Published in final edited form as:
J Res Pers. 2015 April 1; 55: 30–40. doi:10.1016/j.jrp.2014.12.005.

Childhood Sexual Abuse and Impulsive Personality Traits: Mixed Evidence for Moderation by *DRD4* Genotype

K. Paige Harden[1,2], **Marie D. Carlson**[1], **Natalie Kretsch**[1], **William R. Corbin**[3], and **Kim Fromme**[1]

[1]Department of Psychology, University of Texas at Austin, Austin, TX 78712

[2]Population Research Center, University of Texas at Austin, Austin, TX 78712

[3]Department of Psychology, Arizona State University, Phoenix, AZ 85287

Abstract

This project examines associations between childhood sexual abuse (CSA) and two dimensions of impulsivity (sensation seeking and premeditation), and tests whether CSA-personality associations are moderated by the *DRD4* exon III VNTR polymorphism. Sample 1 is from a longitudinal study of university students measured at 10 waves over ages 18–24 years (n = 500). Sample 2 is from a national sample of young adult sibling pairs, ages 18–24, from the National Longitudinal Study of Adolescent Health (n = 2,559). In both samples, CSA was associated with elevated sensation seeking. In Sample 1, the association between CSA and sensation seeking was moderated by *DRD4* genotype; this gene × environment interaction effect, however, was not replicated in Sample 2. Results suggest new avenues for research on CSA in the area of normal-range personality variation.

Keywords

sensation seeking; impulsivity; childhood sexual abuse; DRD4; gene-environment interaction; diathesis stress; differential susceptibility

1. Introduction

Childhood sexual abuse (CSA) figures prominently in theoretical models of personality disorders, particularly borderline personality disorder. In addition, history of CSA is associated with elevated risk for mood and anxiety disorders, substance use disorders, eating disorders, suicide, self-injury, and poorer physical health (for a comprehensive review, see Maniglio, 2009). These associations are evident both when using self-reports of CSA and when using social service agency records. Notably, an effect of self-reported CSA is evident

Address correspondence to: Dr. Paige Harden, 108 E. Dean Keeton Stop #A8000, Austin, Texas 78712, harden@psy.utexas.edu, 512.471.1124.

even when using a discordant twin design to control for the confounding effects of other family background factors and passive gene-environment correlation (Kendler et al., 2000; Nelson et al., 2002). Overall, CSA is robustly associated with a panoply of clinical disorders.

Few studies, however, have examined the association between CSA and normative personality variation. In the current paper, we examine the associations between self-reported history of CSA and impulsive personality traits. Impulsivity is a core feature of personality disorder pathology; for example, one of the *DSM*-5 symptoms for borderline personality disorder is "impulsivity in at least two areas that are potentially self-damaging" (American Psychiatric Association, 2013). Impulsive personality traits are also strongly associated with substance use disorders and can be conceptualized as part of the externalizing spectrum (Krueger, Markon, Patrick, Benning, & Kramer, 2007). Given their associations with an array of clinical pathologies, variation in impulsive personality traits may be a "missing link" that connects specific etiological factors, such as CSA, with a spectrum of clinical diagnoses. Consistent with this hypothesis, Wonderlich and colleagues (2001) found that impulsivity statistically mediated the association between CSA and symptoms of eating disorders. More generally, several theorists have encouraged a synthesis of research on "normal-range" individual differences in personality, on the one hand, and "abnormal" personality and affective disorders (Krueger & Tackett, 2003; Krueger et al., 2007). Examining how CSA – a frequently investigated risk factor for psychopathology – relates to impulsive personality traits in a non-clinical sample is consistent with this broader goal.

1.1. Differentiating Facets of Impulsivity

Although often referred to as unitary, impulsivity (the tendency toward rash action) is a heterogeneous construct. In this paper we focus on *sensation seeking*, defined as the preference for novel, exciting, or physically stimulating events and experiences, and *premeditation*, defined as the tendency to think carefully and plan before initiating actions (Whiteside & Lynam, 2001). These facets of impulsivity are differentially related to Big Five personality traits, with sensation seeking most strongly associated with extraversion, whereas premeditation is most strongly associated with conscientiousness (Whiteside & Lynam, 2001). Moreover, facets of impulsivity differentially predict alcohol and substance use, as well as other health risk behaviors and clinical disorders (Deckman & DeWall, 2011; Quinn & Harden, 2013; Whiteside & Lynam, 2003).

Both cross-sectional and longitudinal studies have found evidence that sensation seeking and "impulse control" (mapping most closely to the construct of premeditation) have distinct developmental trends in adolescence and young adulthood: Premeditation increases monotonically through the early lifespan, whereas sensation seeking initially increases in adolescence and then decreases through early adulthood (Steinberg et al., 2008; Harden & Tucker-Drob, 2011). Behavioral genetic research using adult twins has found that genetic influences on sensation seeking are distinct from genetic influences on lack of premeditation, particularly among females (Ellingson, Verges, Littlefield, Martin, & Slutske, 2013). Finally, one previous study found evidence for facet-specific associations with abuse

history. Specifically, using a sample of African American adolescents, Bornovalova, Gwadz, Kahler, Aklin, and Lejeuz (2008) found that self-reported childhood abuse history was related to higher sensation seeking but not to "impulsivity" (measured by the Eysenck Impulsiveness Scale, which maps most closely to lack of premeditation in the UPPS model, Whiteside & Lynam, 2001). Together, the factor analytic, behavior genetic, developmental, and clinical literatures suggest that it is important to differentiate facets of impulsive personality, as they may have unique etiologies and correlates.

1.2. Moderation by DRD4 Genotype

Dopamine is a neurotransmitter that is crucial for brain systems involved in reward, motivation, and exploration (Bromberg-Martin, Mastumoto, & Hikosaka, 2010; DeYoung, 2013; Depue & Collins, 1999). Differences in dopaminergic functioning have been linked to both lack of premeditation and higher sensation seeking (DeYoung, 2013; Norbury, Manohar, Rogers, & Husain, 2013). For instance, Zald et al. (2008) found that reduced binding to the dopamine D2 autoreceptor – which resulted into greater dopaminergic release in response to amphetamine was associated with both impulsive personality traits. Consequently, in addition to examining the association between CSA and impulsive personality traits, this paper also tests whether this relationship is moderated by *DRD4* (dopamine D4 receptor gene) genotype.

Given dopamine's role in reward and motivation, polymorphisms in dopamine-relevant genes have been hypothesized to be specifically relevant to impulsive personality traits. The most commonly studied polymorphism has been a 48-base-pair variable number tandem repeat (VNTR) polymorphism in exon III of *DRD4* (Van Tol et al., 1992). The number of repeats in *DRD4* VNTR range from 2–11, and the 7-repeat allele is commonly operationalized as the "risky" or "vulnerable" allele because of its association with lower dopamine reception efficiency (Asghari et al., 1995). Initial positive associations between *DRD4* genotype and sensation seeking and/or impulsivity were reported in the human literature (e.g., Becker, Laucht, El-Fladdagh, & Schmidt, 2005; Dreber and colleagues, 2008), and similar associations with dopamine-related genes have also been reported in the animal literature (e.g., Dulawa, Grady, Low, Paulus, & Greyer, 1999; Hall & Waynne, 2012). In addition to candidate gene approaches that focus on the effects of a single polymorphism, genomic profiling approaches that have leveraged the aggregate effect of DA-relevant polymorphisms to predict facets of impulsivity have shown some success (e.g., Derringer et al., 2010; Davis & Loxton, 2013).

Studies documenting a main effect of dopamine-related candidate genes on impulsive personality traits, however, have faced valid criticism (e.g., Powell & Zietsch, 2010; Duncan & Keller, 2011). Most importantly, the promising results of individual studies are tempered by meta-analytic results. Munafo, Binnaz, Willis-Owen & Flint (2008), for instance, did not support an omnibus association between *DRD4* and approach-related personality traits. Similarly, null results were reported in a meta-analysis that examined the relation between *DRD4* and novelty seeking (Kluger, Siegfried, & Ebstein, 2002). Overall, these findings reflect a more general trend of failures to replicate in the candidate gene literature. Thus the heritability of impulsive personality traits detected in twin studies (e.g., Fulker, Eysenck, &

Zuckerman, 1980; Harden & Tucker-Drob, 2011; Hur & Bouchard, 1997; Stoel, DeGeus, & Boomsma, 2006) continues to be largely "missing" in the candidate gene literature (Manolio et al., 2009, p. 747).

