fruitful than the search for direct associations between genotype and outcome. That is, genetic polymorphisms in the dopaminergic system have been found to moderate peer influence: Guo, Roettger, and Cai (2008) examined whether *DRD2* and *DAT1* affect the association between peers' self-reported delinquent behavior and young adult males violent and serious delinquency. Their analyses showed that carriers of the heterozygous variant of *DRD2* (i.e., carrying one A1 and one A2 allele rather than two A1 or two A2 alleles) were more likely to engage in serious and violent delinquency if they affiliated with delinquent friends. Using an experimental design with a drinking confederate, Larsen et al. (2010) showed that carriers of the long allele of *DRD4* were at increased risk for heavy alcohol use in the presence of someone who consumed large amounts of alcohol in a short period of time.

Notably, Van der Zwaluw, Larsen, & Engels (2012 did not find this interaction effect in a longitudinal study of real friends.

Dopamine system genes have also attracted interest from researchers who examined affiliation with deviant peers not as environmental measure as done in the studies discussed above but as outcome. Beaver, Gibson, DeLisi, Vaughn, and Wright (2012), for instance, used data of more than 1000 males within the Add Health sample to examine whether the *DRD2* A1 allele and *DRD4* long allele interacted with neighborhood disadvantage in the prediction of four measures of antisocial phenotype, among them presence of delinquent peers. The peer measure was assessed through adolescent's self-report referred to number of peers who smoked more than one cigarette per day, smoked marijuana more than once a month, and drank alcohol more than once a month. Beaver et al. (2012) showed that *DRD2* significantly predicted affiliation with delinquent peers in adolescents growing up in disadvantaged neighborhoods. They further found that *DRD2* predicted adolescent experiences of victimization (e.g., someone pulling a gun against them) but only among youth with a low number of deviant peers. That is, affiliation with deviant peers buffered the genetic risk for victimization. There is also support for the notion that dopamine polymorphisms interact with environmental characteristics in predicting desistance from delinquency. Beaver, Wright, DeLisi, and Vaughn (2008) showed that *DRD2* and *DRD4* (as well as *MAOA*, described below) moderated the association between marriage and desistance from delinquency.

Serotonin

The role of serotonin for mood, sleep, memory, and behavior is unquestioned, resulting in numerous studies on the effect of serotonergic system genes on a range of psychopathologies, mostly on the internalizing spectrum. Serotonin has also been linked to externalizing behaviors: A meta-analysis by Moore, Scarpa, and Raine (2002) showed that

metabolites (degradation products) of brain serotonin were reduced in antisocial compared to non-antisocial individuals. A number of genes are implied in brain serotonin circuitry, the most popular one probably being the serotonin transporter polymorphism *5-HTTLPR*, which transcribes for a protein that is associated with reuptake speed of serotonin at brain synapses and constitutes an important source of variation in serotonin levels in the brain. Whereas the majority of people of Caucasian descent are carriers of the heterozygous short-long variant (~50%) of this polymorphism, the homozygous short-short variant is observed less often than the homozygous long-long variant (Noskova et al., 2008). Because of its reduced efficiency in terminating synaptic serotonin activity, the short allele has been treated as the risk allele in most studies (but see Glenn, 2011, for a review on psychopathology associated with the long allele). The *5-HTTLPR* short allele has been linked to personality traits such as neuroticism (Sen, Burmeister, & Ghosh, 2004) as well as mood disorders (Rosenthal et al., 1998). The polymorphism is also implicated in behaviors and traits that show overlap with antisocial behavior. For instance, studies have reported associations between the short allele and substance use (Feinn, Nellissery, & Kranzler, 2005). Retz, Retz-Junginger, Supprian, Thome, and Rösler (2004) showed that the short allele was overrepresented in males displaying recurrent and overt violent behavior, whereas Sakai et al. (2010) showed that female carriers of the short allele were more likely to display chronic conduct problems and Vaughn et al. (2009) reported a significant association with pathological criminal behavior in youth. The polymorphism has further been associated with persistent, pervasive childhood aggression (Beitchman et al., 2006) and antisocial traits in young adults (Lyons-Ruth et al., 2007). Moreover, probably because of its importance in the amygdala, *5-HTTLPR* affects perception of emotional stimuli: Beevers, Gibb, McGeary, and Miller (2007) showed that the polymorphism affected the speed at which someone was able to disengage their attention from both happy and sad stimuli. This effect is interesting because it may provide a pointer as

to why some adolescents react differently to cues from their peer environment. That is, peer pressure in form of teasing and threats to ensure compliant behavior may have a stronger impact upon those adolescents who carry the more susceptible variant of *5-HTTLPR*. Surprisingly, no study has tested whether *5-HTTLPR* moderates the influence of peer characteristics on adolescent antisocial behavior, although three studies have tested whether associations between peer relationship quality (i.e., peer victimization) and later depression (Benjet, Thompson, & Gotlib, 2009; Sugden et al., 2010) and delinquency (Kretschmer, Sentse, Dijkstra, & Veenstra, 2014) vary by *5-HTTLPR*. Indeed, all three studies showed that carries or the short allele of the polymorphism were at greater risk to develop problems following victimization by peers.

