Common Genetic Vulnerability for Pathological Gambling and Alcohol Dependence in Men

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Background: In comparison with alcohol dependence (AD), relatively little is known about the causes of pathological gambling (PG). Given the high rate of comorbidity between PG and AD, knowledge about the causes of AD may be applied to understanding those of PG.

Methods: Subjects were adult male twin pairs from the Vietnam Era Twin Registry. Lifetime histories of PG and AD were assessed by structured psychiatric telephone interview. The validity of a continuum of PG liability was tested to determine whether the causes of subclinical PG, or problem gambling, are quantitatively or qualitatively distinct from those of DSM-III-R PG disorder. Genetic model-fitting methods were used to quantify the extent to which the genetic and environmental risk for PG could be explained by the risk for AD.

Results: Tests of the continuity model of PG were all consistent with the hypothesis that subclinical PG and DSM-III-R PG disorder have many, perhaps all, of the same risk factors and thus differ quantitatively rather than qualitatively. Depending on the PG definition, between 12% and 20% of the genetic variation and between 3% and 8% of the nonshared environmental variation in the risk for PG were accounted for by the risk for AD.

Conclusions: Subclinical PG, or problem gambling, may be a milder form of PG, rather than an etiologically distinct syndrome. Risk for AD accounts for a significant but modest proportion of the genetic and environmental risk for subclinical PG and DSM-III-R PG disorder.

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It has been suggested that individuals with pathological gambling (PG) experience and become dependent on achieving an aroused, euphoric state similar to a drug-induced “high.”1-4 Thus, PG has been conceptualized as an addictive disorder, similar to alcohol and drug abuse.1,5-7 The conceptualization of PG as an addictive disorder is embodied in the DSM-III-R8 and DSM-IV9 PG criteria, which include the concepts of preoccupation, loss of control, tolerance, and withdrawal. Perhaps the most well-established finding in the literature consistent with the conceptualization of PG as an addictive disorder is the high rates of alcohol dependence (AD) and other substance use disorders among individuals with PG.10-23 For example, 19% to 50% of individuals in treatment for PG10,19-21 and 45% to 63% of individuals with PG in the community11,22-23 have a history of alcohol abuse or dependence. These results suggest a common underlying vulnerability for PG and other addictive disorders such as alcoholism.

A recent meta-analysis of 119 PG prevalence studies in the United States and Canada14 estimated the lifetime prevalence of diagnosable PG among adults at 1.6%. An additional 3.9% of adults have a history of problem gambling (ie, gambling that has an adverse impact on the individual but that does not meet the criteria for a diagnosis).24 It is important to know whether clinically significant PG and subclinical PG share many of the same risk factors and differ mainly in level of severity, or whether they have distinct causes and thus differ qualitatively from each other.

Family study methods provide a powerful approach to testing questions of diagnostic continuities vs discontinuities by examining the risk of, for example, diagnosable PG and comorbid conditions such as AD among the relatives of individuals with diagnosable PG vs subclinical PG vs no PG symptoms. A continuity model predicts that relatives of individuals with subclinical PG would be at intermediate risk of PG (between relatives of individuals with diagnosable PG and those with no PG problems), whereas the discontinuity model predicts that relatives of individuals with subclinical PG would be at increased risk for diagnosable PG compared with relatives of individuals with no PG problems.

In the present study, we examined in a large registry of adult male twin pairs (1) the evidence for the continuity model of PG and (2) the causes of comorbidity between PG and AD. In a previous study that used this sample, Eisen and colleagues25...
SUBJECTS AND METHODS

SAMPLE

Participants were members of the Vietnam Era Twin Registry, a national sample of male-male twin pairs born between 1939 and 1957 in which both twins served in the military during the Vietnam era (1965-1975). Complete descriptions of registry development and characteristics of participants are given elsewhere. Of 10,253 eligible individuals, 8,169 (80%) were successfully interviewed for the present study. The mean age of the sample was 42 years (SD, 2.8; range, 34-54 years); 94% of the men were non-Hispanic white and 6% were African American; less than 1% completed only elementary school, 33% completed only high school, and 39% graduated from college; 93% were employed full-time and 2% were employed part-time.

