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Teaching Biosocial Criminology I: Understanding Endophenotypes Using Gottfredson and Hirschi's Self-Control Construct

*Matt DeLisi, John Paul Wright, Kevin Beaver and
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Criminology is experiencing a paradigm shift in theory and research that articulates a more interdisciplinary, biosocial mode of inquiry. Unfortunately, however, graduate-level criminal justice education rarely encompasses biosocial training. The current review is the first in a series of works that seeks to fill this biosocial training void by providing instruction on concepts in the biological sciences and neurosciences that have direct relevance to criminal justice education and criminology. Here we introduce endophenotypes and demonstrate their relevance to the study of crime using the self-control construct from Gottfredson and Hirschi's general theory. Because biosocial criminology is in its nascent stage, it is critical that biosocial criminologists provide service and instruction to their social science-trained colleagues.

Introduction

There is burgeoning enthusiasm and acknowledgement that criminology is experiencing a paradigm shift from which it will emerge as a more scientific, interdisciplinary academic discipline (Armstrong, Keller, Franklin, and Macmillan 2009; DeLisi, Wright, Vaughn, and Beaver 2009; Plomin and Rutter 1998; Wright, Beaver, DeLisi, and Vaughn 2008; Wright and Boisvert 2009; Wright, Tibbetts, and Daigle 2008). Gone is the exclusive mode of inquiry where constructs—except for those reducible to the individual—were used to study crime, and in its place is a biosocial perspective that utilizes biological, psychological, and sociological constructs in tandem in its approach to the study of crime. This paradigm shift has caught the attention of some of the leading figures in traditional criminology. For instance, Cullen (2009) recently observed, "Although I have trumpeted its value, I am equally persuaded that sociological criminology has exhausted itself as a guide for future study on the origins of crime" (p. xvi).

Primarily driving this assessment is Cullen's view that criminology "ignores too much [biology] that we know matters" (p. xvi, insert added). In a relatively short period of time, it is today somewhat common to see research rooted in the biological sciences appearing in criminology and criminal justice journals.

Despite the shift in criminology from a mostly sociological to an almost common sense recognition that crime and complex human behaviors are biological and social in nature, there remains a structural problem in higher level criminal justice education that centers on the paucity of training in the biological sciences and neurosciences. Even if prospective criminologists were interested in utilizing biological and biosocial constructs in their research, it is doubtful that graduate students would receive instruction in the area. For example, a recent national survey of 33 Ph.D. granting programs in criminology and criminal justice found that less than 2% of all graduate faculty members responsible for training criminology doctoral students in the USA had any training—very broadly defined—in biology (Wright et al., 2008). The current review aims to partially rectify the lack of biological training in criminal justice education by introducing the concept of endophenotypes and illustrating its relevance to criminology by demonstrating its relation to the accessible and widely known self-control construct advanced in Gottfredson and Hirschi's (1990) general theory of crime.

Endophenotypes

It is often assumed that the causal relationship between a genetic factor (genotype) and some outcome (phenotype) is straightforward and direct. This is because many social scientists assume a model of Mendelian inheritance where a person has a gene "for" a behavior, or does not. Human genetics are far more complicated. Antisocial behaviors are not inherited in a Mendelian way unfortunately and instead follow a pattern of incomplete dominance where alleles that influence a trait are not wholly dominant over other alleles. As a general rule, the more complex and behaviorally oriented the phenotype, the more indirectly it is predicted genetically and the less likely a "gene for" rationale makes sense (Chakravarti and Little 2003; Kidd 1991; Meyer-Lindenberg and Weinberger 2006; Plomin and Caspi 1998; Rutter, Moffitt, and Caspi 2006). In fact, there are several important differences between Mendelian—or simple inheritance—disorders and multifactorial phenotypes such as psychopathology, externalizing behaviors, and crime. For example, Mendelian disorders are caused by rare allelic mutations that produce catastrophic effects, such as the autism spectrum disorder Rett's syndrome which afflicts females and is caused by a mutation in the MECP2 gene located on the X chromosome (the gene mutation is fatal for males who either miscarry before birth or die during early infancy). The essential point to understand is that Mendelian genetic effects are rare and prevent vital functions from occurring.

