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Tourette's syndrome and psychiatric disorders

SIR: Pauls *et al* (*BJP*, February 1994, **164**, 215–221) claim to provide evidence against our proposal (Comings & Comings, 1987; Comings, 1995a,b) that depression and anxiety disorders are genetically related to the *TS* (*Gts*) gene(s). In reality their report provides strong evidence in favour of such a relationship.

Pauls *et al* have consistently maintained that the wide range of psychiatric disorders comorbid to Tourette's syndrome (TS) are all due to proband ascertainment bias, and would not be increased in frequency in the relatives of TS probands. Now they find that in fact the frequency of generalised anxiety disorder, major depressive disorder, panic disorder, simple and social phobia are significantly increased in frequency in 338 relatives of TS probands compared with 113 relatives of controls (Table 2). To extricate themselves from this turn of events, they now retreat to the extraordinary position of claiming that these disorders are unrelated to the *Gts* genes because their frequency is not significantly higher in first degree relatives *without* TS, chronic tics (CT) or obsessive-compulsive disorder (OCD) compared with relatives of controls. However, their own studies state that TS is inherited as an autosomal dominant trait with 100% penetrance in males, and when OCD is included, 71% penetrance in females (Pauls & Leckman, 1986). Thus, only 15% of relatives of TS probands without TS, CT or OCD should be carrying the *Gts* genes. As such, one would not be able to identify any of the pleiotropic effects of the *Gts* genes in a group of subjects where so few carry the genes. This

would be analogous to attempting to claim that the Huntington's disease gene does not cause dementia because none of the relatives *without* HD show dementia.

They next claim that the increased frequency of these behaviours in the relatives that they observed was not due to the *Gts* gene(s), but was instead due to the presence of OCD. Again they seem to have forgotten their own paper (Pauls & Leckman, 1986) showing that the *Gts* gene(s) can cause OCD with or without tics. Thus, they would accept that *Gts* genes cause OCD, and that OCD produces depression and anxiety disorders, but deny the whole equation that *Gts* genes can cause depression and anxiety disorders. The demonstration by Pauls *et al*, of the increased frequency of associated behaviours in relatives with OCD but no tics, totally supports our suggestion that some individuals in the general population without tics may have these conditions because they carry *Gts* genes.

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ECT seizure duration and efficacy

SIR: The study by Fear *et al* (*BJP*, October 1994, **165**, 506–509), their prior correspondence (*BJP*, March 1993, **162**, 421–423), and two previous studies (Mitchell *et al*, 1991; Malsch *et al*, 1992) resurrect the issue of whether ECT seizure duration *per se* is related to the antidepressive efficacy of ECT. For example, Malsch *et al* (1992) state that “one of the unsolved questions of [ECT] is the effect of seizure duration on the efficacy of treatment”.

The notion that ECT seizure duration might be related to its antidepressive efficacy has been advanced by the works of Maletzky (1978) and Kramer (1983). However, neither investigation demonstrated a significant positive correlation