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# Sensation seeking and impulsive traits as personality endophenotypes for antisocial behavior: Evidence from two independent samples

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ABSTRACT

Sensation seeking and impulsivity are personality traits that are correlated with risk for antisocial behavior (ASB). This paper uses two independent samples of twins to (a) test the extent to which sensation seeking and impulsivity statistically mediate genetic influence on ASB, and (b) compare this to genetic influences accounted for by other personality traits. In Sample 1, delinquent behavior, as well as impulsivity, sensation seeking and Big Five personality traits, were measured in adolescent twins from the Texas Twin Project. In Sample 2, adult twins from the Australian Twin Registry responded to questionnaires that assessed individual differences in Eysenck's and Cloninger's personality dimensions, and a structured telephone interview that asked participants to retrospectively report DSM-defined symptoms of conduct disorder. Bivariate quantitative genetic models were used to identify genetic overlap between personality traits and ASB. Across both samples, novelty/sensation seeking and impulsive traits accounted for larger portions of genetic variance in ASB than other personality traits. We discuss whether sensation seeking and impulsive personality are causal endophenotypes for ASB, or merely index genetic liability for ASB.


## 1. Introduction

Antisocial behaviors (ASB) are a constellation of problematic and deviant behaviors that violate laws, social norms or the rights of others. The ASB continuum includes symptoms of DSM-defined psychiatric disorders (e.g., conduct disorder and antisocial personality disorder), as well as less severe behaviors, such as lying to parents or getting in trouble at school. ASB is moderately to highly heritable (Mason and Frick, 1994; Rhee and Waldman, 2002), but the pathway from genotype to ASB phenotype remains largely unknown (Dick et al., 2011; Pappa et al., 2015; Tielbeek et al., 2012; Trzaskowski, Dale, and Plomin, 2013). One approach to help understand how genetic risk is translated into complex behavioral phenotypes, such as ASB, is to identify endophenotypes (Gottesman and Gould, 2003). Endophenotypes are intermediary constructs that bridge the gap between genotype and individual differences in a complex phenotype. In this paper, we consider the hypothesis that sensation seeking and impulsive traits index genetic liability for ASB and, as such, function as personality endophenotypes for ASB. We begin by defining endophenotypes more precisely and discussing why identifying endophenotypes for ASB is a potentially useful endeavor, even if the risk alleles for putative endophenotypes are no more easily identified than those for ASB itself (Flint and Munafò, 2007). We then describe previous correlational and behavior genetic research on the association between personality and ASB, and present evidence from two independent samples that sensation seeking and impulsive traits account for substantial proportions of genetic variance in ASB.

### 1.1. Definition and criteria for an endophenotype

Endophenotypes are biological or psychological constructs that are heritable, hypothesized to be primary to a phenotype of interest, and may vary continuously or manifest as distinct classes. Endophenotypes are "state-independent" (Gottesman and Gould, 2003, pp. 639). That is, for dichotomously classified diseases, such as DSM-defined psychiatric disorders, endophenotypes manifest in individuals regardless of whether

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the associated disorder is present. For example, a person can exhibit a high level of an endophenotype even if they do not meet criteria for Conduct Disorder or Antisocial Personality Disorder. Moreover, endophenotypes should prospectively predict the phenotype of interest in longitudinal studies (Cannon and Keller, 2006; Frederick and Iacono, 2006). There has also been discussion of whether it is necessary that an endophenotype *cause* variation in a complex phenotype or merely provide an index of genetic liability (Kendler and Neale, 2010; Walters and Owen, 2007). Regardless, a putative endophenotype should, at a minimum, share genetic variance with a phenotype of interest.

Contrary to the original conception of endophenotypes, current evidence suggests that the genetic architecture of an endophenotype may be no simpler than that of complex behavioral phenotypes (Flint and Munafò, 2007; Flint, Timpson, and Munafò, 2014; Iacono, Malone, Vaidyanathan & Vrieze, 2014). In other words, the specific alleles that contribute to polygenic risk for an endophenotype may be no fewer or more easily identified than the risk alleles for the "downstream" phenotype of interest. This is certainly the case for personality traits (De Moor et al., 2012; Verweij et al., 2010). As a consequence, identifying personality endophenotypes may not be particularly useful for identifying novel molecular genetic associations with ASB. Yet endophenotypes remain useful for understanding the development of psychopathology by providing more clearly defined links to the biological correlates of complex psychological phenotypes. Emerging prior to the onset of clinical symptoms, personality endophenotypes may help target youth who are at heighted risk for psychopathology. The assessment of externalizing disorders often includes asking children and teens about socially prohibited or illegal behavior; in contrast, measurement of personality, at face value, involves fewer demand characteristics. Finally, identifying personality endophenotypes for ASB may open avenues for research using animal models, which can employ experimental manipulations (e.g. gene knockout, experimental ablation, pharmacological intervention) that are unfit for use with human participants.

