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 influence 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 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 portions of genetic variance in ASB other than personality traits. We discuss whether sensation seeking and impulsive personality are causal endophenotypes for ASB, or merely index genetic liability for ASB.
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 (Flett and Munafo, 2007; Flett, Timpson, and Munafo, 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 heightened 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 unifit 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 perseveration, 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 Gaulnutey, 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 Gaulnutey, 2013) and positively correlate with externalizing behaviors, including substance-use disorders (Verdejo-Garcia, 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 (h² = 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 (Buckholz 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...
of one’s mental health, as well as the motivation and willingness to entertain novel ideas and perspectives.

Alternative measures of personality include the Eysenck Personality Questionnaire (EPQ) (Eysenck and Eysenck, 1975) and Cloninger’s Tri-dimensional Personality Questionnaire (TPQ) (Cloninger, 1986, 1987), which are derived from models that posit three broad dimensions of variation in personality: psychoticism, extraversion, and neuroticism, or alternatively, harm avoidance, novelty seeking, and reward dependence. Thus, in addition to extraversion and neuroticism, the Eysenck personality scheme highlights psychoticism as a dimension of personality that captures tendencies toward psychopathology (e.g., schizophrenia and bipolar disorder), as well as dispositions toward prosocial and environmental overlap between personality traits and ASB in two independent samples. In the Texas sample, we hypothesize that sensation seeking and novelty seeking traits will account for more additive genetic variance in ASB than Big Five personality traits. Similarly, in the Australia sample, we hypothesize that novelty seeking and impulsive traits will mediate more additive genetic variance in ASB than the remaining dimensions in the Eysenck and Cloninger personality schemes.

2. Method

2.1. Samples

2.1.1. Texas twin sample

The Texas sample consisted of 835 adolescents from 410 families (396 twin pairs, 13 sets of triplets and 1 set of quadruplets), ages 13–20 years (mean age = 15.87 years, SD = 1.36 years) from the Texas Twin Project (TXT) (Harden, Tucker-Drob, and Tackett, 2013). Adolescent twins were identified from public school rosters and recruited via invitation by phone call or mailing to visit the University campus to complete a battery of psychological assessments, including questionnaires that assessed individual differences in personality and ASB. Parents and adolescents signed consent prior to participation, and the university IRB approved all testing procedures. Participants were assured that a federal certificate of confidentiality obtained from NIH protects their identifiable information. The racial composition of the sample was approximately 60% non-Hispanic White, 20% Hispanic/Latino, 15% African-American, 1% Native American, 5% Asian and 4% mixed-race/other. Approximately 10% of adolescents’ parents had a high school degree or less, 30% completed some college or trade school, 25% completed a bachelor’s degree and 31% pursued or completed graduate training.

2.1.2. Australia twin sample

The Australia sample consisted of adult twins (99% non-Hispanic White) from the Australian Twin Registry (ATR). The full sample consisted of participants combined across four independent assessments. Details regarding participant recruitment and sample demographics can be found elsewhere (see Lynskey et al., 2002; Meier, Slutske, Heath, and Martin, 2011; Slutske, Blaszczynski, and Martin, 2009; Slutske et al., 1997; Slutske et al., 2002). Personality traits were measured using two mail-based surveys. A total of N = 6979 participants returned the Health and Lifestyle Survey for Twins, which was completed by participants from 1988 to 1991. Of the 6979 twins who returned the survey, 4261 twins had no missing data across study variables (i.e. age, sex, zygosity, and all indicators of personality). A total of N = 6367 participants returned the Australian Health Study of Twins and Families, which was completed by participants from 1990 to 1992. Of the participants that returned the survey, 2513 twins had no missing data across study variables. The Semi-Structured Assessment for the Genetics of Alcoholism (Buchoz et al., 1994) was completed by participants from 1992 to 1993 and by a second cohort from 1996 to 2000. ASB was measured in both cohorts via a structured telephone interview that asked participants to retrospectively report symptoms of conduct disorder. From the 1992–1993 cohort, partial data is available for N = 6894 participants and complete data (i.e. no missing values) for study variables is available for N = 2732 participants. From the 1996–2000 cohort, partial data are available for N = 6007 participants and complete data are available for N = 5840 participants. Participant reports of personality and ASB were matched across studies using a

