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Antonia Abbey

Wayne State University, aabbey@wayne.edu

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# Responsible Integration of Biological and Psychosocial Models: Comments on “Genetic Associations with Intimate Partner Violence in a Sample of Hazardous Drinking Men in Batterer Intervention Programs”

**Antonia Abbey**

Department of Psychology, Wayne State University

5051 Woodward, Detroit, MI, 48202. Tel: 313 577 6686. E-mail: [aabbey@wayne.edu](mailto:aabbey@wayne.edu)

Despite research demonstrating that gene expression differs in response to social environmental circumstances, deterministic views of biology are common. Stuart and colleagues (this issue) encourage readers to think about genetic factors in the same dynamic and probabilistic manner that they consider other causes of intimate partner violence. Given that participants had co-occurring alcohol problems, future studies should evaluate how different genetic polymorphisms uniquely and synergistically contribute to heavy drinking and aggression under different socio-environmental conditions. Psychological expectancies have a powerful impact on behavior, thus extreme caution is required before labeling people as genetically predisposed to violence.

**Keywords:** violence perpetration, genetics, risk factors, alcohol

Like many feminist social scientists, I get nervous when I see the word “genetics” in the title of articles that examine potential causes of violence. Lay people, scientists, and policy makers often give potential biological causes of behavior precedence over psychological, social, cultural, and macro-economic causes. Despite great advances in understanding the synergistic relationships between the mind and the body (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010; Daruna, 2012; Taylor, Lerner, Sage, Lehman, & Seeman, 2004), deterministic views of biology are still

surprisingly common. Stuart, McGeary, Shorey, Knopik, Beaucage, and Temple (this issue) provide an exemplary guide for researchers who want to encourage readers to think about genetic factors in the same probabilistic manner that they consider other causes of intimate partner violence (IPV). Their article begins with an acknowledgement that “The etiology of IPV is multifactorial, with a dynamic interplay of variables that influence the probability of IPV differently across time and situations” (p. 3 of manuscript). Stuart et al. provide empirical and theoretical support for the selected candidate genetic polymorphisms, rather than using a data driven atheoretical approach. The authors interpret their findings cautiously as supporting the argument that some genetic polymorphisms increase the likelihood of violence through their effects on other risk factors such as impulsivity and emotion regulation skills. For all these reasons, I commend the authors for their contribution to the literature.

### **Shared Causes of Violence and Heavy Drinking: Control Away or Explain?**

Participants in Stuart and colleagues’ (this issue) study were 97 men who were arrested for domestic violence, diagnosed as hazardous drinkers, and attended a batterers’ intervention program in Rhode Island. The average educational attainment was less than high school completion and average annual income was less than \$25,000. All studies have external validity limitations and the authors state that it is important to replicate these findings with other populations. It is also important to recognize that research on gene environment interactions and epigenetics demonstrate that gene expression differs in response to social environmental factors, thus different trajectories are possible for people who experience different environmental forces (Caspi et al. 2010; Taylor et al., 2004; Young-Woff, Enoch, & Prescott, 2011).

Because participants had co-occurring alcohol problems, Stuart and colleagues statistically controlled for alcohol and drug problems by including measures of both on the first step in hierarchical regression analysis. Scores on the Alcohol Use Disorders Identification Test (AUDIT) were significantly correlated with psychological and physical violence as assessed by the revised Conflict Tactics Scale, and remained significant in regression analyses when the cumulative genetics score was added to the model. This finding is surprising because of the restriction of range in AUDIT scores in this sample due to the eligibility criteria (AUDIT scores

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of 8 or higher or monthly consumption of 5 or more drinks on one occasion). The reader is left wondering about the severity of the sample's alcohol problems and how it affects the generalizability of the findings.