Focusing on a polymorphism in a serotonin receptor gene (*5-HT$_{2A}$*), Dijkstra, Lindenberg, Zijlstra, Bouma, and Veenstra (2013) examined genetic moderation of the association between boys' aggression and their popularity in the peer group. The study built on two studies by Burt (2008; 2009) that showed how this polymorphism predicts which peers adolescents like and dislike and that this association is mediated by rule-breaking behavior. Extending these findings, Dijkstra and colleagues' showed that the positive association between aggressive behavior and popularity was moderated by *5-HT$_{2A.}$*

Acetylcholine

Acetylcholine is the longest known and most common neurotransmitter and implicated in attention and arousal. Candidate genes that are associated with brain acetylcholine (nicotinic and muscarinic acetylcholine receptor genes) have previously been linked to personality traits (e.g., Hendershot, Bryan, Ewing, Claus, & Hutchison, 2011) and alcohol related disorders (Jung et al., 2011). Nicotinic acetylcholine receptor gene polymorphisms (e.g., *CHRNA4*, *CHRNA5*, *CHRNA7*, *CHRNB3*) have been associated with smoking (Munafò & Johnstone, 2008) and attracted some interest as moderators of peer

group influence. For instance, Latendresse et al. (2011) examined antisocial behavior in the peer group as predictor of developmental trajectories of externalizing behavior. In this study, Latendresse and colleagues showed that adolescents who carried minor alleles of muscarinic acetylcholine receptor gene *CHRM2* and affiliated with deviant peers were at greater risk to show stable or moderately high externalizing behavior. Treating the environmental measure as moderator and based on a main association between *CHRNA5* and nicotine dependence, Johnson et al. (2010) showed that peer smoking reduced the genetic effect. Put differently, the genetic association with nicotine dependence was strongest in the absence of environmental risk.

Monoamine-oxidase-A (MAOA)

Some genetic polymorphisms affect more than one neurotransmitter system. The monoamine-oxidase-A (*MAOA*) gene codes for an enzyme that is important in degrading serotonin and dopamine. A VNTR polymorphism in this gene concerns the number of tandem repeats in the promoter region of the gene with higher expression in carriers of the 3.5 or 4 repeat variant and lower expression in carriers of the 3 (and possibly 5) repeat variant. *MAOA* has repeatedly been linked to antisocial behavior, specifically human aggression (Buckholtz & Meyer-Lindenberg, 2008) and stably interacts with adverse environmental conditions such as maltreatment (Kim-Cohen et al., 2006) in increasing the risk for antisocial outcomes. The less efficient variant of the polymorphism thereby increased the risk for aggression and in most studies also increased vulnerability to adversity in the environment. With regard to moderation of peer effects, however, studies yielded mixed findings. Lee (2011), for instance, examined the interplay between *MAOA* and self-reported peer delinquency with regard to own antisocial behavior using a male subsample of the Add Health study and showed that carriers of the high-activity *MAOA* variant were at greater risk to engage in antisocial behavior if they affiliated with delinquent peers. Also using Add

Health data, Beaver and Holtfreter (2009) showed that *MAOA* moderated the link between delinquent peer affiliation in adolescence and engagement in fraudulent behavior in early adulthood. Again carriers of the high-activity variant of *MAOA* were most vulnerable to the effect of deviant peers.

Brain-derived neurotrophic factor (BDNF)

Stepping out of the neurotransmitter systems, Kretschmer, Vitaro, and Barker (2014) examined the brain-derived neurotrophic factor gene (*BDNF),* which regulates the secretion of brain-derived neurotrophic factor in the brain. The *BDNF* polymorphism consists of a valine to methionine substitution with *BDNF* secretion being reduced in met- compared to val-alleles (Hong, Liou, & Tsai, 2011). *BDNF* affects susceptibility to environmental stressors in the prediction of impulsive aggression (Wagner, Baskaya, Dahmen, Lieb, & Tadić, 2010) with met-allele carriers being more vulnerable to environmental risk than val-val carriers. Moreover, carriers of the met-allele show an increased risk for psychopathological disorders related to aggression (e.g., Spalletta et al., 2010) and impulsivity (Oades et al., 2008). In line with previous research, Kretschmer et al. (2014) showed a stronger effect of peer on adolescents' own aggression in carriers of the met-met variant of this polymorphism. That is, those adolescents who had affiliated with aggressive peers in late childhood and carried two minor alleles were at greatest risk to engage in aggressive behaviors themselves five years later.

μ-opioid receptor (OPRM1)

The *OPRM1* gene encodes for the μ-opioid receptor (MOR, receptor of endogenous opioids including heroin, morphine, and methadone). The *OPRM1* polymorphism consists of an adenine to guanine substitution which affects the reinforcing (thus addictive) effect of drugs in various brain regions (Ray et al., 2011) This polymorphism has been linked to a variety of substance use characteristics including sensitivity to the effects of alcohol (Ray &

Hutchison, 2004) and adolescent alcohol misuse (Miranda et al., 2010), heroin addiction (Shi et al., 2002), and nicotine reinforcement (Ray et al., 2006). *OPRM1* has also been associated with neural sensitivity to social rejection (Way, Taylor, & Eisenberger, 2009).

Moreover, the polymorphism has been found to interact with peer characteristics in predicting alcohol use disorders in adolescence (Miranda et al., 2013) in that carriers of the G-allele who affiliated with deviant peers had an almost eight times increased risk to be diagnosed with alcohol use disorder compared to their homozygous A-allele counterparts. Another study examined whether *OPRM1* was associated with affiliation with peers with a positive attitude towards drinking (Chassin et al., 2012). This study showed that male individuals homozygous for the A-allele were more likely to affiliate with alcohol promoting peers who in turn increased their risk for alcohol use disorder. A different pattern emerged for girls: G-carriers of *OPRM1* were more vulnerable to peer influence than carriers of the A-homozygous variant of the polymorphism.

Peers, molecular genetics, and antisocial behavior: Where do we go from here?