Zygosity was determined by means of responses to questions about similarity of physical appearance supplemented with blood group typing information. Tests of the continuity model of PG were based on the 3,372 complete twin pairs of known zygosity (1,874 MZ pairs and 1,498 DZ pairs). Bivariate model-fitting analyses were based on the 3,372 complete twin pairs as well as 1125 individuals of unknown zygosity from incomplete twin pairs (516 MZ individuals and 609 DZ individuals).

MEASURES

The presence of PG and AD was assessed by structured psychiatric telephone interviews. Interviews were conducted by means of a computer-assisted telephone interview version of the Diagnostic Interview Schedule for DSM-III-R disorders by trained lay interviewers who were monitored by a project director. Interviewers contacted twins and began interviewing after verbal informed consent was obtained, a method approved by the institutional review boards at the participating universities. Lifetime diagnoses of PG and AD were determined by standard Diagnostic Interview Schedule computer algorithms. Symptoms of DSM-III-R PG were assessed only among those individuals who had ever gambled, bet, bought a lottery ticket, or used a slot machine 25 or more times in a year.

In addition to the DSM-III-R diagnosis of PG (DSM-III-R PG), we examined the association between AD and several other definitions of gambling-related problems. Individuals were diagnosed as having "subclinical PG" if they endorsed 1 to 3 lifetime symptoms of DSM-III-R PG. "Problem gambling" included both DSM-III-R PG and subclinical PG and was defined as having at least 1 symptom of PG. A 3-level “multiple-threshold” PG variable was also created by categorizing individuals with no DSM-III-R PG symptoms as a 0, with 1 to 3 DSM-III-R PG symptoms (subclinical PG) as a 1, and with 4 or more DSM-III-R PG symptoms (DSM-III-R PG) as a 2.

DATA ANALYSIS

Tests of the Continuity Model of PG

The continuity model of PG implies that subclinical manifestations of PG (ie, 1, 2, or 3 PG symptoms endorsed) lie on the same liability dimension as the diagnosable DSM-III-R disorder (Figure 1, top). An alternative model, the discontinuity model, implies that the liability for subclinical manifestations of PG is distinct from the liability for diagnosable DSM-III-R PG disorder (Figure 1, bottom). The validity of the continuity model of PG was evaluated in 5

TABLE 1

<table>
<thead>
<tr>
<th>Zygosity</th>
<th>PG MZ Men</th>
<th>PG MZ Women</th>
<th>PG DZ Men</th>
<th>PG DZ Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ</td>
<td>50.4%</td>
<td>52.8%</td>
<td>44.3%</td>
<td>43.9%</td>
</tr>
<tr>
<td>DZ</td>
<td>46.5%</td>
<td>44.3%</td>
<td>35.0%</td>
<td>33.4%</td>
</tr>
</tbody>
</table>

The lifetime rate of AD was 35.2% among the 6,744 individuals from complete twin pairs. Men with subclinical PG were at significantly elevated risk for AD compared with men with no PG symptoms (55.5% vs 33.4%; \( \chi^2 = 84.92, P < .001 \)) and were not at significantly lower risk for AD than men with DSM-III-R PG disorder (55.5% vs 64.9%; \( \chi^2 = 2.78, P = .10 \)). The risk of AD was also significantly elevated among both MZ and DZ cotwins of men with subclinical PG compared with MZ and DZ cotwins of men with no PG symptoms (MZ: 50.4% vs 33.5%, \( \chi^2 = 27.29, P < .001 \); DZ: 44.3% vs 35.0%, \( \chi^2 = 6.79, P = .009 \)) and was not significantly lower than the risk of AD among the MZ and DZ cotwins of men with DSM-III-R PG disorder (MZ: 50.4% vs 52.8%, \( \chi^2 = 0.10, P = .75 \); DZ: 44.3% vs 43.9%, \( \chi^2 = 0.00, P = .97 \), Table 2). The risk for AD was elevated among the MZ and DZ cotwins of men with subclinical PG even when cotwins with a personal history of DSM-III-R PG or any PG symptoms were excluded (Table 2).

