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Genetic Associations with Intimate Partner Violence in a Sample of Hazardous Drinking Men in Batterer Intervention Programs

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Abstract

The etiology of intimate partner violence (IPV) is multifactorial. However, etiological theories of IPV have rarely included potential genetic factors. The purpose of the present study was to examine whether a cumulative genetic score (CGS) containing the *MAOA* and 5-HTTLPR polymorphisms was associated with IPV perpetration after accounting for the effects of alcohol problems, drug problems, age, and length of relationship. We obtained DNA from 97 men in batterer intervention programs in the state of Rhode Island. In the full sample, the CGS was significantly associated with physical and psychological aggression and injuries caused to one's partner, even after controlling for the effects of alcohol problems, drug problems, age, and length of relationship. Two of the men in the sample likely had Klinefelter's syndrome and analyses were repeated excluding these two individuals, leading to similar results. The implications of the genetics findings for the etiology and treatment of IPV among men in batter intervention programs are briefly discussed.

Keywords

Genetics; Intimate Partner Violence; *MAOA*; 5-HTTLPR; Batterer Intervention

The prevalence and frequency of intimate partner violence (IPV) is a major public health concern with significant negative consequences for victims (Jordan, Campbell, & Follingstad, 2010). A study conducted by the World Health Organization demonstrated that the prevalence of physical or sexual domestic violence against women ranged from 15%–71% in ten countries across the globe (Garcia-Moreno, Jansen, Ellsberg, Heise, & Watts, 2006). Within the United States, a large study evaluating couples found that over one-fifth

of the sample reported experiencing IPV within the past year (Schafer, Caetano, & Clark, 1998). Severe relationship violence that carries a high potential for injury (e.g., punching a partner; using a weapon) is also a highly prevalent national problem, with at least 1.3 million women severely victimized annually in the United States (National Center for Injury Prevention and Control, 2003). The medical costs related to IPV against women in the United States are staggering, estimated to range from \$2.3 billion to \$7.0 billion per year (Brown, Finkelstein, & Mercy, 2008).

The etiology of IPV is multifactorial, with a dynamic interplay of variables that influence the probability of IPV differently across time and situations (Chermack & Giancola, 1997; Crowell & Burgess, 1996; Leonard, 1993; 2001; 2005; Stuart et al., 2006; 2008; 2009a; 2009b). Although numerous etiological theories of IPV have been proposed (see Bell & Naugle, 2008 and Shorey, Cornelius, & Bell, 2008 for reviews), genetic factors have generally been neglected from theoretical consideration (Hines & Saudino, 2004). The failure to include genetic factors in etiological theories of IPV is notable given the increasing body of evidence to suggest the potential relevance of genetic factors to general forms of aggression (Pinto et al., 2010).

In a meta-analysis, Miles and Carey (1997) found that up to 50% of the variance in aggression was due to genetic influences, suggesting that specific genes might be identified that contribute to aggressive phenotypes. In a twin study, Coccaro, Bergeman, Kavoussi, and Scroczynski (1997) found that 69% of the variance in general physical assaults was explained by genetic factors. To our knowledge, only one previous study has examined whether there is a genetic component to IPV. In a study of 134 monozygotic and 41 dizygotic same-sex twin pairs, Hines and Saudino (2004) found that genetic factors accounted for 16% of the variance in physical IPV and 22% of the variance in psychological aggression.

Specific candidate genes have also been linked to aggressive behavior. A review of the genetics of aggression identified Monoamine Oxidase A (*MAOA*) as a major contributor (Volavka et al., 2004), with some even labeling it the “warrior gene” due to its relation with aggressive behavior (McDermott, Tingley, Cowden, Frazzetto, & Johnson, 2009). *MAOA*, located on the X chromosome, is a mitochondrial enzyme that metabolizes neurotransmitters including serotonin, dopamine, and norepinephrine (Pinto et al., 2010). There is a putatively functional variable number of tandem repeats polymorphism (*MAOA* uVNTR) with 3.5 and 4 repeat variants showing more efficient transcription rates than the 2 or 3-repeat versions (Huizinga et al., 2006). This *MAOA* variation has been associated with impulsive aggressive behavior, including violent criminality (e.g., Widom & Brzustowicz, 2006), aggression in laboratory paradigms (McDermott et al., 2009), and aggression in primates (Pinto et al., 2010).

