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Molecular genetic underpinnings of self-control: 5-HTTLPR and self-control in a sample of inmates

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ABSTRACT

Purpose: Several studies now show that self-control, as proposed by Gottfredson and Hirschi (1990), is at least moderately heritable. Studies of molecular genetic variation related to serotonergic function suggest that the heritability of self-control may be explained, in part, by the 5-HTTLPR polymorphism.
Methods: The current research tests the association between the 5-HTTLPR polymorphism and self-control as measured by the Grasmick et al. (1993) scale. Analyses were based on a sample of incarcerated males and considered the effect of the 5-HTTLPR polymorphism on the full self-control scale as well as the specific dimensions of self-control.
Results: The s/s genotype interacted with abuse to predict increases in overall self-control, preference for simple tasks and physical activity. Relative to the s/l genotype, the l/l genotype, which has been linked to psychopathy, was directly associated with more self-centeredness.
Conclusions: Results show that molecular genetic variation related to serotonergic function plays a role in the heritability of self-control. Variation in the association between 5-HTTLPR genotype and the distinct dimensions of self-control, while consistent with recent literature (see Yildirim & Derksen, 2013), indicates that self-control as originally presented by Gottfredson and Hirschi (1990) is not a unitary construct.

Introduction

The central premise of Gottfredson and Hirschi's (1990) general theory of crime is that an individual's level of self-control influences the extent to which they are able to refrain from crime and analogous behaviors. Those with low levels of self-control are typically "impulsive, insensitive, physical (as opposed to mental), risk-taking, short-sighted, and nonverbal" (Gottfredson & Hirschi, 1990, p. 90). As a consequence, low self-control leads to an increased likelihood of acts that provide immediate benefit but have the potential for negative consequences in the future (Gottfredson & Hirschi, 2003). Consistent with this premise, research has shown that those with low self-control engage in a range of criminal behaviors including drug use (Baron, 2003), minor delinquency (Cauffman, Steinberg, & Piquero, 2005), violent offending (Piquero, MacDonald, Dobrin, Daigle, & Cullen, 2005), aggression (Archer & Southall, 2009), and fraud (Holtfreter, Reisig, Piquero, & Piquero, 2010).

Gottfredson and Hirschi (1990) assert that individual variation in self-control is primarily determined by parenting practices. To be sure, a line of research has revealed evidence of an association between parenting styles and self-control (Gibbs, Giever, & Higgins, 2003; Perrone, Sulliva, Pratt, & Margaryan, 2004; Pratt, Turner, & Piquero, 2004; Unnever, Cullen, & Pratt, 2003). Other research suggests, however, that parenting may not be the sole determinant of self-control (Hay, 2001; Pratt & Cullen, 2000). The role of parenting in the etiology of self-control is called further into question by behavioral genetic studies showing that parental efficacy has a negligible effect on self-control when genetic factors were taken into account (DeLisi, 2014; Wright & Beaver, 2005).

Subsequently a number of tests have established that self-control is partially heritable (Beaver, DeLisi, Vaughn, Wright, & Boutwell, 2008; Beaver et al., 2009a; Boisvert, Boutwell, Barnes, & Vaske, 2013; Boisvert, Wright, Knopik, & Vaske, 2012; Boisvert, Wright, Knopik, & Vaske, 2013; Wright Beaver, DeLisi, & Vaughn, 2008). Parallel lines of literature in the field of psychology, have also provided evidence concerning the heritability of traits and clinical diagnoses that overlap with Gottfredson and Hirschi's concept of low self-control. Studies have consistently found that at least 70% of the variance in attention

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deficit hyperactivity disorder (ADHD) is the result of genetic factors (Farone et al., 2005; McLaughlin, Ronald, Kuntsi, Asherson, & Plomin, 2007; Nikolas & Burt, 2010; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2003). Evidence for the heritability of traits and clinical diagnoses overlapping with self-control extends also to the trait of impulsivity. A recent meta-analysis based on 41 studies with 56 independent samples found that approximately 50% of the variance in impulsivity is heritable (Bezdjian, Baker, & Tuvblad, 2011, see also Niv, Tuvblad, Raine, Wang, & Baker, 2012).

