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Molecular genetics of alcoholism

SIR: In their impressive review Mullan & Murray (*Journal*, May 1989, **154**, 591–595) have confined themselves to the impact of molecular genetics on understanding and nosology of psychotic disorders. The same techniques, however, can be extended to other biologically determined disorders in psychiatry. We are constantly moving from diagnostic categories with only face and descriptive validity to aetiologically homogeneous nosologic entities, which are defined on the solid foundation of construct and predictive validity. The application of 'new genetics' is a powerful tool in teasing out aetiologically heterogeneous categories which were hitherto lumped together on the basis of superficial chemical resemblance.

In the field of alcoholism, the recent genetic epidemiological work done by Cloninger and his coworkers has supported genetic heterogeneity in alcoholism (Bohman *et al*, 1987; Cloninger, 1987; Gilligan *et al*, 1987). Despite the suggestion that at least some subtypes of alcoholism are genetically mediated, it is not yet certain as to what is inherited. The vulnerability to alcohol use may be transmitted independently of vulnerability to develop alcohol-related pathologies. The most simple model is reductionist but practical, i.e. that of inherited abnormal biochemistry predisposing to the development of physiological addiction. However, there is little evidence that the gross rate of alcohol metabolism is itself associated with the risk for developing alcoholism. Recent pharmacogenetic studies in animals have shown that the relationship between initial sensitivity to ethanol (as measured by ataxia, body temperature, and disruption of motor performance) and the ability of this drug to serve as a positive reinforcer is minimal, thus distinguishing the specific intoxicating effects of ethanol from its reinforcement effects (George & Goldberg, 1989).

It is highly likely that the genetic influence is one of specific predisposition to the development of alcoholism, in view of these findings. The recent genetic epidemiological work advocates the interesting possibility of an aetiologically homogeneous subgroup (Type II) in which most of the liability to alcoholism is transmitted by a single dominant gene (Gilligan *et al*, 1987; Craufurd, 1989). Furthermore, keeping in view that the Type II alcoholism is consistently associated with the male sex and the conspicuous absence of father to daughter transmission of this subtype, one could hypothesise that the dominant gene is probably located on the sex-determining region of the Y chromosome.

This hypothesis would be of great heuristic importance as a basis for future molecular genetic studies. Prior evidence for the hypothesis that the marker is on a particular chromosome can be of great help in localisation and subsequent mapping of a genetic locus (Baron & Rainer, 1988). Anonymous Y-linked DNA probes may be employed to exclude or affirm Y-linkage in those pedigrees that show no father–daughter transmission and where no females are affected. Testing of this hypothesis would be helpful in the development of reliable diagnostic criteria for the correct classification of the alcoholic subtypes, besides providing construct validators for Cloninger's typology.

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Multiple personality disorder

SIR: Fahy (*Journal*, October 1988, **153**, 597–606) provided a useful critical review of the highly peculiar nosological category of multiple personality disorder (MPD). I suggest that, taking into account the many uniquely odd characteristics of this diagnosis, an alternative hypothesis is needed to account for this most rum condition.

More than any other diagnosis, patients allegedly suffering from it have become major media celebrities, fêted in the popular media. In the history of medicine or psychology, has any condition been so reliably rewarding to those supposedly 'suffering' from it; or to the physicians and psychologists who attend them?

The condition arouses excessive interest and excessive claims. Clinicians reporting such cases usually show an infatuation with them. Like new parents, they can never miss an opportunity to show photographs, movies, or videos of their uniquely talented offspring, or to tell you of their latest cute trick.

The distribution of MPD is bizarre. There is no normal distribution of cases. I've never met a clinician who, over any significant period of time, has seen just one case. The vast majority of talented, sensitive, observant clinicians have never seen a case at all. A very small number of clinicians report the great majority of case reports.

Spontaneous remission is probably the norm, unless the patient becomes engaged with a clinician already primed and interested in the condition. It seems to be one of the few conditions which almost invariably get worse in therapy; the extent of the patient's pathology is directly proportional to its amount and intensity, and shows the most evolved and disturbed anomalies in the most intensively studied cases. It appears to be the norm that further 'personalities', often more entertaining and rewarding for the audience, emerge in therapy.

My hypothesis is that MPD is an iatrogenic, largely culture-bound disorder, with some resemblances to folie à deux, arising when a bright, suggestible patient meets a bright, suggestible physician convinced that MPD is an important diagnosis. Selective reinforcement of symptoms, unconscious and conscious, progressively shapes the symptoms and behaviour of the patient, and the depiction of MPD is elaborated and reinforced. Patients usually show clear primary or secondary gain, but this is often not noted or acknowledged by their therapists, whose own secondary (and maybe primary?) gains are similarly covert.

Procedures regularly followed, such as naming the alternative 'personalities' and having long, carefully recorded conversations with them, serve to preserve and reify these otherwise transient situations, locking them into publicly shared 'reality'. Any 'as-if' quality to the original experiences is stamped out. The inevitable audio and videotaping provides a handy record of the nuances of the successful performance, and an aide-memoire for both participants, to enable the maintenance of a consistent portrayal.

There is no convincing evidence that MPD is a naturally occurring condition, let alone a distinct diagnosis. It is a symptom complex that may be superimposed on other psychopathologies, consequent upon the unfortunate matching of a susceptible patient with a susceptible therapist and trainer. The diagnosis is dysfunctional, focusing attention selectively in a way that will almost invariably worsen the condition, rather than improving it. It occurs in the context of the availability of lengthy psychotherapy. Where the health care system or health insurance does not sponsor this indulgence, the condition simply does not occur.

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How many sheltered housing places?

SIR: We recently conducted in Montréal a survey comparable to the Glasgow Rehabilitation Survey (Livingston & Bryson, *Journal*, May 1989, **154**, 620–624), and found a strikingly similar rate (29%) of patients who would be able to live in the community. However, our consultants estimated that more sheltered housing would be needed, especially group homes directly supervised by nursing staff.

Canada's health and social care systems grant universal access to services. In Montréal, all psychiatric in-patient facilities are public. The recently revised provincial mental health policies call for further transfer of mental hospital in-patients to the community. Hôpital Louis-H. Lafontaine is a 2000-bed mental hospital currently covering a catchment population of 330 000. However, until 5 years ago it also served as a long-term facility for the French-speaking population of Greater Montréal (2.8 million