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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

of one's mental life, 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 Tridimensional 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 ASB (e.g., "going your own way rather than acting by the rules"). Principal components and factor analyses indicate that psychoticism aligns closely with low levels of conscientiousness and agreeableness, compared to the other Big Five traits (Aluja, García & García, 2002; Zuckerman, Kuhlman, Joireman, Teta, and Kraft, 1993). In the revised version of the EPQ, additional items were added to measure impulsive personality because the original version was perceived as insufficiently assessing the construct (Eysenck and Eysenck, 1977). Examples of such items include, "Do you stop to think things over before doing anything?" (Reverse scored), "Have people said that you sometimes act too rashly?" and "Do you often make decisions on the spur of the moment?"

Cloninger's personality scheme, on the other hand, posits broad dimensions that capture tendencies toward shyness and fearful apprehension (harm avoidance); exploratory, hasty and impulsive behavior (novelty seeking); and openness to, and dependence on, warm communication with others (reward dependence). Relative to the Big Five framework, harm avoidance is associated with increased neuroticism and decreased extraversion; reward dependence with extraversion, agreeableness, and openness; and novelty seeking with decreased conscientiousness and increased openness (Capanna et al., 2012; De Fruyt, Van De Wiele & Van Heeringen, 2000). Sensation seeking also aligns closely with novelty seeking in Cloninger's personality scheme. In fact, Zuckerman and Cloninger (1996) found that the correlation between sensation seeking and novelty seeking is nearly perfect after correcting for attenuation due to unreliability in both measures, which provides evidence that the scales measure the same construct.

A prodigious body of research has examined associations between broad dimensions of personality and ASB. Miller and Lynam (2001) conducted a meta-analysis of the relations between broad dimensions of personality and ASB, including studies that measured the Big Five, Eysenck's and Cloninger's personality dimensions. A more recent meta-analysis (Jones, Miller, and Lynam, 2011) reviewed a decade of research on associations between personality and ASB, focusing exclusively on the Big Five dimensions of personality. Consistent across meta-analytic results, agreeableness and conscientiousness are the Big Five dimensions of personality that show the strongest associations with ASB (mean correlations span −0.23 to −0.37). Associations between extraversion and ASB, as well as openness to experience and ASB, approximate zero. On average, neuroticism shows a weak positive association with ASB (mean $r = 0.09$) but with a wide range of effect sizes (range of $r = -0.31$ to 0.34). Not surprisingly, in Eysenck's personality scheme psychoticism is most strongly related to ASB (mean $r = 0.39$). In Cloninger's model, novelty seeking and reward dependence tend to positively (mean $r = 0.34$) and negatively (mean $r = -0.12$) correlate with ASB, respectively.

1.4. Goals of the current study

In the current project, we compare the magnitude of genetic variance accounted for by impulsivity and sensation/novelty seeking to that accounted for by other common dimensions of personality. Specifically, we fit bivariate biometric models that test latent additive genetic and environmental overlap between personality traits and ASB in two independent samples. In the Texas sample, we hypothesize that sensation seeking and impulsive 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 multiples 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 (Bucholz 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,[1] 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 allowed coefficients to be freely estimated 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 confirm 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 ($\alpha = 0.81$), agreeableness ($\alpha = 0.75$), conscientiousness ($\alpha = 0.78$), neuroticism ($\alpha = 0.80$) and openness to experience ($\alpha = 0.75$) 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 ($\alpha = 0.73$) 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 ($\alpha = 0.86$), (low) premeditation ($\alpha = 0.84$), and (low) perseverance ($\alpha = 0.83$) 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 ($\alpha = 0.87$) using a 36-item survey adapted from Huizinga, Esbensen, and Weiher (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 violation 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, psychoticism, extraversion, neuroticism and impulsivity were measured using Eysenck's Personality Questionnaire - Revised (Eysenck and Eysenck, 1975). The psychoticism scale ($\alpha = 0.52$) 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 ($\alpha = 0.83$) includes the items "Are you rather lively?" and "Can you usually let yourself go and enjoy yourself at a lively party?" The neuroticism scale ($\alpha = 0.78$) 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 ($\alpha = 0.50$).

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 ($\alpha = 0.74$) 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 ($\alpha = 0.84$) and reward dependence ($\alpha = 0.62$), respectively. All items were rated on a dichotomous scale (0 = No, 1 = Yes) and responses were summed across subscales to form composite scores.

2.2.2.3. ASB. ASB ($\alpha = 0.63$) was measured using retrospective reports of DSM-defined (version III) conduct disorder symptoms assessed during a structured telephone interview. There were 17 symptoms including, "Did you ever steal money or things from your home or family?" "Did you ever damage someone's property on purpose?" and "Were you ever arrested for anything other than traffic violations?" Participants indicated whether they had experienced symptoms prior to 18 years of age and affirmative responses were summed to calculate the total number of reported symptoms.

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 M*plus* 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. Psychoticism (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[3]:

$$a^2_{shared} = \frac{a_{12^2}}{a_{12^2} + e_{12^2} + a_{2^2} + c_{2^2} + e_{2^2}}.$$

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 (c.f. 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.

[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 indexes 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).

[3] Notation corresponds to path coefficients depicted in Fig. 1.

