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On the genetic and environmental reasons why intelligence correlates with criminal victimization

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ABSTRACT

Researchers have expended considerable effort to understand the causes and correlates of criminal victimization. More recently, scholars have focused on identifying individual-level traits that increase the odds of victimization. Generally absent from this line of research, however, is examining the extent to which previously unmeasured genetic and environmental influences contribute to the covariation between victimization and individual-level risk factors. The current study aims to replicate and extend prior research by examining the contribution of genetic and environmental influences on the association between intelligence and victimization by analyzing twin and sibling data from two nationally representative samples of American youth. Quantitative genetic analyses indicate that common additive genetic factors, as well as non-shared environmental factors, explained the phenotypic association between intelligence and victimization. Finally, our results revealed that after correcting for possible familial confounding, the effect of intelligence on victimization experiences remained statistically significant. The findings of the current study replicate and extend prior research on the phenotypic association between indicators of general intelligence and the experience of victimization.

1. Introduction

Personal victimization can have serious consequences, ranging from physical injury and loss of property, to psychological and emotional trauma (Graham & Juvonen, 2001; Menard, 2002). Researchers across a range of disciplines, including criminology, psychology, and sociology, have expended considerable effort to understand the factors that lead to personal victimization, and to construct theories of victimization that integrate findings from this body of research (Cohen & Felson, 1979). To date, however, much of the effort devoted to understanding the risk factors for victimization has focused on identifying environmental factors, such as lifestyle choices, residence in disadvantaged neighborhoods, and exposure to delinquent peers (routine activities/lifestyle theories; e.g., Averdijk & Bernasco, 2014; Schreck & Fisher, 2004; Schreck, Fisher, & Miller, 2004) to explain personal victimization.

Recently, however, studies have begun examining individual-level attributes that may increase the likelihood of personal victimization. Certain cognitive factors and psychological traits, for instance, have been found to increase the risk of being criminally victimized (Beaver, Nedelec, Barnes, Boutwell, & Boccio, 2016; Cohen & Felson, 1979). Self-control, for example, is consistently associated with an increase in the odds of victimization (see Pratt, Turanovic, Fox, & Wright, 2014, for meta-analysis). Several other psychological traits, such as anger, psychopathy, and self-regulation (Finkelhor, Ormrod, & Turner, 2007; Henry, Caspi, Moffitt, & Silva, 1996; Silver, Piquero, Jennings, Piquero, & Leiber, 2011) have also been identified as increasing the risk of personal victimization.

Given the consistent association between victimization and traits such as self-control, scholars have also hypothesized that another individual-level trait—general intelligence—may predict victimization. This hypothesis is derived from research findings indicating that lower intelligence is associated with exposure to criminal peers (Kimonis, Frick, & Barry, 2004; Seals & Stern, 2013), drug culture (Duncan, Kennedy, & Smith, 2000; Latvala et al., 2011), lower self-control

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(Meldrum, Petkovsek, Boutwell, & Young, 2017), and greater risk-taking propensities (Pharo, Sim, Graham, Gross, & Hayne, 2011)—all of which are associated with increased odds victimization. Beaver et al. (2016) directly tested the intelligence-victimization link using nationally representative data from the National Longitudinal Study of Adolescent to Adult Health and documented an association between verbal intelligence and criminal victimization in early adulthood. The results suggest that as scores on an indicator of general intelligence decreased, the odds of falling victim to criminal behavior increased.

Additionally, a growing line of research has begun examining the potential for genetic contributions to victimization experiences (Vaillancourt, Hymel, & McDougall, 2013) by utilizing behavioral genetic designs—adoption, twin, or sibling analyses. These studies have consistently revealed that victimization experiences are at least moderately heritable (Ball et al., 2008; Barnes & Beaver, 2012; Beaver, Boutwell, Barnes, & Cooper, 2009; Beaver et al., 2007; Connolly & Beaver, 2014; Hines & Saudino, 2004). This is particularly important given that the phenotypes correlate with victimization are also, to varying degrees, heritable (see Beaver et al., 2016). Individual differences in levels of self-control, for example, have consistently been shown to be under genetic influence (Beaver, Wright, DeLisi, & Vaughn, 2008). Criminal involvement is also moderately to highly heritable (Barnes, Beaver, & Boutwell, 2011). General intelligence, likewise, is highly heritable—becoming increasingly so as individuals age (Plomin & Deary, 2015). Taken together, this evidence suggests that at least part of the reason why victimization covaries with other phenotypes could be because shared genetic factors influence both traits (see Barnes, Boutwell, Beaver, Gibson, & Wright, 2014 for more discussion).

