
**Reply**

Lawson's letter makes a number of points concerning the importance of spatial autocorrelation in disease data with which I cannot agree. This is an area where generalisation may be dangerous, so I would certainly not wish to dispute the possibility that such autocorrelation may be important in some instances. But by the same token, I believe that there are others where it is not of major concern. I restrict my remarks to diseases for which cases may be taken for granted, independently of one another, conditionally on any underlying variation in risk; the appropriate underlying error model must then be the Poisson process, although we will not necessarily know the true risk at a given point.

In the first place, unrecognised spatial autocorrelation presumably has the effect – at least within the framework of frequentist inference and modelling – of leading to heterogeneity that might be recognised, for example, by a high deviation residual to a fitted model. However, in this respect this heterogeneity is like that resulting from any other factor that has not been properly taken into account in a given model. The crucial question is surely whether this over dispersion leads to misleading conclusions about the factors that have been fitted. Whether it does so will, broadly speaking, depend on whether the factors are themselves associated with whatever spatially varying factors are responsible for the over dispersion. If they are, then of course such factors may be regarded as confounding the effects we are interested in. However, we can never eliminate the possibility of confounding factors anyway, it seems to me to be of limited inferential value to know that such confounding – if it occurs – is mediated through an unidentified spatially varying variable. It is, of course, scientifically much more valuable to try and identify all the factors that matter.

Lawson claims that the heterogeneity induced by spatial autocorrelation is "particularly important in putative source applications". I believe this to be most unlikely. For here the contributions of cases to neighbouring intervals on the distance axis will come in large measure from small areas which are relatively far apart, so that the serial correlation on this axis should be expected to be substantially less than the spatial autocorrelation per unit distance prevailing in two dimensions.

Likewise, the claim that "Leukaemias are known to cluster... around putative sources (nuclear power stations)" is highly suspect. There is very little published evidence relating to adult leukaemias, while, for childhood leukemia in England and Wales, the evidence was recently reviewed in the *British Medical Journal* and found to be largely negative, with the exception of the data relating to Sellafield. The latter excess is extremely hard to interpret because of the small post hoc nature of the observation; numerous papers have addressed the possible aetiology, but it seems clear that a purely geographical explanation is far from convincing.

In fact, contrary to numerous claims in the literature, childhood leukaemia does not exhibit strong spatial auto-correlation. The residual deviance exhibited in my own contribution to the session addressed in Lawson's letter suggests that only little residual heterogeneity to explain, while methodologically sound and intelligible attempts to demonstrate a spatial explanation for any such heterogeneity have generally produced only equivocal results. For adults, there tends to be substantially more heterogeneity, but the small scale spatial component of this again appears to be very weak.

To assume that spatial autocorrelation is important and to build this assumption into a statistical analysis may at first sight appear to be a sensible defensive strategy. But there is a high price to pay for an unnecessarily elaborate model. The situation becomes more difficult and it is likely that estimates of the parameters of primary interest become more precise and stable as we attempt to gain more information from modest data sets. Also, the question of how to measure or "put the builder of a mean" (eg around a stack) scenarios discussed by Lawson (Diggie et al, unpublished data).

While recent methodological developments in complex statistical modelling open up exciting possibilities, it should not be forgotten that all models are wrong, and that parsimony is a sound guiding principle.

**The decline in sex ratios at birth, England and Wales, 1973–90**

Sin – Dickinson and Parker entitled the letter which was published in this journal, "Why is the sex ratio falling in England and Wales?". The short answer to this question is that the sex ratio is obviously falling. So what? The answer is that any of these explanations. It fell more or less continuously from 1973–90, but in the four succeeding years it rose; and in 1993 it reached the highest level since 1983. It is interesting, however, to consider the causes of the fall during the 1970s and 1980s. Dickinson and Parker 1 mention three possibilities – coital rate, pollution, and hormones – and I should like to comment on these.

**Coital rate**

The sex ratios of white births in the USA between 1915 and 1988 seemed to show unexplained oscillations of very roughly 20–30 years. In particular, they rose during the years 1965–75 and fell during the years 1975–88. In that paper, 1 cited evidence that age standardised reported marital coital rates increased by 22% between 1965 and 1975 and fell by 27% between 1975 and 1988. Moreover, I also reviewed there the hypothesis that parental coital rate (weakly) controls the offspring sex ratio. 2 The evidence seems strong so it appears that in the USA from 1965–88 variations in the sex ratio might be explained by secular variations in the coital rate.

The recent decline in the sex ratio in England and Wales parallels a decline in the USA across the same years. It is possible that the
cause is the same. The growing perception of the danger of AIDS has caused a reduction in the rate of change of sexual partners and hence a reduction in the coital rate. There is also the possibility that coital rates are subject to medium term oscillations synchronised with changes in the economy. Fashionably decreed hemline levels and accompanying measures of sexual permissiveness do not move at random but seem to be subject to changes of fashion which, plausibly, also influence coital rates.

