to produce a measure of relative differences in each country. The figure combines the data for neonatal and postneonatal mortality and shows that England and Wales have a steeper and more consistent gradient of mortality across social classes than Sweden. The width of the "steps" is proportional to each class's share of total live births.

The differences in total mortality and its socioeconomic distribution in the two countries are both consistent with the suggestion that Sweden's more egalitarian income distribution might be influential.[3] Mortality in each social class will be affected by the scale of relative deprivation in each. Social classes are heterogeneous with respect to income: there are unemployed and low paid workers in social class I occupations just as there are occasional well paid unskilled workers in social class V. Therefore, while having its greatest impact lower down the social scale, a more egalitarian income distribution may be expected to produce a small reduction in the burden of relative deprivation on mortality even in the upper classes. Though Leon and colleagues seem to assume that smaller differences in income should lead to a relative rather than an absolute diminution in differentials in mortality, if relative deprivation is regarded as a risk factor like any other, an absolute effect may be more plausible. This would also be consistent with its impact on total mortality.

RICHARD G WILKINSON

Trafford Centre for Medical Research,
University of Sussex,
Brighton BN1 9RY

1 Leon DA, Vagero D, Otterblad Olausson P. Social class differences in infant mortality in Sweden: a comparison with England and Wales. *BMJ* 1992;**305**:687-91. (19 September.)
2 Pamuk ER. Social class inequality in mortality from 1921 to 1971 in England and Wales. *Population Studies* 1985;**39**:17-31.
3 Wilkinson RG. Income distribution and life expectancy. *BMJ* 1992;**304**:165-8.

EDITOR,—D A Leon and colleagues found that in the mid-1980s social class differences in infant mortality were largely similar, in relative terms, in Sweden and in England and Wales, even though neonatal and post-neonatal mortality differed considerably in the two areas.[1] Babies born to mothers of lower social class had about an 80% increased risk of death during the first year of life compared with babies born to women of higher social class.

Evidence largely consistent with these findings emerges from analysis of Italian data on all births and infant deaths routinely collected and published by strata of maternal education (a rough approximation to social class) by the Italian Central Institute of Statistics.[2,3] Infant mortality in women reporting no education or only primary education was 23·7/1000 live births in 1975 and 14·8/1000 in the four years 1980/3. In comparison, the rates in women with college education were 9·2 and 8·1/1000 live births respectively (table). The mortality ratio tended to decrease from the mid-1970s to early 1980s, but in 1980-3 it was still 1·8 for babies born to women with no or primary education compared with those born to women with a college education.

These differences between less and more educated women were not attributable to birth weight—for example, in 1980-3 infant mortality adjusted for birth weight was 14·4/1000 for women with no or primary education and 8·7/1000 for women educated at college. The proportion of infant deaths that would have been avoided if all babies experienced the mortality of the babies of women educated at college was 55% in 1975 and 32% in 1980-3; this estimate for 1980-3 is consistent with findings from Sweden and England and Wales.[1]

This evidence suggests that the differences among groups classified by social class or education are largely similar in relative terms in populations characterised by important differences in stillbirth rates and infant mortality (mortality in the first year of life was 13·5/1000 live births in Italy in 1980-3 and 9·5/1000 live births in England and Wales in 1983-5[1,3]). This supports Leon and colleagues' suggestion that differences in the home environments of infants, rather than inequalities in health care, are the main determinants of mortality in the first year of life in babies born to women of lower social class.

FABIO PARAZZINI

Istituto di Ricerche Farmacologiche "Mario Negri,"
20157 Milan,
Italy

1 Leon Da, Vågerö D, Otterblad Olausson P. Social class differences in infant mortality in Sweden: comparison with England and Wales. *BMJ* 1992;**305**:687-91. (19 September.)
2 Parazzini F, Imazio C, Pampallona S, La Vecchia C. Trends in perinatal, neonatal and postneonatal mortality in Italy, 1955-84. *Soz Praventivmed* 1987;**32**:286-90.
3 Parazzini F, Pirota N, La Vecchia C, Bocciolone L, Fedele L. Determinants of perinatal and infant mortality in Italy. *Rev Epidemiol Sante Publique* 1992;**40**:15-24.

Numbers of registered deaths of infants, infant mortality, and mortality ratio according to maternal education in Italy, 1975 and 1980-3

Maternal education (years)	1975			1980-3		
	No of registered deaths	Rate/1000 live births	Mortality ratio	No of registered deaths	Rate/1000 live births	Mortality ratio
College	184	9·2	1*	902	8·1	1*
≥12 (high school)	1 079	11·5	1·3	4 615	8·9	1·1
8-11 (secondary school)	3 510	16·7	1·8	11 255	11·6	1·3
<7 (primary school or none)	11 431	23·7	2·6	17 820	14·8	1·8

*Reference category.

