

Supplemental Materials:

Genetic and Environmental Influences on Gambling Disorder Liability: A Replication and Combined Analysis of Two Twin Studies

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Comparison of Prevalences of Gambling Involvement and Disorder in the Two Cohorts

Data Analysis

The evidence for sampling bias within the two cohorts and differential sampling bias between the two cohorts was examined using SAS survey analysis procedures (SAS Institute, 2015). Sampling bias was examined by comparing the rates of GD among twins from pairs concordant for participation in the survey (“complete pairs”) to the rates of GD among twins whose cotwin did not participate in the survey (“incomplete pairs”). The incomplete pairs provided a window into the non-participating twins. That is, if twins with GD were systematically undersampled, higher rates of GD would be expected among twins whose cotwin did not participate in the interview than among twins concordant for participation in the interview (assuming that GD status is correlated in twin pairs). Differential sampling bias was tested by examining whether there was a significant interaction by pair type (complete or incomplete) and cohort in predicting GD.

Results

In the original cohort, the prevalences of GD were 2.4 and 4.0 among the complete and incomplete pairs ($t = 2.77$, $df = 2888$, $p = .01$), and in the replication cohort they were 2.0 and 2.4 ($t = 0.76$, $df = 2086$, $p = .45$). This suggests that individuals with GD were systematically under-sampled in Cohort II but not Cohort III. However, the levels of bias in the two cohorts did not significantly differ ($t = -1.11$, $df = 4974$, $p = .27$).

Discussion

There were some puzzling differences between the two cohorts in the magnitude of gambling involvement; for example, only 11% of the replication study reported ever gambling weekly compared to 36% of the original study. The most obvious explanation for this would be differences between the studies in the timing of the data collection or the age or birth year of the

participants. However, the data were collected at about the same time, the participants differed by only six years of age on average, and they were born between 1964-71 (original cohort) and 1972-1979 (replication cohort). These differences do not seem to be enough to explain such large differences in gambling behavior. Differential exposure of the two cohorts to gambling venue densities in the state/territory of residence was ruled out by showing that the replication cohort members were actually more likely to be from a state/territory with greater densities of gambling venues than the original cohort. Differential sampling bias was also ruled out by showing that the replication cohort may have actually under-sampled individuals with GD less than the original cohort. Thus, the only plausible explanation that we can offer is that study characteristics such as the assessment protocol may have been responsible for these differences. The fact that the rates of gambling involvement differed substantially suggests that the assessment of gambling in the original study may have “dug deeper” than the replication study in identifying instances of gambling behaviors. These differences did not influence the conclusions drawn in this study, but illustrate how providing more specific examples of gambling behaviors will lead to higher overall prevalences of gambling. It is noteworthy that the rates of DSM-5 GD did not differ in the two twin cohorts.