the exclusion of one pair in which the control had breech labour from the analyses including birthweight, and the exclusion of two additional pairs in which the case had breech labour from the analyses including gestation. For those pairs included in the analyses the unadjusted relative risk estimate associated with breech labour was 9·0 (9/1) for the birthweight analyses and 7·0 (7/1) for those involving gestational age. These contrast with the relative risk of 4·5 (9/2) in the analysis involving all pairs. These differences emphasise the small numbers of breech labours on which our risk estimates are based. Nonetheless, as shown in the table, adjustment for either of these factors did not have a substantial effect on the estimate of relative risk associated with breech labour.

Whether it is appropriate to thus apply statistical adjustments for birthweight and gestation to calculations of the risk of cryptorchidism associated with breech labour depends upon the mechanism(s) of any association of low birthweight with risk of cryptorchidism persisting beyond infancy, and these mechanism(s) are not yet known clearly.

A SWERDLOW
P G SMITH

Epidemiology and measures of disablement

SIR—Recently I have read a short, lucid, and comprehensive new book introducing community medicine[1] and new editions of two outstanding and widely read textbooks.[2 3] The authors are to be congratulated, but I was disappointed at the lack of emphasis given to the assessment of disablement in these books. Disablement should be viewed alongside mortality and morbidity as one of the dimensions in the assessment of "community health", and it should be a component of outcome measures used in clinical audit and the monitoring and evaluation of health services. Diagnostic labels, for example, cerebral haemorrhage, multiple sclerosis, rheumatoid arthritis, diabetes, do not convey the degree of associated disablement. Survival, restoration to normal physiological and biochemical values, changes in the clinical condition, and return to paid employment are insufficient measures of the outcome of many of today's treatments (eg, renal dialysis, amputations in the elderly, use of anti-rheumatic and cytotoxic drugs) and of physiotherapy and other rehabilitation activities. Certainly there are many difficulties in the measurement of disablement, not least those arising from the complex interplay of impairment, personality, and the social and physical environment. Nevertheless students should be introduced to the concepts of impairment, disability, and handicap,[4 5] and to the various approaches (and the shortcomings) that are available to assess their presence and severity.[6 7]

MICHAEL WARREN

References

[1] Florey C duV, Burney P, D'Souza M, Scrivens E, West P. *An introduction to community medicine.* Edinburgh: Churchill Livingstone, 1983.
[2] Alderson M. *An introduction to epidemiology.* 2nd ed. London: Macmillan, 1983.
[3] Abramson JH. *Survey methods in community medicine.* 3rd ed. Edinburgh: Churchill Livingstone, 1984.
[4] Wood PHN, Badley LM (1978). An epidemiological appraisal of disablement. In *Recent advances in community medicine I.* A E Bennett, ed. Edinburgh: Churchill Livingstone, 1978.
[5] World Health Organization *International classification of impairments, disabilities and handicaps.* Geneva: WHO, 1980.
[6] Duckworth D. *The classification and measurement of disablement.* DHSS Research Report No. 10. London: HMSO, 1983.
[7] Duckworth D, Knight R, Warren MD (1984). Using the ICIDH in household surveys. Problems and possibilities. *Statist J United Nations ECE2* 1984 85–96.

Consumption of trans acids in relation to heart disease

SIR—The Institute of Shortening and Edible Oils would like to comment on the recent articles by Thomas *et al.*[1 2] The authors report a higher proportion of "lower" *trans* acids (16:1 and 18:1) in adipose tissue taken post mortem from 136 people who died of heart disease (cases) compared with 95 individuals who died of unrelated causes (controls). Noting that "lower" *trans* acids are more abundant in commercially hydrogenated fats than in ruminant-animal fats, the authors conclude that "the cases consumed a higher amount of hydrogenated fat relative to ruminant-animal fat than did the controls". The authors conclude further that "those hydrogenated fats having higher content of lower *trans* acids will present the greater risk and in this respect it is possible that some hydrogenated vegetable oils may well be more harmful than hydrogenated marine oils". This is sheer speculation without basis in fact.

Thomas *et al* clearly are speculating and do not provide convincing evidence that the heart disease in their cases was directly related to consumption of *trans* acids. The authors claim that the difference in lower *trans* acid levels (16:1 plus 18:1, designated as "T_L") between cases and controls is statistically significant. However, the absolute difference is small and the variability is high. In table 1,[1] the authors present mean T_L values for cases and controls for 10 regions of the UK but do not present overall mean values. From these data I calculated overall (unweighted) mean T_L values to be 3·31% (of total adipose tissue lipid) for cases and 3·08% for controls.

The difference is only 0·23% of the total adipose tissue lipid. While this difference may be statistically significant, it is far too small in the absence of other considerations to justify the strong conclusion that the heart disease in the cases was related to their apparently higher ingestion of hydrogenated fats. Good scientific judgment leads to the conclusion that the authors are playing games with statistics.

The authors also fail to consider the multifactoral nature of heart disease and therefore did not exclude other possible differences related to heart disease risk between cases and controls. For example, were there differences in serum cholesterol, smoking history, hypertension, physical activity, obesity, or family history of heart disease between cases and controls? The authors do not address these possibilities. It is important to point out that, in the case of serum cholesterol, other factors in the diet such as the type of protein and dietary fibre can affect serum cholesterol level independently of the type and amount of dietary fat.