Buckholtz and Meyer-Lindenberg (2008) proposed gene-environment interactions influencing violence involving low-expressing alleles of the *MAOA* u-VNTR (*MAOA*-L); they postulated that *MAOA*-L contributes to biased information processing in a corticolimbic circuit, via its effect on serotonin. In a sample of alcohol dependent men, Samochowiec et al. (1999) found that the low activity variant of the *MAOA* promoter polymorphism was

significantly associated with antisocial personality disorder. In one study, the high activity *MAOA*-linked polymorphism region interacted with alcohol consumption to predict violence longitudinally (Tikkanen et al., 2009). In another longitudinal study, Caspi et al., (2002) found that *MAOA* variation moderated the effect of child maltreatment on aggression-related outcomes. Relative to maltreated males with the high activity *MAOA* genotype, maltreated males with the low activity *MAOA* genotype were more likely to develop conduct disorder, to be convicted of a violent crime, and to have antisocial personality. In other studies, animals with *MAOA* genes knocked out exhibited elevated aggression, and in a birth cohort study, alleles that coded for decreased activity of *MAOA* were associated with violence, violent crimes, and antisociality (Volavka et al., 2004).

In addition to *MAOA*, there is extensive evidence to suggest that serotonin disturbances are related to violence and aggression (Retz et al., 2004). Indeed, serotonin dysfunction has been associated with violence, suicide attempts, and impulsivity (Retz et al., 2004). A review of serotonergic genes and aggressive behavior suggests the 5-HTTLPR in the serotonin transporter gene (*SLC6A4*) as a strong candidate to be associated with violence (Popova, 2006). 5-HTTLPR is a functional polymorphism in the promoter region of *SLC6A4*. The short (S) 5-HTTLPR allele is less transcriptionally efficient than the long (L) allele (Lesch et al., 1996). A third functional allele, L_G , has been identified (Nakamura et al., 2000) and has shown transcriptional capacity comparable to the S allele (Hu et al, 2005). Popova (2006) concluded that the 5-HTTLPR polymorphism modulates aggression. In a sample of 153 males obtaining a forensic evaluation, Retz et al., (2004) found that men with histories of physical violence against others were significantly more likely than nonviolent men to have the short (S) allele and the S/S genotype. This polymorphism accounted for 5% of the variance of violent behavior.

Many studies of genetics and violence have focused on single genetic variants. A problem with this approach is that the examination of any single variant may be obscured when other relevant genetic variants are not included simultaneously in the model (e.g., the effects of one isolated risk allele may be overshadowed by an aggregate effect of several risk and/or protective variants) (Craig & Halton, 2009; Lotsch et al., 2009). Moreover, individual polymorphisms are likely to account for a relatively small amount of variance in a phenotype (McGeary et al., 2012). The use of a cumulative genetic score (CGS), which considers the collective impact of several variants, is a promising approach for dealing with this problem (McGeary et al., 2012). In short, a single score is created that reflects the number of risk alleles across multiple polymorphisms to represent a summed risk while simultaneously accounting for the influence of all variants modeled at once, thereby alleviating issues related to power and multiple comparisons (McGeary et al., 2012). Although additional refinements to this approach are being developed, at present the method employed here assumes a simple additive model, equivalent effects of each constituent allele, does not model epistasis, and relies on prior knowledge of risk alleles (in contrast to the atheoretical approach of genome wide association). Moreover, it has the additional benefit of providing greater statistical power than does modeling each variant individually (Plomin et al., 2009, McGeary et al., 2012).

In the present study, a CGS derived from empirically-selected candidate polymorphisms was formed to examine whether the combination of the MAOA and 5-HTTLPR genes was associated with three forms of IPV: (i) physical violence, (ii) psychological aggression, and (iii) injuries to one's intimate partner. These genetic associations were examined while simultaneously considering the effects of known correlates of violence in a sample of men arrested for domestic violence who were also hazardous drinkers. We controlled for relationship length and age because previous research suggests that violence increases with relationship length (Marcus & Swett, 2002; Shorey, Febres, Brasfield, & Stuart, 2012) and decreases with age (Capaldi, Kim, & Shortt, 2007; O'Leary, 1999). We also controlled for alcohol problems and drug problems, as there is an extensive literature showing a strong relationship between substance abuse and IPV (e.g., Chermack & Giancola, 1997; Leonard, 1993; 2001; 2005; Stuart et al., 2006; 2007; 2008; 2009a; 2009b). Finally, since population stratification can bias estimates of genetic association (Devlin & Roeder, 1999), we repeated all of the regression analyses with only the men who reported that they were Caucasian to determine whether we would obtain similar results while reducing heterogeneity attributable to racial background (Sinha, Larkin, Elston, & Redline, 2006; Tang et al., 2005). We hypothesized that the CGS would be significantly related to the three forms of IPV, even after controlling for the effects of alcohol problems, drug problems, age, and length of relationship.