Screening for Down's syndrome

EDITOR,—Tower Hamlets was one of the districts that participated in the demonstration project for antenatal maternal serum screening for Down's syndrome.[1] When the research funding ended we were asked for £40 000 to continue the service. An audit of Down's syndrome cases in Tower Hamlets revealed a cautionary tale that supports some of the concerns raised by correspondents.[2]

In Tower Hamlets the screening programme did not reduce the prevalence of Down's syndrome during the 18 months of the study. All five cases in this period had had the triple test. One woman requested amniocentesis on the basis of her age alone before the results of her test were known and terminated her pregnancy. Two women were offered amniocentesis on the basis of a high risk estimate (one on age alone) but declined. The remaining two women had a low risk estimate on the triple test but subsequently delivered babies with Down's syndrome. One of these is exploring the possibility of litigation (as in the case of Michelle Huberman[3]).

One major reason for the low detection rate was the low uptake of amniocentesis. The acceptance of amniocentesis following a positive triple test was 53% in Tower Hamlets and 75% in the demonstration project overall. This raises important questions about how the test is being offered, especially to non-English speaking women: nearly half the babies born in the district are to Bangladeshi women.

In a survey of women using the maternity services in Tower Hamlets, less than 10% of non-English women knew they were having a test for a congenital abnormality.[4] In a separate survey conducted by the community health council, only 30% of English speaking women knew they were having a blood test for congenital abnormalities.

The funding quoted does not include the costs of staff training, counselling, costs to women and families, or the opportunity costs. The original estimates quoted in Wald's article also excluded the cost of routinely offering amniocentesis to women 37 years and over. We do not think it is ethical to withdraw a screening programme that detects 100% of cases and replace it with one that detects only 70% of cases, and therefore we will continue to offer amniocentesis to women over the age of 37 years. This increases the costs.

Finally, like other correspondents, we questioned whether the results supported the conclusions that every district in the NHS should give priority to funding the programme.

LUISE PARSONS
J RICHARDS
R GARLICK

Tower Hamlets Health Authority,
London E3 2AN

1 Wald NJ, Kennard A, Densen JW, Cuckle HS, Chard T, Butler L. Antenatal maternal screening for Down's syndrome: results of a demonstration project. *BMJ* 1992;**305**:391-4. (15 August.)
2 Correspondence. Antenatal screening for Down's syndrome. *BMJ* 1992;**305**:768-70. (26 September.)
3 The right to a perfect baby [editorial]. *Independent* 1992 August 22:13.
4 Akin-Deko ALS. *Maternity services liaison scheme: working for the community*. London: MSLS, 1991.

EDITOR,—N J Wald and colleagues' paper on antenatal screening for Down's syndrome[1] has provoked copious correspondence.[2] None of it considers the individual pregnant woman's view. When confronted with the simple information that if her fetus has Down's syndrome the biochemical risk assessment (not a screening test in itself) plus a midtrimester invasive procedure has only a one in two chance of diagnosing this and, even if it hasn't, a one in 20 chance of recommending an amniocentesis, most women decline the test. Population statistics are all very well, but what we are dealing with is one woman and her pregnancy. People want facts, not probabilities.

MARK SELINGER

Nuffield Department of Obstetrics and Gynaecology,
John Radcliffe Hospital,
Oxford OX3 9DU

1 Wald NJ, Kennard A, Densem JW, Cuckle HS, Chard T, Butler L. Antenatal maternal serum screening for Down's syndrome: results of a demonstration project. *BMJ* 1992;**305**:391-4. (15 August.)
2 Correspondence. Antenatal screening for Down's syndrome. *BMJ* 1992;**305**:768-71. (26 September.)

Decreasing quality of semen

EDITOR,—The investigation by Elisabeth Carlsen and colleagues[1] sounds alarm bells and has received worldwide attention. The authors assert that there has been a genuine decline in sperm quality during the past 50 years. However, whether such a conclusion is valid depends on the quality of semen analyses performed more than 30 years ago. How good was quality control in andrology laboratories in the 1940s and 1950s? Today few andrology laboratories have ongoing quality control programmes for sperm concentration determinations, and even using contemporary techniques there can be an error of about 25% in measurements.[2,3]

The authors doubted the existence of a secular trend in technique based on comparison with

haematology results. Such a comparison may be invalid because of the differing viscosities of blood and semen. Unfortunately, white cell pipettes were commonly used for diluting semen specimens for counting in haemocytometers until the 1960s. Eliasson attributed imprecision in sperm concentration determinations to the use of white cell pipettes.[4] Consequently we must conclude that grounds do exist for suspecting a significant bias to methodological error during the period under consideration.