Effects of peer characteristics, irrespective whether they refer to substance use or delinquency, are not uniformly strong and some of the variation is accounted for by genetic make-up. The genetic polymorphisms that have been tested to date are theoretically meaningful in that they mostly refer to neurotransmitters that are implicated in impulsivity, aggression, or response to emotional stimuli, or affect how an individual processes substances like nicotine or alcohol. However, systematic and replicated findings are still scarce. The final part of this chapter aims to provide some pointers that may help to strengthen research into the interplay of peer environment and genetic factors in antisocial behavior research.

Gene-environment correlation studies

Although we know from quantitative behavioral genetics that genetic factors are associated with delinquent peer group affiliation (Brendgen, 2012), only few studies have examined rGE using measured genotype. The studies by Burt (2009), Beaver et al. (2012), and Chassin et al. (2012) are notable exceptions and their findings clearly suggest that adolescent peer selection is partly driven by specific genetic factors. These findings contain a word of warning: If genetic factors influence someone's preferences for affiliation, such associations need to be controlled for in GxE designs. Adolescents' peer groups are very specific environments driven by selection and choice (Veenstra & Dijkstra, 2011) more so than other environments that have played a role in gene-environment interplay studies (e.g., maltreatment, neighborhood). Given the significance of peers in adolescence and findings from social network studies that show how peer similarity in antisocial forms of behavior is to a non-negligible extent driven by selection (individuals affiliate with those who are similar to them, Veenstra, Dijkstra, Steglich, & Van Zalk, 2013), it is surprising that rGE studies are scarce in research on peer effects on antisocial behavior. Future studies are advised to pay more attention to the correlational mechanisms, as these may help to better understand the role of the peer environment in antisocial behavior.

Assessment of the environment

Although we have not broached the issue of validity and reliability of the peer measures, it is crucial to note that the quality of environmental assessment is a driving force in the success of a gene-environment interplay study. Particularly in the field of peer characteristics, shared-rater bias (adolescent reports on their own and their peers' behavior), huge variability in measures used, and ambiguity as to whom "peer group" refers to affect comparability and might determine whether or not a set of findings is replicated or not (see Larsen et al., 2010 and Van der Zwaluw et al., 2012). Of course, this problem is not easy to solve and many of the studies described here are secondary analyses of existing cohort

studies where measures cannot be changed in hindsight. Researchers are advised to revert to peers' own reports of their behavior as much as possible and streamline future studies with successful past ones in terms of measures and definitions (Veenstra & Steglich, 2012; Veenstra et al., 2013).

Peer relationship quality as environmental measure

Importantly, adolescent development is not only affected by peer characteristics such as drinking and delinquency, but the quality of relationships with peers also affects adjustment (e.g., Hanish & Guerra, 2002). Failing to establish positive relationships with peers is predictive of aggression (e.g., Boivin, Vitaro, & Poulin, 2005) and engagement in delinquent behavior (Laird, Jordan, Dodge, Pettit, & Bates, 2001). Peer victimization as a particularly grave form of peer negativity increases the risk for maladjustment (Arseneault, Bowes, & Shakoor, 2010), including aggression and delinquency (Hodges, Boivin, Vitaro, & Bukowski, 1999; Ostrov, 2010). Arguably, rejected or victimized adolescents are less well able to achieve social well being through peer relationships and may turn to other strategies. These may involve antisocial forms of behavior, especially so in adolescence when delinquency to a certain extent contributes to status attainment and involves behaviors that may elicit confirmation from the peer group (Mayeux, Sandstrom, & Cillessen, 2008). Such associations may be genetically moderated as initial research shows (Kretschmer, Dijkstra, Ormel, Verhulst, & Veenstra, 2013). Future studies are needed to replicate and extend these findings in independent samples.

Dual risk or differential susceptibility?

This chapter focused on peer effects in the development of adolescent antisocial behavior and how these are moderated by variations in DNA (or conversely how associations between genetic factors and antisocial behavior differ depending on environmental conditions). The perspective taken assumed a dual-risk model in which the environment

carries risk for a negative outcome and this risk is elevated in the presence of particular genetic variants. While this model is theoretically appealing given antisocial behavior as outcome, recent accounts have begun to challenge the generalizability of the dual-risk model to person-environment interactions (Belsky & Pluess, 2009; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2011). Rather, a differential susceptibility framework in which person factors – including but not limited to genetic polymorphisms – make individuals more susceptible to both negative and positive environmental conditions. For instance, affiliation with deviant peers may elevate the risk for antisocial behavior development in some adolescents more than in others but these same individuals may also benefit more from prosocial peer environments than less susceptible adolescents. Although pro- and antisocial behavior do not constitute opposite ends of one continuum, taking such effects into account will further the field and contribute important information about the role of person factors in understanding peer group effects on adolescent development beyond antisocial behavior.

Re-thinking the use of DNA information

Although studying the interplay between peer environment and measured genes adds valuable information to our understanding of heterogeneity in associations between peer environment and antisocial behavior, the effects that have been published are small and interactions explain only a tiny fraction of variance in antisocial behavior. This problem has been observed in most GxE studies as well as in research on main effects of genetic polymorphisms, notwithstanding quantitative genetic findings that a large proportion of population variance is genetically influenced. How can we account for this problem of "missing heritability" (Maher, 2008)? Discussions of the problem in neighboring disciplines (e.g. Plomin, 2012) suggest that we need to examine more than one polymorphism at a time. Not only are polymorphisms not necessarily inherited independently, they function additively

and interact with each other. Accounting for such interdependencies should become integral in studies on peer environment and gene-environment research in general. Using polygenic scores (indexes of many polymorphisms of small effect) as well as including gene-gene interactions in our models are steps towards this goal (Plomin, 2013). Finally, dopamine, serotonin, and acetylcholine are of course not the only human brain neurotransmitters. *Norepinephrine* is important in the regulation of concentration but can also function as a hormone and as such is important in the regulation of stress and response to fight or flight situations. *GABA* is the most important inhibitory neurotransmitter in the human central nervous system. We are not suggesting that researchers should now pick single candidates from these systems instead, but the different neurotransmitter systems do not operate in isolation. Informed by molecular neuroscience, researchers are advised to account for interactions and combined effects.