Mendelian disorders are rare because the catastrophic mutations are weeded out via natural selection. However, they persist due to mutation-selection

balance which is the process by which new mutations at the disease locus are introduced into the population at a low frequency (Cannon and Keller 2006). In contrast, externalizing disorders (e.g., criminal behavior) and psychiatric conditions are polygenic—caused by many genes—and pleiotropic—where individual genes are associated with multiple phenotypes. These risk alleles produce smaller, incremental effects that affect traits, such as aggressiveness or impulsivity, in quantitative ways. Compared to rare Mendelian disorders, antisocial traits are significantly more common because according to mutation-selection balance, they have higher trait-level mutation rates and the selection against each mutation is much lower. As noted by Cannon and Keller (2006), “the relative commonality of certain mental disorders may simply reflect the much larger number of environmental and genetic factors that contribute to these disorders” (p. 272).

What this means is that there is not “a gene for” or even a set of genes for crime (Kendler 2005). This sets the stage for endophenotypes. Broadly defined, endophenotypes are the intermediary ground that connects genetic liability that underscores psychiatric and antisocial phenotypes (Gottesman and Gould 2003). It is important to recognize, however, that in the psychiatric and neuroscience literatures, endophenotypes have been variously defined. For instance, Castellanos and Tannock (2002) defined endophenotypes as “heritable quantitative traits that index an individual’s liability to develop or manifest a given disease, and they are thought to be more directly related than dichotomous diagnostic categories to aetiological factors” (p. 617). With a more expansive, biological systems approach, Caspi and Moffitt (2006) defined endophenotypes as “heritable neurophysiological, biochemical, endocrinological, neuroanatomical, or neuropsychological constituents of disorders ... assumed to have simpler genetic underpinnings than disorders themselves” (p. 583). From a neuropsychological perspective, Green et al. (2008) defined endophenotypes which they called intermediate phenotypes as a “heritable trait or characteristic that is not a direct symptom of the condition under investigation but that has been shown to be associated with the condition. It might reflect an intermediate step in the pathway between gene and psychological function (or dysfunction)” (p. 717). Finally, Cannon and Keller (2006) suggested:

The use of endophenotypes—intermediate phenotypes that form the causal links between genes and overt expression of disorders—promises to facilitate discovery of the genetic and environmental architecture of common mental disorders and thereby suggest novel strategies for intervention and prevention based on an understanding of the molecular mechanisms underlying risk and manifestation. (p. 268)

Several conditions must be present to suggest that a particular endophenotype serves as the middle ground between genetic risks and psychiatric or behavioral phenotypes. First, the endophenotype must distinguish between those who meet an empirically validated condition or disorder (e.g., high score versus low score or diagnostic criteria). It must be able to separate the clinical

from the non-clinical. Second, the endophenotype should embody the traits or characteristics that typify the phenotype. In the parlance of self-control, an endophenotype of impulsivity, for example, would more generally embody a person with low self-control or reduced self-regulation. The third and related point is there must be a known heritability of the endophenotype—garnered from twin studies—which empirically shows the degree that a specific disorder or characteristic is attributable to genetic factors. For example, the heritability of antisocial behavior is about 50% based on meta-analyses and comprehensive reviews (for a review, see Beaver 2009), but it can be as high as 85%. The heritability of low self-control is even higher with recent estimates suggesting that between 52% and 64% of the variance in low self-control is attributable to genetic factors (Beaver, Wright, DeLisi, and Vaughn 2008). Fourth, the disorder should be more common in family members with greater genetic similarity, for instance monozygotic/identical twins as opposed to dizygotic/fraternal twins. Fifth, the prevalence of the disorder should be higher in non-affected family members of probands than in the general population. In sum, endophenotypes serve as a bridge to link the admittedly complex and convoluted genetic foundations of antisociality through their neural substrates to the ultimate manifestations of that antisociality.

Self-Control \approx Executive Functioning

In their landmark *A General Theory of Crime*, Gottfredson and Hirschi (1990) advanced a self-control construct that had six subdimensions that were theorized to coalesce into a unitary, individual-level construct. These six dimensions were impulsivity, physical activity (as opposed to cognitive pursuits), risk-taking/sensation-seeking, self-centeredness/narcissism, preference for simple tasks/low tenacity, and poor temper (see Figure 1). Gottfredson and Hirschi suggested that bad parenting is characterized by low monitoring, inability or reluctance to recognize childhood deviance, and failure to follow through with disciplining or responding to deviant conduct that occurred during the first decade of life contributed to low self-control. Children who were ineffectively socialized, according to the theory, evinced some combination of the six characteristics of low self-control, and were at risk for a range of maladaptive, delinquent, and imprudent behaviors. These claims about the linkage between parenting and the inculcation of self-control have been empirically supported (for a review, see Cullen, Unnever, Wright, and Beaver 2008).