### 1.2. Personality as endophenotype: impulsivity and sensation seeking

Personality traits are defined as cognitive, affective and motivational tendencies that are relatively consistent across context and time. *Sensation seeking* is a personality trait that reflects the tendency to pursue and enjoy novel and stimulating experiences. *Impulsivity* is a related yet distinct construct that reflects deficits in perseverance, planning, and inhibitory control. Results of multitrait-multimethod analysis provide evidence for high discriminant validity among measures of sensation seeking and impulsive traits (Smith et al., 2007). Furthermore, sensation seeking and impulsivity show different patterns of association with externalizing behaviors (e.g., alcohol-use; Magid, MacLean, and Colder, 2007) and have distinct developmental trajectories (Harden and Tucker-Drob, 2011; Peach and Gaultney, 2013) that map onto dissociable neurobiological systems (Steinberg, 2010; Steinberg et al., 2008). In addition, a recent meta-analysis of self-report and behavioral measures of impulsive personality confirms that sensation seeking and impulsivity comprise distinct factors (Sharma, Markon, and Clark, 2014).

Importantly, sensation seeking and impulsive traits meet the conceptual criteria for endophenotypes (e.g. state-independence). Furthermore, results of cross-sectional and longitudinal studies of the associations between sensation seeking, impulsivity and ASB are consistent with an endophenotype hypothesis. Both traits show concurrent associations with antisocial and delinquent behavior (Mann, Kretsch, Tackett, Harden, and Tucker-Drob, 2015; Peach and Gaultney, 2013) and positively correlate with externalizing behaviors, including substance-use disorders (Verdejo-García, Lawrence, and Clark, 2008) and risky sexual behavior (McCoul and Haslam, 2001), which pose considerable risk to health and well-being, like ASB, but do not fit cleanly into the ASB continuum. There is also considerable evidence supporting the contention that sensation seeking and impulsivity are primary to ASB in the causal chain from genotype to phenotype. Individual differences in sensation seeking and impulsivity emerge early in childhood (Aksan & Kochanska, 2004; Laucht, Becker, and Schmidt, 2006) and prospectively predict ASB and associated health-risk behaviors in longitudinal studies (Caspi et al., 1997; Farrington, 1995; Masse and Tremblay, 1997; Moffitt and Harrington, 1996; Murray and Farrington, 2010; Newcomb and McGee, 1991; Olson, Schilling, and Bates, 1999; Raine, Reynolds, Venables, Mednick, and Farrington, 1998), whereas ASB does not predict future sensation seeking (Harden, Quinn, and Tucker-Drob, 2012).

Evidence from past behavior genetic research is also largely consistent with sensation seeking and impulsive traits functioning as endophenotypes for ASB. For example, both personality traits are moderately to highly heritable (Bezdijian, Baker, and Tuvblad, 2011; Koopmans, Boomsma, Heath & van Doornen, 1995; Stoel, De Geus, and Boomsma, 2006). With respect to impulsive personality, genetic correlations with DSM-defined externalizing disorders have been documented (Blonigen, Hicks, Krueger, Patrick, and Iacono, 2005) and a large ($N > 1000$) multivariate twin study found that impulsivity (or low constraint) loaded positively onto a highly heritable ($h^2 = 90\%$) externalizing factor that captured variance common to conduct disorder, alcohol dependence, drug dependence and ASB (Krueger et al., 2002). With respect to individual differences in sensation seeking, Waldman et al. (2011) found that genetic influences on children's` preference for novelty, intensity, and danger (i.e. "daring" dispositions) were shared with genetic influences on conduct disorder symptoms, even after accounting for common variance attributable to genetic and environmental associations with prosociality and negative emotionality. Furthermore, a nationally representative study of U.S. adolescents found that a substantial portion (>80%) of genetic influences on longitudinal change in delinquency was mediated by genes influencing longitudinal change in sensation seeking (Harden et al., 2012).

Additionally, neurobiological correlates of impulsivity and sensation seeking have been identified (Buckholtz et al., 2010; Casey, Jones, and Somerville, 2011; Congdon and Canli, 2008; Roberti, 2004), and both traits are commonly measured in non-human subjects (Dent, Isles, and Humby, 2014; Fox, Hand, and Reilly, 2008; Zuckerman, 1984). In contrast, the construct of ASB – particularly rule-breaking forms of ASB – involves evaluating behavior with reference to a socially-defined and culturally-specific set of norms and rules, and is thus considerably more difficult to operationalize in non-human animals. To conclude, results from previous studies are consistent with an endophenotype hypothesis by (1) providing evidence for the causal primacy of personality to ASB, (2) highlighting sensation seeking and impulsive traits as longitudinal predictors of ASB and (3) providing evidence that both traits act as (statistical if not causal) mediators of genetic influences on ASB.

### 1.3. Dimensional models of broad personality traits

Previous behavior genetic research on the relationship between sensation seeking, impulsivity, and ASB has typically examined pair-wise associations in isolation, rather than considering them alongside a number of alternative traits. In this section, we describe dimensional models of personality, and then discuss how these models relate to sensation seeking, impulsivity, and ASB.

The Big Five model (i.e. the Five Factor model) describes variation in personality along five broad dimensions: *extraversion*, *agreeableness*, *conscientiousness*, *neuroticism* and *openness to experience*, under which more specific facets are subsumed (John, Naumann, and Soto, 2008). *Extraversion* encompasses socially uninhibited and emotionally expressive tendencies, such as assertiveness, gregariousness, and excitement seeking. *Agreeableness* captures prosocial and group-oriented tendencies, such as altruism, trust, modesty and tender-mindedness. *Conscientiousness* describes cognitive and motivational processes that help facilitate long-term planning and goal-directed behavior, and *neuroticism* describes tendencies toward negative emotionality, including depression and anxiety. *Openness to experience* taps into the depth and complexity