AUTHOR'S REPLY: Dr Godfrey suggests that no single factor can be said to cause eating disorders. I would agree wholeheartedly. Indeed, I do not know of any clinician or researcher who would claim otherwise. In my paper I have outlined the importance of a multifactorial approach to understanding the complex, circular causality involved in anorexia and bulimia nervosa. The potential factors that I stressed are probably more psychological than sociodemographic. For example, I would agree with Dr Hambidge's opinion that parenting and personality are among the factors that are of great interest in understanding possible links between abuse and eating disorders. However, I would not deny that many of the sociodemographic factors that Dr Godfrey mentions are of interest. For example, the women who have reported sexual abuse in my case series to date are significantly older ($n = 56$, mean = 25.9 years, s.d. = 6.20 years) than the women who report no abuse ($n = 58$, mean = 22.2 years, s.d. = 5.80 years), in contrast to the results cited by Dr Godfrey.

In order to understand more fully the interaction between sexual abuse and other factors in the aetiology and maintenance of any psychopathology, large case series are needed. However, that requirement should not prevent all communication of important findings from work in progress while those case series are under development. It is critical that research suggesting that particular factors are worthy of consideration should be available to other clinicians and researchers, in order to further the process of developing our understanding. It was never my intention that this report should have been seen as a definitive explanation of the aetiology of anorexia and bulimia nervosa, as I hope is made clear in the final paragraph of the paper. After all, the title was 'Sexual Abuse as *a* Factor in Eating Disorders'.

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SLE and psychiatric morbidity

SIR: I wish to thank Dr Ong (*Journal*, March 1992, **160,** 420) for his interest in our study (*Journal*, October 1991, **159,** 520–523). Dr Ong commented that age might be a compounding factor and suggested that younger patients facing chronic debilitating illness might suffer a greater psychiatric morbidity. The controls in our study consisted of 29 patients with rheumatoid arthritis. As a group, they tend to be older and hence it was difficult to match exactly for age. It can also be argued that the older the patient, the longer the duration of illness, the more likely they are to develop complications and thus the increased likelihood of psychiatric morbidity. We were aware of the effect of age as a compounding factor, and suggested this as a possible reason for the higher psychiatric morbidity observed in the current series.

The majority of the Chinese population in Singapore is bilingual, being conversant in both Mandarin and English. The selection of English-speaking patients might have unwittingly excluded older patients who were monolingual but not necessarily less educated. English-speaking patients from a lupus clinic were approached for the study as part of the research design to enable us to compare the results from these two culturally diverse samples. Dr Ong has missed the point when he suggests that we use a Chinese version of the questionnaires and investigators well-versed in Chinese for the study.

Our study can be considered as a series of unmatched case-controlled studies. Dr Ong used the chi-squared calculation and assumed that the risks of psychiatric morbidity are the same in the Singapore and the London samples. This is not the case, as the samples were drawn from a culturally diverse population, and they are not directly comparable if we do not take into account the risk to the controls. The relative risks of psychiatric morbidity in SLE patients is therefore different in the two populations. The odds ratio is thus a more appropriate method of analysing our data.

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Head size in dementia

SIR: We read with interest the study on 'Head Circumference in Elderly Long-Stay Patients with Schizophrenia' by Jones & Lewis (*Journal*, September 1991, **159,** 435–438) and wondered whether their use of demented in-patients for comparison was appropriate. It has been suspected for some time that a large percentage of patients with Alzheimer's disease have 'relative microcephaly' (Grünthal, 1927). Observations on non-demented elderly individuals found to have Alzheimer pathology at post-mortem examination have led to the suggestion that patients with larger brains and more neurons may be less susceptible because of their greater reserve, whereas patients starting out with smaller brains are at greater risk of developing clinical deficits early in the course of illness (Katzman *et al*, 1988). Our own computerised tomography (CT)-scan measurements in patients with senile dementia of the Alzheimer type

have shown a significant decrease of intracranial size in comparison to age- and sex-matched controls (Förstl *et al*, 1991). We have since extended our measurements to 210 patients with dementia of the Alzheimer type who were drawn from two different prospective studies – one, a longitudinal study on the course of Alzheimer's disease (Study A: Burns *et al*, 1990) the other a clinical trial on 100 volunteers (study B; Eagger *et al*, 1991).

CT-scans for computer-assisted planimetric measurements were available from 210 patients satisfying NINCDS-ADRDA criteria for 'possible' or 'probable' Alzheimer's disease (study A: 31 males, 101 females; mean age 79.8 (s.d. 6.2) years; study B: 45 male, 33 female; 66.3 (s.d. 8.5) years and 34 controls: 10 male, 24 female; 74.3 (s.d. 5.3) years. The largest horizontal intracranial areas measured parallel to the orbitomeatal line were significantly smaller in the patients from study A (155.0 (s.d. 13.7) cm^2) than in the controls (162.3 (s.d. 12.7) cm^2), whereas the patients from study B had significantly larger intracranial areas than the controls (167.5 (s.d. 11.8) cm^2, $F=20.37$, d.f. 2,241, $P=0.000$. The differences mentioned above remained significant at the 0.05 level after Duncan's test for multiple comparisons, and when men and women were examined separately). No differences from the control group were observed when patient samples A and B were collapsed. Cognitive performance on the 'Mini-Mental State' test were correlated with the intracranial area (total sample: $r=0.287$, $P=0.000$; males: $r=0.250$, $P=0.030$; females: $r=0.149$, $P=0.149$). In our samples a family history of dementia, and age or the year of birth bore no significant relationship to the intracranial area. An analysis of covariance demonstrated a significant relationship between the intracranial area and the type of study (study A/study B; $F=12.9$, d.f. $=1,207$, $P=0.000$) even after correction for the covariate gender.

Sample A can be considered as a representative sample of patients with dementia of the Alzheimer type from a well-defined catchment area (Burns *et al*, 1990). Jones & Lewis have remarked upon the non-representative nature of patients who volunteer for special investigations of treatment trials (study B). Therefore we have reason to believe that the intracranial area in representative patient samples with dementia of the Alzheimer type may indeed be lower than in the normal population (and that smaller skull or brain sizes may contribute to the manifestation of dementia in many of the patients with dementia of the Alzheimer type; Katzman *et al*, 1988). If this is true, Jones & Lewis have possibly underestimated the decrease of head size in their patients.

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SIR: We are interested to hear of the suggestion that we may have underestimated the difference between the head circumference of schizophrenic patients and demented patients as the latter might have smaller heads. However, the measurements of Dr Förstl *et al* were of the largest intracranial areas of computerised tomography scan slices, and we cannot be certain that these correlate well with the external measure of head circumference, the latter including additional contributions of skull and scalp thicknesses.

Secondly, the population estimates from the anthropological literature did not differ significantly from our male patients with dementia (Daniel, 1937), nor from our female patients (Fetter, 1969).

We agree entirely that further work on this vexed question must be based on epidemiologically defined populations, and that ideally a normal control group should be used, although this would involve a mismatch for place of residence.

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