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A New Piece to Understanding the Intimate Partner Violence Puzzle: What Role Do Genetics Play?

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Abstract

Numerous factors prompt intimate partner violence (IPV). Stuart and colleagues examined whether variations in two theoretically relevant genetic variants predispose some people toward perpetrating psychological aggression, physical aggression, and violence that causes the victim serious physical injury. This commentary discusses the importance of considering how the observed genetic risk factors for IPV may be best understood within the context of their interaction with environmental risk factors. By focusing on gene–environment interactions, future work may help identify not only who is at risk for IPV perpetration but also who may be buffered from it.

Keywords

genetics; intimate partner violence

Abdelmalek Bayout is a controversial murderer. He confessed to killing Walter Felipe Novoa Perez, who insulted Bayout's use of religious eye make-up. But this confession is not what gripped headlines and shook the foundation of legal ethics (Baum, 2013). At his appeal hearing, Bayout received a lenient sentence because he possessed a genetic marker associated with a greater propensity toward violence. Controversy erupted. Should genetic markers be used to exculpate people of responsibility for their violent actions? Or should genetic markers only be used to test hypotheses regarding the probability that a person may behave aggressively?

According to Stuart and colleagues (2014), the answers are a definitive *No* and *Yes*. This commentary is separated into four sections. First, it discusses how Stuart and colleagues' study demonstrates how psychologists can avoid current conundrums facing hypothesis-

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driven candidate gene research. Second, it argues that the genetic factors Stuart and colleagues identify likely interact with environmental variables. The third section asserts that in positive environments, scores on Stuart and colleagues' cumulative genetic risk variable may increase a person's propensity toward positive behavior. The final section discusses the role genetics may play in the future of intimate partner violence (IPV) research.

A Snapshot of the Future of Genetics Research?

In their careful study, Stuart and colleagues (2014) tested the hypothesis that a cumulative genetic risk score comprising polymorphisms in the monoamine oxidase A gene (*MAOA*) and the serotonin transporter gene (*5-HTTLPR*) would relate to a greater propensity to perpetrate psychological aggression, physical aggression, and cause serious physical injury to a romantic partner. They collected genetic and self-report data from 97 men who were hazardous drinkers and who were enrolled in a batterer intervention program. Men with higher scores on the cumulative genetic risk score had a higher probability of perpetrating IPV, as indicated by elevated levels of psychological aggression, physical aggression, and injury perpetration.

Why might this approach provide a glimpse into the future of genetics research? The main innovation is that Stuart and colleagues (2014) used a cumulative genetic risk score instead of focusing on single genetic variants. Genetics research has experienced a tremendous crisis of confidence, with many candidate gene findings failing to replicate. Indeed, the editor of *Behavior Genetics* recently summarized this crisis by stating that "it now seems likely that many of the published findings of the last decade are wrong or misleading and have not contributed to real advances in knowledge" (Hewitt, 2012, p. 1). These replication failures occur for many reasons, including the small effect that any particular genetic variant exerts on a given behavior. By focusing on the cumulative effect of two polymorphisms, Stuart and colleagues harnessed the power of the effect that *MAOA* and *5-HTTLPR* exert individually on increasing the probability of IPV perpetration.

The cumulative risk score seeks to increase statistical power for studies with small sample sizes. To put this in perspective, the latest genome-wide association study (GWAS) for major depressive disorder was underpowered with 18,759 participants. The authors determined that 200,000 participants would be necessary to be adequately powered for identifying novel genetic variants (Major Depressive Disorder Working Group of the Psychiatric GWAS Consortium et al., 2013). Requiring such gargantuan sample sizes would halt most research that seeks to identify specific genetic contributors to psychological processes. Hence, cumulative genetic risk scores may prove to be a fruitful approach to better understanding the role of genetics in increasing the likelihood of certain behaviors, including IPV perpetration.

The next question for the field is to determine how many genetic polymorphisms constitute a valid and reliable index. From a psychometric perspective, self-report instruments often benefit from having multiple items to establish reliability and validity. Two-item questionnaires are often chided for low internal reliability (Gosling, Rentfrow, & Swann,

2003). Does the same logic apply to cumulative genetic variables? In their review, Stuart and colleagues (2014) highlighted the wide variability in the percentage of variance that genetic polymorphisms show in predicting aggression. By including multiple genetic variants within a single variable, researchers will not only have greater power to detect genetic influences on aggression but also identify which specific neurochemical systems best predict aggression.

A final point is to consider various conceptual approaches when constructing a cumulative genetic variable. Stuart and colleagues (2014) chose the *MAOA* and *5-HTTLPR* because of their prior connection to aggression. This is a logical approach, but it is based on the outcome variable. Future research may construct cumulative genetic variables on the basis of their relation to specific neural pathways (e.g., dopaminergic, serotonergic) instead of the outcome variable. Using this approach might be most beneficial when similar genetic variants are associated with both positive and negative outcomes.

