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 movements of the economy. Fashionably decreed hemline levels and accompanying measures of sexual permissiveness do not move at random but seem to be subject to forms of homeostatic control which, plausibly, also influence coital rates.

Pollution

Dickinson and Parker[1] 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 heat[3] and to some chemicals like lead,[4] borates,[5] and pesticides[6] causes men to sire a disproportionate number of daughters. And there is evidence that some forms of disease (for example, non-Hodgkin's lymphoma[7] and multiple sclerosis[8] 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 men[9] 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.[8] Moreover the adrenal glands of MS patients are large.[10] Stress produces adrenocorticotrophic hormone 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 industrial 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 sexes equally) cannot be expected to reveal itself in a changed offspring sex ratio. Dickinson and Parker[1] cite Williams *et al* (who reported a lowered sex ratio in association with pollution).[12] But one might mention that Lloyd *et al*[13] 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 16] in contrast to the movements of the sex ratio and coital rates described above.

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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 Congress Services, PO Box 762, FIN-00101 Helsinki, Finland. Fax: +358 9 492 810. Email: kako-ar@cc.helsinki.fi.

European Conference on Costs and Benefits of Occupational Safety and Health 1997, 28, 29, 30 May 1997, The Hague, The Netherlands. For further information, contact: Conference Secretariat: European Conference on Costs and Benefits of Occupational Safety and Health 1997, c/o Holland Organizing Centre, Parkstraat 29, NL-2514 JD The Hague, The Netherlands. Tel: +31 70 365 7850. Fax: +31 70 364 5748. Email: Conference97@hoc.nl.

Conference on Cities and Addiction: (balancing) public health and public order, 21–23 April 1997, Conference centre De Doelen, Rotterdam, The Netherlands. Scientific Secretariat: GGD, Professor HFL Garretsen, PO Box 70032, Schiedamsedijk 95, 3000 LP Rotterdam, The Netherlands; tel: +31 (0) 10 433 96 20; fax: +31 (0) 10 433 94 93. Conference Secretariat: Van Namen & Westerlaken, Congress Organization Services, 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 Epidermo-Epidemiology Society Workshop, 17 January 1997, at the University of Nottingham, England. For further information, contact: Ms Melanie Bowesman, Secretary to Dr Hywel 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 Kongressbüro, Allgemeine Unfallversicherungsanstalt, Adalbert-Stifter-Strasse 65, A-1200 Vienna, Austria; Tel: +43 1 33111 537; fax: +43 1 33111 469.

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 acute 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 column 2 should read ISD and DPC and row 3 column 2 should read ISD and POL.

BOOK REVIEWS

Evaluation of cancer screening. J Chamberlain and S Moss (eds). (Pp 192; dm54.00) Berlin: Springer Verlag, 1996. ISBN 3-540-19957-8.

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. Finally, there are chapters on both the economic, and the relatively neglected, psychological aspects.