The results of fitting the multifactorial threshold model to the twin data suggested that the assumption of a bivariate normal liability distribution underlying the 3 ordered PG categories could not be rejected by goodness-of-fit \( \chi^2 \) test for either MZ (\( \chi^2 = 3.4, P = .33 \)) or DZ (\( \chi^2 = 2.4, P = .503 \)) twins.

Continued on next page
ways. First, we examined the risk of DSM-III-R PG among cotwins of men with subclinical PG compared with cotwins of men with DSM-III-R PG and no PG symptoms. Second, we examined patterns of within-individual comorbidity of AD with subclinical PG compared with DSM-III-R PG. Third, we examined the risk of AD among cotwins of men with subclinical PG compared with cotwins of men with DSM-III-R PG and no PG symptoms. All tests of significance were 2-tailed with an $\alpha$ level of .05.

A fourth test of the continuity model of PG involved testing the goodness of fit of the multifactorial threshold model of PG. The polychoric correlation between the ordered categories of no PG symptoms, subclinical PG, and DSM-III-R PG disorder within twin pairs was calculated from the $3 \times 3$ contingency tables by means of PRELIS. The assumption of a single underlying distribution of liability was tested by comparing the observed frequencies in the $3 \times 3$ contingency tables with the frequencies expected assuming bivariate normal distributions of the underlying latent liability.

A fifth test of the continuity model of PG compared the results of bivariate model fitting of AD with 3 definitions of gambling-related problems (DSM-III-R PG, problem gambling, and multiple-threshold PG) to examine the similarity of the causes of PG liability and the comorbidity between PG and AD. Before model fitting, within-individual odds ratios and polychoric correlations between the 3 definitions of gambling-related problems and AD were computed. Confidence intervals (CIs) around odds ratios were computed with the use of 1000 bootstrapped samples from the original data set to adjust for the nonindependence of observations from twin pairs; polychoric correlations and their CIs were estimated with structural equation models that accounted for the twin structure of the data.

Bivariate Model Fitting of the Association Between PG and AD

In univariate model fitting of twin data, the variation in liability for a single trait is partitioned into that caused by additive genetic influences (A), shared environmental (C) or nonadditive genetic (D) influences, and nonshared environmental influences and measurement error (E). In bivariate model fitting, this is done for each of the 2 traits; in addition, the correlation between the 2 traits is similarly decomposed into that portion resulting from additive genetic influences, shared environmental or nonadditive genetic influences, and nonshared environmental influences and measurement error that is correlated in the 2 traits (Figure 2).

Models were fitted directly to the raw data by the method of maximum likelihood with the Mx program. The goodness-of-fits of a series of nested submodels were evaluated by comparing them with a saturated model that placed no constraints on the elements of the estimated MZ and DZ twin correlation matrices. Selected as the final model was the simplest model that was consistent with the data (see Neale and Cardon, Heath et al, and Kendler for further details about model fitting). After selection of a final model, 95% CIs around parameter estimates were computed to evaluate the following hypotheses: (1) whether the genetic, shared environmental, nonadditive genetic, or nonshared environmental correlations between PG and AD (ie, paths $r_A$, $r_C$ or $r_D$, or $r_E$ from Figure 2) differed significantly from 0; and (2) whether the genetic or environmental correlations between PG and AD differed significantly from 1. Three sets of bivariate models were fit to evaluate the causes of comorbidity of DSM-III-R PG and AD, problem gambling and AD, and multiple-threshold PG and AD.

The correlations between the genetic liabilities for PG and AD were significantly different from 0 for all 3 definitions of gambling-related problems. This suggests that there is at least 1 genetic locus that jointly increases the susceptibility for DSM-III-R PG and AD, for problem gambling and AD, and for multiple-threshold PG and AD. An alternative but less likely explanation is that there is a susceptibility locus for PG and another for AD that are statistically associated. Such an association could occur, for example, if these 2 loci were very close to each other on the same chromosome.