Beyond the particular relevance of dopamine for personality, prominent developmental theorists have posited that polymorphisms in dopamine-related genes confer *differential susceptibility* to environmental influence more generally (Belsky, Bakermans-Kraenburg & van Ijzendoorn 2007). According to differential susceptibility theory, individuals differ in their plasticity to environmental inputs, such that those with greater plasticity will show more positive outcomes in the context of relatively high quality environments *and* more negative outcomes in the context of relatively poor quality environments. In contrast, persons with low plasticity will be largely impervious to the influence of environmental extremes. Consistent with this framework, low dopaminergic efficiency has been linked with decreased reward and attentional mechanisms (Robbins & Everitt, 1999) and, depending on environmental circumstances, low dopaminergic efficiency could be advantageous or disadvantageous (Suomi, 1997). Tests of differential susceptibility theory using measured genes have focused on a small set of dopamine-related genes, including *DRD4*, *DRD2* (dopamine receptor D2 gene), and *DAT1* (dopamine transporter gene). Findings in this domain have been disseminated with a notably optimistic tone. For example, Bakermans-Kranenburg and van Ijzendoorn (2011), concluded "Our meta-analysis confirmed the role of dopamine-related genes as moderators of the association between positive as well as negative environmental factors and developmental outcome … Differential susceptibility based on dopamine-related genotypes appears to be a replicable finding" (p.48).

In contrast to these optimistic conclusions, results from genome-wide association studies suggest that, on average, researchers' ability to select candidate genes *a priori* is poor, and most psychological studies are underpowered to detect biologically-plausible effect sizes (Duncan & Keller, 2011). Moreover, in highly-multivariate datasets, researchers have many degrees of freedom to pick measures of environmental context, genetic risk, and developmental outcome in order to produce a statistically significant result ("*p*-hacking", Simmons, Nelson, & Simonsohn, 2011), a practice that capitalizes on chance and decreases the replicability of results. Nevertheless, the impact of dopamine-related genes generally – and *DRD4* specifically – on impulsivity has been a topic of long-standing research interest; studies of candidate G×E interactions continue to proliferate in the literature; and these results are used as support for popularized theories of human development, such as differential susceptibility theory. Consequently, there is a continuing need for studies that test predictions about G×E (such as the claims about "differential susceptibility based on dopamine-related genotypes") using replication samples.

Putting these lines of research together, the current study addresses two research questions. First, we examine the extent to which CSA is associated with facets of impulsivity in young adulthood, specifically sensation seeking and premeditation. Second, we evaluate the extent to which the association between CSA and impulsive personality traits is moderated by *DRD4* genotype. We address these questions using two independent samples of young adults. Specifically, initial analyses were conducted in a longitudinal sample of students

from a public university, and replication analyses were conducted using publically-available data from a nationally representative sample of young adults.

2. Method

2.1. Participants

2.1.1. Sample 1—Data for Sample 1 were drawn from a multi-wave longitudinal study (the "UT Experience!" study [UTE]), which focused on substance use and risky behavior in a student cohort that matriculated to a large southwestern public university in 2004 (n = 2,245). Study participants were assessed beginning during the summer following their senior year of high school and followed for six years post matriculation. All first-time students, ages 17–19, were invited to participate (n = 6,319). From this pool, 76% of students (n = 4,832) expressed interest and also met the criterion of being unmarried. From this pool, n = 3,046 students were randomly assigned to complete surveys during high school, each semester during their first three years of college, and in the fall of their fourth, fifth, and sixth year after high school. Eligible participants gave informed consent and completed the surveys using a secure Web server (n = 2,245). Further details on participant recruitment for this portion of the study are given by Corbin, Vaughan, & Fromme (2008). Participants completed 10 waves of data collection between 2004 and 2009. In 2012, a subsample of participants, now ages 26–27, was re-contacted to collect additional survey data, as well as salivary DNA. Data collection and genotyping are still in progress. This paper presents initial data on n = 500 participants (65% female) who have been genotyped. The sample is racially and ethnically diverse (62% White/Caucasian, 16.2% Asian, 14% Hispanic/Latino(a), 6.5% Multi-Ethnic, 1.4% Black/African American). Personality inventories related to impulsive traits were completed by participants at Waves 1 (M age = 18.40), 8 (M age = 21.80), 9 (M age = 22.78), and 10 (M age = 23.78). Relative to the full sample at wave 1, the proportion of females and Whites in the genetic sample was slightly higher (χ^2 = 4.9, df = 1, $p < .05$; χ^2 = 45.24, df = 1, $p < .001$) and the proportion of Blacks was slightly lower (χ^2 = 7.6, df = 1, $p < .01$). No significant differences between the two samples emerged for sensation seeking (t = 0.59, df = 478, p = .56), premeditation (t = −0.2, df = 483, p = .84) or age (t = 0.56, df = 497, p = .58).

2.2.2. Sample 2—Replication analyses were conducted using the National Longitudinal Study of Adolescent Health (Add Health; Udry 2003a), a multi-wave longitudinal study focused on adolescent health and risk behaviors. Add Health data was collected using a multi-stage, stratified, school-based cluster design and includes four waves of data (for a full description see Harris et al., 2009). The first wave of data was collected in 1995, when participant age ranged from 12 to 20 years old, the second wave of data in 1996, the third in 2001/2002 and the fourth wave in 2007/2008. Add Health began by identifying all schools in the United States that had at least 30 enrollees (n = 26,000). Schools were then stratified by size and type, geographic region, racial composition and urbanicity. From these strata, a random sample of schools, grades 7–12 or 9–12, were collected. A total of 134 schools (79%) agreed to participate. Ninety-six percent of these schools (n = 129; student n = 90,118) agreed to have students complete an in-school survey during the 1994–1995 academic year. Among these schools, a randomly selected subsample (n = 20,745)

completed a 90-minute In-Home interview (Wave 1 interview). The study continued with follow up waves of in-home interviews as outlined above. The present study focuses on a subset of sibling pairs for whom Add Health collected DNA (n = 2,559; 52% female). To maximize consistency across samples, personality measures were assessed at Wave 3 when mean participant age was 21.9 (SD = 1.7, *range* = 18–26).

2.2. Measures

2.2.1. Personality

Sample 1: During study Waves 1 and 8–10, all UTE participants completed the novelty seeking subscale of the *Tridimensional Personality Questionnaire* (TPQ; Cloninger, 1987) and the impulsive unsocialized sensation seeking subscale of the *Zuckerman-Kuhlman Personality Questionnaire III-R* (ZKPQ; Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993). The TPQ novelty seeking subscale consists of 32 true-false items designed to index an elevated excitatory response to novel stimuli (Cloninger, 1987). Examples items include *"It is difficult for me to keep the same interests for a long time because my attention often shifts to something else,"* and *"I hate to make decisions based on my first impressions."* The ZKPQ impulsive unsocialized sensation seeking scale is a 19-item true-false inventory including items indexing the desire to seek out physiologically arousing experiences, such as *"I like to have new and exciting experiences and sensations,"* and items indexing a tendency to act without thinking and failure to plan ahead, such as*" I tend to begin a new job without much advance planning on how I will do it,*" and *"I usually think about what I am going to do before doing it."* Because the scales contained items that appeared to tap both premeditation and sensation seeking, we conducted a series of exploratory and confirmatory factor analyses of the personality items (described below).

Sample 2: Personality items were drawn from two sections of the AddHealth Wave 3 interview. Participants were presented with seven pairs of sentences and asked to indicate which one described them better. Examples included "*I like wild uninhibited parties* vs. *I like quiet parties with good conversation*." Participants were also presented with a 9-item scale assessing novelty-seeking and impulse control. Items included "*I often try new things just for fun or thrills, even if most people think they are a waste of time*" and "*When nothing new is happening I usually start looking for something exciting*." Participants rated these items on a 5-point scale, ranging from "not true" to "very true." As with Sample 1, we also analyzed a series of measurement models of Sample 2 personality items (described below).

