Environmental lead and children's intelligence

Obvious hypothesis is ignored

EDITOR,—Stuart J Pocock has always been profoundly sceptical of a causal relation between lead and IQ.[1] Accordingly, the recent paper by him and colleagues deserves a cautious welcome, if only because the data show conclusively that a doubling of body lead from 10 to 20 μg/dl of blood (0·48 to 0·97 μmol/l) or 5 to 10 μg/g of tooth is associated with an IQ deficit of 1-2 points after allowance for confounding variables.[2] Unfortunately, the "standards of objectivity and critical appraisal" that the authors find so lacking in others are crucially absent from the discussion of their paper, in which, seemingly, any hypothesis is entertained other than the obvious—namely, that lead reduces IQ.

Although the authors concede that this is plausible, they dismiss neuropsychological studies in animals as providing "only indirect support" but fail to mention the reproducibility of such data.[3] Nor do they consider studies in humans that have shown biochemical effects at blood lead concentrations well within the range studied epidemiologically—studies that include the effects of lead on enzyme systems central to neurological function.[4]

It needs to be remembered that the natural (that is, pre-technological) blood lead concentration in children has been calculated at 0·016 μg/dl (0·0008 μmol/l)[5] and that the concentrations typically found in modern children are greater by three orders of magnitude. Furthermore, it requires only a quadrupling of the concentration to bring children into the range associated with neuropathy, coma, and ultimately death. For no other neurotoxin is there such a narrow gap between what is typical and what is toxic, so no one should be surprised when small deficits in IQ are shown by epidemiological studies.

It is unfortunate that these studies have to rely on tooth lead content or blood lead concentration as an indirect measure of exposure to lead on the far side of the blood-brain barrier. Perhaps Pocock and colleagues should turn their attention to the extent to which epidemiological studies underestimate the true impact of lead on children's intelligence.

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Cleveland study hypothesis was not confirmed

EDITOR,—Stuart J Pocock and colleagues' objective review of the epidemiological evidence with regard to environmental lead and children's intelligence provides a carefully conducted set of analyses of a complex and confusing set of studies.[1] It contrasts sharply with reviews recently published in the United States.

The authors suggest that maternal alcohol dependence contributed to the effectiveness of adjustment for covariates in the prediction of IQ in the Cleveland study, of which I was principal investigator.[2] I wish that it was so. Our hypothesis that fetal exposure to alcohol has a direct effect on IQ and an effect interacting with that of lead was not confirmed. Our otherwise powerful covariate set controlled for childhood illness, including otitis media, and race, which had little or no variance in other studies.

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Taiwan results are not included

EDITOR,—Stuart J Pocock and colleagues' systematic review of recent studies of environmental lead and children's intelligence[1] does not include a cross sectional study from Taiwan.[2] This may be because the study's outcome measure was the score in Raven's coloured progressive matrices test rather than the score on the more commonly used Wechsler intelligence scale for children. As an author of the study in Taiwan I have reanalysed our data, using an appropriate IQ score for each value in the coloured progressive matrices test.

The coloured progressive matrices test is scored from 0 to 36, is free of obvious cultural content, has no time limit, involves visual-spatial reasoning, and correlates strongly with the score on the Wechsler intelligence scale for children and other measurements of IQ. I translated each child's score in the coloured progressive matrices test to the equivalent IQ score to obtain figures directly comparable with those of Pocock and colleagues. I used the Standford-Binet distribution of IQ for schoolchildren in Taipei City to assign each score an IQ score.

The data from the Taiwanese study can be presented in the form used in Pocock and colleagues' table III. The study drew children from urban primary schools and two schools near lead smelters. The group with complete data comprised 458 children aged 6-8 with a mean IQ score of 106. The blood lead concentration of 60 children from the schools near the smelters averaged 13·0 μg/dl (0·63 μmol/l), and their tooth lead content averaged 6·2 μg/g. From this we estimated a mean blood lead concentration of 9·0 μg/dl (0·43 μmol/l) for the entire cohort, whose tooth lead averaged 4·3 μg/g. The significant covariates were parents' education, sex, and grade at school. Forty potential covariates were considered. Adding additional variables did not decrease the lead coefficient. The data were not log transformed.

When the data were analysed as in Pocock and colleagues' table IV, an increase in tooth lead from 5 to 10 μg/g was associated with a change in IQ of −3·6 (SE 1·0) (unadjusted) and −2·2 (0·9) (adjusted) (P=0·02). The population in Taiwan is generally well nourished, and use of alcohol, tobacco, or illicit drugs while pregnant is virtually zero.