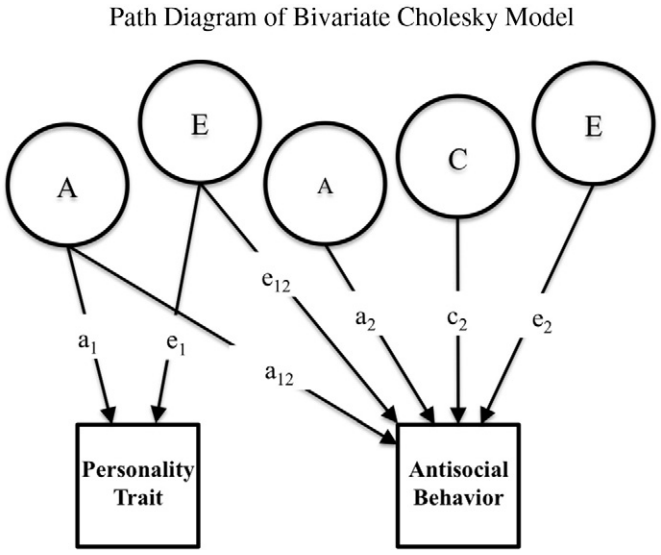

Fig. 1. A secondary phenotype (e.g. ASB) is regressed on the latent genetic and environmental components of a primary phenotype (e.g. personality trait). Path diagram is shown for one twin only.

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 ($r = 0.26$, $SE = 0.021$, $p < 0.001$), 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 χ^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

Table 1
Descriptive statistics and phenotypic correlations between personality traits and antisocial behavior.

Texas sample	*N*	M (SD)	E	A	C	*N*	O	SS	URG	PRE	PER	ASB
Extraversion (E)	835	3.24 (0.73)	1	**.12**	.04	**−.20**	.02	**.21**	.02	**.21**	**−.16**	**.14**
Agreeableness (A)	835	3.72 (0.53)		1	**.25**	**−.29**	.00	−.06	**−.33**	**−.20**	**−.22**	**−.25**
Conscientiousness (C)	835	3.35 (0.61)			1	**−.21**	.02	**−.20**	**−.33**	**−.46**	**−.68**	**−.28**
Neuroticism (N)	835	2.85 (0.68)				1	.01	.00	**.46**	.00	**.22**	**.09**
Openness (O)	835	3.83 (0.51)					1	.08	−.01	−.06	−.06	−.06
Sensation seeking (SS)	835	3.18 (0.70)						1	**.26**	**.36**	.06	**.47**
Urgency (URG)	835	2.13 (0.57)							1	**.25**	**.30**	**.37**
Premeditation (PRE)	835	2.04 (0.48)								1	**.44**	**.31**
Perseverance (PER)	835	1.99 (0.50)									1	**.21**
Delinquency (ASB)	835	6.54 (7.03)										1
Australia sample	Male		Female									
	N	M (SD)	*N*	M (SD)	P	E	*N*	IMP	NS	HA	RD	ASB
Psychoticism (P)	2872	1.13 (0.56)	5203	0.87 (0.57)	1	**.15**	**−.09**	**.21**	**.26**	**−.20**	**−.16**	**.15**
Extraversion (E)	2749	7.93 (3.92)	4990	7.95 (3.91)	**.09**	1	**−.20**	**.32**	**.43**	**−.56**	**.34**	**.16**
Neuroticism (N)	2905	4.60 (3.41)	5251	5.72 (3.46)	−.04	**−.21**	1	**.18**	.01	**.62**	.04	.03
Impulsivity (IMP)	2967	2.66 (1.47)	5347	2.53 (1.51	**.22**	**.29**	**.17**	1	**.52**	**−.11**	**.15**	**.22**
Novelty seeking (NS)	2780	8.09 (3.82)	4974	7.74 (3.55)	**.30**	**.41**	.00	**.52**	1	**−.24**	**.17**	**.23**
Harm avoidance (HA)	2827	6.00 (4.16)	5137	7.93 (4.32)	**−.14**	**−.55**	**.60**	**−.11**	**−.24**	1	**−.12**	**−.09**
Reward dependence (RD)	2829	9.90 (3.25)	5110	11.61 (2.95)	**−.19**	**.38**	.01	**.14**	**.13**	**−.18**	1	.03
Symptom count (ASB)	3064	1.13 (0.53)	4699	0.64 (0.52)	**.15**	**.12**	**.06**	**.20**	**.26**	−-.02	.00	1

Notes. In the Australia sample, correlations for females and males are reported upper and lower off diagonal, respectively, and control for sex-specific linear and quadratic effects of age. In the Texas sample correlations control for linear and quadratic effects of age, sex, age × sex and race. *N* = number of observations. Means (M) and standard deviations (SD) are reported for non-transformed variables. Partial correlations are reported for transformed variables. Estimates highlighted bold are significant at $p < 0.01$.

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.

Table 2
Fit Indices from Cholesky decompositions of bivariate associations between personality traits and antisocial behavior.