The importance of a possible secular trend in methodological error is apparent when one considers that the apparent differences in sperm concentration were found in studies before 1961 and after 1961, as shown in figure 2 in the paper.[1] Differences were not apparent from 1961 to 1991. Early studies comprised 16% of reports and 11% of subjects.

The authors are urged to establish that their comparison of historical data is free of methodological bias; perhaps a more cohesive set (or subset) in which comparable techniques were used could be considered.

Can we conclude that there is or is not a genuine decline in sperm counts? Unfortunately, comparison of historical and contemporary methods may not be reliable. The greenhouse effect, global warming, declining sperm counts—all may be true; we just do not know.

I S TUMMON

Department of Obstetrics and Gynaecology,
University of Western Ontario,
London, Ontario,
Canada N6A 3K7

DAVID MORTIMER

Sydney IVF Pty Ltd,
Sydney 2000,
Australia

1 Carlsen E, Giwerman A, Keiding N, Skakkebaek N. Evidence for decreasing quality of semen during past 50 years. *BMJ* 1992;**305**:609-13. (12 September.)
2 Newinger J, Behre HM, Nieschlag E. External quality control in the andrology laboratory: an experimental multicentre trial. *Fertil Steril* 1990;**54**:308-14.
3 Mortimer D, Shu MA, Tan R, Mortimer ST. A technical note on diluting semen for the haemocytometric determination of sperm concentration. *Hum Reprod* 1989;**4**:166-8.
4 Eliasson R. Parameters of male fertility. In: Hafez ESE, Evans TN, eds. *Human reproduction, conception and contraception.* Hagerstown: Harper and Row, 1973:39-51.

EDITOR,—The recent meta-analysis by Elisabeth Carlsen and colleagues confirms the previously reported trend in declining sperm density over the past five decades but underestimates the potential effect on male fertility.[1] Although sperm density fell from 113 to 66 million/ml, the mean remains within normal limits. Of far more serious import is the reported fourfold increase in men with oligospermia (< 20 million/ml).

Carlsen and colleagues confined their analysis to sperm density, ignoring other changes. Our own data from partners of women undergoing gonadotrophin stimulant therapy showed that the decrease in sperm density was accompanied by increased prevalence of reduced sperm motility and of abnormal morphology.[2] Mean sperm density fell significantly (p=0·0001) over two six year periods (1977-83 and 1983-9) from 101 million/ml to 96 million/ml and the proportion of men with low sperm density (< 40 million/ml) increased from 5·8% to 16·3%. At the same time the proportion of our population with reduced sperm motility (< 50%) increased from 20·7% to 34·4%. Most striking of all was the 12-fold increase in the prevalence of abnormal morphology (> 50% on light microscopy), from 1·0% to 12·3%. Thus it would seem that there is a much greater deterioration in semen quality than is apparent from the data based on sperm density alone provided by Carlsen. Moreover our finding of a 5% fall in sperm density between 1977 and 1989 indicates that the downward trend is continuing unabated.

The reasons for this decline both in sperm density and quality are not known. We too raised the possibility of an environmental factor,[2] but the nature of this remains obscure and will not be elucidated by further meta-analysis.

JEAN GINSBURG

Academic Department of Endocrinology,
Royal Free Hospital School of Medicine,
London NW3 2QG

PAUL HARDIMAN

Department of Obstetrics and Gynaecology,
Whipps Cross Hospital,
London E11

1 Carlsen E, Giwercman A, Keiding N. Skakkebaek N. Evidence for decreasing semen quality during past 50 years. *BMJ* 1992;**305**:609-12. (12 September.)
2 Ginsburg J, Hardiman P. Ovulation induction with human menopausal gonadotrophins—a changing scene. *J Gynecol Endocrinol* 1991;**5**:57-78.

Resettling long stay psychiatric patients in the community

EDITOR,—David Dayson and colleagues' review of patients discharged from long term care in psychiatric hospitals contains a horrifying paragraph detailing some of the acts of violence committed by a minority of patients after discharge.[1] This indicates how important it is to assess critically the current fashion for providing community care for the mentally ill. In medical terms community care is largely general practice care. The role of general practitioners was not mentioned once in Dayson and colleagues' two articles,[1,2] which in this respect were typical of many others written on the subject. For the past four years I have acted as medical officer to a psychiatric nursing home with nearly 40 residents with major mental illness. During this time I have never once been contacted by a psychiatrist to discuss a patient before his or her discharge. The patients are simply discharged on the assumption that a general practitioner will take on their medical care, without any thought being given to whether the general practitioner is willing or able to do this.