1 The Australia sample combined participants across different cohorts of data collection. To assess whether observed patterns of variance and covariance varied as a function of cohort membership, correlations between study variables were estimated in a model that accounted for additive genetic effects across cohorts. This model was compared to a reduced model that estimated correlations between study variables constraining coefficients to be equal across cohorts. Using BIC as an indicator of model fit, the more parsimonious model showed better fit to the data (BIC = 166,591.33), compared to a model that allowed coefficients to be freely estimated across cohorts (BIC = 166,656.46). Additional indexes of model fit confirmed that collapsing estimates across cohort does not result in poor model fit (RMSEA = 0.023; CFI = 0.995; TLI = 0.989; SRMR = 0.024).
unique identification number and combined to create a final dataset. The ages of participants span 17–87 years (mean age = 32.51, SD = 12.49 years).

2.2. Measures

2.2.1. Texas twin sample

2.2.1.1. Big Five personality scale. In the Texas sample, extraversion (α = 0.81), agreeableness (α = 0.75), conscientiousness (α = 0.78), neuroticism (α = 0.80) and openness to experience (α = 0.75) were measured using the Big Five Inventory (BFI), which consists of 44 items comprising five broad factors of personality (John et al., 2008). Adolescents rated items on a 5-point scale ranging from 1 (Strongly Disagree) to 5 (Strongly Agree). The Scale scores for each of the Big Five factors were computed using the method described by Soto, John, Gosling, and Potter (2008), which includes ipsatization to control for individual differences in response sets (i.e., extreme responding and acquiescence). A large body of empirical evidence supports the construct validity of the Big Five, including convergent and discriminant validity across multiple raters and instrument types, as well as predictive validity for important life outcomes (Deary, Weiss, and Batty, 2010; DeYoung, 2006; Judge, Higgins, Thoresen, and Barrick, 1999; Paunonen, 2003; Roberts, Kuncel, Shiner, Caspi, and Goldberg, 2007).

2.2.1.2. Sensation seeking. Sensation seeking (α = 0.73) was measured using the Brief Sensation Seeking Scale (BSSS), which includes items such as “I would like to explore strange places” and “I prefer friends who are excitingly unpredictable.” All items were rated on a scale ranging from 1 (Strongly Disagree) to 5 (Strongly Agree). Previous research has found that the BSSS shows high reliability and construct validity (Stephenson, Hoyle, Palmgreen, and Slater, 2003).

2.2.1.3. Impulsivity. Impulsive personality traits were measured using the urgency (α = 0.86), (low) premeditation (α = 0.84), and (low) perseverance (α = 0.83) subscales of the UPPS Impulsive Behavior Scale, for which the psychometric properties are well established (Whiteside and Lynam, 2001; Whiteside, Lynam, Miller & Reynolds, 2005).

2.2.1.4. ASB. Adolescents provided self-reports of delinquent behavior (α = 0.87) using a 36-item survey adapted from Huizinga, Esbensen, and Weihler (1991). Items were rated on a 3-point scale (1 = Never, 2 = Once, 3 = More than once) and varied in severity from minor violations to relatively severe criminal offenses. Examples of minor violations include, “been loud, rowdy, or unruly in a public place” and “been suspended or expelled from school.” More severe offenses include, “carried a hidden weapon (a knife or a gun).” Honest reporting was encouraged by reminding participants that the study was granted a federal certificate confidentiality, which enables investigators to refuse to disclose information in response to legal demands.

2.2.2. Australia twin sample

2.2.2.1. Eysenck Personality Questionnaire - Revised. In the Australia sample, psychotism, extraversion, neuroticism and impulsivity were measured using Eysenck’s Personality Questionnaire - Revised (Eysenck and Eysenck, 1975). The psychotism scale (α = 0.52) includes items such as, “Would you like others to be afraid of you?” and “Do you prefer to go your own way rather than act by the rules?” The extraversion scale (α = 0.83) includes the items “Are you rather lively?” and “Can you usually let yourself go and enjoy yourself at a lively party?” The neuroticism scale (α = 0.78) includes the items, “Does your mood often go up and down?” and “Are you an irritable person.” On the other hand, “Do you stop to think things over before doing anything?” (R) and “Do you often make decisions on the spur of the moment?” are items that index impulsivity (α = 0.50).

