Blood lead concentrations ($\mu\text{mol/l}$) in young children and their mothers in dwellings on the roadside and in dwellings in cul-de-sacs, etc. Numbers of subjects in brackets

	Roadside	Cul-de-sacs
Children	0.80 SD 0.40 (No = 42)	0.71 SD 0.18 (No = 30)
Mothers	0.43 SD 0.12 (No = 42)	0.43 SD 0.14 (No = 30)

Conversion: SI to traditional units—1 $\mu\text{mol/l} \approx 21 \mu\text{g/100 ml}$.

of one very high concentration (2.8 $\mu\text{mol/l}$ (43 $\mu\text{g/100 ml}$) which, on checking with a further blood sample was 0.62 $\mu\text{mol/l}$ (13 $\mu\text{g/100 ml}$) gives a mean concentration of 0.75 $\mu\text{mol/l}$ (15 $\mu\text{g/100 ml}$) SD 0.25 $\mu\text{mol/l}$ (5 $\mu\text{g/100 ml}$).

It is of interest to compare these results with those obtained from a random sample of children of the same ages, and their mothers, seen in a remote village in North Wales with no industry and only a light traffic flow. These children were seen as part of another study, but the sampling techniques and analytical procedures were identical. In this village the mean concentration in 29 children was 0.85 $\mu\text{mol/l}$ (18 $\mu\text{g/100 ml}$) SD 0.43 $\mu\text{mol/l}$ (9 $\mu\text{g/100 ml}$), and in their mothers 0.37 $\mu\text{mol/l}$ (8 $\mu\text{g/100 ml}$) SD 0.09 $\mu\text{mol/l}$ (2 $\mu\text{g/100 ml}$).

These data give no evidence that a residence in close proximity to heavy traffic is a serious hazard to children in terms of blood lead.

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SIR,—Your leading article on lead in petrol (20 February, p 529) deserves muted applause for drawing attention to the need for scrupulous examination of the evidence on which important decisions of public policy are based. It is clearly true that studies showing an association between body lead burden in children and indices of intelligence do not prove, according to rigorous criteria, that the association is a causal one, and, indeed, the findings may be explained either by lower-than-average intelligence being a predisposing factor in increased lead intake or, alternatively, by a common association with, for instance, social class, for which high blood lead might be only a marker.

At the same time it should be recognised that the only type of study which would in proper scientific fashion distinguish between these alternatives would be one in which a control group could be compared with a test group deliberately exposed to a higher or to a lower lead intake, but carefully standardised to exclude other confusing variables influencing development. Those who reject the idea of administrative action to reduce the present dispersion of lead into the environment on the basis of the evidence, so far available must accept that, by implication, they are advocating a prolonged controlled trial of the type outlined above. To wait for more reliable scientific evidence might, otherwise, be to wait for ever, unless, of course, animal work is accepted as having some bearing on the human situation. It is a matter for balanced judgment how extensive the circumstantial evidence need be

before the British Government should fall into line with those governments which already require all new cars to run on lead-free petrol.

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SIR,—If I was trying to establish the non-toxicity of lead, I would hesitate before mentioning either of the two articles of which Claire Ernhart is coauthor.^{1,2} Both of these studies were carried out on the same group of children, and both studies showed a significant correlation between elevated blood lead concentrations and intellectual deficit. It is true that in her later study Dr Ernhart interprets the data somewhat differently and claims that most of the lead-related deficits can be explained away in terms of parental IQ. This might be an important observation were it not that in her original study Ernhart used the disassociation between parental and child IQ as evidence of lead's toxic action: in the low lead group, the child/parent IQ correlation was as expected ($r=0.52$), whereas in the high lead group the correlation was very weak ($r=0.10$). Dr Ernhart makes no attempt to replicate this particular analysis in her later study.

The simplest method of rationalising Ernhart's conflicting conclusions is to suggest that the initial study influenced the outcome of the later study, in that the more intelligent parents subsequently took precautionary measures to protect their offspring from excessive lead exposure. Dr Ernhart admits that this is a possibility which she never considered (personal communication).