Community care is not a panacea. In recent years the value of care in a humane asylum has been underestimated. But most general practitioners are keen to help make a success of community care, especially if we help choose the patients for whom it is most appropriate. We would be keener still if our role wasn't undervalued or ignored. "The community" is, after all, our field of expertise.

V P SMITH

Lyngford Park Surgery,
Taunton,
Somerset TA2 8SQ

1 Dayson D, Gooch C, Thornicroft G. The TAPS project. 16. Difficult to place, long term psychiatric patients: risk factors for failure to resettle long stay patients in community facilities. *BMJ* 1992;**305**:993–5. (24 October.)
2 Thornicroft G, Gooch C, Dayson D. The TAPS project. 17. Readmission to hospital for long term psychiatric patients after discharge to the community. *BMJ* 1992;**305**:996-8. (24 October.)

Mental health services for children

EDITOR,—Dora Black makes no mention of the contribution made by general and community paediatricians in treating children with behavioural and emotional disorders.[1] Studies of case mix in general paediatric clinics show that 3-4% of new referrals are for behaviour disorders.[2,3] In one study nearly 12% of all referrals were from clinical medical officers, but none referred a child with a behaviour disorder.[2] This finding is significant in the current climate, when general practitioners are replacing clinical medical officers in child health surveillance programmes.

A one year audit of behaviour disorders that I saw in a community "patch" in south Sefton, Merseyside, yielded the following data. Twenty-three children were referred to me with behaviour and conduct disorders (excluding enuresis) from a total child population of 5300 (4·3 referrals per thousand). Referrals included seven from health visitors or school nurses, six direct from schools, four from educational psychologists, three during Education Act assessments, two each from general practitioners and education welfare officers, and one each from a parent and a hospital paediatrician. Only one of these children was referred on to a child psychiatrist and none to a general paediatrician. Six children were, however, referred to a community clinical nurse specialist in behaviour disorders, and one to the educational psychologist. Three others, whose deteriorating behaviour was related to developmental disorders, were referred to the child development centre. Follow up showed that, although seven children defaulted, 12 had improved and another two needed no intervention after the initial consultation.

Many clinical medical officers currently feel threatened by the changes occurring in community services. Their considerable expertise in both child development and behaviour management, together with their extensive networking with other agencies, places them in an ideal position to provide a secondary referral service to general practitioners for children with behaviour disorders. This would release more specialist child psychiatry time for those with more intractable or complex disorders, which should in turn improve the quality of service for both groups of children. Those with simple problems get a local, easily accessible service and those in most need get the specialist attention they require.

CLIONA NI BHROLCHAIN

Northampton General Hospital,
Northampton NN1 5BD

1 Black D. Mental health services for children. *BMJ* 1992;**305**: 971-2. (24 October.)
2 Ni Bhrolchain CM. A district survey of paediatric outpatient referrals. *Public Health* (in press).
3 MacFaul R, Long R. Paediatric outpatient utilisation in a district general hospital. *Arch Dis Child* 1992;**67**:1068-72.

Mouse model for cystic fibrosis research

EDITOR,—Steve Connor's report on the first genetically engineered cystic fibrosis "mouse" which apparently "died soon after birth" and "was not considered to be of any practical use" is inaccurate.[1] In fact the "mouse" represents an expanding population of breeding heterozygote rodents carrying cystic fibrosis whose homozygote offspring have been and will continue to be studied enthusiastically in laboratories across North America. These mice show the defect in ion transport thought to be central to the pathogenesis of pulmonary cystic fibrosis (chlorine ion conductance activated by cyclic AMP) and, unlike the more recently reported mouse in Edinburgh, a proportion of the American mice show a clinical syndrome similar to meconium ileus seen in infants with cystic fibrosis.[2,3]

PIERRE M BARKER

Department of Pediatrics,
University of North Carolina,
Chapel Hill,
NC 27599-7220,
USA

1 Connor S. Mouse model for cystic fibrosis research. *BMJ* 1992;**305**:734. (29 September.)
2 Snouwaert JN, Brigman KK, Latour AM, Malouf NM, Boucher RC, Smithies O, *et al.* An animal model for cystic fibrosis made by gene targeting. *Science* 1992;**257**:1083-8.
3 Clarke LL, Grubb BR, Gabriel SE, Smithies O, Koller BH, Boucher RC. Defective epithelial chloride transport in a gene-targeted mouse model of cystic fibrosis. *Science* 1992;**257**: 1125-7.