TXT sample					
Primary phenotype	χ^2	df	*p*	RMSEA	CFI
Extraversion	31.18	19	.04	.05	.90
Agreeableness	14.43	19	.76	.00	1.0
Conscientiousness	24.95	18	.13	.04	.96
Neuroticism	34.49	19	.02	.06	.86
Openness	16.50	19	.62	.00	1.0
Sensation seeking	21.50	19	.31	.03	.99
Urgency	20.84	19	.35	.02	.99
Premeditation (lack of)	20.42	19	.37	.02	.99
Perseverance (lack of)	23.29	19	.23	.03	.97
ATR sample					
Primary phenotype	χ^2	df	*p*	RMSEA	CFI
Psychoticism	67.23	52	.08	.02	.98
Extraversion	61.57	52	.17	.01	.99
Neuroticism	47.55	52	.65	.00	1.00
Novelty seeking	73.62	52	.03	.02	.98
Impulsivity	66.44	52	.09	.02	.99
Harm avoidance	47.85	52	.64	.00	1.00
Reward dependence	68.33	52	.06	.02	.98

Notes. χ^2 = model chi squared. df = degrees of freedom. RMSEA = root mean squared error of approximation. CFI = comparative fit index. Sibling contrast effects were modeled for conscientiousness.

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

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

TXT sample											
Primary phenotype		A_1	E_1	b_A	b_E	A_2	C_2	E_2	a^2_{shared}	CI.95%	p
Extraversion		.52 (0.08)	.85 (0.05)	.21 (0.09)	.03 (0.04)	.59 (0.13)	.41 (0.15)	.65 (0.04)	.05	.00, 0.12	.22
Agreeableness		.52 (0.08)	.86 (0.05)	−.21 (0.09)	−.15 (0.04)	.56 (0.13)	.45 (0.12)	.64 (0.04)	.04	.00, 0.12	.24
Conscientiousness		.82 (0.06)	.62 (0.05)	−.31 (0.06)	−.08 (0.05)	.56 (0.14)	.40 (0.15)	.64 (0.05)	.09	.03, 0.16	<0.01
Neuroticism		.48 (0.09)	.88 (0.05)	.05 (0.10)	.07 (0.05)	.62 (0.12)	.42 (0.14)	.65 (0.04)	.00	.00, 0.02	.79
Openness		.59 (0.07)	.81 (0.05)	−.03 (0.09)	−.03 (0.06)	.63 (0.13)	.42 (0.15)	.65 (0.05)	.00	.00, 0.01	.88
Sensation seeking		.70 (0.05)	.71 (0.03)	.54 (0.06)	.11 (0.05)	.40 (0.20)	.33 (0.17)	.64 (0.05)	.30	.17, 0.43	<0.001
Urgency		.57 (0.06)	.81 (0.04)	.46 (0.07)	.13 (0.05)	.45 (0.16)	.38 (0.14)	.64 (0.04)	.21	.08, 0.35	<0.01
Premeditation (lack of)		.58 (0.05)	.81 (0.04)	.45 (0.08)	.06 (0.05)	.46 (0.20)	.40 (0.14)	.65 (0.05)	.20	.06, 0.34	<0.01
Perseverance (lack of)		.63 (0.06)	.78 (0.05)	.28 (0.08)	.04 (0.05)	.56 (0.14)	.43 (0.14)	.65 (0.04)	.08	.00, 0.16	.07
ATR sample											
Primary phenotype		A_1	E_1	b_A	b_E	A_2	C_2	E_2	a^2_{shared}	CI.95%	p
Psychoticism-	Female	.59 (0.02)	.82 (0.02)	.19 (0.04)	.05 (0.02)	.59 (0.04)	.19 (0.09)	.75 (0.02)	.04	.01, 0.07	<0.01
	Male	.57 (0.03)	.80 (0.02)	.18 (0.05)	.05 (0.03)	.46 (0.13)	.48 (0.11)	.73 (0.02)	.03	.00, 0.07	.04
Extraversion-	Female	.67 (0.02)	.74 (0.01)	.18 (0.03)	.03 (0.02)	.59 (0.04)	.19 (0.09)	.76 (0.02)	.03	.01, 0.06	<0.01
	Male	.68 (0.03)	.73 (0.02)	.13 (0.04)	.04 (0.03)	.49 (0.12)	.46 (0.12)	.73 (0.02)	.02	.00, 0.04	0.11
Neuroticism-	Female	.66 (0.02)	.75 (0.01)	.08 (0.03)	−.03 (0.03)	.61 (0.04)	.19 (0.09)	.76 (0.02)	.00	.00, 0.02	.22
	Male	.64 (0.03)	.77 (0.02)	.07 (0.04)	.01 (0.03)	.51 (0.12)	.46 (0.12)	.73 (0.02)	.00	.00, 0.00	.40
Novelty seek.-	Female	.62 (0.02)	.78 (0.02)	.28 (0.03)	.07 (0.02)	.54 (0.05)	.22 (0.09)	.75 (0.02)	.08	.04, 0.11	<0.001
	Male	.59 (0.03)	.81 (0.02)	.36 (0.04)	.05 (0.03)	.38 (0.15)	.46 (0.11)	.72 (0.02)	.13	.06, 0.19	<0.001
Impulsivity-	Female	.55 (0.02)	.83 (0.01)	.30 (0.04)	.06 (0.02)	.52 (0.05)	.23 (0.08)	.76 (0.02)	.09	.04, 0.13	.001
	Male	.51 (0.04)	.86 (0.02)	.35 (0.05)	.03 (0.03)	.37 (0.16)	.47 (0.11)	.73 (0.02)	.12	.05, 0.20	<0.001
Harm avoid.-	Female	.64 (0.02)	.77 (0.01)	−.10 (0.03)	−.03 (0.02)	.61 (0.04)	.19 (0.09)	.75 (0.01)	.01	.00, 0.02	.11
	Male	.63 (0.03)	.78 (0.02)	−.07 (0.05)	−.01 (0.03)	.50 (0.12)	.47 (0.11)	.73 (0.02)	.01	.00, 0.02	.42
Reward dep.-	Female	.57 (0.02)	.82 (0.02)	.09 (0.04)	−.01 (0.02)	.61 (0.04)	.20 (0.09)	.76 (0.02)	.01	.00, 0.02	.88
	Male	.63 (0.03)	.80 (0.02)	−.06 (0.05)	.03 (0.03)	.51 (0.12)	.47 (0.11)	.73 (0.02)	.00	.00, 0.01	.63