2.2.2. Childhood sexual abuse

Sample 1: CSA was indexed by coding endorsement of any of the following questions, at any point over Waves 8–10: *"During your childhood, how many times did you experience unwanted sexual contact by guardian, parent, or relative?"; "Before the age of 18, how many times did you experience unwanted sexual contact by adult more than 5 years older than you?"; "Prior to your Senior year in high school, how many times did you feel coerced into having sex (through arguments, pressure, or physical force)?"; "Prior to your Senior year in high school, how many times did you experience unwanted sexual advances or sex*

play (fondling, kissing, petting but not intercourse)?" Twenty-eight percent of participants reported some form of CSA (38% of females and 10% of males).

Sample 2: CSA was indexed by coding any positive endorsement from Waves 3 or 4 to the following question: *"How often had one of your parents or other adult care-givers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?"* Five percent of participants reported CSA (6% of females and 4% of males).

2.2.3. Socioeconomic status—Socioeconomic status (SES) was indexed by taking the mean of residential parents' education. SES was mean-centered and entered as a covariate in all analyses.

Sample 1: Parent education was coded on a 6-point scale ranging from "some education, but did not complete high school or obtain GED" to "professional training beyond a 4-year degree" (M = 4.36 [corresponding approximately to completing junior college or a trade school degree], SD = 1.34).

Sample 2: Parent education was coded on a 9-point scale, ranging from "8th grade or less" to "professional training beyond a 4-year degree" (M = 4.24 [corresponding approximately to completing high school], SD = 2.31).

2.2.4. Genotyping—For both samples, DNA samples were collected and genotyped in collaboration with the Dr. Andrew Smolen at the Institute for Behavior Genetics at the University of Colorado at Boulder. For Sample 1, DNA was collected using Oragene salivary self-collection kits. Participants returned their kits to our lab through the US mail where they were subsequently stored in batches to be sent to Colorado for genotyping, using the same procedures described in Smolen et al. (2013). For Sample 2, DNA was collected using buccal cell swabs according to procedures described in http://www.cpc.unc.edu/projects/addhealth/data/guides/biomark.pdf.

The present analyses focus on the *DRD4* gene, which maps to 11p15.5. This gene contains a 48-base-pair variable number tandem repeat (VNTR) in exon III (Van Tol et al., 1992). The number of repeats ranges from 2–11. Genetic risk is commonly operationalized as being a carrier of the 7-repeat allele due to its association with lower dopamine reception efficiency, which is posited to be linked to decreased attentional and reward mechanisms (Robbins & Everitt, 1999). For all analyses, genotype was coded as the number of 7R alleles (0, 1, or 2). The two samples had similar proportions of individuals with each genotype (*Sample 1:* 67% non-carrier, 28% 7R-heterozygous, 5% 7R-homozygous; *Sample 2:* 63%, 32.5%, & 4.5%, respectively). Both samples were in Hardy-Weinberg Equilibrium (HWE) (*Sample 1:* χ^2 (2) = 3.69, p = .16; *Sample 2*: χ^2 (2) =.645, p = .72).

2.2.5. Controls—Biological sex was coded such that 0 = male and 1 = female. Race and ethnicity were dummy-coded with Caucasians as the reference group. Because Add Health participant ages ranged from 18–26 at Wave 3, it was necessary to control for age in

analyses of Sample 2. In Sample 1, participants were highly age homogenous (within one year) at each wave; therefore, age was not controlled for wave-specific Sample 1 models.

2.3. Analyses

Analyses were conducted using the structural equation software program *Mplus* (Muthen & Muthen, 2013). Full information maximum likelihood was used to account for missing data (Schafer & Graham, 2002). Model fit was assessed with root-mean-square error of approximation (RMSEA), comparative fit index (CFI), and Tucker-Lewis index (TLI). Because the Add Health samples includes sibling pairs, non-independence was corrected using the complex sampling option in M*plus*.

2.3.1. Measurement models of personality—As described in the Introduction, theoretical and empirical work has identified distinct facets of impulsivity, which show divergent developmental trends and may be differentially related to dopamine-related genes (DeYoung, 2013; Harden & Tucker-Drob, 2011; Stautz & Cooper, 2013; Steinberg et al., 2008; Whiteside & Lynam, 2001). Consequently, we first conducted a series of exploratory and confirmatory factor analyses of the personality items in both samples, with the aim of mapping the personality items in the two samples to the sensation seeking versus premeditation constructs.

2.3.2. Candidate gene models—Building off the measurement models of personality, latent factors for sensation seeking and premeditation were regressed on markers of environmental context (CSA and physical abuse), *DRD4*, and interactions between *DRD4* and environmental context, as well as control variables.

3. Results

3.1. Measurement Models of Personality

3.1.1. Sample 1—We first conducted exploratory factor analysis (EFA) of personality items at each wave using geomin rotation in M*plus*. Results were highly similar across all waves, so we focus on Wave 10 results here (results from Waves 1, 8, and 9 available from first author upon request). A scree plot of the eigenvalues indicated a three-factor solution. The first two factors in a three-factor solution corresponded to the constructs of sensation seeking and lack of premeditation, respectively. The third factor, however, was comprised only of three items from the TPQ that referred to money ("I am better at saving money than most people"; "I often spend money until I run out of cash or get into debt from using too much credit;" and "Because I so often spend too much money on impulse, it is hard for me to save money even for special plans like a vacation."). Consequently, we dropped these three money-related items, and performed confirmatory factor analyses (CFA) with the remaining ZKPQ and TPQ items that loaded on the first two factors. We fit separate correlated two-factor models for each of the four waves. Items and standardized factor loadings for these CFA models are presented in Table 1. In each case, overall model fit was good (all RMSEAs < .05, CFIs and TLIs ranged from .90 to .97). Zero-order correlations between all personality items and covariates from Sample 1 are available in the Supplement (Tables S1–S4).

3.1.2. Sample 2—We performed an analogous EFA of Wave 3 personality items from Sample 2. A scree plot of the eigenvalues suggested a one-factor solution, and all items appeared to tap the construct of sensation seeking. These items were subsequently modeled using CFA. Because the sensation seeking items were drawn from two different scales administered at different points in the interview, we fit a bifactor model, in which all items loaded on a general factor, and scale-specific items were allowed to load on a scale-specific factor that accounted for residual covariance among items from the same scale. The CFA model had good fit (RMSEA =.04, CFI =.93, TLI =.90). Items and factor loadings are presented in Table 2. One implication of these measurement model results is that Sample 2 could not be used as a replication sample for analyses of premeditation; rather, Sample 2 is a replication sample only for the construct of sensation seeking. Zero-order correlations between all personality items and covariates from Sample 2 are available in the Supplement (Table S5).

3.2. Regression Models

3.2.1. Sample 1—Regression analyses were conducted separately for each of the four waves. We regressed the latent factors for sensation seeking and premeditation on CSA, *DRD4* genotype, and the interaction between *DRD4* and CSA. All models controlled for race, gender, and SES. All models fit the data well (all model RMSEAs < .05, CFI and TLI's ranging between .90–.95). For sensation seeking (parameter estimates summarized in Table 3), there were significant main effects for both gender and CSA at every wave, such that males and individuals that endorsed CSA had higher levels of sensation seeking. There was no main effect of *DRD4* on sensation seeking, but there were significant interactions between *DRD4* and CSA across waves 1, 9, and 10, with a marginally significant interaction (p = .05) at wave 8. Among individuals who reported a history of CSA, *DRD4* 7R carriers tended to show lower sensation seeking relative to their non-carrier counterparts. Results for sensation seeking are illustrated for all four waves in Figure 1. For premeditation (parameter estimates summarized in Table 4), there was a significant main effect for CSA but only at Wave 1. There were no main effects of gender, race, *DRD4* or genotype, nor were there any significant *DRD4* × CSA interactions at any wave.

