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Dissecting Antisocial Behavior: The Impact of Neural, Genetic, and Environmental Factors

Arielle R. Baskin-Sommers
Departments of Psychology and Psychiatry, Yale University

Abstract

Antisocial behavior is a heterogeneous construct. The adverse consequences of the behaviors and diagnoses related to this construct produce great burden for the perpetrators, victims, family members, and for society at-large. The articles presented in this special series build on decades of research aimed at identifying various factors across neural, genetic, and environmental levels that contribute to antisocial behavior. However, there are several methodological issues plaguing this research and it often fails to address the specificity of certain factors for subtypes of antisocial behavior. Furthermore, most research on antisocial behavior does not provide a good sense of how combinations of factors produce specific behaviors or how these underlying factors achieve a level of durability (e.g., adaptive constancy) that continually promotes chronic antisocial behavior. The articles in this series take an important step toward disaggregating factors and individuals to develop appropriate assessment techniques, characterizations, intervention strategies, and prevention programs.

Keywords

antisocial behavior, neural, genetic, environment, adaptive constancy

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Antisocial behavior is a heterogeneous construct that includes a wide range of behavioral problems and psychopathologies. In terms of behavior, antisocial acts can manifest as cheating, lying, aggression, substance use, theft, and violence. With regard to classification, children and adolescents may be identified as having conduct disorder or callous–unemotional traits, whereas adults may be identified as having antisocial personality disorder, psychopathy, trait externalizing, or other serious forms of psychopathology. The adverse consequences of such acts and diagnoses produce great physical, emotional, and economic burden for the perpetrators themselves, victims, family members, and for society at-large. For example, this past year in the United States alone, official counts of violent and nonviolent crime approached 31,000 per day. The financial impact on society from economic losses to the victims to government expenditures on policing, legal activities, and corrections was estimated to run over $3 trillion for that year (Federal Bureau of Investigation, 2015). Similarly, over 21.6 million individuals ages 12 and older in the United States were classified with a substance use diagnosis, exacting an annual cost of over $700 billion related to crime, lost work productivity, and health care (National Institute on Drug Abuse, 2015; Substance Abuse and Mental Health Services Administration, 2014). The pervasiveness of these behaviors highlights the importance of identifying those specific factors that are etiologically related to the onset and maintenance of such behaviors.

Before proceeding, it is essential to note that some antisocial behaviors are quite normative. For instance, most people have told a lie, violated speed limits, and misused substances; and many have even engaged in more serious behaviors, including interpersonal violence and theft. In the majority of these cases, though, the behavior does not generate public concern. And even for those whose behavior would be considered serious,

Corresponding Author:
Arielle R. Baskin-Sommers, Departments of Psychology and Psychiatry, Yale University, P.O. Box 208205, New Haven, CT 06520
E-mail: arielle.baskin-sommers@yale.edu

desistance generally occurs naturally through maturation. Those who engage in more normative antisocial behaviors or naturally desist are not the focus of the present series. Instead, the articles presented here concentrate on the factors underlying chronic antisocial behavior.

The articles in this special issue build on decades of research aimed at identifying factors related to engagement in antisocial behavior. Substantial progress in understanding these factors has been made in a variety of disciplines, from the natural to the social and behavior sciences. Increasingly, research in these fields documents the influence of various neural, genetic, and environmental factors on broad classes of antisocial behavior. In the following sections, I consider briefly the neural, genetic, and environmental factors that have been replicated across a significant body of the literature. Some of these factors will be common across forms of antisocial behavior and others unique to subtypes of antisocial behavior.

Although current research has identified a number of key factors across levels of analysis, it often fails to address the specificity of certain factors for subtypes of antisocial behavior. Furthermore, it does not provide a good sense of how combinations of factors produce specific antisocial behaviors. In the final section, I highlight how the articles in the present series take an important step toward disaggregating factors and individuals to develop appropriate assessment techniques, etiologically based characterizations, intervention strategies, and prevention programs.

Neural Factors

Research on the neural factors of antisocial behavior, specifically those that use neuroimaging, can be divided into "structural" studies, which assess brain morphology, and "functional" studies, which assess brain activity. These studies identify core neural regions related to salience detection, affect, and controlled cognition, including the amygdala, (pre)frontal cortex, and anterior cingulate cortex.