Pollution

Dickinson and Parker1 also raise the possibility that pollution may be a cause of the decline in sex ratios. However, pollution might have opposing reproductive effects on men and women. There is good evidence that exposure to industrial hazards like heat1 and to some chemicals like lead,4 borates,5 and pesticides6 causes men to sire a disproportionate number of daughters. And there is evidence that some forms of disease (for example, non-Hodgkin's lymphoma7 and multiple sclerosis8 in men are associated with low offspring sex ratios. It seems plausible to suggest that all this variation is hormonally mediated. Low values of testosterone and/or high values of gonadotrophins are associated with many diseases in men9 and with the above mentioned deleterious industrial exposures.

Only limited data exist on the effects of hazardous occupational exposures and diseases to women on offspring sex ratios. The most suggestive line of evidence comes from the sex ratio of offspring of women with MS. This is high. Moreover the adrenal glands of MS patients are large.10 Stress produces adrenal enlargement in non-human primates which lowers testosterone in men and apparently raises it in women.11 It is therefore reasonable to propose that in this disease (and perhaps others) raised adrenal androgens occasion a rise in the sex ratio of offspring born to affected women.

The present line of reasoning suggests that the reproductive effects of disease and of hazardous occupational exposure are similar. If this is accepted, one might expect these exposures and diseases to have opposite effects on the offspring sex ratios of men and women. The upshot is that air pollution (if it affects the hypothalamus?) cannot be expected to reveal itself in a changed offspring sex ratio. Dickinson and Parker1 cite Williams et al12 (who reported a lowered sex ratio in association with pollution). But one might mention that Lloyd et al13 found raised sex ratios in association with pollution. Thus, though pollution might have caused the decline in sex ratios, the hypothesis that it actually did so would only gain plausibility if it were backed by evidence of a particular pollutant which increased between 1973 and 1990, and decreased thereafter.

Hormones

The question nevertheless arises whether the decline in sex ratios in England and Wales from 1973–90 reflected some sort of increasing hazardous environmental exposure to men. In particular, is it to be associated with the recent suggestion relating diminished sperm counts to environmental oestrogen exposure?14 If the USA is anything to go by, this seems not to be the case. There (where the data on secular movements in sperm counts are more abundant than in the UK) sperm counts were apparently declining during the 1970s and possibly stable during the 1980s,15 in contrast to the movements of the sex ratio and coital rates described above.16

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Corrigenda

Society for Social Medicine annual meeting 1996 (vol 50:580–600) — a paper by A Grey, N Fulop, and I Allen entitled, 'Alternatives to academic admission: the national picture', was withdrawn from the meeting at the last minute. As the journal had already gone to press, the abstract still appears on page 586 of the October issue.

Estimating the prevalence of drug misuse in Dundee, Scotland: an application of capture-recapture methods by G Hay and N McKeganey (vol 50:469–73) — there is an error in table 4 on page 471; row 2 columns 2 should be read ISD and DPC and row 3 column 2 should be read ISD and POL.

NOTICES

The 1997 World Congress of the World Federation for Mental Health, 6–11 July 1997, Lahti and Helsinki, Finland. For further information contact: The Secretariat, KaKo Services, PO Box 762, FIN-00101 Helsinki, Finland. Fax: +358 9 492 810. Email: kako-ar@cc.helsinki.fi.


Conference on Cities and Addiction: (balancing) the health and public order. 21–23 April 1997, Conference centre De Dolen, Rotterdam, The Netherlands. Scientific Secretariat: GGFD, Professor HFL Garretsen, PO Box 70032, Schiedamsedijk 95, 3000 LP Rotterdam, The Netherlands; tel: +31 (0) 10 4433 96 20; fax: +31 (0) 10 4433 94 93. Conference Secretariat: Van Namen & Westerkamp, Congress Organisers, PO Box 1558, 6501 BN Nijmegen, The Netherlands; Tel: +31 (0) 24 323 44 71; fax: +31 (0) 24 360 11 59.

Sixth Annual British Epidermol-Epidemiology Society Workshop, 17 January 1997, at the University of Nottingham, England. For further information, contact: Ms Melanie Bowesman, Secretary to Dr Helen Williams, Department of Dermatology, C Floor, South Block, Queen's Medical Centre, Nottingham NG7 2UH, UK; Tel: 0115 924 9924 ext 44539; fax: 0115 970 9003.

Sixth International Symposium of the International Section of the ISSA for the Prevention of Occupational Risks in the Iron and Metal Industry, 20–22 October 1997, Barcelona. For further information, contact: Secretariat of the ISSA Section "Metal", c/o Kongresbüro, Allgemeine Unfallversicherungsanstalt, Adalbert-Süßer-Strasse 65, A-1200 Vienna, Austria; Tel: +43 1 33111 537; fax: +43 1 33111 469.

BOOK REVIEWS


Evaluation of cancer screening opens with a discussion of the broad principles of screening. It then looks in turn (possibly intentionally in descending order of effectiveness?) at screening for cancers of the cervix, breast, colon and rectum, melanoma, ovary, prostate, and "other"—lung, stomach, oral, and neuroblastoma. For each site, there are sections on the epidemiology, aetiology, screening test(s), effectiveness (with reviews of published trials where appropriate), acceptability, and conclusions. Included are movements on both the economic, and the relatively neglected, psychological aspects.
The decline in sex ratios at birth, England and Wales, 1973-90.

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*J Epidemiol Community Health* 1996 50: 690-691
doi: 10.1136/jech.50.6.690

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