3.2.2. Sample 2—Results for Sample 2 are shown in Table 3. The latent factor for Wave 3 sensation seeking was regressed on our contextual predictor (CSA,), our moderator (*DRD4*), and the interaction between *DRD4* and CSA. We controlled for race, gender, SES and age. This model fit the data well (RMSEA = .03, CFI = .93, TLI = .92). We found significant main effects for age, race and CSA. Specifically, younger sample participants, Black individuals, and individuals who endorsed CSA all showed greater sensation seeking on average than their older, Caucasian, and non-abused counterparts. Consistent with findings from Sample 1, there was no main effect of *DRD4* on sensation seeking in Sample 2. In contrast to findings for Sample 1, there was no statistically significant interaction between *DRD4* genotype and CSA. Post-hoc sensitivity analyses in which *DRD4* was coded dichotomously (7R carriers versus non-carriers) were consistent with these null results.

3.3. Sensitivity Analyses

We conducted three sets of sensitivity analyses to address possible explanations for differences in findings across studies (Analyses 1 and 2) and to control for effects of population stratification (Analysis 3). CSA was measured more broadly in Sample 1 than in Sample 2, including two items on unwanted sexual contact with a caregiver or another adult and two more items regarding coerced sex or coerced sexual contact (which was not necessarily with an adult). Thus, we first re-ran the Sample 1 models using an alternative measure of CSA that used only the first two items (regarding unwanted sexual contact with a caregiver or other adult). The percentage of Sample 1 participants endorsing CSA on these two items was more commensurate with the rate seen in Sample 2 (10% in Sample 1 versus 5% in Sample 2). As shown in Table 5, the sensitivity analyses for the alternative coding of CSA were largely consistent with the original results. There was a significant main effect of CSA on sensation seeking at Waves 8, 9, and 10 but not Wave 1. Consistent with primary results, the interaction effects between *DRD4* genotype and CSA were statistically significant for Waves 1, 9, and 10, and the magnitude of the interaction effects were greater in later waves than in earlier waves. Overall, the magnitude of the interaction effects was slightly greater in sensitivity analyses compared to the primary analyses. These results suggest that the discrepancy in results between Sample 1 and Sample 2 is unlikely to be a consequence of including items regarding coerced sex in the operationalization of CSA.

It is also possible that the association between CSA and sensation seeking detected in Sample 1 could reflect a reverse causal association, as (a) sensation seeking in adolescence is associated with earlier age at first sexual intercourse and a greater numbers of sexual partners (e.g., Donohew, Zimmerman, Cupp, Novak, Colon, & Abell, 2000), and (b) greater sexual activity is associated with increased chances of sexual victimization (Eaton, Davis, Barrios, Brener, & Noonan, 2007). To address this concern, we re-ran Sample 1 analyses controlling for lifetime number of sexual partners reported at Wave 1. As shown in Table 5, the pattern of results was largely consistent with the original findings. All waves showed a significant main effect of CSA on sensation seeking. *DRD4* genotype showed significant interactions with CSA at Waves 9 and 10, and interactions approached significance at Waves 1 ($p = .05$) and 8 ($p = .07$). Notably, the magnitude and direction of interaction results was consistent with the primary results.

Finally, to address potential concerns about population stratification in a racially diverse sample, we conducted sensitivity analyses restricted to white participants for all regression models. These results are displayed in Table 5. The magnitudes of the interaction effects were generally consistent with those of the full sample ($\beta_{w1} = -.08$, $\beta_{w8} = -46$, $\beta_{w9} = -.45$, $\beta_{w10} = -.54$) with the exception of Wave 1, which was non-significant and attenuated in magnitude. Wave 9 and 10 interactions were statistically significant at $p < .05$.

4. Discussion

In two independent samples of young adults, self-reports of CSA predicted higher average levels of sensation seeking. Sensation seeking is correlated with a variety of externalizing behaviors and disorders, including alcohol and drug use and sexual risk-taking, as well as suicidality and self-injury (Charnigo et al., 2013; Knorr, Jenkins, & Conner, 2013; Ortin,

Lake, Kleinman, & Gould, 2012; Quinn & Harden, 2013). Thus the current results suggest that sensation seeking is a promising candidate for a "missing link" that connects CSA with an array of clinical conditions. More generally, these results contribute to a growing synthesis between research on normative variation in personality and research on clinically-defined disorders (Krueger & Tackett, 2003).

The current results set the stage for five future lines of inquiry. First, although previous research has linked both CSA and sensation seeking with clinical disorders, and the current study found an association between CSA and sensation seeking, a full mediation model – in which personality is tested as an intermediary of the link between CSA and multiple clinical disorders – has not yet been tested. Neither data set used here has information on DSM-defined clinical conditions, thus this mediation hypothesis awaits testing in future research.

Second, analysis of population-average developmental trends has found that sensation seeking peaks near mid-adolescence and declines through the early 20s (Harden & Tucker-Drob, 2008; Steinberg et al, 2008). As personality data from earlier in adolescence are not available on these participants, it is unclear whether the higher levels of sensation seeking evident in early adulthood would be also be evident earlier in development. Do individuals with a history of CSA experience a larger adolescent "spike" in sensation seeking, or a slower "maturing out" of sensation seeking? Developmental data that spans adolescence would be necessary to address this question.

Third, this study relies on participants' retrospective self-reports of childhood maltreatment. Corroborated reports, such as medical or social services records, may be more "objective" indicators of maltreatment, because they are not subject to participants' biases. However, such records are not available for our sample, nor are they typically not available for the large samples necessary for genetic research. On the other hand, many individuals experience childhood maltreatment but never have contact with any social services agencies. Additionally, a lthough previous research has found that associations between clinical disorders and childhood history of CSA are robust to controls for shared environmental and genetic confounds using a co-twin-control design (e.g., Kendler et al., 2000; Nelson et al., 2002), self-reports of CSA – like any self-reported environmental experience – are necessarily confounded by potential gene-environment correlations. It remains to be seen whether the association between CSA and sensation seeking found here generalizes to the more serious cases of abuse typically captured from corroborated abuse reports or withstands more rigorous quasi-experimental tests of environmental causation.

Fourth, one limitation of the current study is that all facets of impulsivity were not measured; we were limited to sensation seeking and premeditation in Sample 1 and only sensation seeking in Sample 2. Other facets of impulsivity that have been identified include lack of perseverance, defined as the ability to persist until a task is completed despite boredom; negative urgency, defined as the tendency to commit rash actions as a response to negative affect; and positive urgency, defined as the tendency to commit rash actions as a response to positive affect (Whiteside & Lynam, 2001; Cyders & Smith, 2008). Negative urgency, in particular, has been specifically linked with a variety of clinical problems, such as externalizing behaviors and disordered eating (Anestis, Smith, Fink, & Joiner, 2009;

Settles et al., 2012). Future research on the role of childhood maltreatment should examine these clinically important facets of impulsivity.

Fifth, if the association between sexual abuse and sensation seeking represents a causal relationship, the precise mechanism underlying this effect remains unknown. One speculative hypothesis is that these effects are mediated through earlier pubertal maturation. Girls, in particular, with a history of CSA experience menarche at younger ages, on average, than girls who do not have a CSA history (Bergevin, Bukowski, & Karavasilis, 2003; Mendle, Leve, Van Ryzin, Natsuaki, & Ge, 2011; Romans, Martin, Gendall, & Herbison, 2003; Turner, Runtz, & Galambos, 1999; Wise, Palmer, Rothman, & Rosenberg, 2009). CSA has been hypothesized to accelerate pubertal development via its effects on both adrenal and gonadal axes of the endocrine system (Trickett & Putnam, 1993). Interestingly, these effects may be particular for sexual abuse as compared to physical abuse or neglect. In a study of girls from the foster care system, all of whom who had experienced at least one form of maltreatment, Mendle and colleagues (2011) found that sexual abuse (as measured from child welfare case records) uniquely predicted more advanced physical development at baseline and earlier age at menarche. In turn, the neuroendocrine events of puberty – particularly the effects of sex hormones on dopaminergic systems in the brain that undergird reward responses – have been hypothesized to drive adolescent-typical increases in sensation seeking (but to be independent of adolescent gains in premeditation; reviewed in Smith, Chein, & Steinberg, 2013). Future research can test this hypothesis by evaluating a comprehensive model in which the timing of pubertal development is tested as a mediator of the relation between CSA and sensation seeking. A related consideration for females is that impulsivity may be sensitive to hormonal fluctuations that characterize different phases of the menstrual cycle, which was not measured in the present study.

