with this hypothesis (reviewed by Goldin *et al, Journal,* September 1987, **151,** 302–305). Goldin *et al* point out the problems with this methodology, in particular the definition of what constitutes sporadic schizophrenia, a sub-group that may be inherently heterogeneous. We have some data suggesting that the way we define this group affects the differences, if any, between familial and sporadic schizophrenias.

Eighteen consenting patients from the Bethlem Royal and the Maudsley Hospitals (mean age = 33.8 ± 10.8; 13 males and 7 females) were studied. All fulfilled RDC criteria (Spitzer *et al,* 1975) for schizophrenia. The mean duration of illness was 57.2 ± 45.8 months. Six patients were drug-free at the time of the study. None had any major systemic illness, current alcohol or drug abuse, or evidence of neurological disease. Family history of psychiatric illness was obtained from the patient and any available informant. A positive family history required either a hospital summary specifying a clinical diagnosis of schizophrenia or other psychiatric disorder, or a subject or informant volunteering both the diagnosis and a compatible history in a first-degree relative. Six subjects had a family history of schizophrenia in a first-degree relative, three had a family history of affective psychoses or depressive spectrum disorders, and nine subjects had no family history of psychopathology.

Patients were scanned without contrast using the Maudsley Hospital EMI 1010 CT scanner. Five or six contiguous 1 cm slices were taken parallel to the orbitomeatal line to encompass the whole ventricular system. Ventricle-brain ratios were measured using a semiautomated technique on the single slice with the largest-ventricular area (Reveley *et al,* 1982).

The mean VBR in the patient group as a whole was 4.63 (s.d. = 2.99, median = 4.3, range = 1.2–11.4). The non-familial sub-group ($n = 12$) had non-significantly larger VBR than the 6 subjects with a family history of schizophrenia (VBRs = 5.18 ± 3.2 and 3.5 ± 1.6 respectively; $t = 1.47$, NS). However, when the three patients who had a family history of affective disorders were excluded from the former group, the difference became modestly significant (VBRs = 5.85 ± 3.5 and 3.5 ± 1.6 respectively, $t = 1.8$, $P < 0.05$, one-tailed).

Our study was limited by the very small sample size, use of the family history method (which is inferior to the family interview method), and the use of a relatively young patient population which may have resulted in fewer relatives reaching the age of risk for psychiatric disorders. However, our data suggest that the familial sporadic distinction within schizophrenia may be more valid when a 'narrow' definition of sporadic and familial schizophrenias is used. Lewis *et al*'s succinct guidelines for assessment of family history will certainly help future studies intending to pursue their innovative hypothesis further.

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SIR: Kendler's group (Eaves *et al,* 1986; Kendler, 1987) have produced good statistical reasons to be circumspect about the power of the familial-sporadic distinction in small samples. Nevertheless, the majority of studies have shown a familial effect on VBR, and Drs Keshavan & Toone do so in a sample of only 18 patients. Although Drs Keshavan & Toone do not explicitly say so, they may have intentionally selected their patients to ensure equal numbers with and without a family history. This strategy would certainly have increased the power of the distinction. They also allude to young age as a factor which may have acted against them. On the contrary, young age may well increase the power of the familial-sporadic distinction. As more and more time elapses after the onset of schizophrenia, hospital in-patient samples will increasingly represent the poor outcome end of the spectrum. Should, as is likely, either VBR or family history influence outcome in schizophrenia, then the heterogeneity of these variables in hospital samples will therefore become less and less as time goes by. The way round this artificial attrition of heterogeneity with time would either be to confine studies to young patients who are in or near their first episode or to ensure epidemiological (not hospital-based) sampling.

Drs Keshavan & Toone's point about ensuring a 'narrow' definition of sporadic schizophrenia is well made. Certainly, cases with a history of affective disorder in a first-degree relative should not be considered 'sporadic'; the more interesting question is

whether or not to consider them 'familial'. Had Drs Keshavan & Toone chosen to do so, inspection of their data suggests they would have found an even more impressive difference in VBR between their familial and sporadic groups. However, it is important to keep in mind an underlying hypothesis before deciding whether one's glass is really half-full or half-empty. If the dependent variable is VBR, then the critical independent variable is not so much the absence of a family history as the presence of earlier environmentally-mediated brain insults which tend to congregate in the sporadic group.

One last point: perhaps because it jars with current nosological conceptions, schizophrenia with a family history of affective disorder is under-researched. The notion that it represents a distinct biological subgroup is well worth exploring further.

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Deliberate Self-Harm and Out-patient Attendance

SIR: In their report concerning deliberate self-harm in Newcastle, O'Brien *et al* (*Journal*, February 1987, **150**, 246–247) demonstrated that the attendance rate of patients at out-patient appointments one week after the episode was 40%. A survey of deliberate self-harm (DSH) referrals carried out in the Bristol Royal Infirmary (BRI) over a 16-week period in 1986 produced similar findings.

All cases of DSH at the BRI are referred for psychiatric assessment and disposal by a Senior House Officer (SHO) in psychiatry. During the course of our study, each of 88 patients was seen by one of four SHOs. Half of the patients were offered an out-patient appointment at the time they were seen. The reasons for not being offered an appointment were either that the patient was being followed up by another psychiatric team (13 patients), or the patient refused the appointment offered (14 patients), or finally that follow-up by the psychiatric services was not thought appropriate. In the latter case, either the patient was already involved with other agencies or the act of self-harm had produced a positive change in circumstances (15 patients). Two patients were admitted to the psychiatric ward.

Of the group given an appointment for the next available psychiatric clinic place, to be seen by the assessing SHO, only 50% (22 patients) subsequently attended.

This study broadly confirms the findings of O'Brien *et al* and others (Morgan, 1976; Kreitman, 1979) showing a very high drop-out rate from psychiatric care of DSH patients. This phenomenon poses considerable difficulties for research in gathering both adequate numbers and representative samples of patients. As a corollary, it emphasises the need for improvement in the psychiatric management of DSH. We need to clarify whether high default rates imply an inherent limit to what can be offered to DSH patients or reflect deficiencies in treatment styles, some of which may be remediable.

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The BITE: Indices of Agreement

SIR: In reply to the letter from King & Williams (*Journal*, May 1987, **150**, 714), we would like to make the following points regarding the Bulimic Investigatory Test, Edinburgh (BITE) (Henderson & Freeman, 1987). Firstly, Drs King & Williams state that it is unclear whether the BITE is a screening test or a diagnostic instrument. The BITE was designed as a screening test for use in a wide variety of settings to allow the detection of sufferers and potential sufferers of bulimia nervosa. Examination of the thirty items that comprise the symptom sub-scale will show that they provide information on a wide range of types of behaviour associated with binge-eating. By looking at an individual's responses to each item, the user will be able to extract the information they require to answer questions concerning diagnosis. We felt that it was pointless to attempt to produce a diagnostic instrument in an area where there is no agreement as to what constitutes a diagnosis of bulimia. Even the most recent DSM–III–R diagnostic category for bulimia is open to discussion.

The BITE covers all the current criteria for a DSM–III diagnosis of bulimia, as well as those proposed by Russell (1979). It is assumed that any