Correspondence

EDITED BY GREG WILKINSON

Genetic epidemiology of binging and vomiting

Sir: Sullivan *et al* (1998) applied bivariate twin modelling to 1897 female twins born between 1934 and 1971, and appeared to demonstrate a strong association between binging and vomiting, with a high genetic correlation. This assumes a degree of temporal uniformity with regard to bulimia nervosa (i.e. that a subject binging or vomiting in the 1950s exemplifies the same phenotypic trait as a subject in the 1990s).

Re-interpreting original data, Russell (1995) has cogently argued that people binging and vomiting before the late 1970s may differ from those presenting with recognised bulimia nervosa in the 1980s and after. He raises the possibility that bulimia nervosa may have escalated by virtue of its clinical characterisation in 1979 ". . . in vulnerable young women who consequently acquired the illness as if by contagion" (Russell, 1995). Coupled with a low response rate (64%), this calls into question the validity of their findings and a re-analysis is suggested to account for year of birth.

Russell, G. F. M. (1995) Anorexia nervosa through time. In *Handbook of Eating Disorders – Theory, Treatment and Research* (eds G. Szmukler, C. Dare & J. Treasure), p. 15. Chichester: Wiley and Sons.

Sullivan, P. F., Bulik, C. M. & Kendler, K. S. (1998) Genetic epidemiology of binging and vomiting. *British Journal of Psychiatry*, **173**, 75–79.

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Sir: Sullivan *et al* (1998) claim that their data support a genetic contribution to binging and vomiting behaviour, but the statistical interpretation of the results is flawed and the conclusions thereby undermined. They have tested for violations of the equal environment assumption (EEA), which, if present, would invalidate the conclusions, by carrying out a logistic regression analysis of six measures of specified common environment and zygosity, with concordance for either binging or vomiting as the dependent variable. This yields 12 tests, and two of the tests for vomiting are individually significant at $P=0.02$. However, they apply a Bonferroni correction for multiple testing and claim that overall these results are not statistically significant. This is based on the argument that one of the tests would need to reach a P value of 0.004 in order to count as significant, since the probability for at least one of 12 independent tests to be significant at 0.004 by chance is $1-(1-0.004)^{12}=0.047$. The probability for at least one test to be significant at 0.02 by chance is $1-(1-0.02)^{12}=0.22$.

There are two problems with this approach. The first is that the Bonferroni correction assumes that all tests are independent and this is unlikely to be the case. Some of the measures used might plausibly be expected to be correlated, and there is very substantial overlap between binging and vomiting behaviours. If a Bonferroni correction is applied to non-independent tests such as these, then significant results can be wrongly rejected.

The second problem is that not one test is significant at $P=0.02$, but two. The probability for at least one test to be significant at $P=0.02$ is, as already stated, 0.22. To obtain the probability that more than one test will reach this level of significance we simply subtract from this figure the probability for exactly one of the 12 tests to be significant at 0.02, which is the binomial probability $(1-0.02)^{11}\times 0.02\times 12=0.19$. Carrying out this procedure gives us the result that the probability to observe two or more of the 12 tests to be significant at 0.02 by chance is only 0.023. This result is thus unlikely to occur by chance and contrary to the claims of Sullivan *et al* there is significant evidence for violation of the EEA.

Although both the significant tests relate to vomiting rather than binging, the behaviours are highly correlated. The observed violation of the EEA invalidates the conclusion that there is necessarily a genetic contribution to these behaviours.

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Author's reply: Morgan suggests that our findings are invalidated because we did not take age cohort effects into account. Other data certainly suggest that there are important age cohort effects on the lifetime prevalence of bulimia (Bushnell *et al*, 1990; Kendler *et al*, 1991), although detection bias may be operative (Soundy *et al*, 1995). To evaluate Morgan's concern, we stratified our sample via a median split on date of birth and then repeated the univariate twin modelling for lifetime self-reported binging and vomiting. In the older and younger strata, AE (additive genetic and individual-specific effects) models again provided the best fit to the data and the parameter estimates for a^2 ('heritability') and e^2 (environmental influences specific to an individual and thus unshared by members of a twin pair) were similar to those reported in our paper. Thus, in response to Morgan, age cohort effects do not materially alter our findings.

The more substantive issue to which Morgan alludes regards the absence of common environmental effects on the behaviours of binging and vomiting (one conceptualisation of 'contagion' would reveal itself as common environmental effects). We did not detect such effects and discuss this issue at length in our paper (Sullivan *et al*, 1998, p. 78, col. 3).

Curtis is correct in identifying the equal environment assumption (EEA) as an important assumption in twin research. He argues that our application of the Bonferroni correction was incorrect and that, contrary to our interpretation, our EEA analyses invalidate our conclusions.

We suggest that the more critical issue is the magnitude of any possible violation of the EEA rather than simply its presence or absence. This is of particular relevance in fairly sizeable samples such as ours