Although Gottfredson and Hirschi's low self-control construct is theorized to be a constellation of traits that come together within individuals, this does not mean that the assorted elements of low self-control exert equal weight in producing antisocial conduct. Assorted studies based on diverse samples of data have suggested that within the low self-control construct, the cardinal subcomponent was gratification delay (Krueger, Caspi, Moffitt, White, and Stouthamer-Loeber 1996), self-centeredness/narcissism (Vaughn, DeLisi, Beaver, Wright,

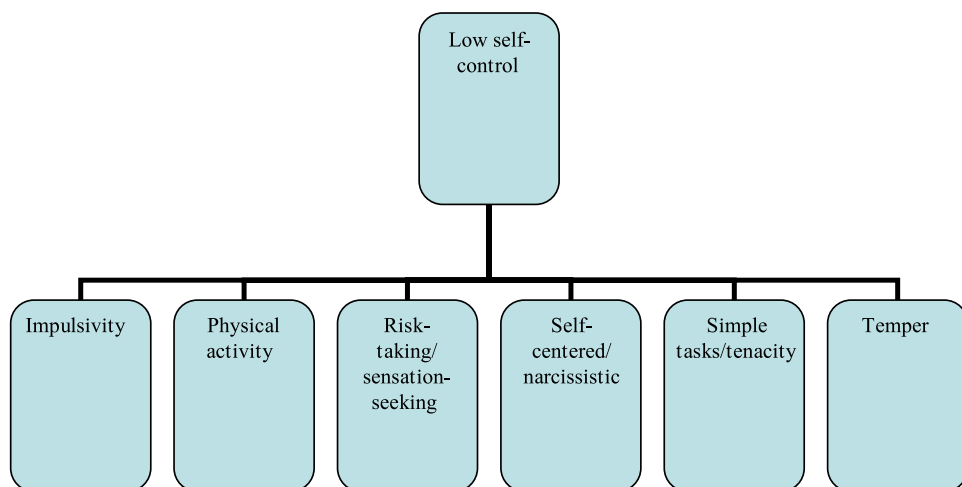


Figure 1 Low self-control and its constitutive parts.

and Howard 2007), temper/disputatiousness (DeLisi, Hochstetler, Higgins, Beaver, and Graeve 2008; DeLisi, Hochstetler, and Murphy 2003), or risk-taking (Arneklev, Grasmick, Tittle, and Bursik 1993). Others have found that the temper and risk-taking/risk-seeking dimensions together were particularly predictive of deviant acts (Hochstetler and DeLisi 2005; Longshore, Turner, and Stein 1996; Piquero and Rosay 1998).

Indeed, disparate elements of self-control account for varying amounts of variance in the overall self-control construct. For example, Vaughn et al. (2007) found that narcissism accounted for nearly two-thirds of the variance in low self-control. Self-control also does not manifest equally across social groups. For instance, LaGrange and Silverman (1999) reported that impulsivity is most strongly related to delinquency for males and risk-taking is most strongly related to delinquency for females. Williams, Fletcher, and Ronan (2007) compared the invariance of self-control among prisoners and male students and found that the self-centeredness and preference for simple tasks were particularly salient among prisoners versus students. For students, impulsivity accounted for the bulk of the variance in self-control.

In their study of the offending and victimization experiences of male homeless street youths, Baron, Forde, and Kay (2007) found that the risk-taking/risk-seeking dimension of self-control was the only component of low self-control that was positively associated with victimization. Conversely, self-centeredness was associated with lower levels of victimization (also see Piquero, MacDonald, Dobrin, Daigle, and Cullen 2005; Schreck 1999; Schreck, Wright, and Miller 2002; Stewart, Elifson, and Sterk 2004). Relatedly, Forde and Kennedy (1997) found that impulsivity, self-centeredness, and preference for simple tasks are negatively associated with victimization. Whereas several investigators have empirically assessed which subcomponent was "the best" element of self-control, others have found that various components of low self-control had relatively low

predictive validity. For example, Arneklev et al. (1993) found that although an attitudinal measure of self-control was predictive of drinking and gambling, the simple tasks and physical activities subcomponents had the weakest predictive power. In their study of academic dishonesty-related deviance, Cochran, Wood, Sellers, Wilkerson, and Chamlin (1998) found that the physical activity part of low self-control was the weakest.

