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Childhood sexual abuse and impulsive personality traits: Mixed evidence for moderation by *DRD4* genotype

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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 ($n = 2559$). 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.

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 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 et al. (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

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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 (Harden & Tucker-Drob, 2011; Steinberg et al., 2008). 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 Lejuez (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, Matsumoto, & Hikosaka, 2010; Depue & Collins, 1999; DeYoung, 2013). 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 & Wu, 1992). The number of repeats in *DRD4* VNTR range from 2 to 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-Faddagh, & Schmidt, 2005; Dreber et al., 2009), and similar associations with dopamine-related genes have also been reported in the animal literature (e.g., Dulawa, Grandy, Low, Paulus, & Geyer, 1999; Hall & Wynne, 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., Davis & Loxton, 2013; Derringer et al., 2010).

Studies documenting a main effect of dopamine-related candidate genes on impulsive personality traits, however, have faced valid criticism (e.g., Duncan & Keller, 2011; Powell & Zietsch, 2011). Most importantly, the promising results of individual studies are tempered by meta-analytic results. Munafo, Yalcin, Willis-Owen, and 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, De Geus, & 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-Kranenburg, & 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. 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 $G \times E$ 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 $G \times E$ (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.