Conclusion

The inclusion of molecular genetic information into studies of associations between peer environment and antisocial behavior is novel and it may be too early to say whether genetic and environmental effects work together systematically in affecting individual risk for antisocial behavior. As with gene-environment studies in other fields, independent replications are sorely needed, as are strategies to investigate cumulative and interacting effects of several polymorphism at a time. However, the studies reviewed in this chapter certainly show that stepping out of the family environment and examining whether individual genetic make-up qualifies the effect of fairly common environmental risks (e.g., affiliating with peers who smoke or engage in deviant behavior) is a meaningful and worthwhile strategy to understand adolescent antisocial development. The knowledge gained in such studies may not be practically applicable in the short-term but being able to identify the adolescents most at risk for antisocial or substance use disorders can eventually help to target

prevention and intervention strategies. Whereas it is not ethical or possible to modify and adjust genetic factors, changing the environment may be particularly important for some young people. In other words, if certain environmental risks, such as peer deviance or peer drinking, are especially harmful for some adolescents (those that carry specific genetic variants), efforts should go into studying how we can prevent or intervene in these adverse environments to ensure healthy development.

References

Arseneault, L., Bowes, L., & Shakoor, S. (2010). Bullying victimization in youths and mental health problems: 'Much ado about nothing'. *Psychological Medicine, 40*(5), 717-729.

Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2006). Gene-environment interaction of the dopamine D4 receptor (*DRD4*) and observed maternal insensitivity predicting externalizing behavior in preschoolers. *Developmental Psychobiology, 48*(5), 406-409.

Beaver, K. M., Gibson, C. L., DeLisi, M., Vaughn, M. G., & Wright, J. P. (2012). The interaction between neighborhood disadvantage and genetic factors in the prediction of antisocial outcomes. In C. L. Gibson, & M. D. Krohn (Eds.), *Handbook of life-course criminology: Emerging trends and directions for future research* (pp. 25-40). New York: Springer Science & Business Media.

Beaver, K. M., & Holtfreter, K. (2009). Biosocial influences on fraudulent behaviors. *Journal of Genetic Psychology, 170*, 101-114.

Beaver, K. M., Wright, J. P., & DeLisi, M. (2008). Delinquent peer group formation: Evidence of a gene x environment correlation. *The Journal of Genetic Psychology, 169*, 227-244.

Beaver, K. M., Wright, J. P., DeLisi, M., & Vaughn, M. G. (2008). Desistance from delinquency: The marriage effect revisited and extended. *Social Science Research, 37*(3), 736-752.

Beevers, C. G., Gibb, B. E., McGeary, J. E., & Miller, I. W. (2007). Serotonin transporter genetic variation and biased attention for emotional word stimuli among psychiatric inpatients. *Journal of Abnormal Psychology, 116*(1), 208-212.

Beitchman, J., Baldassarra, L., Mik, H., De Luca, V., King, N., Bender, D., . . . Kennedy, J. (2006). Serotonin transporter polymorphisms and persistent, pervasive childhood aggression. *American Journal of Psychiatry, 163*(6), 1103-1105.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*(6), 885-908.

Benjet, C., Thompson, R. J., & Gotlib, I. H. (2009). *5-HTTLPR* moderates the effect of relational peer victimization on depressive symptoms in adolescent girls. *Journal of Child Psychology and Psychiatry, 51*(2), 173-179.

Boivin, M., Vitaro, F., & Poulin, F. (2005). Peer relationships and the development of aggressive behavior in early childhood. In R. E. Tremblay, W. W. Hartup & J. Archer (Eds.), *Developmental origins of aggression* (pp. 376-397). New York: Guilford Press.

Brechwald, W. A., & Prinstein, M. J. (2011). Beyond homophily: A decade of advances in understanding peer influence processes. *Journal of Research on Adolescence, 21*(1), 166-179.

Brendgen, M. (2012). Genetics and peer relations: A review. *Journal of Research on Adolescence, 22*(3), 419-437.

Buckholtz, J. W., & Meyer-Lindenberg, A. (2008). *MAOA* and the neurogenetic architecture of human aggression. *Trends in Neurosciences, 31*(3), 120-129.

Burt, A. (2009). A mechanistic explanation of popularity: Genes, rule breaking, and evocative gene–environment correlations. *Journal of Personality and Social Psychology, 96*(4), 783-794.

Burt, S. A. (2008). Genes and popularity: Evidence of an evocative gene-environment correlation. *Psychological Science, 19*(2), 112-113.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., . . . Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*(5582), 851-854.

Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: Joining forces with neuroscience. *Nature Reviews Neuroscience, 7*(7), 583-590.

Chassin, L., Lee, M. R., Cho, Y. I. L., Wang, F. L., Agrawal, A., Sher, K. J., & Lynskey, M. T. (2012). Testing multiple levels of influence in the intergenerational transmission of alcohol disorders from a developmental perspective: The example of alcohol use promoting peers and mu-opioid receptor M1 variation. *Development and Psychopathology, 24*, 953-967.

Cornish, K. M., Manly, T., Savage, R., Swanson, J., Morisano, D., Butler, N., . . . Hollis, C. (2005). Association of the dopamine transporter (*DAT1*) 10/10-repeat genotype with ADHD symptoms and response inhibition in a general population sample. *Molecular Psychiatry, 10*(7), 686-698.