Method

Participants

Ninety-seven men attending batterer intervention programs (BIPs) in the state of Rhode Island participated in the current study. Participants reported a mean age of 31.64 years ($SD = 9.83$), education of 11.78 years ($SD = 1.63$), and annual income of \$22,864 ($SD = \$18,490$). The ethnic composition was as follows: 77.3% non-Hispanic Caucasian, 12.4% African American, 8.2% Hispanic, 4.1% American Indian/Alaskan Native, and 2% other. Four participants did not indicate their race. All men in the sample were in a current intimate relationship with a partner. The average length of the men's current relationships was 5.3 years ($SD = 5.27$). For the men who were married or living with an intimate partner (53% of the sample), the length of time living together was 5.74 years ($SD = 5.59$). The average number of children reported by participants was 1.66 ($SD = 1.98$).

Measures

Intimate Partner Violence—The Revised Conflict Tactics Scales (CTS2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996), a 78-item self-report measure, was used to examine violence that occurred against an intimate partner in the 6 months prior to the BIP. In the current study, we employed the psychological aggression, physical assault, and injury perpetration subscales. Participants rated on a 7-point scale (0 = never; 6 = more than 20 times) the number of times they used a particular form of aggression against their intimate partner in the previous year. For the CTS2, scores are obtained by taking the midpoint for each response (e.g., a score of “4” for the response “3–5 times”) and then summing the frequency of each of the behaviors for each subscale. The score range for each item is 0 to 25 and higher scores correspond to more frequent aggression perpetration (Straus, Hamby,

& Warren, 2003). The CTS2 has demonstrated adequate internal consistency across multiple samples, ranging from .79 to .95 (Straus et al., 1996).

Alcohol Problems—The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuente, & Grant, 1993) was used to examine participant's alcohol use and problems in the 6 months prior to the BIP. The AUDIT, which consists of 10 items, examines the frequency and intensity of alcohol use, symptoms that are characteristic of alcohol tolerance and dependence, and negative consequences that are related to alcohol use (e.g., injuries). Previous research has demonstrated that the AUDIT is superior to other measures in screening for problematic alcohol use (Reinert & Allen, 2002).

Drug Problems—The Drug Use Disorders Identification Test (DUDIT; Stuart, Moore, Kahler, & Ramsey, 2003; Stuart, Moore, Ramsey, & Kahler, 2004) was used to examine drug use and problems in the year prior to the BIP. The DUDIT, which is modeled after the AUDIT, consists of 14 questions that examine the frequency and intensity of drug use and symptoms that may be indicative of tolerance or dependence. Seven different classes of drugs are assessed by the DUDIT, including cannabis, cocaine, hallucinogens, stimulants, sedatives/hypnotics/anxiolytics, opiates, and other substances [e.g., steroids, inhalants]. The DUDIT has demonstrated good reliability and validity across multiple samples (Stuart et al., 2004; 2008).

Genotypes and Cumulative Genetic Score (CGS)

Genotyping was performed using DNA isolated from Oragene kits. Genotyping for the individual polymorphisms in the candidate genes followed published methods. Genomic DNA was collected and extracted from saliva cells using methods described in McGeary (2009). Assays for the *MAOA* and 5-HTTLPR followed methods described in Habertsick et al., (2005) and Beevers et al., (2010). A cumulative genetic score (CGS) was then calculated for each participant. The CGS included genotypes of the two candidate polymorphisms that were identified as potentially important candidates in relation to violence. For the 5-HTTLPR polymorphism, a participant was given a risk allele score of 0, 1, or 2 corresponding to the number of low expressing alleles they possess. For the *MAOA* polymorphism, an allele score of 1 corresponded to the presence of 2-repeat, 3-repeat, or 5-repeat base pairs, indicating a lower transcription rate. Participants with the 3.5 or 4-repeat variants of the *MAOA* polymorphism, indicating a more efficient transcription rate, were assigned an allele score of 0. The scores across each of the four candidate alleles were summed to create a CGS that ranged from 0–3. There were no missing genotype data in this sample.