In sum, research has produced mixed findings about the various predictive validity of self-control and its subcomponents and antisocial behavior. This is suggestive that correlated but discrete personality and sociocognitive dimensions undergird self-control. Moreover, the etiology of self-control is likely more complex than Gottfredson and Hirschi (1990) theorized. Although Gottfredson and Hirschi's general theory is a sociological one, its central construct is congruent to executive functioning. Executive functioning refers to the higher order intellectual tasks that occur in the prefrontal cortex that pertain to behavioral control, modulation of emotions, planning and decision-making and related activities (see Figure 2). For some time, biosocial criminologists have noticed the overlap between self-control as it is conventionally understood to criminology audiences and executive functioning. For instance, Moffitt (1990) observed:

The normal functions of the frontal lobes of the brain include sustaining attention and concentration, abstract reasoning and concept formation, goal formulation, anticipation and planning, programming and initiation of purposive sequences of motor behavior, effective self-monitoring of behavior and self-awareness, and inhibition of unsuccessful, inappropriate, or impulsive behaviors, with adaptive shifting to alternative behaviors. These functions are commonly referred to as "executive functions," and they hold consequent implications for social judgment, self-control, responsiveness to punishment, and ethical behavior. (p. 115)

Recently, Beaver, Wright, and DeLisi (2007) explicitly reformulated the self-control construct from Gottfredson and Hirschi's general theory and recast it as a part of the executive functions that are anatomically housed in the prefrontal cortex. Drawing on data from nearly 3,000 children selected from the Early Childhood Longitudinal Study, Kindergarten Class of 1998-1999 (ECLS-K), Beaver and his colleagues found that neuropsychological deficits were significantly associated with levels of self-control among children in kindergarten and first grade. These effects withstood competing confounds for parental involvement, parental withdrawal, parental affection, family rules, physical punishment, neighborhood disadvantage, gender, race, and prior self-control (also see Beaver, DeLisi, Vaughn, and Wright 2010; Beaver, Vaughn, DeLisi, and Higgins, in press).

That the low self-control construct is largely shorthand for executive functioning comports with research that suggests that self-control has a biosocial—and not entirely social—etiology. To illustrate, Wright and Beaver (2005) analyzed twin data from the ECLS-K and showed that self-control was not