Notes. Model estimator = maximum likelihood with robust standard errors. Secondary variable: antisocial behavior. TXT = Texas Twin Project. ATR = Australia Twin Registry. a^2_{shared} = the proportion of total variance in antisocial behavior due to additive genetic influences shared with each personality trait. CI.95% = 95% confidence intervals for a^2_{shared} estimate. p = probability that a^2_{shared} estimate is greater than zero.

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 (c.f. 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 in pleiotropy with 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 heighted 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 set of underlying genes (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 Australia sample ($N \sim 7500$). There was no racial diversity in the Australia 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

[4] The Australia sample used in the current project was constructed post-hoc for secondary data analysis, i.e. to perform a replication. Consequently, measures of personality were obtained from a mail-based survey prior to obtaining reports of conduct disorder symptoms via telephone interview. The response intervals between measures of personality and conduct disorder symptoms varied from 4 months to 10 years (median response interval = 5.03 years). As a form of sensitivity analysis, trivariate twin models were fit, in which genetic and environmental factor loadings (a_1, e_1, a_2, c_2 & e_2), as well as genetic and environmental cross-paths (a_{12} and e_{12}) for females and males were constrained to interact with individual differences in participants' response interval. Importantly, interaction terms on genetic and environmental factor loadings, as well as genetic and environmental cross-paths were small in magnitude (beta range = −0.09 to 0.10) and not significant ($p > 0.01$) for both females and males.