For decades, the amygdala has been the primary structure of interest in studies of antisocial behavior and psychopathology. The amygdala is a subcortical structure that is important for processes related to responding to threat, attention orienting to salience, and learning from the environment (e.g., fear conditioning; LeDoux, 2000). Generally speaking, structural studies report a thinning or volume reduction in the amygdala across such antisocial subtypes as antisocial personality disorder, psychopathy, conduct disorder, and overall high levels of aggressive behavior (Fairchild et al., 2011; Matthies et al., 2012; Raine, 2006; Yang, Raine, Narr, Colletti, & Toga, 2009). Thus, reductions in the structural integrity of the amygdala may be common across subtypes of antisocial behavior.

However, functional differences in amygdala activation are found across the life span and diverge based on the specific subtypes of antisocial behavior. On the one hand, reactive aggression, conduct disorder, and antisocial personality disorder are associated with increased amygdala responses to affectively evocative stimuli (Blair, 2010; Hyde, Shaw, & Hariri, 2013). This pattern of activation is consistent with the conceptualization of one type of antisocial behavior being related to over-reactivity to threatening or frustrating environments. On the other hand, proactive aggression, callous–unemotional traits, and psychopathy are associated with reductions in amygdala activation, particularly during aversive conditioning, moral decision making, social cooperation, and memory for emotionally salient words (see Frick & White, 2008; Koenigs, Baskin-Sommers, Zeier, & Newman, 2011, for reviews). For the most part, this pattern of activation is interpreted as deficiencies in emotional expression and learning. However, other studies indicate that the amygdala is normatively or even hyperreactive when psychopathic individuals view certain emotionally salient information (Larson et al., 2013; Müller et al., 2003). Moreover, a recent theory of psychopathy emphasizes the importance of differential responses within subcomponents of the amygdala (Moul, Killcross, & Dadds, 2012), suggesting that the heterogeneity of antisocial behavior may be better understood by parsing the different components of the amygdala itself. Overall, however, activation of the amygdala is one etiological factor that differentiates subtypes of antisocial behavior based on divergent affective and salience sensitivities.

Beyond the amygdala, other subcortical regions that are related to the onset and maintenance of antisocial behavior include the insula and ventral striatum. The insula is associated with the integration of an internal state with another's experience, making it important for decision making and empathic functioning (Decety & Jackson, 2006; Naqvi & Bechara, 2009). The ventral striatum is linked primarily to reward and motivation, providing a potentially important substrate for risk taking, impulsive, and self-destructive behaviors (Camille et al., 2010; Robbins, Gillan, Smith, de Wit, & Ersche, 2012). Abnormalities in the structure and function of the insula and ventral striatum (specifically the nucleus accumbens) are found in youth and adults who display conduct problems, aggression, psychopathy, and trait externalizing (Buckholtz et al., 2010; Glenn & Yang, 2012). However, findings on the direction of these abnormalities are equivocal, with some studies reporting increased and others reporting decreased structural integrity and function in these regions. What seems more consistent is that disorders associated with momentary, state-like increases in antisocial, risk taking, behavior like bipolar disorder and borderline personality disorder are associated with

increases in insula and ventral striatum activation (Maletic & Raison, 2014; Ruocco, Amirthavasagam, Choi-Kain, & McMain, 2013), whereas traits associated with persistent antisocial behavior are more variable in their insula and ventral striatum reactivity. Regardless of specific direction, one interpretation may be that both the insula and ventral striatum are not appropriately processing and integrating affective and motivational experiences. That is, it may not be that all individuals who engage in antisocial behavior are hypersensitive to rewards or motivational salience. Instead, it may be that they fail to integrate the presence or absence of this information. This type of evaluative processing can also intersect with additional abnormalities in (pre)frontal regions.