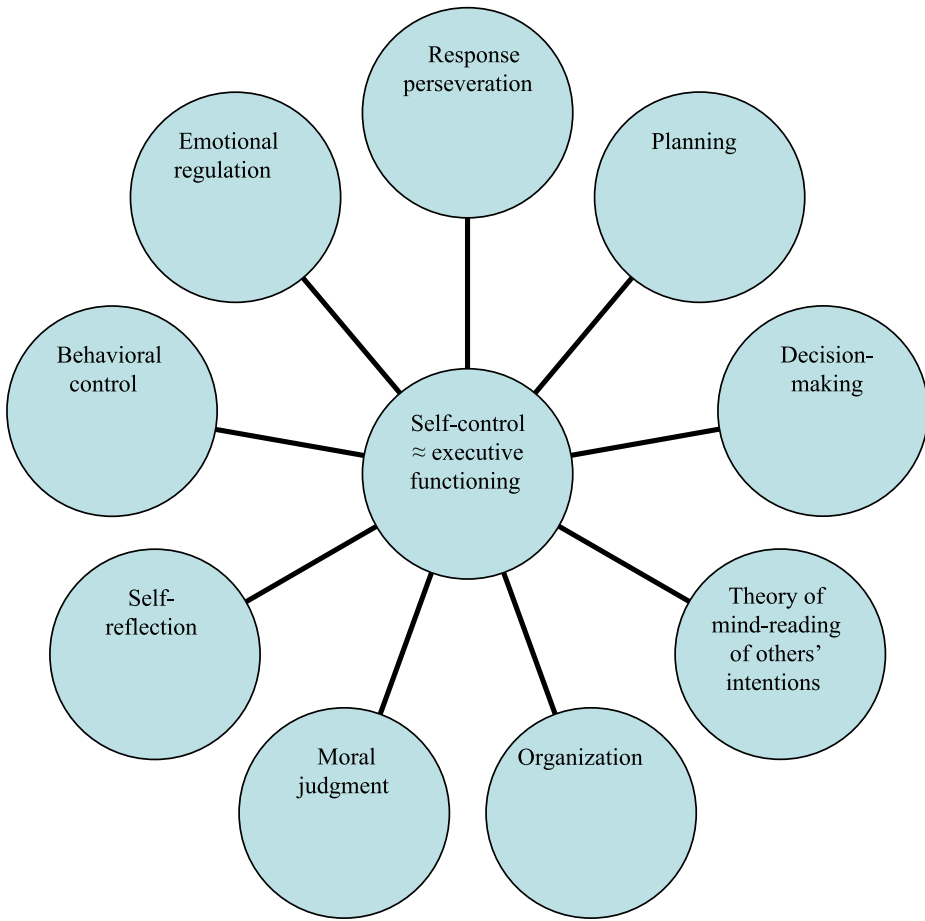


Figure 2 Self-control ≈ executive functioning.

caused by parenting once measures for genetic similarity were included. A follow-up study by Wright et al. (2008) found that only 1% of self-control was the results of parenting practices—which empirically refutes the causal framework advanced by Gottfredson and Hirschi (1990). Moreover, they found that about 25% of self-control was genetic in origin and the rest was caused by non-shared environmental factors, which are influences occurring outside of the family such as peer groups. In addition, Wright et al. (2008) found that parenting accounted for 0% of the variance in delinquency committed during the second wave of data collection and 2% of the variance in crime occurring during the third wave of data collection. In contrast, genes accounted for 44% of the variance in delinquency and 29% of the variance in crime. Overall, this newer area of self-control research shows that self-control is importantly related to delinquency; however, its causes are more genetic than social. Similarly, Nofziger (2008) found that a child’s self-control was predicted by his or her mother’s level of self-control and not necessarily parenting techniques. In fact,

how one parents his or her children was itself caused by one's own level of self-control.

Outside of criminology, prior research has utilized an endophenotypic approach to understand executive functioning, or more generally, self-regulation. For instance, Comings (2003) suggested that deficits in the dorsolateral prefrontal lobes produce symptoms that are essentially those of ADHD. These include inattention, impulsivity, distractability, disinhibition, impaired planning, poor organization, absence of motivation, and poor abstract reasoning. Defects in the orbitofrontal prefrontal lobes produce symptoms that are essentially those of conduct disorder (when observed in children and adolescents) and antisocial personality disorder (when observed in adults). These characteristics include affective disorders, aggression, poor self-control, emotional outbursts, and lack of guilt, remorse, and/or empathy.

Similarly, Castellanos and Tannock (2002) suggested three putative endophenotypes that underscore ADHD. These were: *shortened delay gradient* which is the result of brain abnormalities, including excessive striatal dopamine transporter, striatal lesions, and cerebellar vermis hypoplasia. These brain abnormalities are believed to result in a preference for small, immediate rewards as opposed to larger, delayed rewards. The second, *temporal processing*, refers to the frequent lapses in attention and intention and the moment-to-moment inconsistency in behavioral performance that typifies children with ADHD. Castellanos and Tannock (2002) suggested that temporal processing was the result of the above-mentioned brain abnormalities and prefrontal activity in the COMT Val/Met polymorphism. *Working memory deficits* was hypothesized to be an endophenotype caused by striatal lesions, COMT activity, a possible defect in glial glucose metabolism, and hippocampal dysfunction. The result of working memory deficits is associated with executive dysfunction particularly in regard to problems with focused attention.

An even more fruitful approach to studying the endophenotypes of antisocial traits involves the use of neuroimaging technologies such as functional magnetic resonance imaging (fMRI). The neuroimaging of brain activity during performance of behavioral tests allows a better glimpse into the neural substrates that are responsible for specific endophenotypes (also see Smit et al. 2010). For example, Greene, Braet, Johnson, and Bellgrove (2008) reviewed three executive functions—sustained attention, working memory, and response inhibition—and reported that all were strongly heritable and importantly related to ADHD, autism, and schizophrenia. A commonality of these disorders centers on executive functioning generally and self-control/self-regulation specifically. Indeed, in their review of ADHD endophenotypes, Crosbie, Pérusse, Barr, and Schachar (2008) advised, “executive function deficits could be useful markers of genetic risk in ADHD given the importance of these processes in ADHD, their association with the proposed biological basis of ADHD, and the ease with which these markers could be exploited in clinical and general population samples” (p. 51). The role of genetic markers is explored next.