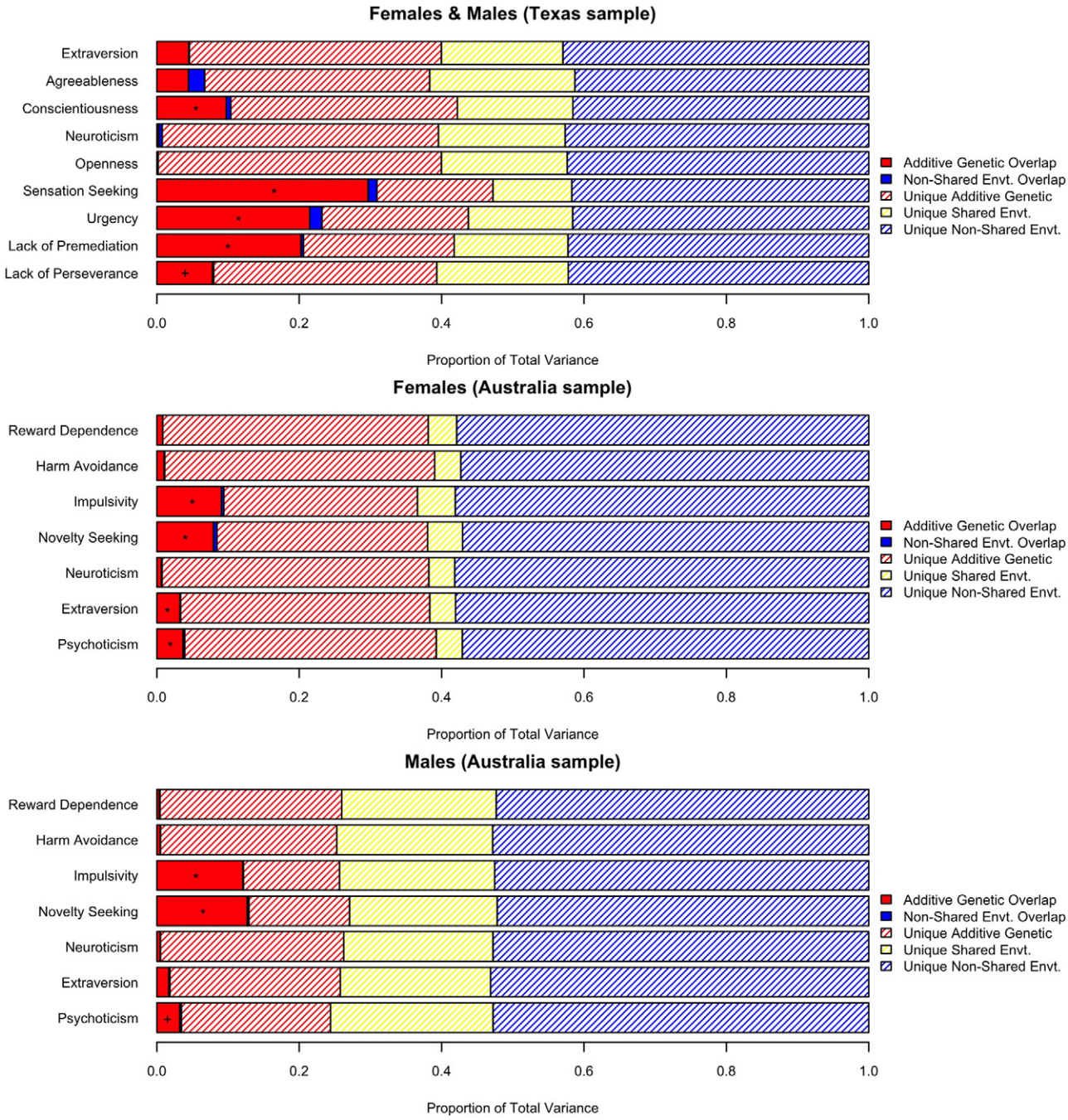

Fig. 2. Latent genetic and environmental overlap between personality & antisocial behavior. Notes. Portions of variance calculated from parameter estimates reported in Table 2. In the Texas sample results control for the linear and quadratic effects of age, sex, age × sex interaction and race. In the Australia sample, results are reported separately for females and males controlling for the sex-specific linear and quadratic effects of age. Asterisks (*) mark estimates of additive genetic overlap that are significantly different from zero at $p < 0.01$. Addition signs (+) mark estimates of additive genetic overlap that are marginally significant from zero at $p < 0.10$.

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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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.paid.2016.09.018.

References

Aksan, N., & Kochanska, G. (2004). Links between systems of inhibition from infancy to preschool years. *Child Development*, *75*(5), 1477–1490.

Aluja, A., Garćia, Ó., & Garćia, L. F. (2002). A comparative study of Zuckerman's three structural models for personality through the NEO-PI-R, ZKPQ-III-R, EPQ-RS and Goldberg's 50-bipolar adjectives. *Personality and Individual Differences*, *33*(5), 713–725.

Arseneault, L., Moffitt, T. E., Caspi, A., Taylor, A., Rijsdijk, F. V., Jaffee, S. R., ... Measelle, J. R. (2003). Strong genetic effects on cross-situational antisocial behaviour among 5-year-old children according to mothers, teachers, examiner-observers, and twins' self-reports. *Journal of Child Psychology and Psychiatry*, *44*(6), 832–848.

Asparouhov, T., & Muthén, B. (2006, August). *Multilevel modeling of complex survey data. In Proceedings of the Joint Statistical Meeting in Seattle, 2718–2726.*

Bezdijian, S., Baker, L. A., & Tuvblad, C. (2011). Genetic and environmental influences on impulsivity: A meta-analysis of twin, family and adoption studies. *Clinical Psychology Review*, *31*(7), 1209–1223.

Blonigen, D. M., Hicks, B. M., Krueger, R. F., Patrick, C. J., & Iacono, W. G. (2005). Psychopathic personality traits: Heritability and genetic overlap with internalizing and externalizing psychopathology. *Psychological Medicine*, *35*(05), 637–648.

Bucholz, K. K., Cadoret, R., Cloninger, C. R., Dinwiddie, S. H., Hesselbrock, V. M., Nurnberger, J. I., Jr., ... Schuckit, M. A. (1994). A new, semi-structured psychiatric interview for use in genetic linkage studies: A report on the reliability of the SSAGA. *Journal of Studies on Alcohol*, *55*(2), 149–158.

Buckholtz, J. W., Treadway, M. T., Cowan, R. L., Woodward, N. D., Li, R., Ansari, M. S., ... Zald, D. H. (2010). Dopaminergic network differences in human impulsivity. *Science*, *329*(5991), 532.

Cannon, T. D., & Keller, M. C. (2006). Endophenotypes in the genetic analyses of mental disorders. *Annual Review of Clinical Psychology*, *2*, 267–290.

Capanna, C., Struglia, F., Riccardi, I., Daneluzzo, E., Stratta, P., & Rossi, A. (2012). Temperament and Character Inventory–R (TCI–R) and Big Five Questionnaire (BFQ): Convergence and divergence. *Psychological Reports*, *110*, 1002–1006.

Casey, B. J., Jones, R. M., & Somerville, L. H. (2011). Braking and accelerating of the adolescent brain. *Journal of Research on Adolescence*, *21*(1), 21–33.

Caspi, A., Begg, D., Dickson, N., Harrington, H., Langley, J., Moffitt, T. E., & Silva, P. A. (1997). Personality differences predict health-risk behaviors in young adulthood: Evidence from a longitudinal study. *Journal of Personality and Social Psychology*, *73*(5), 1052–1063.

Charney, E. (2012). Behavior genetics and postgenomics. *Behavioral and Brain Sciences*, *35*(05), 331–358.

Cloninger, C. R. (1986). A unified biosocial theory of personality and its role in the development of anxiety states. *Psychiatric Developments*, *3*(2), 167–226.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants: A proposal. *Archives of General Psychiatry*, *44*(6), 573–588.