2.2.2.2. Cloninger’s tridimensional personality questionnaire. Novelty seeking, harm avoidance, and reward dependence were measured using Cloninger’s Tridimensional Personality Questionnaire (Cloninger, Przybeck, and Svrakic, 1991; Cloninger, Svrakic, and Przybeck, 1993). Novelty seeking (α = 0.74) is measured by items such as, “When nothing new is happening, I usually start looking for something that is thrilling or exciting”, “I enjoy saving money more than spending it on entertainment or thrills” (R) and “I like to stay at home better than to travel or explore new places” (R). The items “I often stop what I am doing because I get worried, even when my friends tell me everything will go well” and “I am often moved by a fine speech or poetry” measure harm avoidance (α = 0.84) and reward dependence (α = 0.62), respectively. All items were rated on a dichotomous scale (0 = No, 1 = Yes) and responses were summed across subscales to form composite scores.

2.3. Zygosity

All opposite-sex twin pairs are necessarily dizygotic (DZ). In both samples, the determination of zygosity for same-sex pairs was based on questionnaire items regarding physical similarity and ease of being mistaken for the other twin. In the Australia sample, both twins completed these items. In the Texas sample, twins, the twins’ parents and two research assistants following the twins’ laboratory visit completed these items. Responses were analyzed using latent class analysis (LCA), which assigns individuals to subgroups within a population (e.g., monozygotic [MZ] and dizygotic [DZ] twins). Compared to zygosity classification by genotyping, LCA of questionnaire data has been found to have a misclassification rate of < 1% (Heath et al., 2003). In the Texas sample, the LCA solution identified 35% of same-sex pairs as MZ and 65% as DZ and had an entropy statistic of 0.999, which indicates very little uncertainty in classifying pairs. In the Australia sample, approximately 45% and 55% of the sample was identified as MZ and DZ, respectively. Based on a previous study using a subsample of data from the current project (Meier et al., 2011), genotyping of over 200 same-sex twin pairs indicated that zygosity classification had an error rate of only 2.5%.

2.4. Analyses

For both studies, analyses were conducted in three steps using Mplus software version 7.1 (Muthén and Muthén, 2012). Full information maximum likelihood was used to account for missing data (Enders and Bandalos, 2001), which enables the inclusion of data from twins with or without a participating co-twin, as well as twins that provided measures of personality, but not ASB, and vice versa.

First, descriptive statistics and histograms were examined for each variable. Psychotism (in the Australia sample) and both measures of ASB (delinquency and conduct disorder symptoms) were log transformed to correct for positive skew. Next, phenotypic correlations were estimated with standard errors adjusted for nonindependence of data from siblings living in the same household (Asparouhov and Muthén, 2006). This approach was necessary because siblings from the same family contributed nested observations to phenotypic
associations. In the Texas sample, this approach was also necessary for behavioral genetic models, because triplets and quadruplets from the same family provided nested observations to twin pair correlations.

Univariate twin models\(^2\) partition variance in a phenotype into latent additive genetic, shared environmental and non-shared environmental factors. The additive genetic (A) factor represents shared genes (99.99\% in MZ twins and 50\% in DZ twins) that make siblings similar to each other. In addition to shared genes, shared environmental (C) factors that occur at the family-level (such as socioeconomic status, family structure, culture and religion) may also contribute to sibling similarity. Non-shared environmental (E) factors that are uniquely experienced by each twin (such as differential parenting or peer groups) make siblings different from one another. The non-shared environment may also include the effects of measurement error and any genetic differences (e.g., mutations) between identical twins (Charney, 2012).

Bivariate twin models partition the variance in two phenotypes, and their covariance, into latent genetic and environmental components. In the current project, Cholesky decompositions (see Fig. 1) were used to identify personality traits that mediate genetic variance in ASB. Personality traits were modeled primary to ASB to reflect the hypothesis that affective and motivational dispositions (i.e., personality traits) are primary to specific manifest behaviors.

Phenotypic variance in ASB was then decomposed into genetic and environmental factors common to, and unique of, personality traits. Specifically, we calculated the proportion of total variance in ASB due to genetic influences shared with each personality trait using the following equation\(^1\):

\[
a^{2}_{\text{shared}} = a_{12}^2 + e_{12}^2 + a_{2}^2 + c_{2}^2 + e_{2}^2.
\]