Dijkstra, J. K., Lindenberg, S., Zijlstra, L., Bouma, E., & Veenstra, R. (2013). The secret ingredient for social success of young males: A functional polymorphism in the *5HT2A* serotonin receptor gene. *PloS One, 8*(2), e54821.

Dishion, T. J., & Tipsord, J. M. (2011). Peer contagion in child and adolescent social and emotional development. *Annual Review of Psychology, 62*, 189-214.

Dmitrieva, J., Chen, C., Greenberger, E., Ogunseitan, O., & Ding, Y. (2011). Gender-specific expression of the DRD4 gene on adolescent delinquency, anger and thrill seeking. *Social Cognitive and Affective Neuroscience, 6*(1), 82-89.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & Van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. *Development and Psychopathology, 23*(1), 7-28.

Faraone, S. V., Doyle, A. E., Mick, E., & Biederman, J. (2001). Meta-analysis of the association between the 7-repeat allele of the dopamine D4 receptor gene and attention deficit hyperactivity disorder. *American Journal of Psychiatry, 158*(7), 1052-1057.

Feinn, R., Nellissery, M., & Kranzler, H. R. (2005). Meta-analysis of the association of a functional serotonin transporter promoter polymorphism with alcohol dependence. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 133*(1), 79-84.

Ferguson, C. J. (2010). Genetic contributions to antisocial personality and behavior: A meta-analytic review from an evolutionary perspective. *The Journal of Social Psychology, 150*(2), 160-180.

Filbey, F. M., Ray, L., Smolen, A., Claus, E. D., Audette, A., & Hutchison, K. E. (2008). Differential neural response to alcohol priming and alcohol taste cues is associated with *DRD4* VNTR and *OPRM1* genotypes. *Alcoholism, 32*(7), 1113-1123.

Forbes, E., Brown, S., Kimak, M., Ferrell, R., Manuck, S., & Hariri, A. (2007). Genetic variation in components of dopamine neurotransmission impacts ventral striatal reactivity associated with impulsivity. *Molecular Psychiatry, 14*(1), 60-70.

Glenn, A. L. (2011). The other allele: Exploring the long allele of the serotonin transporter gene as a potential risk factor for psychopathy: A review of the parallels in findings. *Neuroscience & Biobehavioral Reviews, 35*(3), 612-620.

Guo, G., Roettger, M. E., & Cai, T. (2008). The integration of genetic propensities into social-control models of delinquency and violence among male youths. *American Sociological Review, 73*(4), 543-568.

Hanish, L. D., & Guerra, N. G. (2002). A longitudinal analysis of patterns of adjustment following peer victimization. *Development and Psychopathology, 14*(01), 69-89.

Hendershot, C. S., Bryan, A. D., Ewing, S. W. F., Claus, E. D., & Hutchison, K. E. (2011). Preliminary evidence for associations of *CHRM2* with substance use and disinhibition in adolescence. *Journal of Abnormal Child Psychology, 39*, 671-681.

Hodges, E. V., Boivin, M., Vitaro, F., & Bukowski, W. M. (1999). The power of friendship: Protection against an escalating cycle of peer victimization. *Developmental Psychology, 35*(1), 94.

Hong, C., Liou, Y., & Tsai, S. (2011). Effects of *BDNF* polymorphisms on brain function and behavior in health and disease. *Brain Research Bulletin, 86*(5), 287-297.

Hutchison, K. E., LaChance, H., Niaura, R., Bryan, A., & Smolen, A. (2002). The *DRD4* VNTR polymorphism influences reactivity to smoking cues. *Journal of Abnormal Psychology, 111*(1), 134.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Dodge, K. A., Rutter, M., Taylor, A., & Tully, L. A. (2005). Nature× nurture: Genetic vulnerabilities interact with physical maltreatment to promote conduct problems. *Development and Psychopathology, 17*(1), 67-84.

Johnson, E. O., Chen, L., Breslau, N., Hatsukami, D., Robbins, T., Saccone, N. L., . . . Bierut, L. J. (2010). Peer smoking and the nicotinic receptor genes: An examination of genetic and environmental risks for nicotine dependence. *Addiction, 105*(11), 2014-2022.

Jung, M., Park, B., Lee, B., Ro, Y., Park, R., Shin, H., . . . Choi, I. (2011). Association of *CHRM2* polymorphisms with severity of alcohol dependence. *Genes, Brain and Behavior, 10*(2), 253-256.

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W., & Moffitt, T. E. (2006). MAOA, maltreatment, and gene–environment interaction predicting children's mental health: New evidence and a meta-analysis. *Molecular Psychiatry, 11*(10), 903-913.

Knafo, A., & Jaffee, S. R. (2013). Gene–environment correlation in developmental psychopathology. *Development and Psychopathology, 25*(01), 1-6.

Kretschmer, T., Dijkstra, J. K., Ormel, J., Verhulst, F. C., & Veenstra, R. (2013). Dopamine receptor D4 gene moderates the effect of positive and negative peer experiences on later delinquency. *Development and Psychopathology, 25,* 1107-1117.

Kretschmer, T., Sentse, M., Dijkstra, J. K., & Veenstra, R. (2014). The interplay between peer rejection in pre- and early adolescence, serotonin transporter gene, and antisocial behavior in late adolescence - the TRAILS study. *Merrill-Palmer Quarterly, 60,* 193-216.

Kretschmer, T., Vitaro, F., & Barker, E. D. Association between peer and own aggression is moderated by *BDNF* val-met polymorphism. *Journal of Research on Adolescence, 24,* 177-185.