Cloninger, C. R., Przybeck, T. R., & Svrakic, D. M. (1991). The tridimensional personality questionnaire: US normative data. *Psychological Reports*, *69*(3), 1047–1057.

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, *50*(12), 975–990.

Congdon, E., & Canli, T. (2008). A neurogenetic approach to impulsivity. *Journal of Personality*, *76*(6), 1447–1484.

De Fruyt, F., Van De Wiele, L., & Van Heeringen, C. (2000). Cloninger's psychobiological model of temperament and character and the five-factor model of personality. *Personality and Individual Differences*, *29*, 441–452.

De Moor, M. H. M., Costa, P. T., Terracciano, A., Krueger, R. F., de Geus, E. J. C., Toshiko, T., ... Boomsma, D. I. (2012). Meta-analysis of genome-wide association studies for personality. *Molecular Psychiatry*, *17*, 227–249.

Deary, I. J., Weiss, A., & Batty, G. D. (2010). Intelligence and personality as predictors of illness and death how researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities. *Psychological Science in the Public Interest*, *11*(2), 53–79.

Dent, C. L., Isles, A. R., & Humby, T. (2014). Measuring risk-taking in mice: Balancing the risk between seeking reward and danger. *European Journal of Neuroscience*, *39*(4), 520–530.

DeYoung, C. G. (2006). Higher-order factors of the Big Five in a multi-informant sample. *Journal of Personality and Social Psychology*, *91*(6), 1138–1151.

Dick, D. M., Aliev, F., Krueger, R. F., Edwards, A., Agrawal, A., Lynskey, M., ... Bierut, L. (2011). Genome-wide association study of conduct disorder symptomatology. *Molecular Psychiatry*, *16*(8), 800–808.

Enders, C. K., & Bandalos, D. L. (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling*, *8*(3), 430–457.

Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck Personality Questionnaire.* London: Hodder & Stoughton.

Eysenck, S. B., & Eysenck, H. J. (1977). The place of impulsiveness in a dimensional system of personality description. *British Journal of Social and Clinical Psychology*, *16*(1), 57–68.

Farrington, D. P. (1995). The development of offending and antisocial behaviour from childhood: Key findings from the Cambridge Study in Delinquent Development. *Journal of Child Psychology and Psychiatry*, *6*(36), 929–964.

Flint, J., & Munafò, M. R. (2007). The endophenotype concept in psychiatric genetics. *Psychological Medicine*, *37*(2), 163–180.

Flint, J., Timpson, N., & Munafò, M. (2014). Assessing the utility of intermediate phenotypes for genetic mapping of psychiatric disease. *Trends in Neurosciences*, *37*(12), 733–741.

Fox, A. T., Hand, D. J., & Reilly, M. P. (2008). Impulsive choice in a rodent model of attention-deficit/hyperactivity disorder. *Behavioural Brain Research*, *187*(1), 146–152.

Frederick, J. A., & Iacono, W. G. (2006). Beyond the DSM: Defining endophenotypes for genetic studies of substance abuse. *Current Psychiatry Reports*, *8*(2), 144–150.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry*, *160*(4), 636–645.

Harden, K. P., & Tucker-Drob, E. M. (2011). Individual differences in the development of sensation seeking and impulsivity during adolescence: Further evidence for a dual systems model. *Developmental Psychology*, *47*(3), 739–746.

Harden, K. P., Quinn, P. D., & Tucker-Drob, E. M. (2012). Genetically influenced change in sensation seeking drives the rise of delinquent behavior during adolescence. *Developmental Science*, *15*(1), 150–163.

Harden, K. P., Tucker-Drob, E. M., & Tackett, J. L. (2013). The Texas Twin Project. *Twin Research and Human Genetics*, *16*, 385–390.

Heath, A. C., Neale, M. C., Kessler, R. C., Eaves, L. J., & Kendler, K. S. (1992). Evidence for genetic influences on personality from self-reports and informant ratings. *Journal of Personality and Social Psychology*, *63*(1), 85–96.

Heath, A. C., Nyholt, D. R., Neuman, R., Madden, P. A., Bucholz, K. K., Todd, R. D., ... Martin, N. G. (2003). Zygosity diagnosis in the absence of genotypic data: An approach using latent class analysis. *Twin Research*, *6*, 22–26.

Huizinga, D., Esbensen, F. A., & Weiher, A. W. (1991). Are there multiple paths to delinquency? *The Journal of Criminal Law and Criminology*, *82*, 83–118.

Iacono, W., Malone, S., Vaidyanathan, U., & Vrieze, S. I. (2014). Genome-wide scans of genetic variants for psychophysiological endophenotypes: A methodological overview. *Psychophysiology*, *51*(12), 1207–1224.

John, O. P., Naumann, L. P., & Soto, C. J. (2008). Paradigm shift to the integrative Big Five trait taxonomy: History, measurement, and conceptual issues. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality* (pp. 114–158). New York, NY: Guilford Press.

Jones, S. E., Miller, J. D., & Lynam, D. R. (2011). Personality, antisocial behavior, and aggression: A meta-analytic review. *Journal of Criminal Justice*, *39*(4), 329–337.