Collectively, the behavioral genetic research outlined above indicates that a substantial portion of the variance in self-control is accounted for by genetic factors. What is less clear, however, are the specific sources of genetic variation that underlie these non-zero heritability estimates. One possibility suggested by molecular genetic research is that the 5-HTTLPR polymorphism in the serotonin transporter gene (SLC6A4) plays a role in the development of self-control. Studies have shown that the 5-HTTLPR polymorphism is associated with both ADHD (Gizer, Ficks, & Waldman, 2009) and impulsivity (Paaver et al., 2007; Walderhaug et al., 2007). Beaver, Ratchford, and Ferguson (2009), using Add Health data, found direct evidence for a link between 5-HTTLPR and self-control wherein 5-HTTLPR interacted with delinquent peer association to explain levels of self-control. Other evidence indicates that 5-HTTLPR may interact with other environmental risk factors to explain impulsivity. This suggests that 5-HTTLPR may interact with other environmental risks, such as childhood abuse, to explain between-individual variation in self-control.

5-HTTLPR and Behavior

Variants of the 5-HTTLPR polymorphism regulate the extent to which the serotonin transporter gene (SLC6A4) is expressed. More specifically, serotonin (5-HT) is held on the dendrites of neurons until released into the intersynaptic cleft by neuron activation. After release, 5-HT reuptake, or return to dendrite terminal, is undertaken by serotonin transporter (5-HTT). The 5-HTTLPR polymorphism in SLC6A4 has two commonly occurring variants, referred to as the short (*s*) and long (*l*) alleles (Heils et al., 1996; Murphy, Lerner, Rudnick, & Lesch, 2004). The 484 base pair *s* allele is associated with reduced transcriptional efficacy as compared to the 512 base pair *l* allele (Heils et al., 1996; Lesch et al., 1996; Murphy et al., 2004). Reduced transcriptional efficiency means that the gene is being 'read' less often resulting in fewer serotonin transporters, which in turn leads to higher levels of serotonin remaining in the synaptic left (Daws & Goudl, 2011). This has neurological, psychological, and behavioral consequences (Hariri & Holmes, 2006). For example, individuals with the s allele of 5-HTTLPR have increased stress system responses, such as increased basal cortisol and hypothalamic-pituitary-adrenal axis response to stress (Chen et al., 2009; Goodyer et al., 2009; Gotlib et al., 2008; Jabbi et al., 2007; Wankerl et al., 2010; Way & Taylor, 2010), and increased amygdala response to threat (Hariri & Holmes, 2006; Hariri & Weinberger, 2003; Hariri et al., 2006), which can lead to maladaptive responses when stressed.

The neurophysiological consequences of the 5-HTTLPR polymorphism that are outlined above likely underpin the association between the polymorphism and a number of antisocial behaviors including violence and aggression (Beitchman et al., 2006; Gerra et al., 2005; Haberstick, Smolen, & Hewitt, 2006; Liao, Hong, Shih, & Tsai, 2004; Retz, Retz-Junginger, Supprian, Thome, & Rosher, 2004) as well as substance use (Feinn, Nellissery, & Kranzler, 2005; Gerra et al., 2005). Psychological conditions that have been linked with the 5-HTTLPR polymorphism include negative affect (Fox et al., 2005; Pauli-Pott, Friedl, Hinney, & Hebebrand, 2009), anxiety (Jorm, Sanson, Smart, Zhang, & Easteal, 2000), neuroticism (Lesch et al., 1996; Sen, Burmeister, & Ghosh, 2004), and risk seeking (Crisan, Pana, Vulturar, Heilman, Szekely, Druga, et al., 2009; Kuhnen & Chiao, 2009). It should be noted that within this literature studies often fail to find direct effects of the 5-HTTLPR polymorphism on antisocial behaviors (see for example, Davidge et al., 2004). One possible reason for this is that genes might impact trait variation in a non-additive manner, interacting with variation in the environment of the organism (Belsky & Beaver, 2011). Thus, measures of genetic risk have been conceptualized as markers of environmental sensitivity or plasticity whose presence increases the impact of risky environments (Belsky & Beaver, 2011). Nonetheless direct effects do occur in the literature and there is some indication that in certain cases specific alleles associated with decreased environmental sensitivity may have direct effects on phenotypes associated with antisocial behavior and antisocial behaviors themselves (see Yildirim & Dersken, 2013).