Molecular Genetic Associations and Self-Control/Executive Functioning Endophenotypes

An endophenotype approach to the low self-control construct permits a disaggregated and interdisciplinary approach where social, biosocial, and biological constructs can be studied for their contribution to self-regulation. Because behavioral phenotypes or outcomes, such as delinquency, crime, and violence, are so complex, it is necessary to decompose the nature of these complex, multifactorial phenotypes in order to explore their genetic underpinnings and social influences on development and course. Once the endophenotypic structure of complex traits or behaviors is understood, investigators can explore candidate genes for the psychopathological conditions that actuate the disorder. To put this into theoretical perspective, genetic associations would be explored for their relation to the self-control endophenotypes which in turn culminate in the low self-control phenotype (see Figure 3). The figure contains a mere few examples of genes that have been linked to one of more self-control endophenotypes. To review, pleiotropic effects occur when single genes are associated with multiple traits or conditions, and polygenic effects speak to multiple genes bearing on a single trait or condition.

Using a range of methodologies, data sources, and candidate genes, genetics researchers have produced evidence for self-control endophenotypes. For impulsivity, Fineberg et al. (2010) reviewed the molecular genetic and neural substrates associated with compulsive and impulsive behaviors. They concluded that self-regulation disorders represent a balance between serotonin receptor gene (5-HT2A and 5-HT2C) activity in the ventromedial prefrontal (VMPFC) and orbitofrontal cortices (OFC), regions which regulate response inhibition and dopaminergic activity in the ventral anterior cingulate cortex (ACC) and ventral striatum/nucleus accumbens which regulate reward and reinforcement

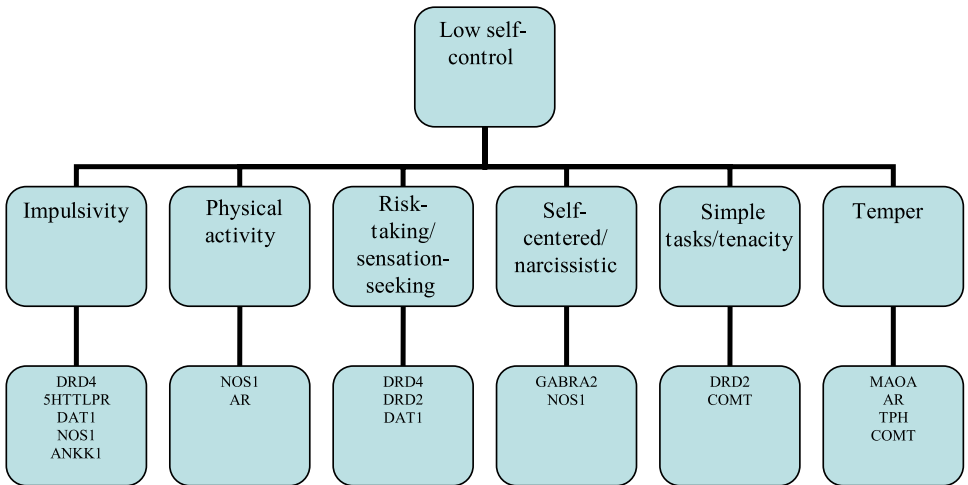


Figure 3 Genotype→ endophenotype→ phenotype conceptual model.

behavior. In this way, the difficulty in delaying gratification and choosing immediate small rewards despite their negative long-term consequences reflects deficits in the neural systems linking the orbitofrontal cortex and subcortical connections which in turn are affected by neurochemistry in the serotonergic and dopaminergic systems. Similarly, White, Morris, Lawford, and Young (2008) examined the A1 allele of the ANKK1 TaqIA polymorphism which is associated with reduced dopamine receptor density in the striatum and implicated in addiction. In their study, 72 healthy young adults were randomly assigned to either a relaxation induction condition or an acute stress condition and behavioral phenotypes of impulsivity were measured using a card-sorting index of reinforcement sensitivity and computerized response inhibition and delay discounting tasks. Those with the A1 ANKK1 allele displayed deficits relating to reinforcement-learned and evinced an impulsive behavioral style compared to those with the A2 allele. Moreover, these genetic effects were independent of stress.

