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My name is Donald A. McCaughran. I am a professor of biostatistics at the University of Washington. During the past four years I have been involved in research on the mathematical relationship of blood lead concentration to air lead concentration. This topic unfortunately is one of great controversy and confusion.

A large part of the problem stems from the difficulty in obtaining the appropriate kind of data to determine such a relationship. The other major problem is that the few mathematical relationships that have been proposed, Goldsmith and Hexter (1967) and Knelson, J. H., Johnson, R. J., Coulston, F., Goldberg, L. and Griffen, T. (1972) are biologically unreasonable and with parameters estimated from inappropriate data sets. Since the audience for these models is largely the medical profession whose members are not trained in mathematical modelling, the models have largely escaped the criticism which should have been brought against them. A formal review of these models is enclosed in the paper; Air lead-Blood lead Models by D. A. McCaughran to be published in the Proceedings of the Symposium on Heavy Metals in the Environment, Toronto, October, 1975.

These models have been badly misused by many groups, for example, the Staff report of the State of California Air Resources Board reproduces a table from Knelson et al (1972)

giving predicted increases in blood lead for representative exposures based on the Knelson et al (1972) model. The values in that table are merely extrapolations from an incorrect model based on reported controlled chamber air lead concentrations that are highly suspect. The table presents blood lead increases for varying days of exposure from 25 to 100. The model predicts the following values for blood lead at 10 ug/m^3 air lead:

	Days of Exposure				
Air lead	25	50	75	100	10000 (27.4 years)
10 ug/m^3	7.9	10.9	12.7	13.9	33.92

When extrapolated further 27.4 years the model predicts air increase of 33.92 although the data of the study showed an equilibrium level was reached after 150 days by many of the subjects. Add to this the average pre-exposure level of 18 ug/100 ml and the individuals blood lead will be 50 ug/100 ml and rising. Obviously this shows that this model, and its use have no scientific justification!

The second major criticism of these models is that they cannot be used to estimate blood lead concentrations in the absence of air lead. In fact the Goldsmith-Hexter model predicts zero blood lead in the absence of air lead and with the Knelson et al (1972) model blood lead increase goes to minus infinity as air lead goes to zero. Both models are incorrect since there is known to be lead entering the blood from the diet.

Differences in sources of lead other than air are known to play a major role in blood lead concentrations. When groups of people from areas of higher lead exposure are compared to groups from areas of lower air lead and found

to have slightly higher blood lead it is always blamed on air lead, but when groups exposed to the higher air leads have lower blood leads that information is ignored! As an example, Table 9 of Goldsmith, J.R., (1974)

	Mean Blood Lead	
	Away from Traffic	Near Traffic
San Diego	17.4	14.4
National City	11.0	16.0

Both sets of values are significant at the 0.06 significance level and National City at 0.05 level but only the National City result is considered significant! The proper interpretation of these values is that there are other sources of lead that are more important than air lead that confound the results of small sample epidemiological studies such as these.

The best epidemiological data collected to date has been those of Azar, A., Snee, R.D. and Habibi, K., (1975). They used personal air samples to get measurements of average air lead concentration. A sample of thirty Los Angeles cab drivers were exposed to mean air lead concentrations as high as 9.12 ug/m^3. They used the log-log model for statistical convenience. They found no statistically significant correlation between air lead and blood lead. The proper interpretation of these results is: that sources other than airborne lead cause such variability in blood lead values that the small increase in blood lead from airborne sources could not be detected with a sample size of 30. The other very important result in Azar et al (1975) is that different groups have different baseline blood lead concentrations (due to non-air lead sources). These different base levels of blood lead exhibited by different groups of people are very often incorrectly blamed on air lead levels (Goldsmith, 1974).

It is unsound experimental design to compare groups by their place of residence for the purpose of testing the hypothesis; increased air lead in respiratory air causes increases in blood lead concentrations. An experiment of this kind has the treatment effects confounded i.e. one cannot determine what factors in the place of residence causes differences in blood lead values because there is more than one contributing source of lead to the blood.

It is my opinion the airborne lead does contribute lead to the body (McCaughran, 1975). It is also my opinion that a very small amount of blood lead can be attributed to air lead. In fact, after a careful review of Table 2 Adjusted Particulate Lead Concentrations Monthly Averages at Selected Locations, Staff Report State of California Air Resources Board (1975) one can conclude that with the exception of the Lennox Location that a maximum concentration of 2 or 3 ug/100 ml could be contributed to the blood from the air. If people are in fact breathing the identical average lead concentration as reported at the Lennox Location the air lead contribution to their blood would be a maximum of 4 ug/100 ml.

One cannot escape the conclusion based on a thorough analyses of the air lead blood lead relationship that if the air lead concentrations were reduced to zero in California the reductions in blood lead concentrations would be less than several ug/100 ml and would require a very large sample of subjects to be able to statistically show the reduction. The small percentage of people in the 40-50 ug/100 ml range of blood lead concentration will certainly not change by the removal of all airborne lead.

The real question is: whether or not the small increase in body lead as measured by an elevated blood lead of a few ug/100 ml has any demonstrable detrimental effect on public health.

References

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