Much like the amygdala, research on the role of the prefrontal cortex (PFC) in antisocial behavior also has a long history. Initially, it was thought that antisocial behavior stemmed from a purported dysfunction in the PFC combined with dysfunction in subcortical regions. This "gas-breaks model" conceptualized this combination as being responsible for chronic failures to inhibit antisocial behavior and to maintain appropriate behavior. However, our understanding of the complexity of the PFC has improved, and as a result, so has our conceptualization of its role in chronic antisocial behavior. Distinct regions of the PFC exist that span the orbitofrontal cortex (OFC), ventromedial PFC (vmPFC), and dorsolateral and -medial PFC (dlPFC, dmPFC). The OFC and vmPFC serve to integrate information related to affective values and decision making (Grabenhorst & Rolls, 2011; Rolls, 2004). The dlPFC and dmPFC are involved in executive functions, execution of long-term goals, and value-based decision making (Dalley, Cardinal, & Robbins, 2004; Rangel, Camerer, & Montague, 2008).

In terms of antisocial behavior, specific PFC structural and functional abnormalities are found and replicated across a wide variety of studies. For instance, violent offenders and individuals diagnosed with antisocial personality disorder have reductions in the structural integrity and functionality of the OFC, vmPFC, dlPFC, and dmPFC (Blair, 2010; Glenn & Raine, 2011). However, these abnormalities are not observed across all antisocial syndromes. For example, a growing body of literature indicates that both youth and adults with high psychopathic traits show normative or enhanced activation in subcomponents of the PFC, such as the lateral and dlPFC (Decety, Chen, Harenski, & Kiehl, 2013; Finger et al., 2008; Glenn, Raine, Schug, Young, & Hauser, 2009; Larson et al., 2013), but abnormalities in subcomponents such as the vmPFC (Blair, 2010). The imbalance between dorsal and ventral processing in psychopathic individuals may serve to inhibit neural regions related to affective functioning and account for the failure of these individuals to integrate information across neural circuits and environmental settings. Thus, regarding the PFC, not only might subtypes of antisocial behavior be differentially related to the PFC, but subdomains of the PFC also may be primarily associated with subtypes of antisocial behavior.

Finally, another neural region to receive considerable attention in the literature on antisocial behavior is that of the anterior cingulate cortex (ACC). The ACC is tagged as a region involved in processing information about emotional states and changing contingencies in the environment. The ACC plays a role in error detection and correction (monitoring when outcomes differ from what was expected; Botvinick, Cohen, & Carter, 2004), with dorsal regions implicated in cognitive processing and ventral regions involved in assessing the salience of emotion and motivational information (Bush, Luu, & Posner, 2000). Studies in youth with conduct disorder and adults with violent tendencies, antisocial personality disorder, or psychopathy report reduced ACC structural integrity and functioning (Blair, 2010; Davidson, Pizzagalli, Nitschke, & Kalin, 2003; Glenn, Yang, Raine, & Colletti, 2010; Lockwood et al., 2013; Marsh et al., 2013; Yang & Raine, 2009). Thus, it is possible that deficits related to the ACC are common across all subtypes of antisocial behavior.

Taken together, the neural factors involved in antisocial behavior have been studied intensively with imaging methodology. Although abnormalities in neural regions may be common across antisocial subtypes (e.g., ACC, structural integrity of amygdala), abnormalities in other neural regions may be an indicator of divergent etiological factors for subtypes of antisocial behavior (e.g., amygdala, insula, ventral striatum, subcomponents of PFC activation). The identification of common and unique neural factors provides a foundation for understanding antisocial behavior. However, the brain does not operate in isolation; therefore, research should explore the role of neural factors in concert with genetic, environmental, and behavioral factors.

Genetic Factors

Various lines of research, including behavioral genetics, temperament, and epidemiological studies, emphasize the genetic basis of antisocial behavior. Some studies focus on specific genetic variants (e.g., single nucleotide polymorphisms or polygenic, multiple genes). Others use imaging genetics to provide a neural substrate through which genes affect behavior. Broadly speaking, genetic influences have been reported for aggression and criminality, temperament and personality factors (e.g., disinhibited, impulsive, callous–unemotional), and psychiatric outcomes related to antisocial behavior (e.g., substance use disorders, borderline personality disorder, bipolar

disorder). Across these studies, there is strong evidence that genetic factors, at least to a degree, are key in explaining individual differences in antisocial behavior. And increasingly, gene–environment studies emphasize the connection between experience and the genome in the development of antisocial behavior (e.g., genetic epidemiology).