For action/non-cognitive pursuits, Mill et al. (2006) examined the linkages between the 7-repeat allele of DRD4 and 10-repeat allele of DAT1 on ADHD and intellectual functioning using data from two birth cohorts, the E-Risk Study of 2,232 children in England and the Dunedin (New Zealand) study of 1,037 children. They found that DRD4 and DAT1 predicted intellectual functioning and ADHD which although not identical are consistent with the action-orientation and non-cognitive disposition found in Gottfredson and Hirschi's (1990) self-control construct. Moreover, in the Dunedin study, subjects were followed up to age 26 and evaluated for 10 adult outcomes, including violent conviction, nonviolent conviction, substance abuse diagnosis, psychiatric diagnoses, aggression against partner, aggression against minors, no high school qualification, out-of-wedlock parenthood, government welfare benefits, and long-term unemployment of more than six months. These outcomes additionally comport with the low self-control disposition (DeLisi 2003; Kreek, Nielsen, Butelman, and LaForge 2005; Lynam and Miller 2004).

The trait endophenotype novelty/sensation-seeking was the first to be linked to a specific gene: DRD4. The 7-repeat allele of DRD4 has been shown to be associated with novelty/sensation-seeking personality traits (Benjamin et al. 1996; Ebstein et al. 1996; Cloninger, Adolphson, and Svrakic 1996). In their review of the first decade of research on DRD4, Hubert Van Tol—whose research was instrumental in discovering the gene—and his colleagues advised that “numerous reports of linkage or a weak association between the 7-repeat/long alleles of DRD4 and novelty-seeking, drug and alcohol abuse, ADHD, and Tourette syndrome may indicate that the dopamine D4 receptor polymorphism is one of several genetic contributions to these traits or disorders” (Oak, Oldenhof, and Van Tol 2000, p. 316). In a more recent review, Ebstein (2006) assessed, “We deduce with some measure of certainty that DRD4 indeed contributes to personality and behavioral traits related to the Novelty Seeking phenotype” (p. 435).

For temper, Manuck et al. (1999) explored the associations between aggressive/antagonistic behavior and a polymorphism of the gene coding for tryptophan hydroxylase (TPH) which is implicated in serotonin biosynthesis. In a

community sample of 251 adults, Manuck et al. (1999) found that persons possessing alleles of an intronic polymorphism in the TPH gene, specifically the A218C U allele, scored significantly higher on aggression, propensity to unprovoked anger, and outward expressions of anger than persons with the alternate L allele. Williams et al. (2009) recently studied electrical brain activity and antisocial personality traits among 210 participants with low or high MAOA alleles. They found that those with the low-activity MAOA genotype showed alterations in brain electrical activity that was most apparent for negative emotions such as overt anger. In addition, these individuals had higher scores on an index of antisocial traits.

For global low self-control (not its constitutive parts) and based on genetically sensitive data derived from the National Longitudinal Study of Adolescent Health (Add Health), Beaver et al. (2010) reported an interaction between the low-activity allele of the MAOA gene and neuropsychological deficits in the prediction of not only low self-control, but also adolescent delinquency. This is important because neuroimaging studies have shown that persons with the low-activity allele of MAOA have reduced limbic volume and reduced neural activity in the prefrontal cortex. Moreover, the low-activity allele of MAOA has been cited as a candidate gene or risk marker for impulsive antisocial behavior (Meyer-Lindenberg et al. 2006). Similarly, Retz, Reif, Freitag, Retz-Junginger, and Rösler (2010) recently reported a variant in the neuronal nitric oxide synthase gene (NOS1) that was significantly associated with impulsivity, venturesomeness, and reduced empathy in a sample of 182 male criminal offenders. Other genes that have been linked to low self-control include the 5HTTLPR polymorphism in the serotonin transporter gene (Beaver, Ratchford, and Ferguson 2009).

Molecular genetic association studies are both a bane and a blessing in understanding antisocial traits and behaviors. Cynics caricature the 'gene for' implications that stem from the external validity of their findings viz. that a polymorphism in a gene is associated with some trait, behavior, outcome, or phenotype. Supporters identify genes that are involved in particular neurotransmitter systems and neurophysiological processes that give rise to the disorders. In the neuroscience literature, there is clear evidence that personality traits (such as those that comprise low self-control) are associated with individual differences in neural circuits involved in emotional processing, emotional regulation, and behavioral inhibition (Canli, Ferri, and Duman 2009). Thus, if low self-control represents a cortical inability to reign in subcortical impulses, an understanding of the candidate genes described here and the specific neural pathways that they innervate will lead to a greater understanding of self-control's endophenotypic structure.