Despite the possible genetic correlation between victimization and associated risk factors, little empirical work has directly addressed the issue of shared genetic etiology for explaining victimization, more generally. Barnes and Beaver (2012) examined the victim-offender overlap using a nationally representative sample and reported that genetic factors explained between 51% and 98% of the association between offending and victimization. Similar results have been documented for the association between victimization and low self-control (Boutwell et al., 2013), and for the association between violent victimization and criminal behavior (Vaske, Boisvert, & Wright, 2012). It is therefore reasonable to hypothesize that the effect of certain risk factors, such as intelligence, on victimization might exist not because one is necessarily causing the other, but also because the two traits correlate at a genetic level. Both general intelligence and victimization are heritable traits and, as mentioned above, Beaver et al. (2016) documented a negative association between verbal intelligence and criminal victimization experiences in early adulthood. What the analysis by Beaver et al. (2016) did not reveal was the extent to which the phenotypic correlation between intelligence and victimization may be accounted for by shared—or correlated—genetic and environmental influences.

The current study aims to replicate and extend prior research on the common genetic and environmental influences on intelligence and victimization. We analyzed data from two nationally representative samples of American youth to first estimate the phenotypic correlation between indicators of general intelligence and criminal victimization. If both traits correlate at the phenotypic level—which we hypothesize based on findings from Beaver et al. (2016)—we will then extend the analysis and estimate a series of behavioral genetic models to examine the relative contribution of genetic and environmental influences on the phenotypic correlation between multiple indicators of general intelligence and criminal victimization.

2. Method

2.1. Data

To further examine the link between indicators of general intelligence and victimization, we utilized data drawn from the National Longitudinal Survey of Youth 1997 (NLSY97) and the Children of the National Longitudinal Survey of Youth 1979 (CNLSY).

2.2. NLSY97 sample

The NLSY97 is a nationally representative sample of youth born between 1980 and 1984 living in the United States during the initial survey wave (see also, Connolly & Beaver, 2016). Participants were first surveyed in 1997, and then assessed annually from 1997 to 2011. The NLSY97 sample was the product of a stratified multistage cluster probability sampling design where over 90,000 households were initially selected using probability sampling methods. After this step, NLSY staff identified a target sample of 9808 age-eligible youth for participation in the study. Youth between the ages of 12 and 16 years as of December 31, 1996 were asked to participate in the NLSY97. There were multiple youth between the ages of 12 and 16 years from the same household who agreed to participate in the NLSY97, resulting in many participants in the NLSY97 being biologically related to one another. Previous research has taken advantage of questions asking respondents about their biological or social relationship with other household members to identify levels of biological relatedness between participants in the NLSY97 (Connolly & Beaver, 2016). To validate the kinship links from this method, a series of biometric analyses were conducted using measures of height. Because height is a highly heritable phenotypic trait (heritability estimates ranging from $h^2 = 0.80$ to $h^2 = 0.90$; Silventoinen et al., 2003), height scores were standardized by average heights in the NLSY97 based on age and sex sample norms. Results indicated strong convergent validity between heritable estimates for male and female height in the NLSY97 and those reported in other heterogeneous sibling samples (for more information, see Connolly & Beaver, 2016).

Once sibling pairs of varying levels of genetic relatedness in the NLSY97 were identified, one sibling pair per household was randomly selected to be included in the sample. Because the NLSY97 is a nationally representative sample of youth and staff did not over sample for twins, full siblings represent close to 90% of the sibling sample. A random sample of full-sibling pairs was therefore taken from this population to be included in the final NLSY97 sibling sample. After randomly selecting a sample of full-sibling pairs, the final analytic sample included $n = 1085$ sibling pairs that included 22 monozygotic (MZ) twin pairs (who share 100% of their segregating genes), 30 dizygotic (DZ) twin pairs (who share, on average, 50% of their segregating genes), 947 full-sibling pairs (who share, on average, 50% of their segregating genes), and 86 half-sibling pairs (who share, on average, 25% of their segregating genes). All siblings included in the final analytic sample provided valid information on each measure examined in the current study.

2.3. CNLSY sample

The CNLSY is a sample of youth born to a nationally representative sample of women between the ages of 14 and 21 years in 1979 (NLSY79). Beginning in 1986, children born to women from the NLSY79 were surveyed to create the CNLSY. Children in the CNLSY have been surveyed biennially, beginning in 1986, and completed measures designed to assess cognitive, emotional, and behavioral development. In 1994, children age 15 years and older were administered a self-report survey that asked questions about age-appropriate behaviors, including sexual intercourse, deviance, personal relationships, substance use, and victimization. Because the CNLSY sampled multiple children from the same mother, many participants are biologically related to one another. Although information about levels of biological relatedness between participants was not originally collected, Rodgers, Rowe, and May (1994) has used self-report information on the type of relationship shared between each participant and other household members to develop a linking algorithm that assigns children from the CNLSY a sibling status and genetic relatedness score.