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

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