These Taiwanese data are consistent with the overall pattern described by Pocock and colleagues. Adding them to the review increases the total number of children included, improving the statistical resolution slightly. More importantly, however, these data extend downward the range of lead concentrations for which an effect is seen. If a threshold for lead's effects on intelligence is to be found it is at yet lower concentrations.

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Hair lead studies were excluded

EDITOR,—In their meta-analysis to measure the magnitude of the relation between IQ in children aged ⩾5 and the body burden of lead Stuart J Pocock and colleagues excluded studies that used hair lead because "it is recognised as an unreliable measure of body burden."[1] The idea that hair is not a reliable indicator comes mainly from the possibility of exogenous contamination of hair and the fact that most studies have shown no or weak links between blood lead concentration and hair lead content. We showed that exogenous contamination of hair was not as high as expected when meticulous and thorough washing procedures were used.[2]

Hair and blood provide complementary but not exclusive information about body lead content. With regard to exposure during pregnancy, blood lead concentrations provide information on the degree of exposure currently and recently but not during earlier, possibly more vulnerable, periods of development. Analysis of hair is a potentially more attractive means of assessing long term exposure and seems useful in estimations of exposure in utero, when central nervous system cells are highly vulnerable. Lead is concentrated in head hair, which can be collected without injury and easily preserved and analysed. For measurement of past or current exposure, hair seems better than blood. Our estimation of intrauterine exposure to lead by analysis at birth of neonates' hair and their mothers' hair in a six year prospective study showed that such exposure had a significant negative correlation with the main scores of the McCarthy scales of children's abilities. This study was performed on 81 children, which is a smaller sample than that required in studies of blood.[3] The fact that a relation was found proves that a powerful and useful tool—hair—was used.

Another physiological argument supports the use of hair lead content. A study by our group assessing the influence of environmental lead on membrane ion transport in a French urban male population showed that blood lead concentration was related to cotransport of sodium and potassium ions and was not related to Na^+, K^+ ATPase activity while hair lead was related to Na^+, K^+ ATPase activity.[4] Because Na^+, K^+ ATPase is the main mechanism of the action potential in nervous system cells, we undertook a prospective study. The aim of this study was to evaluate whether the relation between exposure to lead in utero (as estimated from the blood concentration and hair lead content) and subsequent neuropsychological development (at 9 months and (measured with the McCarthy scales) 3 years) could be partly mediated by Na^+, K^+ ATPase activity. The preliminary results (unpublished data) of this study seem to agree with this hypothesis.

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Living close to industry

EDITOR,—Ian Harvey describes some of the difficulties of determining whether living close to industry harms people's health.[1] Scottish studies that colleagues and I carried out during the 1970s and 1980s used several techniques for sharpening the focus on this type of epidemiology of small areas.[2,3]

Firstly, when studying air pollution from local industries, beware overreliance on traditional fixed monitoring sites, which are usually too few and inappropriately located. Concentrations of air pollutants from a fume stack change dramatically within short distances, so that "precise" results from such monitoring sites can lead to misclassification of exposed and non-exposed populations; furthermore, such techniques rarely enable one industry's local pollution pattern to be distinguished from that of another. Keynes's dictum about economics—that it is better to be approximately right than precisely wrong—is also true in environmental epidemiology, and approximate patterns of pollution (notably of metals) can be obtained by use of low technology samplers, including soils and moss bags.[4] Such samplers are inexpensive, are independent of power supplies, are ignored by vandals, provide a detailed and extensive network of sampling sites where data on pollution are most relevant, can sample for long enough for seasonal variations to be discerned, and can assess past pollution patterns. Low technology samplers indicate the approximate locations of pollution hot spots (and hence the most exposed populations); thereafter, the precise details of toxicological relevance may be obtained from high technology equipment installed where most appropriate.

Next, beware the circular area around a source of pollution. This is much favoured by armchair epidemiologists to define the so called exposed population, but its simple geometry ignores fundamental realities determining exposure to pollution: the directions of winds associated with the pollution; local topography; the height of the fume stack; the consequent extent of the "umbrella effect"; and the pattern gleaned from observations of physicians, environmental health officers, residents, farmers, and veterinarians. Such information permits even the most funds starved epidemiologists to demarcate exposed neighbourhoods reasonably realistically and a priori.[5] This pilot epidemiology may persuade local authorities to fund follow up studies.

Thirdly, apply Hill's criteria for causation, but be aware that the realities on which these guidelines operate may need probing. For example, a factory that has operated for decades may change its processes or raw materials radically, thereby causing an unsuspected release of pollutants with new toxicological potentials.