At both theoretical and empirical levels and across animal and human studies, lower serotonin (5-HT) levels are related to higher levels of antisocial behavior, including aggression and impulsivity (Baker, Bezdjian, & Raine, 2006). Genetic variants of 5-HT are associated with aggressive and violent behavior, linked to functioning in the amygdala and PFC, and demonstrate discriminability based on certain environmental experiences (see Hyde et al., 2013, for review). For example, individuals expressing low MAOA alleles display increased functional activity in the left amygdala and decreased response across various cortical areas (e.g., lateral OFC and insula; Buckholtz & Meyer-Lindenberg, 2008), which is then associated with aggressive and impulsive tendencies. In addition, several gene–environment interaction studies demonstrate links between individual differences in variants of MAOA among individuals who engage in antisocial behavior and who have experienced maltreatment (Caspi et al., 2002; Kim-Cohen et al., 2006; Weder et al., 2009). Specifically, low expressing MAOA alleles, especially in the presence of early maltreatment, are associated with greater amygdala reactivity and later reactive antisocial behavior (Dannlowski et al., 2012; Hanson et al., 2010; Márquez et al., 2013; McCrory, De Brito, & Viding, 2011; Tottenham et al., 2011; Viding & Frith, 2006). However, high expressing alleles are linked with proactive aggression and callous–unemotional traits. Supporting this allelic dissociation, another 5-HT variant, 5-HTTLPR, is related to impulsive and psychopathic behavior, such that individuals homozygous for the short versus long allele demonstrate greater impulsivity; but youth with the homozygous-long genotype, in low socioeconomic environments, display the highest callous–unemotional and narcissistic traits. Furthermore, genetic variants in serotonin also appear to reveal dissociable patterns based on subtypes of antisocial behavior, whereby individuals with low MAOA or short 5-HTTLRP alleles are more likely to engage in reactive, impulsive antisocial behavior, but individuals with high MAOA or long 5-HTTLRP alleles are more proactive and psychopathic in their behavior. Together, these studies show the importance of the intersection of multiple factors.

In addition to serotonin, other genes and neurochemicals are connected to antisocial behavior. For example, enhanced dopamine is associated with impulsivity, reward sensitivity, ventral striatum functioning (Buckholtz et al., 2010; Forbes et al., 2009), and substance abuse disorders (Comings & Blum, 2000; Le Foll, Gallo, Le Strat, Lu, & Gorwood, 2009). Furthermore, the catechol O-methyltransferase (COMT) enzyme plays a major role in modulating PFC dopamine levels, which then associated with subtypes of antisocial behavior (Caspi et al., 2008; Hirata, Zai, Nowrouzi, Beitchman, & Kennedy, 2013; Nemoda et al., 2010). Specifically, the COMT valine/methionine polymorphism is predictive of higher levels of conduct problems, aggressive behavior, criminal behavior, and callous–unemotional traits. COMT is a modifying gene that appears to play a role in determining interindividual variability in the proclivity for antisocial behavior, in both individuals with major psychiatric conditions and those without.

Across genetic studies, there is clear evidence that specific genotypes confer risk for antisocial behavior. However, there is equally clear evidence that the interaction between genetic and environmental factors is of major importance in explaining individual differences in antisocial behavior (Simons et al., 2011). Genetic studies have the potential to parse the heterogeneity of antisocial behavior based on differential levels of risk at the various levels of other factors; however, they should be viewed in combination with research on neural and environmental factors.

Environmental Factors

Environmental factors can exert influence on antisocial behavior at various levels, from the community to the family to the peer context. Typically, studies using ethnographic, survey, archival, or network analysis support the strong association between environmental factors and antisocial behavior.