The fact that the correlations between the genetic liabilities for all 3 definitions of gambling-related problems and AD were significantly different from 1 suggests that there are also genetic susceptibility loci that are specific for PG and AD. Overall, genetic factors accounted for 64% of the overlap between DSM-III-R PG and AD, 75% of the overlap between problem gambling and AD, and 74% of the overlap between multiple-threshold PG and AD.

The correlations between the nonshared environmental liabilities for PG and AD were significantly different from 0 and significantly different from 1 for all 3 definitions of gambling-related problems. Thus, the hypotheses that PG and AD do not share any nonshared environmental risk factors and that the nonshared environmental liabilities for PG and AD are perfectly correlated...
were both ruled out for all 3 definitions of gambling-related problems. Overall, nonshared environmental factors accounted for much less of the overlap between PG and AD than did genetic factors.

An examination of the proportion of genetic, nonshared environmental, and total variation in PG risk accounted for by the variation in AD risk (Table 6) suggests that AD risk accounts for a significant but relatively modest proportion of the variation in PG risk.

In comparison with AD, relatively little is known about the causes of PG. To the extent that the causes of PG and AD overlap, knowledge about the causes of AD can be applied to understanding the causes of PG. The present investigation represents the first study to quantify the extent to which PG shares genetic and environmental risk factors with AD, by comparing the patterns of familial coaggregation of PG and AD in MZ vs DZ twin pairs.

All previous studies that examined the familial coaggregation of PG and AD used the family history method,10,17-19,39,40 most used treatment-seeking samples,10,17-19,40 and most did not include an appropriate control group,10,17-19,40 making the interpretation of results difficult. In the present study, the familial coaggregation of PG and AD was examined by directly interviewing the relatives (ie, MZ and DZ cotwins) of individuals with PG to determine their personal history of AD. The results of the present study clearly demonstrated that the cotwins of men with DSM-III-R PG have elevated rates of AD compared with cotwins of men with no PG problems, and this was true even among twin siblings who did not have a personal history of PG. Although the differences in the rates of AD among the MZ vs DZ cotwins of men with DSM-III-R PG were not statistically significant, MZ cotwins had consistently higher rates of AD than DZ cotwins, which suggests a genetic cause of the familial coaggregation. More powerful model-fitting meth-
ods confirmed that the familial coaggregation of PG and AD was more likely caused by their having common genetic rather than family environmental risk factors.

In a previous study of this twin cohort, Eisen et al. found that 62% of the variation in risk for DSM-III-R PG could be explained by familial risk factors, but because of the relatively low prevalence of diagnosable DSM-III-R PG disorder in the sample, it was not possible to discern whether the familial aggregation of DSM-III-R PG was caused by genetic or family environmental effects. In the present study, the familial transmission of DSM-III-R PG was clarified when examined in the context of AD, a much more common disorder with a more clearly discernible mode of familial transmission. The pattern of cross-trait, cross-twin correlations between PG and AD was much more consistent with the familial aggregation of PG being caused by genetic rather than family environmental effects.

The magnitude and sources of familial coaggregation between PG and AD were similar regardless of the definition of gambling-related problems examined, which suggests that AD and subclinical PG are correlated to the same extent and for the same reasons that AD and DSM-III-R PG are correlated. Additional tests of the continuity model of PG were all consistent with the hypothesis that subclinical PG and DSM-III-R PG have many, per-

### Table 2. Lifetime Prevalence of DSM-III-R Alcohol Dependence as a Function of a Lifetime History of DSM-III-R PG Disorder, Subclinical PG, or No Symptoms of PG Within Twins, in MZ Cotwins, and in DZ Cotwins*