Procedures

Men participating in the current study were enrolled in a randomized clinical trial examining the efficacy of a BIP adjunctive treatment for reducing alcohol use (Stuart et al., in press). To be eligible for the current study, men had to be enrolled in a batterer intervention program, in a current intimate relationship with a partner, and meet criteria for hazardous drinking. Hazardous drinking was defined as scoring 8 or higher on the AUDIT or by consuming 5 or more drinks per occasion at least once per month (National Institute of

Alcohol Abuse and Alcoholism, 1995). Exclusion criteria for the current study included having psychosis or cognitive impairment; having attended 6 or more BIP sessions prior to being screened for the study; not speaking English; being incarcerated for half or more of the previous 6 months; or consuming alcohol at a level that may place them at risk for withdrawal symptoms (i.e., 12 drinks daily).

After determining eligibility for the current study, all men provided informed consent and completed self-report questionnaires prior to randomization to treatment condition. All self-report measures were completed individually with a research assistant present to answer any questions. Participants were breathalyzed prior to the completion of self-report measures to confirm that they were alcohol-negative. Each participant was informed that their participation was voluntary, that none of the information provided would be shared with their respective BIP facilitators or anyone within the criminal justice system, and that all information would be protected by a Certificate of Confidentiality. All participants were compensated for their participation. Salivary DNA was collected with Oragene collection kits. Ninety-seven of the 104 men approached provided DNA samples (93.3%).

Results

All statistical analyses were conducted using SPSS version 20.0. We first examined the distribution of scores among variables to check for positive skew and kurtosis. Results showed that the physical assault and injury perpetration subscales of the CTS2 were positively skewed and kurtotic. Thus, log transformations of these variables were performed prior to analyses to reduce positive skew and kurtosis. Correlations, means, and standard deviations among study variables are presented in Table 1. Raw means and standard deviations for physical assault and injury perpetration are presented for clarity purposes.

We examined whether the CGS was uniquely associated with violence after controlling for alcohol and drug use, relationship length, and age. Hierarchical multiple regression analyses were used for these analyses. In the first step of the analyses, AUDIT total score, DUDIT total score, relationship length, and age were entered as predictors of violence. In the second step, the CGS was added as a predictor.

Tables 2–4 display the results of the regression analyses for each form of violence. First, for psychological aggression (Table 2), results demonstrated that AUDIT score was positively associated with aggression perpetration in the first step of the hierarchical regression. The addition of the CGS accounted for unique predictive variance in psychological aggression perpetration and was positively and significantly associated with more frequent psychological aggression ($\beta = .26, p = .02, R^2 = .18, R^2 = .06$). Similar results were also found for physical assault (Table 3) and injury perpetration (Table 4). The addition of the CGS accounted for significant unique variance in the prediction of physical assault perpetration ($\beta = .29, p = .001, R^2 = .22, R^2 = .08$) and violence resulting in injuries to partners ($\beta = .22, p = .01, R^2 = .20, R^2 = .04$).

Upon inspection of the *MAOA* allele frequencies, two men were heterozygous for the *MAOA* promoter polymorphism and rerunning the genotyping assay yielded the same results.

Although we did not pursue aggressive molecular characterization to make a more definitive diagnosis, this suggests the presence of two X-chromosomes, and the possibility of Klinefelter's syndrome (Saito et al., 2002). We repeated the regression analyses described above excluding these two individuals. Results of the regression analyses were similar with these two individuals removed, with the exception of injury perpetration. With these two men excluded from analyses, the CGS was no longer significantly associated with increased injury perpetration. However, the CGS was positively and significantly associated with psychological aggression ($\beta = .21, p = .04, R^2 = .16, R^2 = .04$) and physical aggression perpetration ($\beta = .24, p = .02, R^2 = .19, R^2 = .05$).

Finally, we conducted all of the regression analyses with only the Caucasian men ($N=75$) to reduce the potential impact of population stratification on the results (Devlin & Roeder, 1999). These analyses were consistent with the overall results, in that the CGS was positively and significantly associated with psychological aggression ($\beta = .39, p = .001, R^2 = .25, R^2 = .14$), physical aggression ($\beta = .30, p = .01, R^2 = .24, R^2 = .09$), and violence resulting in injuries to partners ($\beta = .25, p = .02, R^2 = .21, R^2 = .06$).