We found mixed evidence for the hypothesis that the *DRD4* genotype moderates the association between CSA and sensation seeking. Notably, even though we detected a statistically significant G×E interaction in Sample 1, the shape of the interaction was not consistent with the prediction that the *DRD4* is a “susceptibility” gene. If the 7R/7R genotype conferred susceptibility to adverse environmental experiences, then one would expect minimal differences between the CSA and no maltreatment groups (i.e., a flat “slope” for CSA) among individuals with zero 7R alleles, but a sharp positive slope among individuals who are 7R homozygotes (and an intermediate slope for 7R heterozygotes). Instead, we observed a contrastive interaction, such that CSA was *negatively* associated with sensation seeking among 7R/7R homozygotes but positively associated with sensation seeking among individuals with zero 7R alleles. This interaction shape was observed across all waves. Because few people experienced CSA *and* have the 7R/7R genotype, one could argue that it is more prudent to focus on the 7R heterozygotes, as they comprise the bulk of the individuals with “risky” or “susceptible” genotypes. Comparing the 7R heterozygotes to individuals with zero 7R alleles, the 7R heterozygotes also appear *less* susceptible to the effects of CSA.

Additionally, although the CSA × DRD4 genotype interaction was evident at all waves in Sample 1, this result was not replicated in Sample 2. The divergent results could be simply due to sampling error around a true effect size that is null or very small. Alternatively, the

differences across samples could be attributable to differences in how CSA was assessed. Specifically, in Add Health, CSA was assessed rather narrowly as forced touching or forced sexual intercourse by a parent or adult caregiver. In contrast, the UTE assessment of CSA was broader, including experiences up through adolescence (before senior year of high school) and not necessarily restricted to a parent or caregiver, or even to an adult. Not surprisingly, given this broader assessment, a substantially greater proportion of the UTE sample endorsed CSA (28%) than in the Add Health sample (5%). The less frequent endorsement of CSA in the Add Health sample may have compromised statistical power to detect the interaction. However, sensitivity analyses, in which we restricted the definition of CSA in the UTE sample and thus considerably reduced the number of individuals classified as CSA+, continued to detect a *DRD4* × CSA interaction in the UTE sample. This suggests that differences in measurement cannot entirely account for the differences in results across samples.

Finally, across both samples, the main effects of *DRD4* genotype on sensation seeking and premeditation were minimal and inconsistent in direction. The results of our candidate gene analyses further underscore the difficulty of uncovering replicable genetic correlates of complex psychological traits. General conclusions regarding “differential susceptibility based on dopamine-related genotypes” are premature, or at least require additional specification regarding which developmental outcomes and environmental contexts are implicated in this process.

Both differential susceptibility and classical diathesis-stress theories posit that susceptible or vulnerable individuals will be at increased risk for negative outcomes, relative to less vulnerable individuals, in the face of environmental stress or disadvantage. An alternative hypothesis, described as *vantage sensitivity theory* by Pluess & Belsky (2013), posits that certain factors are growth-promoting in response to environmental enrichment, but do not have an impact on responses to environmental disadvantage. This theory would predict that individuals with the 7R allele of *DRD4* will fare particularly well when raised in exceptionally positive family environments. The current study focused only on differentiating adverse childhood experiences from “good-enough” environments, but did not characterize the positive side of the environmental spectrum. Studies that include the full range of environmental quality from very negative to very positive – are necessary to evaluate fully the range of possible interactions with any putative “vantage sensitivity” factor, including *DRD4* genotype.

5. Conclusions

This study presents initial evidence in support of the hypothesis that CSA is associated with elevated sensation seeking – but not premeditation in – late adolescence and early adulthood. This finding is consistent with previous literature connecting both CSA and sensation seeking with an array of clinical disorders, and further bridges clinical research with research on normative personality variation. At the same time, polymorphisms in dopamine-relevant genes are frequently investigated in relation to reward- or approach-related personality traits, and prominent developmental theorists have hypothesized that certain variants, including the 7R allele of *DRD4*, confer increased susceptibility to

environmental experiences. Following recommendations for candidate gene research (Hewitt, 2012), we tested the hypothesis that the association between CSA and sensation seeking is moderated by *DRD4* genotype using two independent samples, and failed to find consistent evidence for gene × environment interaction. Ultimately, the role of specific genetic variants in complex psychological traits, including personality, will be resolved only by the accumulation of multiple studies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by grant 1-R01-AA020637 to Dr. Kim Fromme, grant 1-R21-AA020588 to Dr. Paige Harden, grant 5-R24-HD042849 to the Population Research Center at the University of Texas at Austin, and grant T32-AA07471to the Waggoner Center for Alcohol and Addiction Research.

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill and funded by Grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due to Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from Grant P01-HD31921 for this analysis.

References

Anestis MD, Smith AR, Fink EL, Joiner TE. Dysregulated eating and distress: Examining the specific role of negative urgency in a clinical sample. Cognitive Therapy and Research. 2009; 33:390–397.

Asghari V, Sanyal S, Buchwaldt S, Paterson A, Jovanovic V, Van Tol HH. Modulation of intercellular cyclic AMP levels by different human dopamine D4 receptor variants. Journal of Neurochemistry. 1995; 65:1157–1165. [PubMed: 7643093]

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5. Arlington, VA: American Psychiatric Publishing; 2013.

Bakermans-Kranenburg MJ, van Ijzendoorn MH. Differential susceptibility to rearing environment depending on dopamine-related genes: new evidence and a meta-analysis. Development and Psychopathology. 2011; 23:39–52.10.1017/S0954579410000635 [PubMed: 21262038]

Becker K, Laucht M, El-Faddagh M, Schmidt MH. The dopamine D4 receptor gene exon III polymorphism is associated with novelty seeking in 15-year-old males from a high-risk community sample. Journal of Neural Transmission. 2005; 112:847–858.10.1007/s00702-004-0223-y [PubMed: 15517431]

Belsky J, Bakermans-Kranenburg MJ, Van Ijzendoorn MH. For better and for worse: Differential susceptibility to environmental influences. Current Directions in Psychological Science. 2007; 16:300–304.10.1111/j.1467-8721.2007.00525.x

Bergevin, TA.; Bukowski, WM.; Karavasilis, L. Childhood sexual abuse and pubertal timing: implications for long-term psychosocial adjustment. In: Hayward, C., editor. Gender differences at puberty. Cambridge, UK: Cambridge University Press; 2003. p. 187-216.

Bornovalova MA, Gwadz MA, Kahler C, Aklin WM, Lejuez CW. Sensation seeking and risk-taking propensity as mediators in the relationship between childhood abuse and HIV-related risk behavior. Child Abuse and Neglect. 2008; 32:99–109.10.1016/j.chiabu.2007.04.009 [PubMed: 18155295]

Bromberg-Martin ES, Matsumoto M, Hikosaka O. Dopamine in motivational control: Rewarding, aversive, and alerting. Neuron. 2010; 68:815–834.10.1016/j.neuron.2010.11.022 [PubMed: 21144997]

Charnigo R, Noar SM, Garnett C, Crosby R, Palmgreen P, Zimmerman RS. Sensation seeking and impulsivity: Combined associations with risky sexual behavior in a large sample of young adults. Journal of Sex Research. 2013; 50:480–488.10.1080/00224499 [PubMed: 22456443]

Cloninger CR. A systematic method for clinical description and classification of personality variants. Archives of General Psychiatry. 1987; 44:573–588. [PubMed: 3579504]

Corbin WR, Vaughan EL, Fromme K. Ethnic differences and the closing of the sex gap in alcohol use among college-bound students. Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors. 2008; 22:240–248.10.1037/a0013335 [PubMed: 18540721]

Cyders MA, Smith GT. Emotion-based dispositions to rash action: positive and negative urgency. Psychological Bulletin. 2008; 134:807–828. [PubMed: 18954158]

Davis C, Loxton NJ. Addictive behaviors and addiction-prone personality traits: Associations with a dopamine multilocus genetic profile. Addictive Behaviors. 2013; 38:2306–2312.10.1016/j.addbeh.2013.02.012 [PubMed: 23584190]

Deckman T, Nathan DeWall C. Negative urgency and risky sexual behaviors: A clarification of the relationship between impulsivity and risky sexual behavior. Personality and Individual Differences. 2011; 51:674–678.