Finally, public health practitioners should respond constructively to residents' concerns and to requests for advice or collaboration from environmental health departments. Too often have such opportunities for benefiting the public's health been missed.

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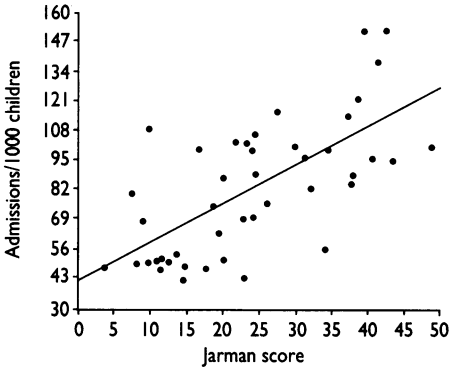
Monitoring effects of deprivation on health

EDITOR,—Recent reports have restated the link between socioeconomic deprivation and experience of poor health.[1,2] Children are sensitive indicators of deprivation, and their health experience reflects the health inequalities in the wider adult population.[3] In our district we have been looking at ways of monitoring the effects of deprivation on the community through the experiences of children, using data from the routine contract minimum dataset.

There were 4037 hospital admissions among 41 152 children (aged 0-15) resident in Kensington, Chelsea and Westminster Health Commissioning Agency from April 1993 to March 1994. The rate of admission was 98·1/1000 children, ranging from 68/1000 to 118/1000 in six localities. Further work to examine these differences looked at admission rates in children under 16 in each of 44 electoral wards within our district. The admission rates varied more than threefold, ranging from 43·5/1000 children to 153·4/1000. When mapped these variations seemed to be related to the variation in the Jarman score for each ward.[4] This was confirmed when the data were plotted and supported by a highly significant correlation coefficient (r=0·68, 95% confidence interval 0·48 to 0·81; figure).



Admission rates per 1000 children aged 0-15 plotted against Jarman scores for 44 electoral wards in London, April 1993 to March 1994

These results show the wide variations in admission rates in children and that these correlate closely with deprivation. This finding may be due to increased use of health care in deprived areas because of social factors or to actual increased morbidity in the child population. Regardless of which point of view is taken, children in deprived areas clearly receive hospital care more frequently than those in other areas, with the attendant disruption of family life and consumption of health resources.

Health authorities need to target deprived areas to examine these trends in more detail. The use of hospital discharge data linked to other sources of routine data presents an opportunity to develop routine ways of monitoring the effects of deprivation on morbidity and complements the use of mortality indicators for similar purposes.[5]

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Teenage pregnancy and deprivation

EDITOR,—Philip McLoone and F A Boddy highlight the differences in mortality experienced by residents of areas characterised by different degrees of deprivation.[1] Using the same classification as part of a needs assessment of teenage pregnancy (Scottish needs assessment programme), we have studied conception rates among teenagers and the outcome of these pregnancies in relation to deprivation.

The conception rate among teenagers in Scotland rose from 40/1000 teenagers in 1983 to 51/10000 in 1991, falling to 46/1000 in 1993. Data about teenage conceptions (births, miscarriages, and terminations) were obtained from the Scottish morbidity records (SMR2 (maternity hospital discharge records)). Anonymised information about terminations of pregnancy was also obtained from the morbidity records (SMR1) (general hospital discharge records)). The age of a young woman at conception was calculated by subtracting the estimated gestation at the end of pregnancy from her age at delivery and then adding two weeks. Where length of gestation was missing it was assumed to be 40 weeks for births and 12 weeks for pregnancies ending in miscarriage. The deprivation category was derived from the postcode with Carstairs and Morris's 1991 classification of deprivation for Scotland and was assigned to 98·7% of the teenage pregnancies. Population totals of teenage girls by age were taken from data in the 1991 census.

During the three years 1990-92, 33 275 teenage pregnancies were recorded. At all ages (13-19 years) the pregnancy rate increased with deprivation, with a fourfold to fivefold difference in the rate between the women living in deprivation category 1 (most affluent) and those in deprivation category 7 (most deprived) (table). The outcome of pregnancy also varied. The proportions of pregnancies that resulted in the birth of a baby were 36% (260/731) in women in deprivation category 1

Teenage pregnancy rates per 1000 teenagers by age and deprivation category, Scotland 1990-92

Deprivation category	Age (years)				
	13-15	16	17	18	19
1*	3	17	24	31	37
2	5	26	40	53	61
3	7	35	53	67	72
4	9	49	75	94	103
5	12	68	95	116	123
6	11	66	104	122	137
7†	15	88	137	172	170

*Most affluent. †Most deprived.