these issues nationally have, likewise, been weakly supported. Networks are established and then closed down. Training in Health and Race (which was supported by the Health Education Council (now the Health Education Authority) and others) and the black health unit (which was at the National Community Health Resource (now Community Health UK)) are both now defunct. At present national initiatives include the King's Fund Centre's "share project" and the Health Education Authority's black and ethnic minority groups database, which provide information resources. These are welcome developments but suffer from insecure funding. Even the Department of Health's new ethnic health unit, which offers more general support, has been established for only three years.

Knowledge of good practice needs to be consolidated over time. To make this possible there must be consistent infrastructural support to help the health service, local authorities, and the voluntary sector to avoid reinventing the wheel.

SOPHIE LAWS
Research officer, policy division
CHRISTOPHER CUNINGHAME
Community health adviser, London division
Save the Children,
London SE5 8RD

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Ethnicity revolves around culture

EDITOR,—Both the editorial and the article on the place of race, ethnicity, and culture in medical research incline to the view that race, even as a biological notion, does not exist.[1][2] This is self evidently an overstatement. In so far, however, as there are no specific qualities that anyone possesses simply by virtue of his or her race alone, race itself falls short of meeting the criteria for a sound epidemiological variable.

As both articles agree that ethnicity is simply a matter of group identification, I am surprised that Peter A Senior and Raj Bhopal go on to propose its elevation to the status of an epidemiological variable.[2] Taken in relation to their criteria for such a variable, ethnicity is not measurable and does not itself distinguish between populations in any characteristics relevant to health. Besides, ethnicity, being a matter of self definition, cannot be assigned by others. Ethnicity is not in its own right a valid epidemiological variable.

Culture, which is "the totality of socially transmitted behaviour patterns, arts, beliefs, institutions, and all other products of human work and thought characteristic of a community or population,"[3] does differentiate human populations in characteristics relevant to health. McKeown makes a sound case for the centrality of culture in the origins of human diseases by showing the different patterns of disease in hunter-gatherer, agrarian, and industrialised societies.[4]

But culture is expressed in geographical locations, and the environment in which people live contains the greatest threats to their health. Given this consideration, race and ethnicity are still relevant, for ethnic subgroups of racial groups tend to live together and evolve a culture in relatively defined geographical regions. In accepting, and being accepted by, a community an individual shares in the benefits and risks of that community's culture and environment.

In summary, culture and geographical location, defined as one factor, provide the most valid and most important epidemiological variable available

today. The current development of language permits us, however, to name cultures only by using racial, ethnic, or national labels. Thus ethnicity is relevant as part of an epidemiological variable if, and only if, we equate it with culture: after all, the group identification that constitutes ethnicity revolves around culture.

IKECHUKWU O AZUONYE
Consultant psychiatrist
Claybury Hospital,
Woodford Bridge,
Essex IG8 8BY

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No secular decline in fecundity has occurred in Sweden

EDITOR,—Stephen Farrow's editorial highlights the important epidemiological issue of whether human fertility is threatened by environmental pollution or occupational exposure.[1] The methodological problems in studies of this are well recognised and include the study design, measures of exposure, reproductive and developmental outcomes, interaction, participation, and recall bias.[2] How then could we address the hypothesis of a possible secular decline in fertility?

A decline in the twinning rate has been suggested to be related to increased rates of miscarriage and environmental pollution.[3] The changed distributions of age and parity of mothers, however, explain the secular decline in the twinning rate from the 19th century until 1960 in Sweden, while the continued decline during the 1960s remains unexplained.[4]

Time to pregnancy, with live birth as the outcome variable, could be a more valid and consistent measurement for analysis of secular trends in fecundity. We have studied fecundity among Swedish peasant women in the 19th century,[5] and a sample of Swedish women born in 1936-60,[6] and have also studied the fecundity of women in Umeå who gave birth to a liveborn child in 1992 within the collaboration project of the European study of infertility and subfecundity. When we compared the different birth cohorts there was no secular decline in the cumulative incidence of pregnancy within one year among Swedish women born during the 19th century, 1936-45, 1946-60, or 1963-72 and having their first child at the age of 20-29. In addition, there was no negative time trend when the fecundity of women in the three last cohorts was compared (table). Thus we did not observe any secular decline in fecundity in Sweden.

ULF HÖGBERG
Senior lecturer
Department of Obstetrics and Gynaecology,
Umeå University,
S-901 87 Umeå,
Sweden
MARIANNE WULFF
Senior registrar
Department of Epidemiology and Public Health,
Umeå University

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Value of data provided for health promotion programmes

EDITOR,—Family health services authorities have recently received an unprecedented amount of clinical data from general practitioners as part of the new health promotion and chronic disease management programmes. Yet the value of these data remains undetermined. For monitoring purposes general practitioners are required to provide data about risk factors, interventions offered, the prevalence of some major chronic diseases, and, for chronic disease management, the numbers of sufferers and treatments provided.

In theory the provision of such data should, in the long term, not only enable monitoring of the practices' level of preventive activity but also give information on the prevalence of major chronic diseases and of "risky" behaviours. As nearly 80% of patients visit their general practitioner in a given year,[1] these programmes could provide data on behaviour that are greatly superior in terms of coverage to those gained by local surveys.

This benefit, however, remains theoretical. Lack of clear definitions for the data has meant that practices enter and extract information in different ways, and consequently comparisons among practices are largely meaningless. Some far sighted family health services authorities may have specified their requirements in detail; most will not have done.

A cogent example is provided by the asthma chronic disease management programme. The standard fees and allowances specify that numbers of "current asthma sufferers" should be provided. Many software companies have developed specific programs to search for the data, but a personal survey of seven major software companies established that the two biggest companies differed substantially in their criteria for identifying patients: one used patients who had ever had a diagnosis of asthma recorded, while the other used patients treated within the previous year. Such different criteria could produce figures varying by up to 30% for populations with the same actual prevalence. Similarly, the criteria for raised blood pressure and coronary heart disease are unclear.

Use of the data in aggregated form for planning, epidemiology, or other purposes could be seriously misleading. Yet a considerable amount of time, energy, and money is being spent in primary care on recording and extracting this information. There is a tremendous opportunity to encourage general practitioners to record information on computer that will be of use clinically and to gain access to extensive data on the prevalence of disease and risk factors that have not previously been available. Much of this opportunity, how-

Life table analysis of fecundity per month in women aged 20-29 by density method (rates adjusted by Mantel-Haenszel method)

	1936-45	1946-60	1963-72
No of live births	1202	1694	385
Person time (months)	5645	8148	1770
Crude rate per month	0·21	0·21	0·22
Adjusted rate	0·21	0·20	0·26
Relative risk (95% confidence interval) (%)	100	94 (87 to 102)	120 (107 to 135)