The association is now piloting sex education modules in schools. It will certainly draw strength from Mellanby and colleagues' work, particularly with regard to the use of specific targeted methods and systematic evaluation.

Sex education has a formidable task to achieve. It is not just about sex: it is also about comprehensive education in family life. This includes interpersonal and decision making skills and positive attitudes about self and family, with the hope that the young person will develop a sense of responsibility for his or her actions.

Critics of sex education claim that evaluations of outcome must show change in the social and sexual behaviour of students outside the classroom. This is a tall order: it is like evaluating civics classes according to their ability to make students into better citizens. I am delighted that the work of Mellanby and colleagues proves the critics wrong. Promoters of sex education programmes around the world must persevere in their efforts. More importantly, they must constantly evaluate their programmes and publicise their findings to dispel myths and misconceptions about sex education.

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Migraine runs in families . . .

. . . but is it inherited?

EDITOR,—Michael Bjørn Russell and Jes Olesen report an increased familial risk of migraine.¹ Before migraine is accepted as an inherited disorder, as it is in the heading to the paragraph about the paper on the "This week in *BMJ*" page, however, all studies and possible biases should be considered.

The paper concentrates on the high prevalence of migraine in first degree relatives of probands with migraine. Surprisingly, however, the figures show that the first degree relatives of probands who had never had migraine had a prevalence of migraine with aura of 5.3% and of migraine without aura of 17.2% (table II). This latter figure is higher than that expected in the general population (table IV). If migraine does aggregate in families one would expect the first degree relatives of probands who had never had migraine to have a significantly lower prevalence than the general population.

Telling all probands in writing that "the object was to study the frequency and heredity of migraine" could have biased the familial study, which seems overall to have shown a higher prevalence of migraine than did the population study, which used the same interviewer, methods, and population.² In Russell and Olesen's study the response rates were high, but some bias may have arisen as the proportion of probands who did not allow their family to be interviewed was 3% for migraine without aura, 6% for migraine with aura, but 11% for those who never had migraine; of the probands in the study, those with migraine had an average of 2.8 relatives whereas those who never had migraine had an average of 3.1 relatives (calculated from table II).

I undertook my study on familial prevalence³ long before the diagnostic criteria of the International Headache Society were available.⁴ Russell and Olesen are wrong to imply that my study suffered "from lack of direct interview of relatives." A trained interviewer administered a questionnaire to over 99% of first degree relatives living in a defined area (south Wales).³ Several large twin studies, also completed before the

new diagnostic criteria were available, gave surprisingly little evidence of heredity.⁵ Perhaps the jury assessing the importance of heredity and environment in the prevalence of migraine should still be considering its final verdict.

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- 3 Waters WE. Migraine: intelligence, social class, and familial prevalence. *BMJ* 1971;iii:77-81.
- 4 Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia* 1988; 8(suppl 7):1-96.
- 5 Waters WE. Headache: Series in Clinical Epidemiology. London: Croom Helm, 1986:92-101.

Authors' reply

EDITOR,—Since migraine without aura aggregates in families of probands with migraine without aura, it is expected that migraine without aura is less common in families of probands who have never had migraine. We calculated the population relative risk, adjusting for sex and age, since migraine without aura depends on these factors. If the population relative risk significantly exceeds one an increased family aggregation is implied, whereas a result significantly below one implies a decreased family aggregation.

W E Waters states that the risk of migraine without aura among the first degree relatives of probands who had never had migraine was expected to be considerably lower than the figure we found. This is not correct. We calculated that the expected population relative risk was 0.87 (95% confidence interval 0.62 to 1.12) and found that it was 1.11 (0.83 to 1.39). These results are not significantly different. Thus random variation may explain our result. The calculation of the expected population relative risk was based on the increased risk of migraine without aura in first degree relatives of probands with migraine without aura, the overall prevalence of migraine without aura, and a similar number of first degree relatives of probands who had never had migraine to that in our study.

Waters suggests that the disclosure of the objective of the family study could have biased the study, since the overall prevalence of migraine was higher in the family study than in the general population.¹ Firstly, these prevalences are not comparable, since migraine aggregates in some families and the prevalence of migraine in first degree relatives depends on the relative proportion of probands with migraine and probands who have never had migraine. Secondly, responders and non-responders were not significantly different regarding migraine. A minority of probands did not allow their families to be interviewed, but this is not likely to have caused bias in the family study since migraine as assessed by probands' report was not significantly different in these families and those included in the family study. Probands who had never had migraine had a slightly higher average number of first degree relatives than probands with migraine, which is probably due to chance.

Although Waters pioneered epidemiological studies of migraine, the methods section in his study that we cited is not completely clear. It states that "relatives were visited and a short administered questionnaire on headache was completed."² This can easily be taken as indicating that the relatives, rather than the trained interviewer, filled in the questionnaire. Our results indicate the importance of genetic factors in migraine without

aura and migraine with aura. This is supported by previous twin studies.³ Studies of unselected twins interviewed blind by a physician will, however, be important, and the final confirmation will be identification of the gene(s).

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Screening for intracranial aneurysms

Short natural course makes screening impracticable

EDITOR,—Jacoline E C Bromberg and colleagues report an increased risk of subarachnoid haemorrhage in first degree relatives of patients presenting with this condition and suggest that these relatives might be screened for unruptured aneurysms.¹ Though the accuracy of telephone interviews to diagnose aneurysmal subarachnoid haemorrhage may be questioned, the authors' findings are not surprising. Subarachnoid haemorrhage generally occurs sporadically in the population, but most neurosurgeons have encountered families with a history of the condition, and there is a large literature on such cases.² The question of whether to screen all first degree relatives of patients presenting with subarachnoid haemorrhage is more debatable.

Screening requires a knowledge of the natural course of unruptured aneurysms. This is lacking in at least two respects. Firstly, what are the risks of an asymptomatic unruptured aneurysm bleeding? The figure of 1% a year has been quoted but is based on a retrospective analysis of data.³ The likelihood of aneurysms bleeding is currently being investigated by the international study of unruptured intracranial aneurysms. Secondly, over what period does an aneurysm develop? This is complex: the size of an aneurysm measured angiographically varies depending on the intraluminal thrombus it contains, as well as with the absolute volume of the aneurysm sac. Certainly, we have seen an aneurysm increase in apparent size in a worrying and dramatic fashion over two weeks. Screening is pointless if the period over which an aneurysm develops and ruptures is short, and without such knowledge we cannot say what the interval should be between repeat screening tests.

The practicality of screening also depends on the sensitivity, specificity, and risks of the screening investigation. The gold standard remains cerebral angiography, which requires catheterisation of the carotid and vertebral arteries. This may carry a risk of causing a persistent neurological deficit of 0.5%.³ In the series of Bromberg and colleagues, if 1259 first degree relatives were investigated to identify 10 people who would develop subarachnoid haemorrhage, persistent deficits might be caused in 6. To this the morbidity and mortality associated with treating aneurysms (which might never haemorrhage) would have to be added. The alternative screening investigation, magnetic resonance angiography, is non-invasive but may be insensitive in detecting aneurysms less than 5 mm in diameter.⁴