Depue RA, Collins PF. Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. The Behavioral and Brain Sciences. 1999; 22(3):491–517.10.107/S0140525x99002046 [PubMed: 11301519]

Derringer J, Krueger RF, Dick DM, Saccone S, Grucza RA, Agrawal A, Bierut LJ. Predicting sensation seeking from dopamine genes: A candidate-system approach. Psychological Science. 2010; 21:1282–1290. [PubMed: 20732903]

DeYoung CG. The neuromodulator of exploration: A unifying theory of the role of dopamine in personality. Frontiers in Human Neuroscience. 2013; 7:Article 762.10.3389/fnhum.2013.00762

Dreber A, Apicella CL, Eisenberg DT, Garcia JR, Zamore RS, Lum JK, Campbell B. Evolution and Human Behavior. 2009; 30:85–92.

Donohew L, Zimmerman R, Cupp PS, Novak S, Colon S, Abell R. Sensation seeking, impulsive decision-making, and risky sex: Implications for risk-taking and design of interventions. Personality and Individual Differences. 2000; 28:1079–1091.

Dulawa SC, Grandy DK, Low MJ, Paulus MP, Geyer MA. Dopamine D4 receptor-knock-out mice exhibit reduced exploration of novel stimuli. The Journal of neuroscience: The Official Journal of The Society for Neuroscience. 1999; 19:9550–9556. [PubMed: 10531457]

Duncan LE, Keller MC. A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. American Journal of Psychiatry. 2011; 168:1041–1049.10.1176/appi.ajp.2011.11020191 [PubMed: 21890791]

Eaton DK, Davis KS, Barrios L, Brener ND, Noonan RK. Associations of dating violence victimization with lifetime participation, co-occurrence, and early initiation of risk behaviors among US high school students. Journal of Interpersonal Violence. 2007; 22:585–602. [PubMed: 17429024]

Ellingson JM, Verges A, Littlefield AK, Martin NG, Slutske WS. Are bottom-up and top-down traits in dual-systems models of risky behavior genetically distinct? Behavior Genetics. 2013; 43:480–490. [PubMed: 24065563]

Ersche KD, Turton AJ, Pradhan S, Bullmore ET, Robbins TW. Drug addiction endophenotypes: Impulsive versus sensation-seeking personality traits. Biological Psychiatry. 2010; 68:770–773. [PubMed: 20678754]

Fulker DW, Eysenck SB, Zuckerman M. A genetic and environmental analysis of sensation seeking. Journal of Research in Personality. 1980; 14:261–281.10.1016/0092-6566(80)90033-1

Hall NJ, Wynne CDL. The canid genome: Behavioral geneticists best friend? Genes, Brain and Behavior. 2012; 11:889–90.10.1111/j.1601-183X.2012.00851.x

Harden K, Tucker-Drob EM. Individual differences in the development of sensation seeking and impulsivity during adolescence: Further evidence for dual systems model. Developmental Psychology. 2011; 47:739–746.10.1037/a0023279 [PubMed: 21534657]

Harris, KM.; Halpern, CT.; Whitsel, E.; Hussey, J.; Tabor, J.; Entzel, P.; Udry, JR. The National Longitudinal Study of Adolescent Health: Research Design. 2009. [WWW document]. URL: http://www.cpc.unc.edu/projects/addhealth/design

Hewitt JK. Editorial policy on candidate gene association and candidate gene-by-environment interaction studies of complex traits. Behavior Genetics. 2012; 42(1):1–2. [PubMed: 21928046]

Hur YM, Bouchard TJ. The genetic correlation between impulsivity and sensation seeking traits. Behavior Genetics. 1997; 27:455–463. [PubMed: 9336082]

Kendler KS, Bulik CM, Silberg J, Hettema JM, Myers J, Prescott CA. Childhood sexual abuse and adult psychiatric and substance use disorders in women: An epidemiological and cotwin control analysis. Archives of General Psychiatry. 2000; 57:953–959.10.1001/archpsyc.57.10.953 [PubMed: 11015813]

Kluger AN, Siegfried Z, Ebstein RP. A meta-analysis of the association between DRD4 polymorphism and novelty seeking. Molecular Psychiatry. 2002; 7:712–717. [PubMed: 12192615]

Knorr AC, Jenkins AL, Conner BT. The role of sensation seeking in Non-suicidal self-injury. Cognitive Therapy and Research. 2013; 37:1276–1284.

Krueger RF, Markon KE, Patrick CJ, Benning SD, Kramer M. Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. Journal of Abnormal Psychology. 2007; 116:645–666. [PubMed: 18020714]

Krueger RF, Tackett JL. Personality and psychopathology: Working toward the bigger picture. Journal of Personality Disorders. 2003; 17(2):109–128. [PubMed: 12755325]

Maniglio R. The impact of child sexual abuse on health: A systematic review of reviews. Clinical Psychology Review. 2009; 29:647–657.10.1016/j.cpr.2009.08.003 [PubMed: 19733950]

Mendle J, Leve LD, Van Ryzin M, Natsuaki MN, Ge X. Associations between early life stress, child maltreatment, and pubertal development among girls in foster care. Journal of Research on Adolescence. 2011; 21:871–880. [PubMed: 22337616]

Munafo MR, Yalcin B, Willis-Owen SA, Flint J. Association of the dopamine d4 receptor (drd4) gene and approach-related personality traits: meta-analysis and new data. Biological Psychiatry. 2008; 63:197–206.10.1016/j.biopsych.2007.04.006 [PubMed: 17574217]

Muthén, LK.; Muthén, BO. User's guide (Version 7.11). Los Angeles, CA: Muthén & Muthén; 2013. Mplus: Statistical analysis with latent variables.

Nelson EC, Heath AC, Madden PF, Cooper L, Dinwiddie SH, Bucholz KK, Martin NG. Association between self-reported childhood sexual abuse and adverse psychosocial outcomes: Results from a twin study. Archives of General Psychiatry. 2002; 59:139–145.10.1001/archpsyc.59.2.139 [PubMed: 11825135]

Norbury A, Manohar S, Rogers RD, Husain M. Dopamine modulates risk-taking as a function of baseline sensation-seeking trait. The Journal of Neuroscience: The Official Journal of the Society for Neuroscience. 2013; 33:12982–6.10.1523/JNEUROSCI.5587-12.2013 [PubMed: 23926253]

Manolio TA, Collins FS, Cox NJ, Goldstein DB, Hindorff LA, Hunter DJ, Visscher PM. Finding the missing heritability of complex diseases. Nature. 2009; 461:747–753. [PubMed: 19812666]

Ortin A, Lake AM, Kleinman M, Gould MS. Sensation seeking as risk factor for suicidal ideation and suicide attempts in adolescence. Journal of Affective Disorders. 2012; 143:214–222.10.1016/j.jad.2012.05.058 [PubMed: 22921521]

Pluess M, Belsky J. Vantage sensitivity: Individual differences in response to positive experiences. Psychological Bulletin. 2013; 139:901–916. [PubMed: 23025924]

Powell JE, Zietsch B. Predicting sensation seeking from dopamine genes: Use and misuse of genetic prediction. Psychological Science. 2011; 22:413–415. [PubMed: 21270448]

Quinn PD, Harden KP. Differential changes in impulsivity and sensation seeking and the escalation of substance use from adolescence to early adulthood. Development and Psychopathology. 2013; 25:223–239. [PubMed: 22824055]

Robbins TW, Everitt BJ. Interaction of the dopaminergic system with mechanisms of associative learning and cognition: Implications for drug abuse. Psychological Science. 1999; 10:199–202.10.1111/1467-9280.00134

Romans SE, Martin JM, Gendall K, Herbison GP. Age of menarche: The role of some psychosocial factors. Psychological Medicine. 2003; 33:933–939. [PubMed: 12877408]