Judge, T. A., Higgins, C. A., Thoresen, C. J., & Barrick, M. R. (1999). The Big Five personality traits, general mental ability, and career success across the life span. *Personnel Psychology*, *52*(3), 621–652.

Keller, M. C., Coventry, W. L., Heath, A. C., & Martin, N. G. (2005). Widespread evidence for non-additive genetic variation in Cloninger's and Eysenck's personality dimensions using a twin plus sibling design. *Behavior Genetics*, *35*(6), 707–721.

Kendler, K. S., & Neale, M. C. (2010). Endophenotype: A conceptual analysis. *Molecular Psychiatry*, *15*(8), 789–797.

Koopmans, J. R., Boomsma, D. I., Heath, A. C., & van Doornen, L. J. (1995). A multivariate genetic analysis of sensation seeking. *Behavior Genetics*, *25*(4), 349–356.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*, *111*(3), 411–424.

Lahey, B. B., & Waldman, I. D. (2003). A developmental propensity model of the origins of conduct problems during childhood and adolescence. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *The causes of conduct disorder and serious juvenile delinquency* (pp. 76–117). New York, NY: Guilford Press.

Lahey, B. B., & Waldman, I. D. (2005). A developmental model of the propensity to offend during childhood and adolescence. In D. P. Farrington (Ed.), *Advances in criminological theory. Vol. 13.* (pp. 15–50). Piscataway, NJ: Transaction.

Laucht, M., Becker, K., & Schmidt, M. H. (2006). Visual exploratory behaviour in infancy and novelty seeking in adolescence: Two developmentally specific phenotypes of DRD4? *Journal of Child Psychology and Psychiatry*, *47*(11), 1143–1151.

Lewis, G. J., Haworth, C. M. A., & Plomin, R. (2014). Identical genetic influences underpin behavior problems in adolescence and basic traits of personality. *Journal of Child Psychology and Psychiatry*, *55*(8), 865–875.

Lynskey, M. T., Heath, A. C., Nelson, E. C., Bucholz, K. K., Madden, P. A. F., Slutske, W. E. E. A., ... Martin, N. G. (2002). Genetic and environmental contributions to cannabis dependence in a national young adult twin sample. *Psychological Medicine*, *32*(02), 195–207.

Magid, V., MacLean, M. G., & Colder, C. R. (2007). Differentiating between sensation seeking and impulsivity through their mediated relations with alcohol use and problems. *Addictive Behaviors*, *32*(10), 2046–2061.

Mann, F. D., Kretsch, N., Tackett, J. L., Harden, K. P., & Tucker-Drob, E. M. (2015). Person × environment interactions on adolescent delinquency: Sensation seeking, peer deviance and parental monitoring. *Personality and Individual Differences*, *76*, 129–134.

Mason, D. A., & Frick, P. J. (1994). The heritability of antisocial behavior: A meta-analysis of twin and adoption studies. *Journal of Psychopathology and Behavioral Assessment*, *16*(4), 301–323.

Masse, L. C., & Tremblay, R. E. (1997). Behavior of boys in kindergarten and the onset of substance use during adolescence. *Archives of General Psychiatry*, *54*, 62–68.

McCoul, M. D., & Haslam, N. (2001). Predicting high risk sexual behaviour in heterosexual and homosexual men: The roles of impulsivity and sensation seeking. *Personality and Individual Differences*, *31*(8), 1303–1310.