Laird, R. D., Jordan, K. Y., Dodge, K. A., Pettit, G. S., & Bates, J. E. (2001). Peer rejection in childhood, involvement with antisocial peers in early adolescence, and the development of externalizing behavior problems. *Development and Psychopathology, 13*(2), 337-354.

Larsen, H., van der Zwaluw, C. S., Overbeek, G., Granic, I., Franke, B., & Engels, R. C. M. E. (2010). A variable-number-of-tandem-repeats polymorphism in the dopamine D4 receptor gene affects social adaptation of alcohol use: Investigation of a gene-environment interaction. *Psychological Science, 21*, 1064-1068.

Latendresse, S. J., Bates, J. E., Goodnight, J. A., Lansford, J. E., Budde, J. P., Goate, A., . . . Dick, D. M. (2011). Differential susceptibility to adolescent externalizing trajectories: Examining the interplay between *CHRM2* and peer group antisocial behavior. *Child Development, 82*(6), 1797-1814.

Laub, J. H. (2004). The life course of criminology in the united states: The american society of criminology 2003 predidential address. *Criminology, 42*(1), 1-26.

Lee, S. S. (2011). Deviant peer affiliation and antisocial behavior: Interaction with monoamine oxidase a (*MAOA*) genotype. *Journal of Abnormal Child Psychology, 39*(3), 321-332.

López León, S., Croes, E. A., Sayed-Tabatabaei, F. A., Claes, S., Broeckhoven, C. V., & van Duijn, C. M. (2005). The dopamine D4 receptor gene 48-base-pair-repeat polymorphism and mood disorders: A meta-analysis. *Biological Psychiatry, 57*(9), 999-1003.

Lyons-Ruth, K., Holmes, B. M., Sasvari-Szekely, M., Ronai, Z., Nemoda, Z., & Pauls, D. (2007). Serotonin transporter polymorphism and borderline/antisocial traits among low-income young adults. *Psychiatric Genetics, 17*(6), 339.

Magnusson, D. (1985). Implications of an interactional paradigm for research on human development. *International Journal of Behavioral Development, 8*(2), 115-137.

Maher, B. (2008). The case of the missing heritability. *Nature, 456*(7218), 18-21.

Mayeux, L., Sandstrom, M. J., & Cillessen, A. H. (2008). Is being popular a risky proposition? *Journal of Research on Adolescence, 18*(1), 49-74.

McCrory, E., De Brito, S. A., & Viding, E. (2012). The link between child abuse and psychopathology: A review of neurobiological and genetic research. *Journal of the Royal Society of Medicine, 105*(4), 151-156.

Miranda, R., Reynolds, E., Ray, L., Justus, A., Knopik, V. S., McGeary, J., & Meyerson, L. A. (2013). Preliminary evidence for a GeneEnvironment interaction in predicting alcohol use disorders in adolescents. *Alcoholism: Clinical and Experimental Research, 37*, 325-331.

Miranda, R., Ray, L., Justus, A., Meyerson, L. A., Knopik, V. S., McGeary, J., & Monti, P. M. (2010). Initial evidence of an association between *OPRM1* and adolescent alcohol misuse. *Alcoholism: Clinical and Experimental Research, 34*(1), 112-122.

Moffitt, T. E. (2005). The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. *Psychological Bulletin, 131*(4), 533.

Monahan, K. C., Steinberg, L., & Cauffman, E. (2009). Affiliation with antisocial peers, susceptibility to peer influence, and antisocial behavior during the transition to adulthood. *Developmental Psychology, 45*(6), 1520.

Moore, T. M., Scarpa, A., & Raine, A. (2002). A meta-analysis of serotonin metabolite *5-HIAA* and antisocial behavior. *Aggressive Behavior, 28*(4), 299-316.

Munafò, M. R., & Johnstone, E. C. (2008). Genes and cigarette smoking. *Addiction, 103*(6), 893-904.

Munafò, M. R., Yalcin, B., Willis-Owen, S. A., & Flint, J. (2008). Association of the dopamine D4 receptor (*DRD4*) gene and approach-related personality traits: Meta-analysis and new data. *Biological Psychiatry, 63*(2), 197-206.

Nacak, M., Isir, A. B., Balci, S. O., Pehlivan, S., Benlier, N., & Aynacioglu, S. (2012). Analysis of dopamine D2 receptor (*DRD2*) gene polymorphisms in cannabinoid addicts*. *Journal of Forensic Sciences, 57*(6), 1621-1624.

Noskova, T., Pivac, N., Nedic, G., Kazantseva, A., Gaysina, D., Faskhutdinova, G., . . . Kovacic, D. K. (2008). Ethnic differences in the serotonin transporter polymorphism (*5-HTTLPR*) in several european populations. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 32*(7), 1735-1739.

Oades, R. D., Lasky-Su, J., Christiansen, H., Faraone, S. V., Sonuga-Barke, E. J., Banaschewski, T., . . . Ebstein, R. P. (2008). The influence of serotonin-and other genes on impulsive behavioral aggression and cognitive impulsivity in children with attention-deficit/hyperactivity disorder (ADHD): Findings from a family-based association test (FBAT) analysis. *Behavioral and Brain Functions, 4*(1), 48.

Oak, J. N., Oldenhof, J., & Van Tol, H. H. M. (2000). The dopamine D4 receptor: One decade of research. *European Journal of Pharmacology, 405*(1–3), 303-327.