Much research on the environmental factors associated with antisocial behavior points to community disadvantage (Anderson, 1994; Beyers, Loeber, Wikström, & Stouthamer-Loeber, 2001; Haynie, Silver, & Teasdale, 2006) as a key etiological factor. Community disadvantage is typically found in neighborhoods where there is a spatial concentration of poverty, reliance on public assistance, female-headed households, joblessness, density of children, residential segregation, social disorder, and lack of political influence. However, community disadvantage does not affect all residents, equally. Therefore, there is some debate as to whether it is community disadvantage, itself, that is directly linked to antisocial behavior or whether there are other factors that transmit such disadvantage, but only to individuals with specific vulnerabilities.

Some studies on community disadvantage and antisocial behavior identify exposure to community violence as an important means through which disadvantage is transmitted (Aisenberg & Herrenkohl, 2008; Baskin & Sommers,

2013; Baskin-Sommers et al., in press; Gorman-Smith & Tolan, 1998; Lynch, 2003). In general, both cross-sectional and longitudinal research finds that exposure to community violence places youth at risk for antisocial behavior (Baskin & Sommers, 2015; Fowler, Tompsett, Braciszewski, Jacques-Tiura, & Baltes, 2009; Javdani, Abdul-Adil, Suarez, Nichols, & Farmer, 2014) and antisocial psychopathology (see Fowler et al., 2009, for meta-analysis). Moreover, earlier exposure has been linked to greater and more chronic adverse consequences (Guerra, Huesmann, & Spindler, 2003; Huesmann & Guerra, 1997), including persistent academic underachievement (Delaney-Black et al., 2002), earlier displays of aggression (e.g., fighting; Durant, Pendergrast, & Cadenhead, 1994), somatic symptoms (e.g., difficulty sleeping, headaches; Bailey et al., 2005), and justice system involvement (Hawkins et al., 2000). It is important to note that negative consequences from exposure to violence are particularly pronounced for youth with conduct disorder–related symptoms (i.e., conduct problems; Javdani et al., 2014) and other antisocial syndromes, including callous–unemotional and psychopathic traits (Baskin-Sommers et al., in press; Fowler et al., 2009; Kimonis, Centifanti, Allen, & Frick, 2014). For these youth, exposure helps to establish serious criminal offending trajectories that continue well into adulthood (Baskin & Sommers, 2013). Overall, then, exposure to violence in the context of community disadvantage appears to increase the risk for chronic antisocial behavior across the life span and across diagnostic categories.

In addition to exposure to community violence, family dysfunction, again in the context of disadvantage, is an important factor related to antisocial behavior. Although neglect, maltreatment, and parental psychopathology exist across communities, their impact appears to be affected by, or at least is made more pernicious in the presence of, community disadvantage (Liu & Heiland, 2009; Sampson, 2008; Waldfogel, Craigie, & Brooks-Gunn, 2010; Walsh et al., 2012). However, other family factors, such as harsh, negative, coercive, or inconsistent parenting, seem to predict higher levels of antisocial behavior, particularly in youth high on callous–unemotional traits or in individuals with borderline personality disorder, regardless of environment or disadvantage (see Waller, Gardner, & Hyde, 2013, for review). Examination of environmental factors also suggests that there are some factors common across antisocial behaviors (e.g., community disadvantage and exposure to violence), whereas other factors are more specific to subtypes of antisocial behaviors (e.g., harsh parenting style predominantly influencing callous–unemotional traits).

Beyond the family, peers may be a particularly important factor for the initiation and maintenance of antisocial behavior. Although peer influence can promote initiation into antisocial behavior, antisocial behavior can also affect the selection of friends (Jessor & Jessor, 1977; Monahan, Steinberg, & Cauffman, 2009; Poulin, Kiesner, Pedersen, & Dishion, 2011). For that matter, involvement in some antisocial behaviors occurs within highly social contexts and these social activities may actually be engaged in precisely due to the expectation that antisocial activities will occur (Osgood, Wilson, O'Malley, Bachman, & Johnston, 1996). These social contexts can take the form of "peer clusters" (Oetting & Beauvais, 1987) in which attitudes and beliefs about antisocial behavior are developed by cluster members. This form of "deviancy training" can escalate to the point where social interactions become increasingly focused on antisocial behavior, to the exclusion of other activities and topics. The influence of peers on antisocial behavior seems to appear across subtypes of antisocial behaviors and environmental contexts.