McGue, M., & Bouchard, T. J., Jr. (1984). Adjustment of twin data for the effects of age and sex. *Behavior Genetics*, *14*(4), 325–343.
Meier, M. H., Slutske, W. S., Heath, A. C., & Martin, N. G. (2011). Sex differences in the genetic and environmental influences on childhood conduct disorder and adult antisocial behavior. *Journal of Abnormal Psychology*, *120*(2), 377–388.
Miller, J. D., & Lynam, D. (2001). Structural models of personality and their relation to antisocial behavior: A meta-analytic review. *Criminology*, *39*(4), 765–798.
Moffitt, T. E., & Harrington, H. L. (1996). *Delinquency: The natural history of antisocial behaviour. Child to Adult*. Silva, PA Stanton, WR: Oxford University Press.
Murray, B. A. J., & Farrington, D. P. (2010). Risk factors for conduct disorder and delinquency: Key findings from longitudinal studies. *Canadian Journal of Psychiatry*, *55*(10), 633–642.
Muthén, L. K., & Muthén, B. O. (2012). *Mplus. The comprehensive modeling program for applied researchers: User's guide, 5*.
Newcomb, M. D., & McGee, L. (1991). Influence of sensation seeking on general deviance and specific problem behaviors from adolescence to young adulthood. *Journal of Personality and Social Psychology*, *61*(4), 614–628.
Olson, S. L., Schilling, E. M., & Bates, J. E. (1999). Measurement of impulsivity: Construct coherence, longitudinal stability, and relationship with externalizing problems in middle childhood and adolescence. *Journal of Abnormal Child Psychology*, *27*(2), 151–165.
Pappa, I., St Pourcain, B., Benke, K., Cavadino, A., Hakulinen, C., Nivard, M. G., ... Tiemeier, H. (2015). A genome-wide approach to children's aggressive behavior: The EAGLE consortium. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 1–11.
Paunonen, S. V. (2003). Big Five factors of personality and replicated predictions of behavior. *Journal of Personality and Social Psychology*, *84*, 411–424.
Peach, H. D., & Gaultney, J. F. (2013). Sleep, impulse control, and sensation-seeking predict delinquent behavior in adolescents, emerging adults, and adults. *Journal of Adolescent Health*, *53*(2), 293–299.
Raine, A., Reynolds, C., Venables, P. H., Mednick, S. A., & Farrington, D. P. (1998). Fearlessness, stimulation-seeking, and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. *Archives of General Psychiatry*, *55*(8), 745–751.
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), 490–529.
Roberti, J. W. (2004). A review of behavioral and biological correlates of sensation seeking. *Journal of Research in Personality*, *38*(3), 256–279.
Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science*, *2*(4), 313–345.
Sharma, L., Markon, K. E., & Clark, L. A. (2014). Toward a theory of distinct types of "impulsive" behaviors: A meta-analysis of self-report and behavioral measures. *Psychological Bulletin*, *140*(2), 374–408.
Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A., Bucholz, K. K., Dunne, M. P., ... Martin, N. G. (1997). Modeling genetic and environmental influences in the etiology of conduct disorder: A study of 2682 adult twin pairs. *Journal of Abnormal Psychology*, *106*(2), 266–279.
Slutske, W. S., Heath, A. C., Madden, P. A. F., Bucholz, K. K., Statham, D., & Martin, N. G. (2002). Personality and the genetic risk for alcohol dependence. *Journal of Abnormal Psychology*, *111*, 124–133.
Slutske, W. S., Blaszczynski, A., & Martin, N. G. (2009). Sex differences in the rates of recovery, treatment-seeking, and natural recovery in pathological gambling: Results from an Australian community-based twin survey. *Twin Research and Human Genetics*, *12*(05), 425–432.
Smith, G. T., Fischer, S., Cyders, M. A., Annus, A. M., Spillane, N. S., & McCarthy, D. M. (2007). On the validity and utility of discriminating among impulsivity-like traits. *Assessment*, *14*(2), 155–170.
Soto, C. J., John, O. P., Gosling, S. D., & Potter, J. (2008). The developmental psychometrics of Big Five self-reports: Acquiescence, factor structure, coherence, and differentiation from ages 10 to 20. *Journal of Personality and Social Psychology*, *94*, 718–737.
Steinberg, L. (2010). A dual systems model of adolescent risk-taking. *Developmental Psychobiology*, *52*(3), 216–224.
Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology*, *44*(6), 1764–1778.
Stephenson, M. T., Hoyle, R. H., Palmgreen, P., & Slater, M. D. (2003). Brief measures of sensation seeking for screening and large-scale surveys. *Drug and Alcohol Dependence*, *72*, 279–286.
Stoel, R. D., De Geus, E. J., & Boomsma, D. I. (2006). Genetic analysis of sensation seeking with an extended twin design. *Behavior Genetics*, *36*(2), 229–237.
Tielbeek, J. J., Medland, S. E., Benyamin, B., Byrne, E. M., Heath, A. C., Madden, P. A., ... Verweij, K. J. (2012). Unraveling the genetic etiology of adult antisocial behavior: A genome-wide association study. *PloS One*, 7–10.
Trzaskowski, M., Dale, P. S., & Plomin, R. (2013). No genetic influence for childhood behavior problems from DNA analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*, 1048–1056.
Verdejo-García, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience & Biobehavioral Reviews*, *32*(4), 777–810.
Verweij, K. J., Zietsch, B. P., Medland, S. E., Gordon, S. D., Benyamin, B., Nyholt, D. R., ... Wray, N. R. (2010). A genome-wide association study of Cloninger's temperament scales: Implications for the evolutionary genetics of personality. *Biological Psychology*, *85*, 306–317.
Waldman, I. D., Tackett, J. L., Van Hulle, C. A., Applegate, B., Pardini, D., Frick, P. J., & Lahey, B. B. (2011). Child and adolescent conduct disorder substantially shares genetic influences with three socioemotional dispositions. *Journal of Abnormal Psychology*, *120*(1), 57–61.
Walters, J. T. R., & Owen, M. J. (2007). Endophenotypes in psychiatric genetics. *Molecular Psychiatry*, *12*(10), 886–890.
Whiteside, S. P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, *30*(4), 669–689.
Whiteside, S. P., Lynam, D. R., Miller, J. D., & Reynolds, S. K. (2005). Validation of the UPPS impulsive behaviour scale: A four-factor model of impulsivity. *European Journal of Personality*, *19*(7), 559–574.
Zuckerman, M. (1984). Sensation seeking: A comparative approach to a human trait. *Behavioral and Brain Sciences*, *7*(03), 413–434.
Zuckerman, M., & Cloninger, C. R. (1996). Relationships between Cloninger's, Zuckerman's, and Eysenck's dimensions of personality. *Personality and Individual Differences*, *21*, 283–285.
Zuckerman, M., Kuhlman, D. M., Joireman, J., Teta, P., & Kraft, M. (1993). A comparison of three structural models for personality: The Big Three, the Big Five, and the Alternative Five. *Journal of Personality and Social Psychology*, *65*(4), 757–768.