Correlational studies do not allow alternative hypotheses to be ruled out, even when statistical controls are used (Shadish, Cook, & Campbell, 2002). Given the extensive literature that examines genetic contributions to alcoholism (see Young-Wolff et al., 2011 for a review), the authors could have considered participants' propensity to alcoholism as an important explanatory variable, rather than trying to control away its impact statistically. It would help readers interpret the findings if the authors described the research that examines the co-occurrence of the genetic variants linked to alcoholism and violence and whether they might have synergistic effects on men's likelihood of engaging in partner violence. The authors' postulation that low-expressing MAOA alleles contribute to biased information processing associated with serotonin metabolism suggests potential overlap in mechanisms as well. Acute alcohol consumption impairs many higher order cognitive functions, including the ability to recognize and process a wide range of information (Fillmore, Dixon, & Schweizer, 2000; Peterson, Rothfleisch, Zelazo, & Pihl, 1990; Pihl, Paylan, Gentes-Hawn, & Hoaken, 2003). Alcohol administration researchers typically explain increased aggression in intoxicated as compared to sober participants in laboratory paradigms as being partially due to biased perceptions of the cues in the situation, such that cues which encourage an aggressive response are more salient than cues that discourage an aggressive response (see Giancola, 2000 for a review). Serotonin has been linked to a wide range of social behaviors associated with both cooperation and aggression. Stuart and colleagues have started an important line of research by exploring some of these links. In future research, it is important to use a wider lens to determine the extent to which the mechanisms that have previously been identified as linking alcohol consumption and interpersonal violence overlap with those identified here. Thus, I echo the authors' caveat that this research needs to be replicated with perpetrators of intimate partner violence who are not heavy drinkers.

## Implications for Treatment, Prevention, and Research

Approximately half of the perpetrators of a wide range of types of violence are under the influence of alcohol during the incident (Abbey,

2011). Thus alcohol is a powerful risk factor, but it is clearly not necessary if half of the violence occurs when the perpetrator is sober. Stuart et al. end their article with the suggestion that future research may allow the tailoring of prevention and treatment programs to individuals with different genetic characteristics. Clearly, much more research is needed before people are labeled regarding their genetic predisposition to violence. Expectancies have a powerful impact on people's behavior, and other people's perceptions of their behavior (Snyder & Stukas, 1999). Thus these labels could have unexpected negative consequences if they create self-fulfilling prophecies.

One unanswered question concerns the specificity of genetic polymorphisms. In this study, there were similar findings for physical and emotional violence perpetrated against a steady partner. Although the authors assessed sexual violence perpetration, they did not report if the pattern of results was comparable. Theories are needed that elucidate when different forms of violence should be considered manifestations of the same underlying psychological states and when they are likely to arise from different needs and motives. We still do not know much about similarities and differences between individuals who perpetrate different types of interpersonal violence, not just different forms of intimate partner violence (e.g., emotional, physical, sexual) but also bullying, sexual harassment, stalking, child abuse, and elder abuse. This information is required to develop targeted treatment and prevention programs. If there are common underlying genetic contributors, as well as psychological, social, cultural, and structural contributors, this provides support for multi-faceted violence prevention and treatment programs. If these genetic contributors also overlap with genetic contributors for alcoholism, this provides strong support for more meaningful integration of violence and alcohol programs.

Current thinking about alcohol's role in interpersonal violence is in many ways similar to Stuart and colleagues' (this issue) description of the role of genetic factors in interpersonal violence. Intoxication increases the likelihood of aggression among individuals predisposed to behave aggressively in a particular context due to personality, attitudinal, and experiential factors (Abbey, 2011; Giancola, 2000). Stuart and colleagues suggest that polymorphisms associated with violence are most likely to affect the behavior of individuals who are extremely impulsive and individuals who have poor emotion regulation skills. Both of these

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variables are associated with an increased likelihood of heavy alcohol consumption, providing another example of the close connection between the mechanisms discussed by Stuart and colleagues linking violence to genetic variants and the mechanisms discussed in the alcohol field linking alcohol and violence. Further exploration of shared and unique pathways is essential for the development of effective programs.

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