LETTERS TO THE EDITOR

Effect of ethnic origin of mother on fetal outcome

EDITOR,—The recent paper by Lyon and colleagues¹ concerning the effect of ethnic origin on fetal outcome illustrates some of the pitfalls of research involving the categorisation of ethnic groups.^{2,3}

Race, ethnicity, and nationality are different concepts and it is difficult to produce categories that represent them accurately.⁴ In this study, the authors do not describe how their racial or ethnic categories are either defined or assigned to the women, and this also makes it difficult to compare the findings with other research.

It is not always appropriate to combine African and West Indian groups as these are not genetically or culturally homogenous categories. There is a large population of African refugees in Croydon who will be very different from British born black women in many ways. While the authors recognise the diversity of the Asian women in their discussion, the fact that the comments are equally applicable to the black women is not acknowledged.

In pursuing their argument, the authors refer to studies from the USA, but do not acknowledge the difficulties involved in generalising from American studies, because of differences in characteristics, distribution and definitions of race and ethnic groups, and of social class and deprivation. Speculation about the role of sexual activity in chorioamnionitis cannot be justified by reference to a study that compares sexual behaviour in 'black' women in the USA with native Americans, particularly in the absence of any information about the sexual behaviour of women in Croydon or the UK. Furthermore, the comment that race may be an independent variable betrays a lack of understanding of the limits of race as a biological entity.⁵

Because social class, a major confounder, is not measured or controlled for in the analysis, it becomes difficult to comment on the possible cause of the observed difference between ethnic groups. Socioeconomic factors are referred to but not discussed adequately in comparison with the space devoted to possible biological differences between the babies of black women and those of other groups.

The high rate of intrauterine death in the babies of black women between 28 and 36 weeks' gestation is based on only eight deaths in total. If three babies had not died, the rate would have been similar to that in the white mothers. Likewise, the comments on the gestation specific neonatal mortality rates refer to four deaths among the Asian women and one death among the babies born to black women. With such small numbers, the influence of chance becomes important, and correct categorisation and controlling for confounders becomes vital. Furthermore, no mention is made of any allowance for the multiple tests of significance which were performed, such as a Bonferroni correction, given the high probability of a type 1 statistical error.

In their discussion, the authors do not appear to recognise either the limits of their data or the problems involved in categorising black or ethnic minority groups, resulting in conclusions that cannot be justified by their findings.

N S CROWCROFT
Department of Public Health Medicine,
South West Thames Regional Health Authority,
London W2 3QR

K J MCKENZIE
Institute of Psychiatry,
de Crespigny Park,
London SE5 8AF

- 1 Lyon AJ, Clarkson P, Jeffrey I, West GA. Effect of ethnic origin of mother on fetal outcome. *Arch Dis Child* 1994; 70: F40-3.
- 2 Chief Medical Officer. *The nation's health*. (1992 annual report.) London: HMSO, 1992.
- 3 Sheldon TA, Parker H. Race and ethnicity in health research. *J Public Health Med* 1992; 14: 104-10.
- 4 Sillitoe K. *The search for an ethnicity question for the census*. London: Office of Population Censuses and Surveys, June 1987.
- 5 Jones JS. How different are human races? *Nature* 1981; 293: 188-90.

Dr Lyon and colleagues comment:

Crowcroft and McKenzie quite correctly highlight problems in the analysis of our data but we would not agree that our conclusions cannot be justified. Looked at independently the West Indian and African groups still had significantly more deaths. In particular there was a strikingly high rate of late fetal deaths (20-23 weeks) in the West Indian group which persists whatever statistical manipulation is done (13/1000 compared with 3/1000 total deliveries in the white mothers). This was an important finding as this would not have been reflected in standard perinatal mortality statistics and it also confirmed observations made by the obstetricians in Croydon—these observations being the initial reason for the study. There are no comparable studies in UK and comparisons with the American literature were to highlight that in other countries racial differences in fetal outcome have been found. Even when allowance is made for socioeconomic status and deprivation, large differences persist between black and white in the USA—and hence the observation that ethnicity itself may be an independent variable.¹ We accept that we cannot make direct comparisons of sexual behaviour between the USA and UK but this was merely a speculation on possible reasons for higher infection rates in the black groups. Again there are no comparable data in Europe and we doubt we will ever be allowed to ask the sort of questions needed to obtain it!