In any event it is not important. Those who dispute the evidence against lead are continually reminding me that epidemiological studies do not provide proof of cause and effect. Why then does the *BMJ* quote a study showing a correlation between lead and intellectual deficit as evidence of lead's non-toxicity (20 March, p 902)?

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¹ Perino J, Ernhart CB. *Journal of Learning Disabilities* 1974;7:26-30.

² Ernhart CB, Landa B, Schell N. *Paediatrics* 1981;67:911-9.

* * * We do not assert that lead is not toxic. Our leading article stated that "there is no convincing evidence" linking blood lead concentrations in children below 1.7 $\mu\text{mol/l}$ (35 $\mu\text{g/100 ml}$) with effects on intelligence and behaviour. Professor Needleman and Dr Russell Jones quote some recent studies suggesting toxic effects at lower doses, but these do not, in our view, represent "substantial new data." Indeed, at present the consensus view on both sides of the Atlantic remains that raised blood lead concentrations in children need cause medical concern only when above 1.5-1.7 $\mu\text{mol/l}$ (30-35 $\mu\text{g/100 ml}$)—see, for example, the recent report¹ on the response by the public health authorities in Boston to environmental contamination from lead paint flaking from the Mystic River bridge.—Ed, *BMJ*.

¹ Landrigan PJ, Baker EL Jr, Himmelstein JS, Stein GF, Weddig JP, Straub WE. *N Engl J Med* 1982;306:673-6.

Oestrogen receptors and survival in early breast cancer

SIR,—Recent correspondence (20 February, p 597) has again focused attention on the value of oestrogen receptor state as a prognostic factor in patients with breast cancer. This prognostic effect would appear to be independent of that of axillary node involvement,¹ and several studies which have examined the relationship between receptor status and node status have failed to demonstrate an association.¹⁻⁷

In a study of 281 patients, we found that 57% of 67 women with receptor-negative tumours (<5 fmol/mg protein) had involved axillary nodes, whereas only 46% of the 216 patients with receptor-positive tumours had nodal involvement. Although this difference is not statistically significant, it prompted us to re-examine the previous reports.

It was notable that although the seven studies comparing oestrogen receptor status with node status could demonstrate no significant relationship,¹⁻⁷ the overall trend did suggest a tendency for receptor-negative tumours to be associated with nodal involvement. We therefore combined these data with the Edinburgh figures and found the association to be statistically significant (table).

Relationship between oestrogen receptor state and node state in 1994 patients with breast cancer. (Data pooled from Edinburgh figures and references 1-7)

Oestrogen receptor status	Axillary lymph node status	
	Positive	Negative
Positive	618 (53%)	542 (47%)
Negative	396 (47%)	438 (53%)

$\chi^2 = 6.51$ ($p < 0.02$).

This finding does not imply that the prognostic effect of oestrogen receptor level relies on node state, but it does show that the independence of these two factors cannot be regarded as being absolute.

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¹ Croton R, Cooke T, Holt S, George WD, Nicholson R, Griffiths K. *Br Med J* 1981;283:1289-91.

² Knight WA, Livingston RB, Gregory EJ, McGuire WL. *Cancer Res* 1977;37:4669-71.

³ Lippmann ME, Allegra JC. *Cancer* 1980;46:2829-34.

⁴ Furmanski P, Saunders DE, Brooks SC, Rich MA. *Cancer* 1980;46:2794-6.

⁵ Blamey RW, Bishop HM, Blake IRS, et al. *Cancer* 1980;46:2765-9.

⁶ Saman NA, Buzdar AV, Aldinger KA, et al. *Cancer* 1981;47:554-60.

⁷ Howat JMT, Barnes DM. *Lancet* 1981;i:1317.

Mapping cancer mortality

SIR,—The use by Dr M J Gardner and others of variations in cancer mortality among local authority areas as a pointer towards possible environmental causes is important and interesting research (13 March, p 784). The use, however, of death records from the Office of Population Censuses and Surveys can be misleading.

Thus one of the author's groups of malignancies, cancer of the nose and accessory sinuses (ICD 160), is not accorded a separate heading in the death returns for West Cumbria. I presume this is because of the small absolute