Roussos P, Giakoumaki SG, Bitsios P. Cognitive and emotional processing in high novelty seeking associated with the L-DRD4 genotype. Neuropsychologia. 2009; 47:1654–1659.10.1016/j.neuropsychologia.2009.02.00 [PubMed: 19397860]

Schafer JL, Graham JW. Missing data: our view of the state of the art. Psychological Methods. 2002; 7:147–177. [PubMed: 12090408]

Settles RE, Fischer S, Cyders MA, Combs JL, Gunn RL, Smith GT. Negative urgency: a personality predictor of externalizing behavior characterized by neuroticism, low conscientiousness, and disagreeableness. Journal of Abnormal Psychology. 2012; 121:160–172. [PubMed: 21859164]

Simmons JP, Nelson LD, Simonsohn U. False-Positive Psychology: Undisclosed flexibility in data collection and analysis allows presenting anything as significant. Psychological Science. 2011; 22:1359–1366.10.1177/0956797611417632 [PubMed: 22006061]

Smith AR, Chein J, Steinberg L. Impact of socio-emotional context, brain development, and pubertal maturation on adolescent risk-taking. Hormones and Behavior. 2013; 64(2):323–332. [PubMed: 23998675]

Smolen, A.; Whitsel, EA.; Tabor, J.; Killeya-Jones, LA.; Cuthbertson, CC.; Hussey, JM.; Halpern, CT.; Harris, KM. Add Health Wave IV documentation: Candidate genes. Chapel Hill, NC: Carolina Population Center, University of North Carolina; 2013. http://www.cpc.unc.edu/projects/addhealth/data/guides/DNA_documentation.pdf

Stautz K, Cooper A. Impulsivity-related personality traits and adolescent alcohol use: A meta-analytic review. Clinical Psychology Review. 2013; 33:574–592.10.1016/j.cpr.2013.03.003 [PubMed: 23563081]

Steinberg L. A social neuroscience perspective on adolescent risk-taking. Developmental Review. 2008; 28:78–106.10.1016/j.dr.2007.08.002 [PubMed: 18509515]

Steinberg L, Albert D, Cauffman E, Banich M, Graham S, Woolard J. Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: evidence for a dual systems model. Developmental Psychology. 2008; 44:1764–1778.10.1037/a0012955 [PubMed: 18999337]

Stoel RD, De Geus EJ, Boomsma DI. Genetic analysis of sensation seeking with an extended twin design. Behavior Genetics. 2006; 36:229–237.10.1007/s10519-005-9028-5 [PubMed: 16550452]

Trickett PK, Putnam FW. Impact of child sexual abuse on females: Toward a developmental, psychobiological integration. Psychological Science. 1993; 4:81–87.

Turner PK, Runtz MG, Galambos NL. Sexual abuse, pubertal timing, and subjective age in adolescent girls: A research note. Journal of Reproductive and Infant Psychology. 1999; 17:111–118.

Udry, JR. The National Longitudinal Study of Adolescent Health (Add Health), Waves I & II, 1994–1996; Wave III, 2001–2002 [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill; 2003.

Van Tol HM, Wu CM. Multiple dopamine D4 receptor variants in the human population. Nature. 1992; 358:149–152. [PubMed: 1319557]

Whiteside SP, Lynam DR. The five factor model and impulsivity: Using structural model of personality to understand impulsivity. Personality and Individual Differences. 2001; 30:669–689.

Whiteside SP, Lynam DR. Understanding the role of impulsivity and externalizing psychopathology in alcohol abuse: Application of the UPPS impulsive behavior scale. Experimental and Clinical Psychopharmacology. 2003; 11:210–217. [PubMed: 12940500]

Wise LA, Palmer JR, Rothman EF, Rosenberg L. Childhood abuse and early menarche: findings from the Black Women's Health Study. American Journal of Public Health. 2009; 99:S460–S466. [PubMed: 19443822]

Wonderlich SA, Crosby RD, Mitchell JE, Thompson KM, Redlin J, Demuth G, Haseltine B. Eating disturbance and sexual trauma in childhood and adulthood. International Journal of Eating Disorders. 2001; 30:401–412. [PubMed: 11746301]

Zuckerman M, Kuhlman D, Joireman J, Teta P, Kraft M. A comparison of three structural models for personality: The big three, the big five, and the alternative five. Journal of Personality and Social Psychology. 1993; 65:757–768.10.1037/0022-3514.65.4.757

Zald DH, Cowan RL, Riccardi P, Baldwin RM, Ansari M, Rui L, Kessler RM. Midbrain dopamine receptor availability is inversely associated with novelty-seeking traits in humans. Journal of Neuroscience. 2008; 28:14372–14378.10.1523/JNEUROSCI.2423-08.2008 [PubMed: 19118170]

Highlights

- We test the relation between childhood sexual abuse and personality in young adults.
- In two samples, sexual abuse was associated with higher sensation seeking.
- The dopamine D4 receptor gene did not consistently moderate the effect of sexual abuse.
- Sensation seeking may link sexual abuse with risk for multiple mental disorders.

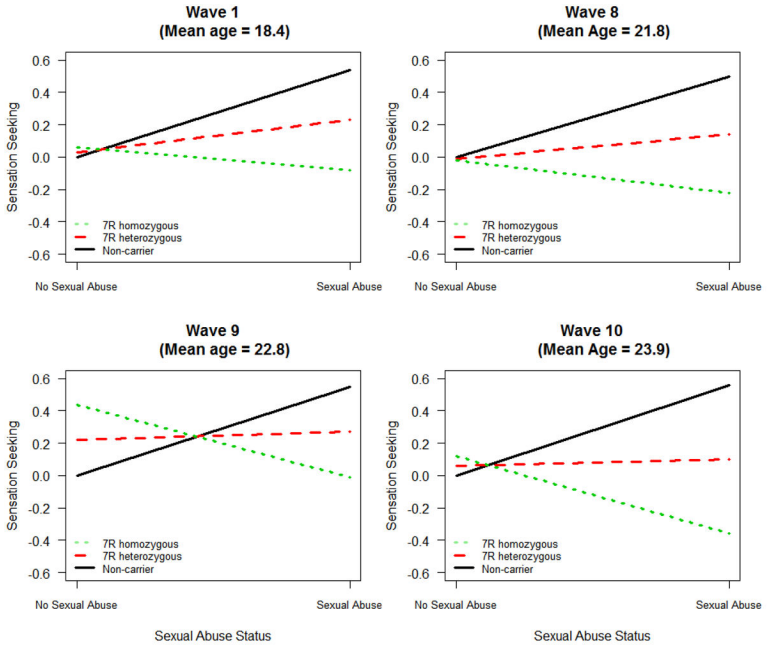

Figure 1. Results from DRD4 × Sexual Abuse Interaction Model in Sample 1 (UTE)

Note. Sensation seeking scores are standardized (*M*=0, *SD*=1). Sensation seeking not measured at study waves 2–7.

Table 1

Confirmatory Factor Analysis Loadings by Wave for Sample 1

	Factor loadings							
	Wave 1		Wave 8		Wave 9		Wave 10	
Items	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***
I often do things on impulse. (ZKPQ)	0.36	0.59	0.47	0.56	0.42	0.49	0.46	0.56
I like to have new and exciting experiences and sensations, even if they are a little frightening. (ZKPQ)	0.74		0.77		0.76		0.73	
I enjoy getting into new situations where you can't predict how things will turn out. (ZKPQ)	0.73		0.74		0.72		0.72	
I like doing things just for the thrill of it. (ZKPQ)	0.75		0.80		0.73		0.80	
I sometimes like to do things that are a little frightening. (ZKPQ)	0.64		0.58		0.64		0.67	
I'll try anything once. (ZKPQ)	0.43		0.46		0.47		0.56	
I would like the kind of life where one is on the move and traveling a lot, with lots of change and excitement. (ZKPQ)	0.43		0.64		0.65		0.62	
I sometimes do "crazy" things just for fun. (ZKPQ)	0.63		0.74		0.73		0.75	
I like to explore a strange city or section of town by myself, even if it means getting lost. (ZKPQ)	0.40		0.57		0.57		0.60	
I prefer friends who are so excitingly unpredictable. (ZKPQ)	0.58		0.69		0.67		0.78	
I often get so carried away by new and exciting things and ideas that I never think of possible complications. (ZKPQ)	0.72		0.81		0.81		0.84	
I am an impulsive person. (ZKPQ)	0.48	0.49	0.57	.56	0.57	.47	0.60	0.46
I like wild "uninhibited" parties. (ZKPQ)	0.58		0.59		0.65		0.58	
I often do things based on how I feel at the moment, without thinking	0.39	0.35	0.44	0.44	0.53	0.32	0.48	0.42

