Letters to the Editor

Residence in the London area and sperm density

SIR—The disturbing trend of decreasing sperm count and seminal volume over the past 50 years,[1] and the concomitant increase in cryptorchidism and testicular cancer,[2,3] have been attributed to oestrogenic environmental pollution.[4]

We found adverse changes in sperm function in partners of 260 infertile women who received gonadotropin therapy between 1984 and 1989 compared with that recorded between 1978 and 1983. Mean sperm density fell from 101 to 96 million per mL, the proportion of men with abnormal sperm motility (<50%) increased from 20·7 to 34·4%, and those with abnormal sperm morphology (>50%) rose from 1 to 12·3%.[5] There had been no changes in the pattern of referral to our clinic, in the mean age, clinical or socio-economic characteristics of the couples in the two cohorts, or in the laboratory techniques over this period.

To investigate possible environmental influences, we have reviewed our data with reference to the water supply of the area where the couples lived. The Royal Free Hospital is situated within the Thames Water supply area (TWA)[6] and, since more of our patients live within this area than in any other water authority area, we compared sperm data from partners of women who received gonadotropin therapy and lived within the TWA with data from partners of women living outside this area. We excluded those from outside the UK, those with temporary addresses, and those living on the border of the TWA. This left 183 couples (group A), of whom 104 were resident outside the TWA (subgroup B) whereas 79 resided within the area (subgroup C). Since the variables analysed—sperm density, sperm motility, percentage of abnormal spermatozoa, and motile sperm density (density × motility per 100)—were normally distributed, parametric tests (one-way analysis of variance and *t* test) were applied.

As shown in the table, the mean (SE) percentage of abnormal forms increased in the second 6-year period (1984–1989) in all subjects irrespective of where they lived (group A, from 19 [1·1] to 31 [1·8]%, p<0·0005; group B, from 18 [1·3] to 32 [2·5]%, p<0·0005; and group C, from 19 [1·7] to 30 [2·4]%, p<0·005). In contrast, sperm density and motile sperm density fell significantly only in those living within the TWA (group C), from 105 (9·7) to 76 (7·5) × 10^6 per mL (p<0·05), and from 61·7 (6·5) to 40·8 (4·9) × 10^6 per mL (p<0·05), respectively, whereas in those living outside the TWA (group B) sperm density and motile sperm density remained unchanged. Similarly, comparison of the changes in mean sperm density and in motile sperm density with time between the two groups (B and C) showed significant (p<0·05) differences.

	Group A		Group B		Group C	
	1978–83	1984–89	1978–83	1984–89	1978–83	1984–89
No of couples	80	103	46	58	34	45
Sperm density (million per mL)	101 (6·1)	95 (6·9)	99 (8·0)	110 (10·5)	105 (9·7)	76 (7·5)*
Sperm motility (%)	56 (1·5)	54 (1·8)	55 (2·4)	55 (2·3)	57 (1·6)	52 (2·9)
Sperm morphology (% abnormal)	19 (1·1)	31 (1·8)‡	18 (1·3)	32 (2·5)‡	19 (1·7)	30 (2·4)†
Motile sperm density (×10^4/mL)	58·6 (4·1)	52·9 (4·6)	56·3 (5·3)	62·3 (7·0)	61·7 (6·5)	40·8 (4·9)*

*p<0·05, †p<0·005, ‡p<0·0005; p value for difference from previous cohort.

Table: **Comparison of mean (SE) semen quality between two 6-year cohorts in partners of infertile women according to place of residence**

These data relate solely to the partners of women treated for anovulatory infertility and so are not necessarily representative of the general population. Nevertheless, the evidence worldwide of falling sperm counts[1] suggests that the phenomena we have observed are only part of a general adverse change in semen quality. We have no knowledge of the precise effect changes in semen quality may exert on male fertility. It is, however, of interest that polychlorinated biphenyl congeners were found in semen and that their concentrations correlated inversely with sperm motility.[7] In addition, environmental factors affecting male fertility could also influence ovarian activity and hence female fertility.

We emphasise that we do not assume that the only environmental factor peculiar to those living within the TWA was their water supply. However, despite exhaustive inquiries, we were unable to ascertain whether there had been any change in TWA testing of water supplies at source and delivery after 1983. Furthermore, some general environmental factor appears to be involved in that the increase in abnormal sperm morphology was not related to geographical area. Analysis of further years will show whether this adverse trend continues, but if aspects of altered semen function can be linked to a specific environmental influence, it should be susceptible to correction.

J Ginsburg, S Okolo, G Prelevic, P Hardiman
Division of Clinical Pharmacology, Royal Free Hospital School of Medicine, London NW3 2QG, UK

1 Carlsen E, Giwercman A, Keiding N, Skakkebaek NE. Evidence for decreasing quality of semen during past 50 years. *BMJ* 1992; **305:** 609–13.
2 Giwercman A, Skakkebaek NE. The human testis: an organ at risk? *Int J Androl* 1992; **15:** 373–75.
3 Boyle P, Kaye SN, Robertson AG. Changes in testicular cancer in Scotland. *Eur J Cancer Clin Oncol* 1987; **23:** 827–30.
4 Sharpe RM, Skakkebaek NE. Are oestrogens involved in falling sperm counts and disorders of the male reproductive tract? *Lancet* 1993; **341:** 1392–95.
5 Ginsburg J, Hardiman P. Ovulation induction with human menopausal gonadotrophins: a changing scene. *Gynecol Endocrinol* 1991; **5:** 57–78.
6 Map of water supply zones, Issue 2. Reading Bridge House, Reading: Thames Water Utilities Limited, Jan, 1993.
7 Bush B, Bennett AH, Snow JT. Polychlorobiphenyl congeners, p,p'-DDE and sperm functions in humans. *Arch Environ Contam Toxicol* 1986; **15:** 333–41.

Dental enamel defects in coeliac disease

SIR—Coeliac disease is a condition in which there is an abnormal jejunal mucosa that improves morphologically when the patient is treated with a gluten-free diet. The incidence in the UK has been estimated to be 1 in 2000 but screening studies suggest that the incidence may be as high as 1 in 266 because many patients with mild or no symptoms remain undiagnosed.[1] These patients should be identified and treated to correct nutritional deficiencies and reduce the increased risk of