Letters to the Editor

SIR—The essential facts are (a) both 16:1 trans and 18:1 trans (sum of both designated T_L) are higher for cases in 8 of the 10 areas studied, (b) mean levels of these acids differ significantly from area to area by amounts greater than case versus control differences. In such circumstances, overall (unweighted) mean values are of no import, and appropriate statistical treatment is required to determine how much of the variability is due to area differences and how much to case versus control differences. It is therefore the weighted mean case minus control differences which are reported in table 2.[1] Dr Hunter may be of the opinion that the 10 differences of table 1[1] are small, but the question is whether the weighted (overall) mean difference is statistically significant.

In fact, in respect of 16:1 trans the difference is highly significant. He is, of course, at liberty to interpret this result in any way he may think fit, but hopefully by employing "good scientific judgement".

There is no contradiction in our papers. In respect of "higher" trans acids T_H (equals total trans acids T minus T_L) such as 20:1 trans etc, weighted case and control mean values are virtually identical, and it is only for "lower" trans acids that a difference is apparent.

Dr Hunter has failed to bring out the fact that odd-number and branched-chain acids (L)—which are "more abundant" in ruminant animal fat—are present in significantly higher amount in the *control* group. The relativity of trans acids to L acids can then be taken as a measure of relative amounts of the two types of fat. We agree that regression of the ratio T/L on H is a "complex statistical manipulation" but the "confusion" is of Dr Hunter's making, The argument[2] led only to a presumption that a case versus control difference might exist in T_L–a conclusion which was justified experimentally in subsequent work[1] in which T_L was measured separately from the other trans components.

Such factors as smoking, hypertension, etc, may or may not be risk factors, and our claim is only that a risk factor previously unrecognised has been identified. It is very unlikely by the very nature of collection of our specimens that case/control differences in smoking, etc, will be other than random. It may be well to add that "employment grade" has been claimed to be "a stronger predictor of a man's risk of dying from IHD than any of the more familiar risk factors"[3], and in this respect our selection showed no significant difference in social class status between cases and controls.[4]

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References
[1] Thomas LH, Winter JA, Scott RG. Concentration of 18:1 and 16:1 trans-unsaturated fatty acids in the adipose body tissue of decedents dying of ischaemic heart disease compared with controls: analysis by gas-liquid chromatography. *J Epidemiol Community Health,* 1983; **37:** 16–21.
[2] Thomas LH, Winter JA, Scott RG. Concentration of trans-unsaturated fatty acids in the adipose body tissue of decedents dying from ischaemic heart disease compared with controls. *J Epidemiol Community Health,* 1983; **37:** 22–4.
[3] Editorial *The Lancet,* 1981; **2:**347.
[4] Ischaemic heart disease and the proportions of hydrogenated fat and ruminant-animal fat in adipose tissue at post-mortem examination: a case control study. *J Epidemiol Community Health,* 1981; **35:** 251–5.

SIR—Gillies *et al*[1] document the excess perinatal mortality and congenital malformation in Asians in Bradford and suggest a number of causes for this, including consanguinity.

Because of the British belief that our culture is "right" and all variations are "inferior", there is a strong tendency in this country to attribute ethnic minority health problems to deficiencies in the ethnic minority culture rather than to material factors, discrimination, or inappropriate social responses to minority cultures.

It is therefore important that the idea of consanguinity as the cause of the high rate of congenital malformations in Asians should be subjected to particularly close scrutiny.

Gillies *et al* point out that first cousin marriages constitute 48% of Pakistani marriages as opposed to 8% of Indian marriages and 0·5% of non-Asian marriages. Therefore, if consanguinity accounted for the excess Asian congenital malformations, it would be expected that the excess would be greater in the Pakistani community than in the Indian community. However, the paper points out that 77% of Bradford Asian families were of Pakistani origin whereas 74% of the Asian deaths associated with congenital abnormality occurred in that community. At first sight therefore the community in which consanguinity is most prevalent does not suffer a disproportionate share of the Asian excess mortality. The paper provides no support for the consanguinity hypothesis, even if it does not refute it.

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Reference
[1] Gillies DRN, Lealman GT, Lumb KM, Congdon P. Analysis of ethnic influence on stillbirths and infant mortality in Bradford 1975–81. *J Epidemiol Community Health* 1984; **38:** 214–7.