In a subsample of the current data, Meier et al. (2011) found sex differences in the etiology of conduct disorder. In the Texas sample, on the other hand, there was no evidence for qualitative or quantitative sex differences in the genetic and environmental influences on adolescents’ delinquent behavior (see Table S1). Moreover, past behavior genetic research provides mixed results for sex differences in the etiology of ASB (cf. Meier et al., 2011). Therefore, given that exploring sex differences was not a goal of the current manuscript, we fit two-group (MZ and DZ) models in the Texas sample that analyzed male and female twin pairs together, controlling for the linear and quadratic effects of age, sex, age × sex interaction and race (McGue and Bouchard, 1984). In the Australia sample, we fit five-group models that analyzed female, male and opposite-sex twin pairs separately, which allows parameter estimates to vary across biological sex, controlling for the sex-specific linear and quadratic effects of age. Thus, in the Texas sample, results of phenotypic and behavior genetic analyses collapse across sex, whereas in the Australia sample, results are reported separately for females and males.

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\(^{2}\) In both samples univariate twin models were fit to all phenotypes. For the sake of brevity, these results were omitted from the body of the manuscript. Model fit statistics and parameter estimates can be found in the online supplement. First, ACE models were fit to provide a baseline model for comparing alternative biometric structures. When MZ twin-pair correlations were more than double DZ twin-pair correlations, dominance genetic influences (D) were estimated in lieu of shared environmental influences (C). The model that maximized predictive fit (ACE or ADE) was then compared to a more parsimonious model that only estimated additive genetic and non-shared environmental influences (AE). Models were compared using \(\chi^2\) difference tests, Akaike Information Criteria (AIC) and Bayesian information criteria (BIC). When model fit indices led to equivocal decisions regarding the best-fitting models, more parsimonious models were carried forward for subsequent analyses. Substantial shared environmental variances in ASB and non-additive genetic variances in personality traits are consistent with those reported in previous behavior genetic studies (Keller, Coventry, Heath, and Martin, 2005; Lewis, Haworth, and Plomin, 2014; Rhee and Waldman, 2002).

\(^{1}\) Notation corresponds to path coefficients depicted in Fig. 1.

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### 3. Results

#### 3.1. Phenotypic analyses

In the Texas sample, ASB showed the strongest associations with sensation seeking (\(r = 0.47\), \(SE = 0.04\), \(p < 0.001\)), and urgency (\(r = 0.37\), \(SE = 0.03\), \(p < 0.001\)), a facet of impulsivity. In the Australia sample, ASB showed the strongest associations with novelty seeking for females (\(r = 0.23\), \(SE = 0.02\), \(p < 0.001\)) and males (\(r = 0.26\), \(SE = 0.02\), \(p < 0.001\)) as well as impulsivity for females (\(r = 0.22\), \(SE = 0.02\), \(p < 0.001\)) and males (\(r = 0.20\), \(SE = 0.02\), \(p < 0.001\)). Table 1 summarizes the descriptive statistics and partial phenotypic correlations between study variables.

#### 3.2. Behavioral genetic analyses

Bivariate Cholesky decompositions were used to identify genetic and environmental influences on ASB shared with personality traits. In both samples, ASB was regressed on the latent genetic and environmental factors that influence personality traits, and additionally allowed to have unique genetic and environmental components. Fit indices (model \(\chi^2\), RMSEA and CFI) are reported in Table 2, and parameter estimates are reported in Table 3.

In the Texas sample, of the total variance in ASB there were significant portions (\(p < 0.01\)) of additive genetic variance shared with lack of conscientiousness (9\%), sensation seeking (30\%), urgency (21\%) and lack of premeditation (20\%). Portions of additive genetic variance in ASB shared with lack of perseverance (8\%) approached, but did not reach, statistical significance. These results are shown in the top panel of Fig. 2. Recast in terms of the genetic variance in ASB, approximately 23\% was shared with genetic influences on lack of conscientiousness and 65\% was shared with genetic influences on sensation seeking, 51\% was shared with genetic influences on urgency, and 49\% was shared with genetic influences on lack of premeditation.

In the Australia sample, of the total variance in female ASB there were significant portions (\(p < 0.01\)) of additive genetic variance shared with psychoticism (4\%), extraversion (3\%), novelty seeking (8\%) and impulsivity (9\%). These results are shown in panel 2 of Fig. 2. Of the genetic variance in female ASB, 9\% was shared with genetic influences on...
psychoticism and 8% was shared with genetic influences on extraversion; 21% and 25% was shared with genetic influences on novelty seeking and impulsivity, respectively. Similar results were found for males in the Australia sample (see panel 3, Fig. 2); of the total variance in male ASB there were significant portions (p < 0.01) of additive genetic variance shared with psychoticism (3%), novelty seeking (13%) and impulsivity (12%). Of the genetic variance in male ASB, approximately 13% was shared with genetic influences on psychoticism, 47% was shared with genetic influences on novelty seeking and 47% was shared with genetic influences on impulsivity.