It is clear that the role of environmental factors is important for the development of antisocial behavior, whether it is one of disadvantage, exposure to violence, patterns of familial engagement, or peer influence. However, these factors often fail to provide specificity in terms of the subtypes of antisocial behaviors. It is only in concert with neural or genetic factors that we begin to achieve predictive specificity. For example gene-by-environment interaction studies tend to focus on factors such as maltreatment and parenting and find interesting patterns of interactions that provide specification based on subtypes of antisocial behavior (see the earlier discussion). Many of these studies, though, do not consider the broader environment (e.g., disadvantage, exposure to violence) or peer relationships. Environmental factors are a necessary condition for antisocial behavior. But the neural and genetic factors that influence affective and information processing may shape how certain types of individuals view their environment. Therefore, considering antisocial behavior through a multifactor lens across levels of analysis is essential for developing comprehensive models for both the broad class and specific subtypes of antisocial behaviors.

The Future of Research on Antisocial Behavior

Currently, research on antisocial behavior identifies a number of important neural, genetic, and environmental factors. Across these studies, some factors, such as dysfunction in the ACC, exposure to violence, and community disadvantage, appear to be important predictors of the broad class of antisocial behaviors. However, there are also factors that differentiate subtypes of antisocial behaviors and disorders, such as activation in the amygdala, specific genotypes, and familial interactions. Nonetheless, there remains a paucity of theoretical

conceptualizations that integrate across factors or levels of analysis and methodological approaches that test integrative theory.

Theoretically, although it is possible that individuals who engage in antisocial behavior are simply at a neural or genetic disadvantage, this perspective fails to consider the importance of environmental factors and fluctuations in behavior over time. Thus, antisocial behavior may be best characterized as an outgrowth of adaptations in various factors that develop to survive. Moreover, interactions of specific factors across levels of analysis may help us understand these chronic trajectories of antisocial behavior.

For example, imagine a child who early in life is exposed to violence, who is taught by parents and others that displaying emotions is a sign of weakness, and who finds his or her own heightened arousal distracting and overwhelming. To flourish in this environment, the child may learn to disconnect from emotions and dampen his or her reactivity. This response may momentarily yield a more stable internal experience and a more reliable way to attain external goals. Over time though, this child will fail to have experiences that reinforce healthy neural development, will fail to build elaborative associations between emotion and behavior, and will find himself or herself in situations that violate behavioral norms that are typically informed by affective input. This child may then take on the behavioral characteristics of a psychopathic individual, initiated and continually reinforced by environmental factors and adaptations in neural regions (e.g., dlPFC, ACC, amygdala) and circuitry (e.g., dorsal lateral prefrontal-cingulate-parietal network, corticolimbic, frontostriatal) responsible for integrating cognitive and affective information. Thus, it is not that the psychopathic individual is incapable of processing or engaging with the complexities associated with the human experience, but that through constant adaptations to maximize control and survival the psychopathic individual is constrained in his or her ability to process, reflect, and respond to various experiences (see also Glenn, Kurzban, & Raine, 2011).

A second example may be a child who genetically has a low expression of MAOA and is raised in an environment of neglect and continual abuse/maltreatment. To survive in this environment with his or her genetic susceptibility, the child may need to be hypervigilant to detect threats in the environment. The constant state of hypervigilance would reinforce amygdala reactivity and constrain the integration of cues that indicate relative value, choice, or conflict. Ultimately, without intervention or change in environment, this child will continually respond to his or her environment in a reactive, impulsive manner even when cues in the environment do not engender threat. Thus, to survive in an unstable and unpredictable environment, the individual in this example must develop an adaptation that yields a constant state of alertness and caution.

In each of these examples, though the individual may be at greater susceptibility for developing antisocial behavior through environmental context, genetic risk, or neural insult, the chronicity of that behavior does not become established until the adaptations in these factors become constant. This constancy may then establish a new point of adaptive homeostasis so that the individual can now survive within the proximal environment, but paradoxically be maladaptive in the larger social landscape over time. This reciprocal influence whereby the brain, genes, environment, and behavior synergistically promote the maintenance of antisocial behavior, ultimately, produces stable traits and chronic forms of psychopathology (see Fig. 1).