Environment Matters

Genes matter, but they do not exist in a vacuum. Environmental factors interact with genetic polymorphisms to influence a variety of behaviors and processes (Caspi & Moffitt, 2006). Stuart and colleagues (2014) are correct that numerous studies have shown that the putative low expression alleles in the *MAOA* gene (*MAOA-L*) are associated with greater aggression. But there are also studies indicating that the low expression alleles are associated with lower aggression (e.g., Manuck, Flory, Ferrell, Mann, & Muldoon, 2000), suggesting that there are likely one or more moderators that influence the effects of *MAOA-L* on aggression. For example, this increased risk for aggression is found in conditions of provocation. One study showed that *MAOA-L* carriers, compared with their high expression *MAOA* counterparts, behaved more aggressively only when that person had provoked them (McDermott, Tingley, Cowden, Frazzetto, & Johnson, 2009). Unprovoked *MAOA-L* participants in that study behaved as aggressively as other participants in the absence of provocation. Similar moderating effects of *MAOA-L* on violence have been observed within the context of child maltreatment (Caspi et al., 2002).

Stuart and colleagues (2014) did not examine how their results were moderated by relevant environmental factors, which may limit the impact of their findings. Scientists and practitioners do not have a starting place from which to target interventions that may reduce aggression. Which environmental factors may have exacerbated the link between the cumulative genetic risk score and aggression in their sample? Researchers would need to conduct an additional study to identify a reliable environmental factor that moderates the effect of the variants that comprise Stuart and colleagues' cumulative genetic variable and their relationship to greater aggression. Until that information is in place, it will be difficult to use Stuart and colleagues' findings to design precise interventions that can modify those environmental factors and thereby reduce aggression.

Is it appropriate to use history of violence and substance abuse as environmental factors that may moderate the relationship between a genetic risk score and aggression? Stuart and colleagues' (2014) sample consisted of men with a history of domestic violence and

substance abuse. These men may have been exposed to more adverse environments during their development compared with men without a history of domestic violence and substance abuse. Although imprecise, Stuart and colleagues may have included a control group of men without a history of domestic violence and substance abuse to determine whether elevated cumulative genetic scores would have also related to greater aggression. Based on prior work demonstrating the importance of gene–environment interactions, we would predict that Stuart and colleagues’ findings would replicate only among a sample of men with a history of exposure to adverse events.

One Side of the Genetic Coin

Given the right circumstances, the most aggressive men in Stuart and colleagues’ (2014) sample might also be the nicest. Stuart and colleagues framed their argument on the basis of the well-supported diathesis-stress model (Monroe & Simons, 1991), in which some people demonstrate greater vulnerability to stressors than others because of a putative risk factor (e.g., a higher score on a cumulative genetic risk variable). An alternative perspective that has gained momentum in recent years, *differential susceptibility* (Belsky & Pluess, 2009), argues that researchers need to develop a new frame of mind when conceptualizing factors that may predispose people toward extreme responses. People may have greater biological sensitivity to the environmental context, which may increase the likelihood of negative or positive responses (Boyce & Ellis, 2005; Way & Taylor, 2010).

This perspective has received support in numerous domains. In one recent study, married couples completed a series of interactions in which they discussed either points of disagreement (a negative interaction) or a personal issue that one partner wanted to change (a positive interaction; Schoebi, Way, Karney, & Bradbury, 2012). Before and after each interaction, both members of each couple reported their levels of positive affect and negative affect. Both members of each couple also provided a saliva sample that was genotyped for the *5-HTTLPR* gene. Participants with the short allele variant of the *5-HTTLPR* gene were more responsive to their partner’s positive affect. This is the very same allele that Stuart and colleagues (2014) scored as a risk allele. These findings demonstrate that under positive conditions, genetic factors thought to increase risk for negative outcomes are linked to positive outcomes.

From a differential susceptibility perspective, Stuart and colleagues (2014) constructed a cumulative genetic variable that increases biological sensitivity to negative and positive environmental contexts. In this way, Stuart and colleagues identified people who are not only at greater risk for behaving aggressively in their romantic relationships but also most likely to behave positively in a good environment. By examining how people respond to both negative and positive situations, Stuart and colleagues will have a broadened perspective on the meaning their cumulative genetic variable may have in understanding how to foster positive and lasting romantic relationships.

What Does the Future Hold?

Our genes are a small, yet important, part of our identity. Genetic mutations can increase our chances of developing Alzheimer’s disease, Huntington’s disease, and Ogdan Syndrome,

among many other ailments (Plomin, Owen, & McGuffin, 1994). They can also increase our risk for engaging in various behaviors, both positive and negative. Stuart and colleagues (2014) provide one of the first attempts to wed behavior genetics with IPV research. The result is a compelling case that much can be gained from understanding a person's genetic background when attempting to identify who is at risk for behaving aggressively.

Despite this advance in the literature, more questions remain. The future of research in this domain will need to understand how genetic variants interact with environmental factors to influence IPV. A critical step for understanding these interactions is measuring not only negative aspects of the environment but also positive aspects. This will help to identify genetic variants that predispose some people toward enhanced biological sensitivity to the environment, which can predispose people toward violent or benevolent actions.

The case of Abdelmalek Bayout shook the fields of genetics, legal ethics, and psychological science. Understanding why people commit murder is almost impossible. To lessen a murderer's responsibility based on his genetic profile is fraught with danger. But these legal mishaps should not stand in the way of brave psychological scientists, such as Stuart and colleagues, from ushering in a new wave of research aimed at uncovering another piece of the puzzle as to why people perpetrate IPV.

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