Furthermore, the conclusion presented in the current papers contradicts the conclusion reached previously by these authors using the *same* adipose tissue specimens.[3] In this earlier paper, the authors note that adipose tissue levels of *trans* unsaturated acids tended to be higher in cases than in controls but that the differences were *not* statistically significant. In the more recent papers,[1 2] the authors offer no explanation of this discrepancy other than their use (for the current work) of a GLC procedure "using a more recently developed highly polar liquid phase". Another possible explanation is that a more complex (and confusing) statistical manipulation of the data in the current papers resulted in a statistically significant difference between cases and controls that was not observed previously. This particularly is notable in the second paper[2] where there are no significant differences noted until the authors state, "when the means of T/L (total trans acids/the sum of 14:1, 15:0, 15:0 br, 15:1, 16:0 br, 17:0 17:1 fatty acids) are now adjusted for the regression on H (the sum of 20:0, 20:1, 20:2, 20:3, 22:0, 22:1 fatty acids) the difference between cases and controls becomes highly significant".

Thomas *et al* fail to cite or comment on the recent work of Ohlrogge, Emken, and Gulley[4] which showed *no* preferential accumulation of 18:1 positional isomers in human tissue total lipids (including adipose tissue) among eight subjects aged 21 to 80. Two of the eight subjects died of myocardial infarction. On the other hand, Ohlrogge *et al* reported adipose tissue levels of *trans* 18:1 (2·0–5·8% of total fatty acids in all subjects) that were similar to those reported by Thomas *et al* (0·87–4·35% of total fatty acids; [unweighted] means for cases = 2·55% and for controls = 2·40%). They also ignored the results of Heckers *et al*[5 6] who concluded that there was no correlation between tissue *trans* isomer levels and atherosclerosis in males dying either from atherosclerotic heart disease or from other causes. In addition, Thomas *et al* failed to consider numerous published studies in which high levels of *trans* acids were fed to animals for long periods of time without any overall adverse effects.[7–10]

In conclusion, Thomas and coworkers have *not* presented data which support convincingly their hypothesis that consumption of *trans* acids is directly related to the development of heart disease. On the contrary, numerous reliable literature accounts reviewed by Applewhite[11] support the conclusion that *trans* acids do not pose any hazard to man in a normal diet.

J EDWARD HUNTER
Biological Subcommittee,
Institute of Shortening & Edible Oils,
Washington, DC, 20006, USA

References

[1] Thomas LH, Winter JA, Scott RG. Concentration of 18:1 and 16:1 trans-unsaturated fatty acids in the adipose tissue of decedents dying of ischaemic heart disease compared with controls: analysis by gas chromatography. *J Epidemiol Community Health* 983; **37:** 6–21.

[2] Thomas LH, Winter JA, Scott RG. Concentration of transunsaturated fatty acids in the adipose tissue of decedents dying of ischaemic heart disease compared with controls. An analysis of covariance. *J Epidemiol Community Health* 1983; **37:** 22–4.

[3] Thomas LH, Jones PR, Winter JA, Smith H. Hydrogenated oils and fats; the presence of chemically-modified fatty acids in human adipose tissue. *Am J Clin Nutr* 1981; **34:** 877–86.

[4] Ohlrogge JB, Emken EA, Gulley RM. Human tissue lipids: occurrence of fatty acid isomers from dietary hydrogenated oils. *J Lipid Res* 1981; **22:** 955–60.

[5] Heckers H, Korner M, Tuschen TWL, Melcher, FW. Occurrence of individual trans-isomeric fatty acids in human myocardium, jejunum and aorta in relation to different degrees of atherosclerosis. *Atherosclerosis,* 1977; **28:** 389–98.

[6] Heckers H, Melcher FW, Dillmar K. Daily consumption of trans-isomeric fatty acids—a calculation based on composition of commercial fats and of various human depot fats. *Fette-Seifen-Anstrichmittel* 1979; **81:** 217–26.

[7] Alfin-Slater R, Aftergood L. Nutritional role of hydrogenated fats (in rats). In: Emken EA, Dutton HJ, eds. *Geometrical and positional fatty acid isomers,* AOCS Monograph No. 6, Champaign, IL, American Oil Chemists' Society, 1979; 53–74.

[8] Mattson FH. An investigation of the essential fatty acid activity of some of the geometrical isomers of unsaturated fatty acids. *J Nutr* 1960; **71:**366–70.

[9] Alfin-Slater RB, Wells AF, Aftergood L, Deuel HJ. Nutritive value and safety of hydrogenated vegetable fats as evaluated by long-term feeding experiments with rats. *J Nutr* 1957; **63:** 241–61.

[10] Aaes-Jorgensen E, Funch JP, Dam JH. The role of fat in the diet of rats. 8. Influence on growth of shortening products, emulsifiers and polymerized linseed oil. *Br J Nutr* 1956; **10:** 317–24.
[11] Applewhite TH. Nutritional effects of hydrogenated soya oil. *J Am Oil Chem Soc* 1981; **58:** 260–9.

Note: This letter was shown to the authors of the article who do not wish to reply but they would agree with the last sentence of the letter above. *Editor.*

Effects of tap water lead, water hardness, alcohol, and cigarettes on blood lead concentrations

SIR—This recent contribution by S J Pocock *et al* (March 1983, 1–7) clearly shows the role of tap water and personal habits in raised blood lead concentrations. The authors, however, conclude by stating that "because direct evidence of a comparable nature has not been shown for lead in petrol . . .lead in water should be given priority . . . ." Petrol lead sales in Boston, Massachusetts, have been shown to correlate very well with blood lead concentrations.[1] We suggest that reduction in lead exposure requires attention to both air and water.

MICHAEL RABINOWITZ
Children's Hospital Medical Center, Boston, Massachusetts 02115, USA

References

[1] Rabinowitz M, Needleman HL. Petrol lead sales and umbilical cord blood lead levels in Boston, Massachusetts. *Lancet* 1983; i: 63.

Note: This letter was shown to the authors of the article who do not wish to reply but they would agree with the last sentence of the letter above. *Editor.*