The notion of adaptive constancy represents the process by which changes in neural, genetic and environmental factors achieve a level of durability that continually promotes chronic antisocial behavior. These adaptations are important for defining subtypes of antisocial behavior. However, to test theories that differentiate subtypes of antisocial behavior based on factors across multiple levels of analysis, a number of changes in the research endeavor must occur.

First, it is essential for researchers to appropriately identify the traits or behaviors being assessed and to use assessments that actually tap those behaviors. For example, some studies assess impulsive and antisocial traits as if they were equivalent to psychopathic traits. Each trait is worthy of assessment in terms of its relationship to antisocial behavior, but the bottom line is that researchers must call the constructs being assessed what they are rather than using terms loosely. Failure to do so has diluted the body research in a way that has reduced the meaningfulness of key constructs and measures. Second, researchers must select appropriate samples. That is, samples should be representative of individuals who engage in the behaviors of interest and not individual who were selected for their convenience (e.g., undergraduate samples, healthy community participants). In addition, samples should be diverse (e.g., race, ethnicity, sex/gender, socioeconomic status). In this way, we can identify whether certain factors are operating across subtypes of individuals, as well as their behaviors. To achieve appropriate sample diversity, individuals should be selected not based on diagnostic or behavioral category, but on levels of specific antisocial traits, behaviors, or factors of interest. Third, research should use a multimethod approach. That is, in a single study antisocial behavior should be explored in different ways (e.g., various self-report measures, manipulating experimental conditions, several assays) and examined at several levels (e.g.,