4. Discussion

In both samples, sensation seeking and impulsive personality traits were more strongly associated with ASB than other broad dimensions of personality, and accounted for greater proportions of genetic variance in ASB. These results are consistent with conceptualizing sensation seeking and impulsivity as personality endophenotypes for ASB.

There are two predominant classes of endophenotype models that have been discussed in the behavior genetic literature: a liability-index model and a mediation model (Kendler and Neale, 2010; Walters and Owen, 2007). A liability-index model of endophenotypes, also known as a risk indicator model, holds that a common set of genes contribute to variance in both a complex phenotype and a hypothesized endophenotype. A mediation model of endophenotypes holds that genes contribute to variance in a complex phenotype via prior effects on hypothesized endophenotypes; thus, what distinguishes the two models is that a mediation model posits a causal relationship between endophenotypes and the downstream phenotype of interest and a liability-index model does not. Nonetheless, both models predict that endophenotypes and associated phenotypes will have overlapping portions of genetic variance.

Past research has found evidence that sensation seeking is causally primary to ASB (Harden et al., 2012), suggesting that a mediation model best captures the relationship between the two constructs. The current study found that sensation seeking and impulsive traits are the primary drivers of genetic overlap between personality risk and ASB. Given the use of cross-sectional data, however, the current project is unable to distinguish between liability-index and mediation models of endophenotypes. The theoretical corollary is that we are unable to determine whether sensation seeking and impulsive traits are causal mechanisms that link polygenic risk to ASB; results of the current project are equally consistent with interpreting sensation seeking and impulsive traits as alternative phenotypic expressions of the same underlying set of genes that contribute to individual differences in ASB. Put differently, it is quite possible that sensation seeking and impulsivity do not cause ASB but rather are non-clinical or sub-threshold expressions of polygenic risk for ASB. Future research efforts focused on testing a personality endophenotype hypothesis should replicate these results, as well as test whether impulsive traits are
causally primary to ASB. This may be achieved by conducting an autoregressive cross-lagged analysis of impulsivity and ASB measured longitudinally in a panel design (cf. Harden et al., 2012).

Results of the current project are also consistent with a dual systems model of risk-taking behavior (Steinberg, 2010). This model posits that adolescents, compared to young children and adults, experience heightened imbalance in two distinct neurobiological systems thought to underlie sensation seeking and impulsive behavior (Steinberg et al., 2008). In turn, high levels of sensation seeking and impulsivity are hypothesized to cause an increased propensity toward risk-taking behaviors, including those typically considered part of the ASB continuum. Thus, by documenting significant portions of additive genetic overlap between sensation seeking, impulsivity and ASB, results of the current project demonstrate that dual systems processes are at play in ASB, that is, a significant portion of genes that contribute to variance in dual systems processes also contributes to variance in antisocial forms of risk-taking behavior.

However, the current results stand in contrast to predictions derived from a socioemotional model of ASB. Specifically, Lahey and Waldman (2003, 2005) predict that children with a tendency toward negative emotionality (similar to high levels of neuroticism) are at heightened risk for ASB. In support of this hypothesis, a recent study found that variance shared between negatively emotionality and conduct disorder symptoms was largely attributable to a common underlying factor (Waldman et al., 2011). In the current study, however, there was no evidence of genetic overlap between neuroticism and ASB. Perhaps such disparate findings may be expected given the wide range of effect sizes (range of $r = -0.31$ to 0.34) found for the phenotypic association between neuroticism and ASB (Miller and Lynam, 2001). Future research efforts stand to benefit from identifying the constellation of moderating factors, both internal and external, that explain why neuroticism confers risk for ASB at certain times, but not others.

There are a number of limitations to the current project. In particular, the Texas sample was only moderate in size ($N = 835$). This limitation was overcome by replicating results in the Australian sample ($N = 7500$). There was no racial diversity in the Australian sample, and measures of personality and ASB were non-concurrent$^4$ and combined across cohorts. The Texas sample, on the other hand, was racially diverse and measures of personality and ASB were obtained concurrently. In these respects, the strengths and weakness unique to each sample are complementary to each other.