Discussion

To our knowledge, this is the first study to examine the association between specific candidate genes and intimate partner violence (IPV). Consistent with our hypotheses, the CGS containing the *MAOA* and 5-HTTLPR polymorphisms was significantly associated with IPV, above and beyond the effects of alcohol problems, drug problems, age, and length of relationship. Specifically, the CGS accounted for an *additional* 8% of the variance in physical IPV, 6% of the variance in psychological aggression, and 4% of the variance in injuries to one's partner. These findings are consistent with the amount of variance accounted for in general aggression that has been previously found in the literature with *MAOA* (e.g., Buckholtz & Meyer, 2008; Caspi et al., 2002; Samochowiec et al., 1999; Volavka et al., 2004) and 5-HTTLPR (e.g., Popova, 2006; Retz et al., 2004).

Indeed, data from the present study add to the recent body of literature highlighting the importance of biological correlates of IPV perpetration (Pinto et al., 2010) and suggest the potential role of genetic factors in the etiology of intimate partner violence. Researchers have identified candidate genes that may contribute to these phenotypes, and the present study highlights the importance of two of these polymorphisms (*MAOA* uVNTR and 5-HTTLPR) that are theorized to be related to neurobiological processes associated with aggression potentially by way of altering prefrontal serotonergic function (e.g., Buckholtz & Meyer-Lindenberg, 2008; Takahashi et al., 2011). Thus, the results significantly contribute to our understanding of the biological underpinnings of IPV.

There are several limitations to this study. First, the sample size was very small for genetic analyses. Second, the participants were hazardous drinking men in batterer intervention programs, and results may generalize only to that population. Future researchers should attempt to replicate these findings in other samples, including partner violent men and women recruited from the community and women participating in batterer intervention programs. Ideally, such replication studies would recruit much larger sample sizes. In

addition, the CGS approach has limitations. The CGS score in this study assumed equal weights for both candidate genes, as sufficient data on the relative weighting of genetic predictors of violence were not available. Further, the CGS approach used in this study assumes additive effects and does not allow for epistasis.

As sufficient sample sizes are gathered to allow more atheoretical variant finding approaches such as genome wide association and next generation sequencing to be performed, the utility of the CGS approach will be further enhanced as currently unidentified variants will likely be added to the score (McGeary et al., 2012). As further refinements are made in disentangling single genetic markers with reliable effects on violence, the utility of the CGS approach will potentially increase. The use of a systems-biology approach that more fully characterizes the genes related to the implicated neurotransmitter and/or neuroanatomical regions may also increase the predictive value of a CGS approach.

Indeed, the present study included only 2 variants in our CGS. However, future researchers could add other promising polymorphisms, which would likely further increase the variance accounted for in violence. For example, there is evidence of an association between other serotonergic genes and aggression. The serotonin 1B receptor (*HTR1B*) gene has been linked to aggression in animal studies, aggressive behavior in children, and antisocial alcoholism (Conner et al., 2009). Jensen et al. (2009) found molecular differences in *HTR1B* such that individuals with the A-allele (rs13212041) have reduced *HTR1B* expression due to impaired interaction with miR-96 and greater risk of aggressive behavior compared to individuals with the G-allele. Another serotonergic gene, *TPH2*, has been implicated in the efficiency that the dorsal raphe nuclei (DRN) supply serotonin to the forebrain (McGeary, unpublished data) and may be an important determinant of serotonergic tone related to aggressive behavior (Takahashi et al., 2010). Future aggregate genetic risk scores that include these and other promising variants would likely further explain variance in violent behavior.

As with all studies of the genetics of complex behavioral phenotypes such as impulsivity or aggression, caution is urged against over-interpreting or misinterpreting the implications of these preliminary findings. The CGS approach employed here has been associated with a nontrivial but importantly non-deterministic amount of phenotypic variance. Any interpretation of these data that suggests exculpation for a violence perpetrator or justification of the violence has misunderstood the true import of this research. These findings provide initial evidence that these genetic variants may increase the probability of aggressive acts (perhaps even more so in alcohol-involved incidents given the study population). The history of human genetics research suggests that audiences who lack sophistication in understanding the relationship between genetics and behavior or medical disease can fail to distinguish between deterministic and probabilistic influences in a fashion that can lead to great harm. Given this potential for misinterpretation, it is crucial that future research be conducted to improve our confidence in these findings whilst simultaneously being mindful of the potential ethical, legal, and political implications of this work.