Ostrov, J. M. (2010). Prospective associations between peer victimization and aggression. *Child Development, 81*(6), 1670-1677.
Plomin, R. (2012). Child development and molecular genetics: 14 years later. *Child Development, 84*, 104-120.
Plomin, R. (2013). Commentary: Missing heritability, polygenic scores, and gene–environment correlation. *Journal of Child Psychology and Psychiatry, 54*(10), 1147-1149.
Propper, C., Willoughby, M., Halpern, C. T., Carbone, M. A., & Cox, M. (2007). Parenting quality, DRD4, and the prediction of externalizing and internalizing behaviors in early childhood. *Developmental Psychobiology, 49*(6), 619-632. doi:10.1002/dev.20249
Raine, A. (2008). From genes to brain to antisocial behavior. *Current Directions in Psychological Science, 17*(5), 323-328.
Ray, L. A., Bryan, A., MacKillop, J., McGeary, J., Hesterberg, K., & Hutchison, K. E. (2009). The dopamine D4 receptor (*DRD4*) gene exon III polymorphism, problematic alcohol use and novelty seeking: Direct and mediated genetic effects. *Addiction Biology, 14*(2), 238-244.
Ray, L. A., & Hutchison, K. E. (2004). A polymorphism of the μ-Opioid receptor gene (*OPRM1*) and sensitivity to the effects of alcohol in humans. *Alcoholism: Clinical and Experimental Research, 28*(12), 1789-1795.
Ray, R., Jepson, C., Patterson, F., Strasser, A., Rukstalis, M., Perkins, K., . . . Lerman, C. (2006). Association of *OPRM1* A118G variant with the relative reinforcing value of nicotine. *Psychopharmacology, 188*(3), 355-363.
Ray, R., Ruparel, K., Newberg, A., Wileyto, E. P., Loughead, J. W., Divgi, C., . . . Lerman, C. (2011). Human mu opioid receptor (*OPRM1* A118G) polymorphism is associated with brain mu-opioid receptor binding potential in smokers. *Proceedings of the National Academy of Sciences, 108*(22), 9268-9273.
Retz, W., Retz-Junginger, P., Supprian, T., Thome, J., & Rösler, M. (2004). Association of serotonin transporter promoter gene polymorphism with violence: Relation with personality disorders, impulsivity, and childhood ADHD psychopathology. *Behavioral Sciences & the Law, 22*(3), 415-425.
Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin, 128*(3), 490.
Rosenthal, N. E., Mazzanti, C. M., Barnett, R. L., Hardin, T. A., Turner, E. H., Lam, G. K., . . . Goldman, D. (1998). Role of serotonin transporter promoter repeat length polymorphism (*5-HTTLPR*) in seasonality and seasonal affective disorder. *Molecular Psychiatry, 3*(2), 175-177.
Rutter, M. (1997). Developmental psychopathology as an organizing research construct. In D. Magnusson (Ed.), *The lifespan development of individuals. behavioral, neurobiological, and psychosocial perspectives.* (pp. 394-413). Cambridge: Cambridge University Press.
Sakai, J. T., Lessem, J. M., Haberstick, B. C., Hopfer, C. J., Smolen, A., Ehringer, M. A., . . . Hewitt, J. K.Case-control and within-family tests for association between *5-HTTLPR* and conduct problems in a longitudinal adolescent sample.
Schinka, J. A., Letsch, E. A., & Crawford, F. C. (2002). *DRD4* and novelty seeking: Results of meta-analyses. *American Journal of Medical Genetics, 114*(6), 643-648.
Sen, S., Burmeister, M., & Ghosh, D. (2004). Meta-analysis of the association between a serotonin transporter promoter polymorphism (*5-HTTLPR*) and anxiety-related personality traits. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 127*(1), 85-89.

Shi, J., Hui, L., Xu, Y., Wang, F., Huang, W., & Hu, G. (2002). Sequence variations in the mu-opioid receptor gene (*OPRM1*) associated with human addiction to heroin. *Human Mutation, 19*(4), 459-460. doi:10.1002/humu.9026

Spalletta, G., Morris, D., Angelucci, F., Rubino, I., Spoletini, I., Bria, P., . . . Bernardini, S. (2010). BDNF Val66Met polymorphism is associated with aggressive behavior in schizophrenia. *European Psychiatry, 25*(6), 311-313.

Steinberg, L., & Morris, A. S. (2001). Adolescent development. *Journal of Cognitive Education and Psychology, 2*(1), 55-87.

Sugden, K., Arseneault, L., Harrington, H., Moffitt, T. E., Williams, B., & Caspi, A. (2010). Serotonin transporter gene moderates the development of emotional problems among children following bullying victimization. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(8), 830-840.

Van der Zwaluw, C. S., Larsen, H., & Engels, R. C. M. E. (2012). Best friends and alcohol use in adolescence: The role of the dopamine D4 receptor gene. *Addiction Biology, 17*, 1036-1045.

Van Lier, P. A., Wanner, B., Vitaro, F., Blishen, B., Carroll, W., Moore, C., . . . Bongers, I. (2007). Onset of antisocial behavior, affiliation with deviant friends, and childhood maladjustment: A test of the childhood-and adolescent-onset models. *Development and Psychopathology, 19*(1), 167.

Vassos, E., Collier, D., & Fazel, S.Systematic meta-analyses and field synopsis of genetic association studies of violence and aggression. *Molecular Psychiatry,* doi:10.1038/mp.2013.31

Vaughn, M. G., Beaver, K. M., & DeLisi, M. (2009). A general biosocial paradigm of antisocial behavior. *Youth Violence and Juvenile Justice, 7*(4), 279-298.

Veenstra, R., & Dijkstra, J. K. (2011). Transformations in adolescent peer networks. In B. Laursen, & W. A. Collins (Eds.), *Relationship pathways: From adolescence to young adulthood* (pp. 135-154). Los Angeles: Sage.