<table>
<thead>
<tr>
<th>Diagnosis of Twin</th>
<th>All Cotwins</th>
<th>Cotwins Without DSM-III-R PG</th>
<th>Cotwins Without Any PG Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Twins</td>
<td>MZ Cotwins</td>
<td>DZ Cotwins</td>
</tr>
<tr>
<td>DSM-III-R PG</td>
<td>64.9% (94)</td>
<td>52.8% (53)</td>
<td>43.9% (41)</td>
</tr>
<tr>
<td>Subclinical PG</td>
<td>55.5% (420)</td>
<td>50.4% (228)</td>
<td>44.3% (192)</td>
</tr>
<tr>
<td>No PG symptoms</td>
<td>33.4% (6230)</td>
<td>33.9% (3467)</td>
<td>35.0% (2763)</td>
</tr>
</tbody>
</table>

* Lifetime prevalence of DSM-III-R alcohol dependence in the entire sample is 35.2%. PG indicates pathological gambling; MZ, monozygotic; and DZ, dizygotic. Superscript letters indicate that entries in the same column with different superscripts differ from each other. None of the within-subgroup and row comparisons of MZ vs DZ cotwins are significantly different. Numbers in parentheses are the number of twins included in the analysis.
subclinical levels of PG to better understand the full spectrum of this disorder.

The finding of significant shared genetic vulnerability for PG and AD in this study might be accounted for by the greater sharing of etiologically relevant experiences by MZ twin pairs than by DZ twin pairs. To address this concern, we conducted a series of analyses in which we predicted twin concordance for PG and AD from several measures of shared childhood and adult experiences, controlling for zygosity. None of the measures of shared experiences predicted twin concordance for both PG and AD, suggesting that greater shared environmental experiences of MZ compared with DZ twin pairs is unlikely to have resulted in an overestimate of the genetic covariation between PG and AD.

It is also possible that part of the association between PG and AD may result from a causal effect of PG on AD or of AD on PG. For example, many gambling facilities provide free alcohol to customers while they are gambling, and studies of individuals with PG confirm that engaging in gambling and alcohol use at the same time is common. Thus, it is likely that involvement in gambling may lead to heavier drinking or maintain or exacerbate existing alcohol-related problems. Conversely, AD may play a role in the initiation, maintenance, or exacerbation of PG. For example, several experimental studies have demonstrated that subjects will gamble more when under the influence of a moderate dose of alcohol than when given a placebo. In addition to a common genetic vulnerability for PG and AD, there may also be reciprocal causal effects between PG and AD, with PG increasing the likelihood of alcohol use and alcohol use increasing the likelihood of PG. Unfortunately, the men in our study were not asked about their ages of onset of gambling behavior and symptoms of PG, so it was not possible to rule out alternate causal interpretations of the association between PG and AD.

The present study is limited in that it was based on middle-aged male veteran twin pairs who were assessed in 1992, before the recent surge in the availability of lottery, casino, and Internet gambling. Thus, the results may not be completely generalizable to other populations, such as women, adolescents, and certain minority groups, and different conclusions about the causes of comorbidity between PG and AD may have been reached if we had assessed PG more recently.

Previous studies of this Vietnam Era Twin Registry cohort have examined the causes of comorbidity of AD with other addictive disorders, in particular, nicotine and marijuana dependence. A comparison of the results of the present study with these previous studies may be informative. Previous studies suggested that both nicotine and marijuana dependence share genetic risk factors with AD, with 46% of the genetic variation in the risk for nicotine dependence and 17% of the genetic variation in the risk for marijuana dependence being accounted for by genetic variation in common with the risk for AD. In the present study, we found that PG also shared genetic risk factors with AD, with 12% to 20% of the genetic variation in the risk for PG being accounted for by genetic variation in common with the risk for AD. Although the genetic association between PG and AD is not nearly as strong as the genetic association between AD and nicotine dependence, the strength of the genetic association between PG and AD of similar

### Table 5. Estimates of the Proportion of Variation in Problem or Pathological Gambling and Alcohol Dependence Liability Accounted for by Additive Genetic and Nonshared Environmental Factors