In our opinion, genetic variants do not *cause* aggression or IPV; rather, certain genetic variants may increase the risk or probability of violence through their impact on other

factors (e.g., predisposition to poor emotion regulation, impulsivity). Thus, genetic studies of IPV may provide us with an understanding of risk factors that could be targeted in IPV treatment, or the ability to match individuals to the treatment that may be most effective for their individual needs, potentially increasing our effectiveness in treating and reducing aggressive behavior.

The results of this study raise some important questions regarding treatment of IPV. Given that genetics are related to multiple forms of IPV, it is critical that future researchers examine whether there are genetic predictors of IPV treatment outcome. Ideally, in the long term, researchers will be able to target pharmacological or behavioral interventions to individuals with specific genetic backgrounds. Ultimately, this line of research could facilitate the development of more robust treatments and prevention strategies.

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Table 1

Correlations, Means, and Standard Deviations among Study Variables.

	1.	2.	3.	4.	5.	6.
1. Psychological Aggression	---	.65***	.45***	.32***	.16	.26**
2. Physical Assault		---	.72***	.36***	.14	.27**
3. Injury Perpetration			---	.36***	.14	.17
4. AUDIT				---	.20*	.03
5. DUDIT					---	-.06
6. CGS						---
<i>M</i>	31.89	6.22	1.40	17.98	12.21	1.36
<i>SD</i>	26.92	15.12	4.61	7.61	11.91	.88

Note: AUDIT = Alcohol Use Disorders Identification Test; DUDIT = Drug Use Disorders Identification Test; CGS = Cumulative Genetic Score

*
 $p < .05$,

**
 $p < .01$,

 $p < .001$

Table 2

Hierarchical Regression Analyses Predicting Psychological Aggression Perpetration.

	β	<i>SE</i>	<i>B</i>	R^2	<i>F</i>
Model 1				.12	3.08, $p = .02$
AUDIT	.31**	.36	1.11		
DUDIT	.08	.23	.19		
Age	-.08	.05	.00		
Relationship Length	.00	.31	-.23		
Model 2				.18	4.02, $p = .02$
AUDIT	.35**	.35	1.02		
DUDIT	.12	.22	.26		
Age	-.00	.31	-.01		
Relationship Length	-.01	.05	-.00		
CGS	.26*	3.04	8.04		

Note: AUDIT = Alcohol Use Disorders Identification Test; DUDIT = Drug Use Disorders Identification Test; CGS = Cumulative Genetic Score

*
 $p < .05$,

**
 $p < .01$,

 $p < .001$

Table 3

Hierarchical Regression Analyses Predicting Physical Assault Perpetration.

	β	<i>SE</i>	<i>B</i>	R^2	<i>F</i>
Model 1				.14	3.13, $p = .01$
AUDIT	.34**	.01	.05		
DUDIT	.07	.01	.01		
Age	-.03	.01	-.00		
Relationship Length	.09	.00	.00		
Model 2				.22	4.02, $p < .001$
AUDIT	.31**	.01	.05		
DUDIT	.11	.01	.01		
Age	.06	.01	.01		
Relationship Length	.08	.00	.00		
CGS	.29	.13	.39		

Note: AUDIT = Alcohol Use Disorders Identification Test; DUDIT = Drug Use Disorders Identification Test; CGS = Cumulative Genetic Score

*
 $p < .05$,

**
 $p < .01$,

 $p < .001$

Table 4

Hierarchical Regression Analyses Predicting Injury Perpetration.

	β	<i>SE</i>	<i>B</i>	R^2	<i>F</i>
Model 1				.16	423, $p < .01$
AUDIT	.32**	.01	.03		
DUDIT	.09	.01	.01		
Age	.12	.01	.00		
Relationship Length	.04	.00	.01		
Model 2				.20	4.58, $p < .01$
AUDIT	.30**	.01	.03		
DUDIT	.12	.01	.01		
Age	.18	.01	.01		
Relationship Length	.03	.00	.00		
CGS	.22*	.08	.19		

Note: AUDIT = Alcohol Use Disorders Identification Test; DUDIT = Drug Use Disorders Identification Test; CGS = Cumulative Genetic Score

* $p < .05$,

** $p < .01$,

*** $p < .001$