Letters to the Editor

Postneonatal mortality

SIR—The article “Why did postperinatal mortality rates fall in the 1970s?”, 1986, **40**, 228–31, by Sunderland, Gardner, and Gordon, is an important contribution. The strong relation between the fall in the birth rate and the death rate from infections during the postperinatal period confirms the clinical impressions of many doctors. A similar analysis of postneonatal mortality rates and birth rates during the period 1968–82 in the City of Nottingham shows a statistically significant correlation ($p < 0.05$) between the two measurements.¹

This was the period when discussion about family planning became much more open, culminating in the acceptance of responsibility for family planning services by the NHS. It seems reasonable to assume that a smaller number of more planned children were better looked after, especially by their parents, and also by health professionals. Although many factors have played a part in the fall in mortality in both cities, it seems that some factors have been overemphasised, for example, the impact of birth scoring systems, whereas the real hero(in)es may well have been the unsung workers of the family planning service.

The article goes on to describe the need for an objective method of identifying deprived, high risk children and communities, and states that “statistical scoring systems provide that objectivity”. This is not our experience in Nottingham. We began a birth scoring system in January 1978,² and discontinued it, after rigorous evaluation,³ in March 1985. As part of this exercise we analysed carefully all the papers published about the Sheffield birth scoring system, plus routine OPCS data from that city. We shared the scepticism expressed when the results of this scheme were presented at a symposium of the Royal Statistical Society.⁴

The problem with birth scoring systems is that they explain only a low percentage of the variance, typically around 20–25%. The commonly used risk factors are necessarily very crude and often lack plausibility, for example “short second stage of labour”, or are far-fetched, “blood group of mother”. The more credible ones, low birthweight, will be taken into account by any competent practitioner as part of normal clinical practice.

It is important that the debate about the best allocation of resources is not allowed to become a question of “birth scoring systems or nothing”. There are other alternatives, for example, mapping zones of low birthweight in a given health district. A recent exercise by the Nottinghamshire County Council⁵

showed large variations in this measurement in different zones of the county, and a strong correlation between low birthweight and indicators of primary poverty such as unemployment, single parenthood, and being in receipt of free school meals.

Such data are very useful in planning services and delivering care to individuals. They are much easier to collect than bothering with birth scoring systems. They also suggest the possibility of strategies aimed at the primary prevention of low birthweight, by various types of anti-poverty policy. In the long run, these may well be the most effective way of reducing mortality further.

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Seasonal variation in birthdates of men with testicular cancer

SIR—Knox and Cummins reported in a recent issue of the *Journal* (39: 237–43, 1985) data which, in their opinion, provided “strong evidence of a temporal cycle” in the birth dates of men with testicular cancer in Britain. This cycle had a four-month period and was interpreted to be significantly different from an expected distribution obtained from national birth data for the year 1950 after correction for secular trend in natality.

This paper prompts us to report on an analysis we conducted on the month of birth of the Hawaii-born testicular cancer cases reported between 1960 and 1983 to our population-based tumour registry. These data are presented in the figure (identified as cases) and also suggest a seasonal pattern but with a six-month cycle instead of the four-month cycle seen

by Knox and Cummins. While we agree with these authors that cyclical patterns are difficult to interpret, we do not think that their use of birth data for a single calendar year constitutes an appropriate comparison for the cases (who were born over a 30 or 40-year period), since the corresponding distribution in the general population may very well have changed during this interval. For example, modern forms of birth control have given parents greater control over the time of conception, so that birth patterns in more recent years have been influenced by such various factors as vacation, climate, etc. Therefore, we chose a case-control approach, as a preferred alternative, to assess the significance of the apparent seasonal pattern in our data.

The 86 Hawaii-born testicular cancer cases were individually matched on sex, race, and birth-year (± 2 years) to four controls who had been randomly selected from the population for a 1975–80 survey.¹ The distributions of the cases and controls by month of birth are reasonably similar (figure) and the chi-square test with 11 degrees of freedom is not significant ($p=0.83$). Thus we are unable to confirm the existence of a seasonal pattern for the birth dates of men with testicular cancer in Hawaii.

The study by Knox and Cummins was an attempt to explore the possibility that an infectious or iatrogenic exposure might explain the recent increase in incidence of testicular cancer. Our analysis, on the other hand, was prompted by the possibility that the seasonal pattern reported by Czeizel *et al.*² for births with undescended testes (increase during March–May and decrease during August–December) might also be observed for patients with testicular cancer, since the two conditions have been strongly associated in epidemiological studies.^{3,4} First trimester exposure to abnormally high levels of endogenous sex hormones has been implicated in the aetiology of both cryptorchidism and testicular cancer.⁵ Consistent with an hormonal aetiology, this seasonal birth pattern

might be explained by the effect in early pregnancy of seasonal changes in the mother's production of melatonin and/or pituitary gonadotropins related to the duration of daylight. Since Hawaii is located in the Tropics, the seasonal variations in duration of daylight are reduced; thus our results do not totally exclude the possibility that such an effect might be seen in testicular cancer cases. This interpretation should certainly be considered, along with a possible infectious aetiology, if cyclical birth patterns for testicular cancer are confirmed in more northern populations. The four-month cycle observed by Knox and Cummins in Britain would be too short to fit with the daylight hypothesis. However, these authors' findings cannot be properly interpreted without secular trend data on the monthly distribution of births in the British population.

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Parental occupations and cancer

SIR—The recent article by Arundel and Kinnier-Wilson¹ serves as a focus to highlight once again the problems of studying the potential relation of parental occupational exposures to childhood cancer. As they point out in their first sentence, little is known about the aetiology of cancer in children, despite the importance of cancer as a cause of morbidity and death in childhood. They also explain the rationale for examining the relation to parental occupation. However, they go on to state that the 14 reports they will review were conducted by a similar method and have only the "minor differences" of source of information, type of case, and categorisation of parental occupation. We believe that these are major

Distribution by month of birth of testicular cancer cases and controls, Hawaii, 1962–83.