	Factor loadings							
	Wave 1		Wave 8		Wave 9		Wave 10	
Items	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***	**Factor 1** ***Sensation Seeking***	**Factor 2** ***Lack of Premeditation***
about how they were done in the past. (TPQ)								
I often break rules and regulations when I think I can get away with it. (TPQ)	0.40		0.46	*.24*	0.53	*.17*	0.35	*.19*
It is difficult for me to keep the same interests for a long time because my attention often shifts to something else. (TPQ)	0.44		0.42	*.25*	0.52		0.50	*.16*
I often follow my instincts, hunches, or intuition, without thinking through all the details. (TPQ)	0.41	0.29	0.31	0.61	0.38	0.52	0.45	0.39
I can usually do a good job of stretching the truth to tell a funnier story or to play a joke on someone. (TPQ)	0.26		0.29		0.30		0.32	
I tend to begin a new job without much advance planning on how I will do it. (ZKPQ)		0.55		0.59		0.69		0.80
I usually think about what I am going to do before doing it. (ZKPQ)		−0.86		−0.85		−0.80		−0.87
I very seldom spend much time on the details of planning ahead. (ZKPQ)		0.62		0.59		0.84		0.79
Before I begin a complicated job, I make careful plans. (ZKPQ)		−0.63		−0.73		−0.60		−0.72
I usually think about all the facts in detail before I make a decision. (TPQ)		−0.80		−0.80		−0.72		−0.73
I nearly always think about all the facts in detail before I make a decision, even when other people demand a quick decision. (TPQ)		−0.69		−0.58		−0.46		−0.47
I hate to make decisions based only on my first impressions. (TPQ)		−0.26		−0.15		−0.28		−0.28
I like to pay close attention to details in everything I do. (TPQ)		−0.66		−0.63		−0.65		−0.55

Note. Factor loadings are in standardized units. All loadings are statistically significant at $p < .001$ except italics which are significant at $p < .05$. ZKPQ = Zuckerman-Kuhlman Personality Questionnaire III-R. TPQ = Tridimensional Personality Questionnaire.

Table 2

Confirmatory Factor Analysis Loadings for Sample 2

Items	Factor 1 *Sensation Seeking*
I like wild uninhibited parties vs. I like quiet parties with good conversation.*	0.50
You like to take risks.	0.51
I like to have new and exciting experiences even if they are a little frightening, unconventional, or illegal vs. I am not interested in experience for its own sake.*	0.51
I like to date people who are physically exciting vs. I like to date people who share my values.*	0.42
I often try new things for the thrill of it, even if others tell me it's a waste of my time.	0.73
When nothing new is happening I usually start looking for something exciting.	0.71
I can usually get people to believe me, even when what I'm saying isn't quite true.	0.52
I often do things based on how I feel at the moment.	0.59
I sometimes get so excited I lose control over myself.	0.52
I like it when people can do whatever they want, without strict rules and regulations.	0.61
I often follow my instincts, without thinking through all the details.	0.54
I can do a good job of "stretching the truth" when I'm talking to people.	0.48
I change my interest a lot, because my attention often shifts to something else.	0.49
A person should have considerable sexual experience before marriage vs. It's better if two married people begin their sexual experience with each other.*	0.35

Note. Factor loadings are in standardized units. Factor loadings are in standardized units.

*Positive factor loadings correspond to endorsement of first response option in the pair. All loadings are statistically significant at $p < .001$.

Table 3

Results from Sensation Seeking Models by Study Sample and Wave

	Sample 1				Sample 2
	Wave 1	**Wave 8**	**Wave 9**	**Wave 10**	**Wave 3**
Gender [female]	−.48***	−.31*	−.50***	−.49***	.00
Hispanic	.20	−.05	.13	.22	−.03
Black	.94	.36	.52	.30	−.19**
Asian	.10	−.06	.19	−.06	.00
Native American	--	--	--	--	−.08
Multiethnic	.06	.20	.38	.26	--
Age	-	-	-	-	−.09***
SES	.02	.03	.07	.03	.00
Sexual abuse	.54***	.50**	.55***	.56***	.40***
DRD4	.03	−.01	.22	.06	.00
DRD4 × Sexual Abuse	−.34*	*−0.35*	−.50**	−.52**	.00
Sample *N*	487	407	428	459	2388

Note. Sample 1 corresponds to "*UTE!*" data and sample 2 corresponds to Add Health data. All values are standardized with respect to sensation seeking. Reference gender category is male. Reference racial category is non-Hispanic White;

*** $p < .001$,

** $p < .01$,

* $p < .05$,

italics $p = .05$.

Table 4

Results from Lack of Premeditation Models by Sample 1 Study Wave

	Wave 1	Wave 8	Wave 9	Wave 10
Gender [female]	−.10	−.20	−.11	−.13
Hispanic	.21	.28	.12	.05
Black	.77	*1.2*	.77	.83
Asian	−.18	−.05	.07	.11
Multiethnic	.20	.14	.05	.16
SES	.01	.04	.02	−.06
Sexual abuse	.32*	.12	.27	.28
DRD4	−.14	−.04	−.05	.19
DRD4 × Sexual Abuse	−.04	−.02	−.08	−.28
Sample *N*	487	407	428	459

Note. All values are standardized with respect to lack of premeditation. Reference gender category is male. Reference racial category is non-Hispanic White;

*** $p < .001$,

** $p < .01$,

* $p < .05$.

Table 5

	Sample 1												Sample 2
	Wave 1			Wave 8			Wave 9			Wave 10			Wave 3
	Cauc. only	CSA 2- item	Ctrl Nsex	Cauc. only	CSA 2-item	Ctrl Nsex	Cauc. only	CSA 2-item	Ctrl Nsex	Cauc. only	CSA 2-item	Ctrl Nsex	Cauc. only
Gender [female]	−.34*	−.35**	−.49***	−.42**	−.31*	−.33**	−.46**	−.42***	−.52***	−.63***	−.41***	−.50***	−.70***
Hispanic	--	.24	.18	--	−.05	−.04	--	.18	.13	--	.27	.19	--
Black	--	.98	.95	--	.36	.32	--	.43	.53	--	.22	.31	--
Asian	--	−.09	−.05	--	−.06	−.02	--	.17	.23	--	−.07	−.03	--
Native American	--	--	--	--	--	--	--	--	--	--	--	--	--
Multiethnic	--	−.04	.02	--	.20	.25	--	.39	.41*	--	.27	−.30	--
Age	--	--	--	--	--		--	--	--	--	--	--	−.10***
SES	.03	.02	.02	.01	.03	.03	.01	.07	.07	.01	−.04	.04	.01
Sexual abuse	.38*	.29	.47**	.55*	.50**	.42**	.56**	.50*	.47**	.61**	.61*	.50***	.63***
DRD4	−.08	.00	.02	.03	.01	.01	.19	.12	.21**	.16	.00	.05	−.10
Nsex	--	--	.14**	--	--	.10**	--	--	.16**	--	--	.12*	--
DRD4 × Sexual Abuse	−.08	−.54*	*−.34*	−.46	−.35	−.36	−.45*	−.63**	−.52**	−.54*	−.88**	−.52**	.09
Sample *N*	303	487	486	257	407	408	261	428	428	286	459	458	261

Note. Sample 1 corresponds to "*UTE!*" data and sample 2 corresponds to Add Health data. All values are standardized with respect to sensation seeking. Reference gender category is male. Reference racial category is non-Hispanic White; Nsex = sensitivity analyses that control for number of sexual partners reported senior year of high school; Cauc only = sensitivity analyses limited to Caucasian subset; CSA 2-item = sensitivity analyses using 2-item childhood sexual abuse composite;

*** $p < .001$,

** $p < .01$,

* $p < .05$,

italics $p = .05$.