All constructs in the current project were measured using self-report questionnaires completed by either adolescent or adults. Although good agreement has been found between self- and informant-reports of personality (Heath, Neale, Kessler, Eaves, and Kendler, 1992), previous behavioral genetic research on ASB using multiple informants (parents, teachers, experimenters and twins) indicates that there is systematic variance in ASB both common and specific to different informants and, moreover, are differentially influenced by genetic factors (Arseneault et al., 2003). Therefore, results of the current study should be considered preliminary prior to replication using multiple informants.

Because the current study used a classical twin design, we had only limited power to differentiate between nonadditive genetic effects and shared environmental influences shared with each personality trait. CI.95% = 95% confidence intervals for $R^2$ estimate. $p =$ probability that $R^2$ estimate is greater than zero.

\[ R^2 = \text{explained variance} = \frac{\text{explained variance}}{\text{total variance}} \]

**Table 3.** Parameter estimates from Cholesky decompositions of bivariate associations between personality traits and antisocial behavior.

<table>
<thead>
<tr>
<th>Trait</th>
<th>Female</th>
<th>Male</th>
<th>$R^2$</th>
<th>CI.95%</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extraversion</td>
<td>0.52 (0.08)</td>
<td>0.59 (0.03)</td>
<td>0.85 (0.05)</td>
<td>0.21 (0.09)</td>
<td>0.03 (0.04)</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>0.52 (0.08)</td>
<td>0.59 (0.03)</td>
<td>0.86 (0.05)</td>
<td>0.21 (0.09)</td>
<td>0.15 (0.04)</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>0.52 (0.08)</td>
<td>0.58 (0.04)</td>
<td>0.92 (0.06)</td>
<td>0.18 (0.05)</td>
<td>0.08 (0.05)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>0.48 (0.09)</td>
<td>0.59 (0.05)</td>
<td>0.85 (0.05)</td>
<td>0.05 (0.10)</td>
<td>0.07 (0.05)</td>
</tr>
<tr>
<td>Openness</td>
<td>0.59 (0.07)</td>
<td>0.59 (0.05)</td>
<td>0.81 (0.05)</td>
<td>0.03 (0.01)</td>
<td>0.03 (0.06)</td>
</tr>
<tr>
<td>Sensation seeking</td>
<td>0.59 (0.07)</td>
<td>0.59 (0.05)</td>
<td>0.71 (0.03)</td>
<td>0.54 (0.06)</td>
<td>0.11 (0.05)</td>
</tr>
<tr>
<td>Urgency</td>
<td>0.63 (0.07)</td>
<td>0.63 (0.04)</td>
<td>0.81 (0.04)</td>
<td>0.46 (0.07)</td>
<td>0.13 (0.05)</td>
</tr>
<tr>
<td>Premeditation (lack of)</td>
<td>0.56 (0.08)</td>
<td>0.58 (0.05)</td>
<td>0.81 (0.04)</td>
<td>0.45 (0.08)</td>
<td>0.06 (0.05)</td>
</tr>
<tr>
<td>Perseverance (lack of)</td>
<td>0.63 (0.06)</td>
<td>0.63 (0.05)</td>
<td>0.78 (0.05)</td>
<td>0.28 (0.08)</td>
<td>0.04 (0.05)</td>
</tr>
</tbody>
</table>

Notes. Model estimator = maximum likelihood with robust standard errors. Secondary variable: antisocial behavior. TXT = Texas Twin Project. ATR = Australia Twin Registry. $\Delta R^2$ = the proportion of total variance in antisocial behavior due to additive genetic influences shared with each personality trait. CI.95% = 95% confidence intervals for $\Delta R^2$ estimate. $p =$ probability that $\Delta R^2$ estimate is greater than zero.
shared environmental effects. Additional limitations of a classical twin design include inattention to epigenetic effects (i.e., gene × environment interactions) and assortative mating, which decreases heritability estimates if present. Nevertheless, the current study provides evidence that genetic variance in ASB, specifically conduct disorder and delinquent behavior, is partially accounted for by genetic variance in sensation seeking, impulsive, and non-conscientious personality traits. Psychoticism and extraversion (only for females) also accounted for significant, albeit small, portions of genetic variance in ASB. To the extent that there was variance in ASB distinct from personality risk, it reflected additive genetic, shared and non-shared environmental influences on the individual (plus measurement error). These results were consistent across two independent samples, across different measures of personality and across biological sex.

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