<table>
<thead>
<tr>
<th>Gambling Definition</th>
<th>Gambling-Related Diagnosis</th>
<th>Alcohol Dependence</th>
<th>Correlations Between Latent Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>E</td>
<td>A</td>
</tr>
<tr>
<td>DSM-III-R</td>
<td>0.64 (0.44-0.78)</td>
<td>0.36 (0.22-0.56)</td>
<td>0.55 (0.50-0.60)</td>
</tr>
<tr>
<td>Problem gambling†</td>
<td>0.49 (0.39-0.58)</td>
<td>0.51 (0.42-0.61)</td>
<td>0.55 (0.50-0.60)</td>
</tr>
<tr>
<td>Multiple threshold‡</td>
<td>0.51 (0.42-0.60)</td>
<td>0.49 (0.40-0.58)</td>
<td>0.55 (0.50-0.60)</td>
</tr>
</tbody>
</table>

* A indicates additive genetic effects; E, nonshared environmental effects; $r_A$, the correlation between additive genetic effects for the gambling-related diagnosis and alcohol dependence liability; and $r_E$, the correlation between nonshared environmental effects for the gambling-related diagnosis and alcohol dependence liability. Numbers in parentheses are 95% confidence intervals around parameter estimates given the assumptions of the final reduced models (ie, that nonadditive genetic and shared environmental effects are not operative). N = 1874 complete monozygotic twin pairs, 516 single monozygotic twins, 1498 complete dizygotic twin pairs, and 609 single dizygotic twins.

†At least 1 symptom of DSM-III-R pathological gambling disorder.

‡A 3-level diagnosis of no pathological gambling symptoms, 1 to 3 pathological gambling symptoms, and 4 or more pathological gambling symptoms.

### Table 6. Estimates of the Percentage of Genetic, Nonshared Environmental, and Total Variation in Risk for Problem or Pathological Gambling (PG) Accounted for by Variation in Alcohol Dependence Risk

<table>
<thead>
<tr>
<th>Gambling Definition</th>
<th>% of Variation in PG Risk Accounted for by Variation in AD Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Genetic Variation</td>
</tr>
<tr>
<td>DSM-III-R</td>
<td>12 (3-28)</td>
</tr>
<tr>
<td>Problem gambling†</td>
<td>20 (11-33)</td>
</tr>
<tr>
<td>Multiple threshold‡</td>
<td>19 (10-30)</td>
</tr>
</tbody>
</table>

* AD indicates alcohol dependence. Numbers in parentheses are 95% confidence intervals around parameter estimates given the assumptions of the final reduced models (ie, that nonadditive genetic and shared environmental effects are not operative). N = 1874 complete monozygotic twin pairs and 516 single monozygotic twins, 1498 complete dizygotic twin pairs and 609 single dizygotic twins.

†At least 1 symptom of DSM-III-R PG disorder.

‡A 3-level diagnosis of no PG symptoms, 1 to 3 PG symptoms, and 4 or more PG symptoms.
lar magnitude as the genetic association between another addictive disorder, that is, marijuana dependence, and AD.

It has been argued that addictive disorders are functionally equivalent, with the form of the addiction (e.g., to alcohol, nicotine, marijuana, or gambling) being dictated more by environmental circumstances than individual genetic vulnerability. If true, this would explain the high rate of comorbidity among the addictive disorders, including PG and AD. The results of the present study, however, do not fully support this conclusion. Although PG and AD do share genetic vulnerability factors, there are additional factors that make an individual vulnerable to PG and not AD, and factors that make an individual vulnerable to AD and not PG. Perhaps the most unexpected finding of the present study is the extent to which the vulnerabilities for PG and AD are distinct. This is surprising because researchers have been much more successful at identifying risk factors common to the addictions and have had less success at identifying risk factors that are unique to a given addictive disorder. The results of the present study suggest that it is worth searching for risk factors that PG does not share with AD, as well as risk factors that PG does share with AD, to better understand the causes of PG.

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REFERENCES


