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Christmas disease and major affective disorder

SIR: We were interested to read Gill *et al*'s description of a pedigree cosegregating Christmas disease and major affective disorder (*Journal*, January 1992, **160**, 112–114). We would like to report a further family in which both disorders are segregating.

The proband, a 44-year-old man, has laboratory-proven Christmas disease and DSM–III–R bipolar disorder. His 40-year-old sister is a carrier of the Christmas disease gene and has DSM–III–R lifetime diagnoses of major depression and alcohol abuse. Her eldest son, who is 19 years old, has laboratory-proven Christmas disease and DSM–III–R major depression and alcohol abuse. The proband's father, who is 67 years old, is haematologically normal but had a single episode of DSM–III–R major depression at the age of 47 years. The proband's mother, who is 68 years old, is a carrier of the Christmas disease gene and had a two-year episode of depression at the age of 30 years which necessitated psychiatric referral but which only satisfies DSM–III–R criteria for depression, not otherwise specified. The proband has a haematologically normal son and four daughters who are obligate carriers of the Christmas gene. The proband's sister has two other sons in addition to the one already described: one who has Christmas disease and the other who is haematologically normal. None of these seven children has expressed affective disorder but all are below the age of 18 years and, thus, they are only starting to approach the risk period.

This pedigree is consistent with genetic linkage between the Christmas disease gene and a susceptibility locus for affective disorder but does not offer strong support for this hypothesis because of: (a) the small size of the pedigree, and (b) complications in interpretation introduced by assortative mating between the proband's parents. Like Dr Gill *et al* we examined the proband's karotype and performed molecular genetic studies on the proband's DNA in the region of the factor IX gene and found no evidence of any abnormality. Thus, it is unlikely that a single mutation such as a large deletion caused both disorders. Clearly our small pedigree may have arisen by chance but like several other pedigrees in the literature it focuses interest on the possibility of an affective disorder susceptibility locus in the Xq27–28 region.

Assuming no population association between Christmas disease and bipolar disorder, we estimate that there should be at least five men with both disorders in the United Kingdom. Drs Gill *et al* have reported one and we have reported another. Perhaps investigation of the families of some of the other doubly affected probands may further inform the continuing debate about the existence of X-linked susceptibility loci in affective disorder (Hebebrand, 1992).

HEBEBRAND, J. (1992) A critical appraisal of X-linked bipolar illness. Evidence for the assumed mode of inheritance is lacking. *British Journal of Psychiatry*, **160**, 7–11.

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Survivors of disaster

SIR: Dr Plummer's critique (*Journal*, March 1992, **160**, 420–421) of our study of causal attributions and psychiatric symptoms in survivors of the Herald of Free Enterprise disaster (*Journal*, October 1991, **159**, 542–546) is based on a misunderstanding of our work and on misapplications of statistical principles.

He begins, for example, by listing a number of "obvious methodological problems". Our sample, which we described carefully and in some detail, was certainly small and unrepresentative, but it is not clear how this fact alone could account for the pattern of findings. He then reproaches us for using correlation rather than regression analysis, omitting to mention that we used *partial* correlation analysis (regression is based on partial correlations). This confusion between correlations and partial correlations may also account for his puzzling charge that we had