Traditionally, the *s* allele of the 5-HTTLPR polymorphism has been considered the 'risk' allele. Consistent with this proposition, studies have found the *s* allele interacts with environmental adversity, such as childhood abuse, to predict antisocial behavior, aggression, impulsivity, violent crime and substance use (Covault et al., 2007; Gerra et al., 2005; Lesch & Merschdorf, 2000; Manuck, Kaplan, & Lotrich, 2004; Nilsson et al., 2005; Reif et al., 2007). Recently, in a comprehensive review of studies linking serotonergic function to neurophysiology and behavior Yildirim and Derksen (2013) argued that the *s* allele interacts with childhood adversity to contribute to the development of a temperament characterized by increased reactive emotional arousal. Yildirim and Derksen (2013) also hypothesized that the *l* allele can contribute to increased risk for antisocial behavior characterized by a lack of emotional arousal and callous unemotional traits. The potential link between the *l* allele and callous unemotional traits was also highlighted recently by Glenn (2011) who argued the *l* allele is associated with increased risk for psychopathy.

The potential contribution of both the *l* allele and the *s* allele of the 5-HTTLPR polymorphism to antisocial behaviors through distinct traits is well illustrated by the recent findings of Sadeh and colleagues (2010). In this study, the homozygous *l*/*l* genotype was related to callous unemotional and narcissistic psychopathic traits while the *s* allele was related to impulsivity. This finding is consistent with the argument that the *s* allele of the 5-HTTLPR polymorphism is associated with behavioral phenotypes consistent with impulsivity and environmental reactivity while the *l* allele is associated with callous unemotional and psychopathic traits.

Such a consideration is likely important for the current study. Analyses presented here explore the link between the 5-HTTLPR polymorphism and the Grasmick et al. (1993) self-control scale. The Grasmick et al. (1993) self-control scale is designed to measure the various dimensions of self-control as outlined by Gottfredson and Hirschi (1990). These include impulsivity, preference for simple rather than complex tasks, orientation toward risk seeking, preference for physical rather than cognitive tasks, self-centered orientation, and temper. Studies have demonstrated that some of the traits encompassed in this measure are distinct, with unique neurophysiological underpinnings and unique associations with antisocial behavior (see for example Burt & Simons, 2013; Dadds et al., 2005; Marcus, 2004; Yildirim & Derksen, 2013). The independence of the traits encompassed in self-control is further underscored by evidentiary and theoretical arguments for the mutidimensionalty of self-control as articulated by Gottfredson and Hirschi (1990) and as measured by the Grasmick et al. (1993) scale (Burt, Sweeten, & Simons, 2014; Conner, Stein, & Longshore, 2009; DeLisi, Hochstetler, & Murphy, 2003; DeLisi, Hochstetler, & Murphy, 2003; Longshore, Stein, & Turner, 1998a; Longshore, Turner, & Stein, 1998b; Longshore, Turner, & Stein, 1996; Marcus, 2004; Vazsonyi, Pickering, Junger, & Hessing, 2001).

Evidence for the multidimensionality of the Grasmick et al (1993) self-control scale and studies showing that the various traits encompassed by the scale have unique neurophysiological underpinnings suggest that the various dimensions of the scale may have unique genetic antecedents. In the case of the 5-HTTLPR polymorphism, studies