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## Neuroleptic Malignant Syndrome (NMS) and Tardive Dyskinesia

SIR: Haggerty & Gillette (*Journal*, January 1987, **150**, 104–105) seem to have concluded that NMS developed while the patient was having reserpine and lithium. The details they give do not support such a conclusion, as they mention in their discussion. For example, they do not mention any alteration in consciousness after lithium and reserpine were started: besides rigidity, hyperpyrexia and autonomic dysfunction, alteration of consciousness in varying degrees is a *sine qua non* for the diagnosis of NMS (Caroff, 1980). Here, only autonomic dysfunction was noticed after starting lithium and reserpine. Moreover, the progression of NMS is known to be very rapid after onset. In more than 90% of 120 cases reviewed, the full syndrome developed within 48 hours of the first symptoms (Shalev & Munitz, 1986). It is naïve to conclude that mild NMS developed over a period of two weeks and that the full-blown syndrome began later, when thioridazine and haloperidol were started.

What is of interest in this report is the unusually long duration of the NMS episode (seven weeks). Generally, it is presumed that NMS lasts for 5–10 days after discontinuation of oral neuroleptics (Sternberg, 1986). I have come across another patient in whom the syndrome persisted for three weeks after cessation of haloperidol and remitted after a 17-day course of amantidine (Woo *et al*, 1986). Treatment was continued for a further period of five months. Such an abnormally long duration may have something to do with the patient's metabolic characteristics, as differences in absorption and first pass metabolism are known to cause wide inter-individual variations in the plasma levels of neuroleptics. Moreover, neuroleptics, being highly lipophilic, can be detected for weeks after discontinuation.

Caution is required before concluding that unexplained fever is due to NMS. This is clear when one considers the authors' suggestion that reports by family of transient diaphoresis and elevations of temperature in the months prior to admission were probably because of NMS in "milder form". In the absence of any alteration of consciousness such an interpretation is not justified. At the moment, there is not enough clinical data to justify the spectrum concept of neuroleptic toxicity (Corlon, 1986) which the authors seem to have in mind.


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## Continuum of Psychosis and the Gene

SIR: Crow's theory (*Journal*, October 1986, **149**, 419–425) bears a striking similarity to the theory of Britain's best-known psychologist, Hans Eysenck, to the effect that a heritable trait of psychoticism (*P*) underlies much psychotic (and indeed psychopathic) disturbance (Eysenck & Eysenck, 1976, 1985). Like Crow's continuum, Eysenck's *P* has sometimes been considered to be related to cerebral lateralisation of function (Brand, 1981) and to forms of creativity and achievement (Claridge, 1985) that might explain the persistence of high-*P* genes in the population despite the lowered average fertility of people who suffer outright psychotic disturbance.

I wonder, could the theories of Crow and Eysenck by any chance be related?


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