This study was a retrospective audit of data and we stressed that care must be taken in its interpretation. Routinely collected data such as this can, at best, point to possible trends but is never complete enough for proof. The results, however, confirm a clinical impression and show a definite and significant tendency of increased fetal loss among black mothers, particularly the West Indian group. A prospective study is needed to determine if ethnic origin is a major factor in fetal outcome and, as this has important implications in targeting health care, we hope that South West Thames Regional Health Authority will maybe take up this challenge and hopefully not hide behind arguments over statistical significance.

- 1 Behrman RE. Premature births among black women [editorial]. *N Engl J Med* 1987; 317: 763-5.

Immunohistochemical localisation of epidermal growth factor and its receptor in the developing human stomach

EDITOR,—Epidermal growth factor (EGF) is a 53 amino acid polypeptide present in many mammalian species and produced in the human by salivary glands, the pancreas, Brunner's glands in the duodenum and in the stomach after gastric ulceration where it has a role in repairing mucosal damage.¹ In an *in vitro* study Britton *et al* clearly showed that EGF was not denatured by gastric acid allowing it to pass into the small intestine or to bind to receptors within the stomach.²

In vivo work on neonatal rats has demonstrated that EGF leads to increased growth of the gastric mucosa but not to functional maturation³ and that anti-EGF antiserum given to newborn mice leads to retardation of gastric ontogeny.⁴ EGF has a clearly documented role in many other species and it seems likely that EGF may have a role during the period of rapid gastrointestinal growth and maturation in fetal and early infant life.

In humans, EGF is also found in large concentrations in breast milk⁵ and in amniotic fluid from the second half of gestation,⁶ with levels increasing to term, a time when the developing fetus has been shown to swallow large volumes of liquor.

We have, therefore, looked for EGF and its receptor in the fetus and newborn infant. Thin sections (8 micron) from 15 fetal (13 to 26 weeks' gestation) and five infant (2 to 21 weeks' postnatal age) stomachs were stained using antibodies to EGF and its receptor (Sigma, Poole). Fetal specimens were obtained from therapeutic terminations and spontaneous abortions in pregnancies with no evidence of congenital abnormality. Infants who died suddenly and unexpectedly after a full term pregnancy were the source of the other specimens. Consent was obtained by the district ethics committee.

EGF activity was not detected in any of the fetal or infant stomachs examined. However, EGF receptors were detected in all fetal and infant stomachs studied from 18 weeks' gestation, a time when cellular differentiation in the stomach is complete. In addition, in all cases, we noted that the EGF receptors were localised to the luminal aspect of the gastric mucosa in both the body and antrum.

The demonstration of EGF receptors on the luminal aspect of the gastric mucosa in the fetus who is swallowing large volumes of liquor containing EGF provides further circumstantial evidence for an important ontogenic role for EGF, acting as a lumone, in the developing human stomach.

E J KELLY
S J NEWELL
K G BROWNLEE
J N PRIMROSE
Academic Unit of Paediatrics and Child Health and
Surgery,
St James's University Hospital,
Leeds LS9 7TF

- 1 Wright NA, Pike CM, Elia G. Ulceration induces a novel epidermal growth factor-secreting cell lineage in human gastrointestinal mucosa. *Digestion* 1990; 46: 125-33.
- 2 Britton JR, George-Nascimento C, Udall JN, Koldovsky O. Minimal hydrolysis of epidermal growth factor by gastric fluid of preterm infants. *Gut* 1989; 30: 322-7.
- 3 Pollock PF, Goda T, Colony C, *et al*. Effects of enterally fed epidermal growth factor on the small and large intestine of the suckling rat. *Regul Pept* 1987; 17: 121-32.
- 4 Zschiesche W. Retardation of growth and epithelial differentiation in suckling mice by anti-EGF antisera. *Biomed Biochim Acta* 1989; 48: 103-9.