Discussion

In a recent criminological study of Gottfredson and Hirschi's (1990) theory, Baron et al. (2007) observed:

Self-control does not appear to be a unidimensional construct. Rather, it appears to be six distinct elements that have differing effects on imprudent behavior, propensities for violence, violent acts, and victimization. There is little evidence that the six elements are important in each case. Instead, the results show that one or more of the elements emerge as an important predictor depending on the behavior examined. (p. 130)

Within criminology, a sizeable literature has developed surrounding the measurement of self-control. Much of this work has focused on the dimensionality of the low self-control construct (for a review, see Piquero 2008). Unfortunately, this vein of research—common in psychology—becomes fixated on measurement issues to such an extent that the more important substantive and theoretical issues become clouded. An endophenotypic approach to the study of crime and related constructs guards against this devolvement into the minutiae of measurement, and echoes the optimistic, disaggregation approach suggested by Baron et al. (2007).

Self-control theory is likely without peer in terms of the interest it has commanded and scrutiny with which it has been subjected (DeLisi and Vaughn 2008). Although there remain ancillary issues (e.g., whether self-control operates the same for males and females, whether attitudinal or behavioral measures or both, whether self-control has been conflated with or is a personality facet, etc.) to be resolved, there is no question empirically that self-control is inversely related to antisocial conduct. The empirical strength of Gottfredson and Hirschi's (1990) theory stands to reason because it aligns with even more vast psychological and psychiatric literatures which have linked temper/aggression, impulsivity, (hyper)activity, narcissism, preference for simple tasks/lack of tenacity, and sensation-seeking/risk-taking/novelty-seeking with deviant, imprudent, antisocial, and criminal outcomes.

So although Gottfredson and Hirschi could not have been more right to introduce an omnibus, individual-level variable like self-control to criminology, they could not be more wrong about the etiology of that construct. The theory suggests that self-control is produced by parenting, exclusively. Not only is self-control produced by other environmental factors such as schools (Turner, Piquero, and Pratt 2005), but also it is produced by genes and other biosocial factors (Nofziger 2008; Ratchford and Beaver 2009; Turner, Hartman, and Bishop 2007). Indeed, more than half of the variance in self-control is produced by genes. For this reason, children with the most significant impairments in self-control are also those with the most significant neuropsychological deficits (Vaughn, DeLisi, Beaver, and Wright 2009). The next generation of self-control studies will likely refine and correct Gottfredson and Hirschi's etiological account of self-control.

But, as explored at the outset of this study, genes and the brain are not the usual study material for professional criminologists; indeed almost no graduate programs in criminology and criminal justice provide such training. As such, the current study demonstrated the relevance of endophenotypes by showing that the subcomponents of the low self-control construct are mediated genetically

and neurologically. By parceling self-control into its constitutive parts, more parsimonious explanations can be advanced to understand the ways that putative genes are associated with constructs such as temper, impulsivity, narcissism, and others. In a recent essay, Plomin, Haworth, and Davis (2009) suggested that the study of polygenic disorders will lead to a focus on quantitative dimensions rather than qualitative disorders. With endophenotypes, criminologists can study those six dimensions of Gottfredson and Hirschi's (1990) theoretical construct and quantitatively demonstrate its multifactorial basis. Only then, will the true generality of the general theory be understood.

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Glossary

Endophenotype: The intermediate traits or characteristics that connect genes to outcomes and are genetically simpler than those outcomes.

Executive functioning: Refers to the higher order intellectual tasks that occur in the prefrontal cortex that pertain to behavioral control, modulation of emotions, planning and decision-making and related activities.

Heritability: The proportion of phenotypic variance in a population that is due to genetic variation.

Mendelian trait: A trait that is controlled by a single genetic locus and follows the simple inheritance pattern proposed by Gregor Mendel.

Multifactorial: A phenotype that is produced by genes, environment, and their interaction, such as low self-control.

Mutation-selection balance: Process by which new mutations at the disease locus are introduced into the population at a low frequency.

Oligogenic: Gene by gene interaction to produce a phenotype.

Phenotype: An observable trait or characteristic that is an expression of genes, environment, and their interaction.

Pleiotropic: Single genes that predict or affect multiple phenotypes.

Polygenic: Phenotypes that are predicted or affected by multiple genes.

Polymorphism: Multiple alleles or variants of a single gene in a population.