Veenstra, R., Dijkstra, J. K., Steglich, C., & Van Zalk, M. H. (2013). Network–Behavior dynamics. *Journal of Research on Adolescence, 23*(3), 399-412.

Veenstra, R., & Steglich, C. (2012). Actor-based model for network and behavior dynamics. In B. Laursen, T. D. Little & N. A. Card (Eds.), *Handbook of developmental research methods* (pp. 598-618). New York: Guilford Press.

Wagner, S., Baskaya, Ö, Dahmen, N., Lieb, K., & Tadić, A. (2010). Modulatory role of the brain-derived neurotrophic factor Val66Met polymorphism on the effects of serious life events on impulsive aggression in borderline personality disorder. *Genes, Brain and Behavior, 9*(1), 97-102.

Wang, F., Simen, A., Arias, A., Lu, Q., & Zhang, H. (2013). A large-scale meta-analysis of the association between the *ANKK1*/*DRD2* Taq1A polymorphism and alcohol dependence. *Human Genetics, 132*(3), 347-358.

Way, B. M., Taylor, S. E., & Eisenberger, N. I. (2009). Variation in the μ-opioid receptor gene (OPRM1) is associated with dispositional and neural sensitivity to social rejection. *Proceedings of the National Academy of Sciences, 106*(35), 15079-15084.

Wu, T., & Barnes, J. C. (2013). Two dopamine receptor genes (*DRD2* and *DRD4*) predict psychopathic personality traits in a sample of american adults. *Journal of Criminal Justice, 41*(3), 188-195.

Young, S. E., Smolen, A., Corley, R. P., Krauter, K. S., DeFries, J. C., Crowley, T. J., & Hewitt, J. K. (2002). Dopamine transporter polymorphism associated with externalizing behavior problems in children. *American Journal of Medical Genetics, 114*(2), 144-149.

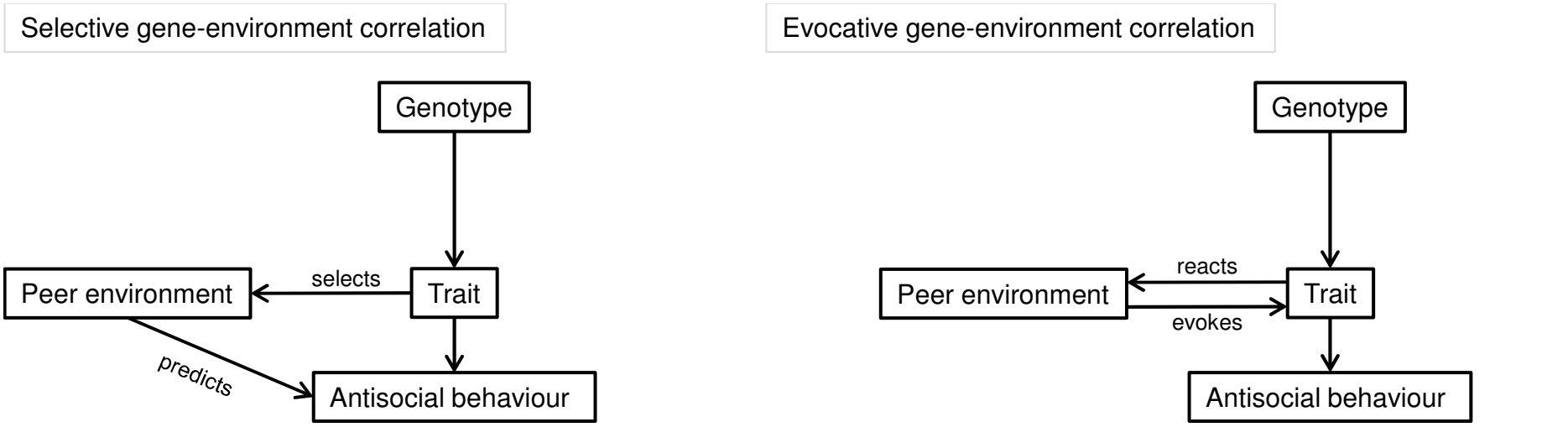

Figure 1: Gene-environment correlation

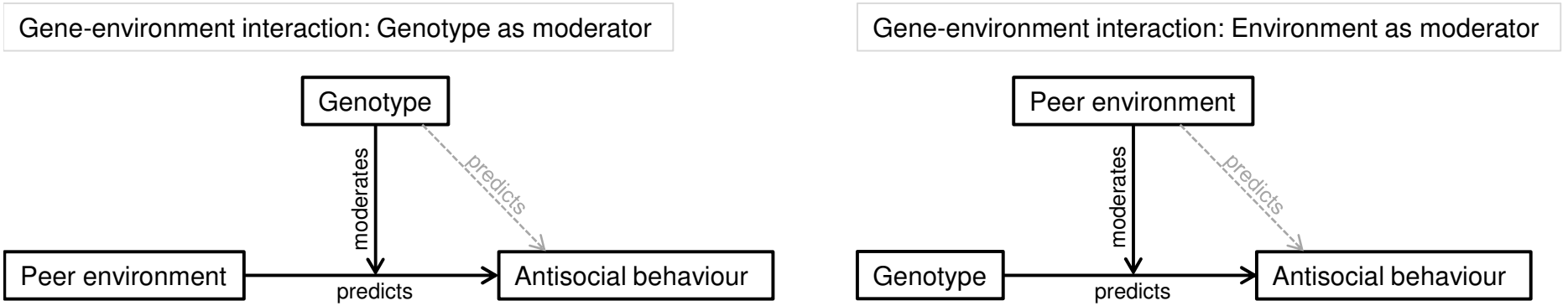

Figure 2: Gene-environment interaction