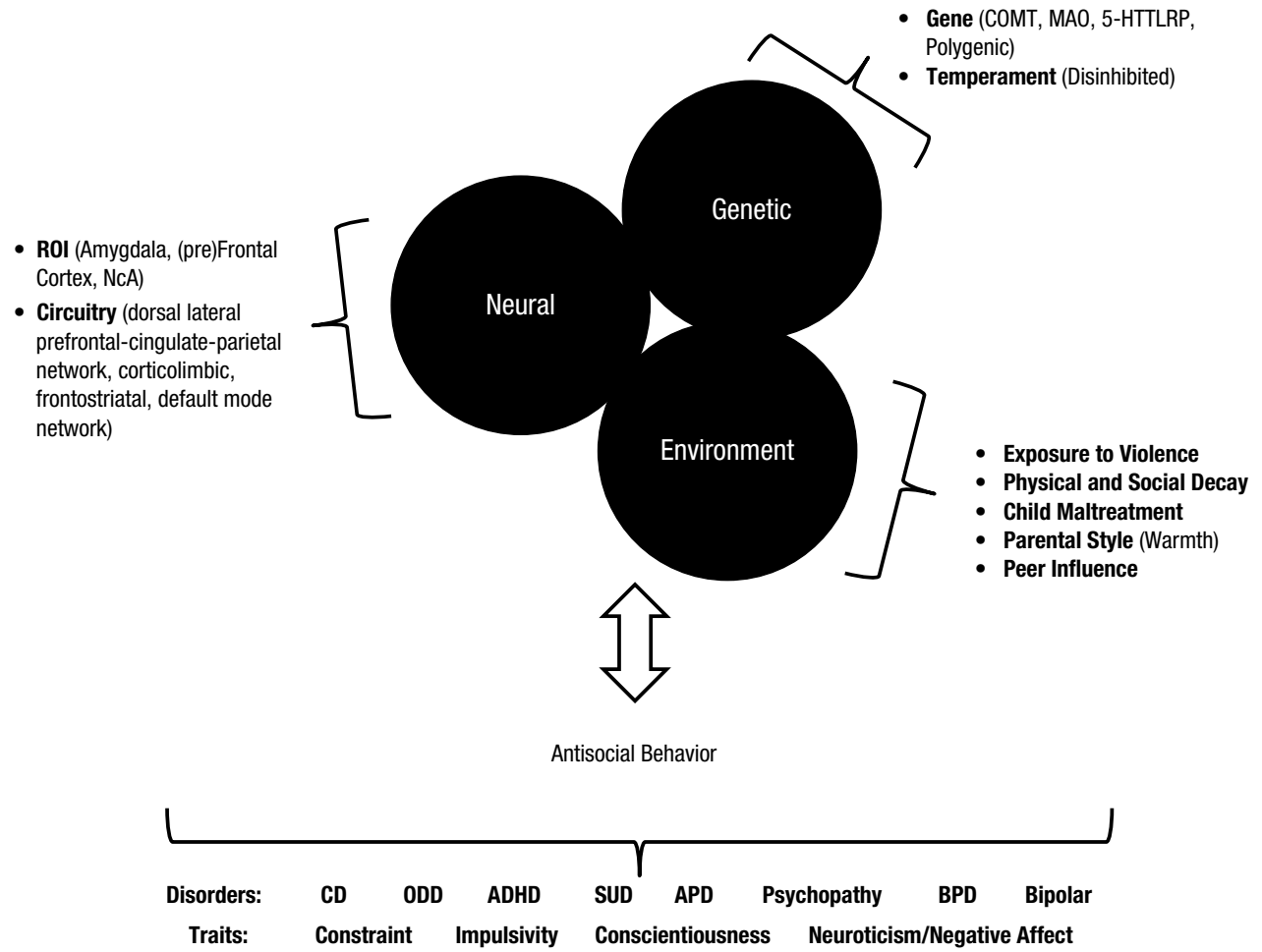

Fig. 1. Summary of potential neural, genetic, and environmental factors contributing to antisocial behavior and its related pathologies. Note: ADHD = attention-deficit/hyperactivity disorder; APD = antisocial personality disorder; BPD = borderline personality disorder; CD = conduct disorder; COMT = catechol O-methyltransferase; MAO = monoamine oxidase; NcA = nucleus accumbens; ODD = oppositional defiant disorder; ROI = region of interest; SUD = substance use disorder.

neural, genetic, environmental). By moving toward more specific assessment, diverse samples, and multiple measures of factors at various levels we can uncover the independent and unique factors that predict and shape multiple pathways toward antisocial behavior.

The current series highlights research moving in the direction of a multilevel methodological framework that is focused on understanding antisocial behavior and its related factors. The five articles included in the series are consistent with changes in the research methods that were proposed earlier.

The Burt et al., Hyde et al., Sadeh et al., Buckholtz et al., and Lynam et al. articles demonstrate the increased specificity achieved by using representative samples and integrative assessments across neural, genetic, or environmental levels to parse the factors associated with subtypes of antisocial behaviors and syndromes. First, Burt and colleagues use a twin sample to examine the genetic and neighborhood factors that contribute to two subtypes of antisocial behaviors, rule breaking and aggression. Second, the article by Hyde and colleagues assesses the impact of community factors associated with low-income neighborhoods and neural factors associated with amygdala development on antisocial behavior. Third, in veterans, Sadeh and colleagues explore the effects of genetic polygenic risk and executive dysfunction on externalizing psychopathology. Fourth, in a sample of inmates, Buckholtz and colleagues parse the neural underpinnings of interference suppression and response inhibition in psychopathic and externalizing individuals. Finally, using a longitudinal sample of at-risk youth, Lynam and colleagues discuss the need for ensuring that the psychometric properties of measures align with the theoretical conceptualization of the construct, specifically with regard to the association between fearless dominance and psychopathy. Together

these studies demonstrate how identifying specific factors at multiple levels of analysis facilitates the identification of variables that need to be controlled for or addressed in experimental design, for developing integrative theory that addresses issues of convergence and divergence, and for designing interventions that are increasingly more efficacious.

Antisocial behavior produces suffering, for the individual, for their family members, for their community, and for society at large. It is important that the underlying factors tell us *why* that individual continues to engage in these behaviors despite the persistence of suffering. Furthermore, a focus on factors at multiple levels of analysis highlights potential targets for alleviating that suffering (see work by Baskin-Sommers, Curtin, & Newman, 2015; Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012; Kazdin, 1997).

Author Contributions

A. R. Baskin-Sommers is the sole author of this article and is responsible for its content.

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