

Abstract

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Air Lead-Blood Lead Models

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Two air lead-blood lead models have been constructed:

1) a model to describe the increase in blood lead from an abrupt increase in air lead as experienced by persons in controlled environment experiments; 2) a model to describe the contribution to the blood of airborne lead in the environment.

I. Abrupt Increase Model

This model describes the average change in blood lead concentration following an abrupt change in the concentration of airborne lead. The average rate of change of blood lead is described by

$$\frac{du}{dt} = A - Bu$$

A is the average intake rate into the blood from all sources ($\mu\text{g}/100 \text{ ml}$)

Bu is the average removal rate from the blood from all sources ($\mu\text{g}/100 \text{ ml}$)

Solving, and reparametrizing, the model for the average blood lead concentration at time t is:

$$u(t) = \begin{cases} L_p & \text{for } t < t_0 \text{ (pre-exposure)} \\ L_E - (L_E - L_p) \exp\left(-\frac{A_E}{L_E}(t - t_0)\right) & \text{for } t_0 < t < t_f \text{ (during exposure)} \\ L_p + (L_E - L_p) \exp\left(-\frac{A_P}{L_P}(t - t_f)\right) (1 - \exp\left[-\frac{A_E}{L_E}(t_f - t_0)\right]) & \text{for } t > t_f \text{ (post exposure)} \end{cases}$$

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$L_E = \lim_{t \rightarrow \infty} u(t)$ is the average asymptotic blood level for the given exposure level ($\mu\text{g}/100 \text{ ml}$).

L_P is the average blood lead concentration prior to exposure to the new air lead concentration ($\mu\text{g}/100 \text{ ml}$).

A_P is the average intake rate pre- and post-exposure ($\mu\text{g}/100 \text{ ml/day}$).

A_E is the average intake rate during exposure ($\mu\text{g}/100 \text{ ml/day}$).

The data from Albany prisoner study of Coulston, et al², are used for parameter estimation. The model describes the data for individuals extremely well.

II. Blood Lead-Environmental Air Lead Model

The model describes the average stable blood lead concentration of persons exposed to constant levels of airborne lead. The model proposed for the rate of change in steady state blood lead concentration with changing air lead concentration is:

$$\frac{dL_B}{dL_A} = K(L_E - L_B) \quad \begin{array}{l} L_B = \text{blood lead concentration} \\ L_A = \text{air lead concentration} \end{array}$$

The assumptions are:

- 1) A maximum rate of intake through the lungs is approached as the air lead concentration increases, due to the finite internal surface area of the lungs.
- 2) At the maximum rate of intake the body is capable of removing lead from the blood at a similar rate, i.e., a maximum equilibrium blood lead concentration is reached (L_E).
- 3) When $L_A = 0 \rightarrow L_B = L_D$ the blood lead concentration due to dietary sources.

Solving the above equation results in

$$L_B = L_E - C_1 e^{-C_2 L_A} \quad \begin{array}{l} C_1, C_2 \text{ are parameters.} \\ L_E - C_1 = L_D \end{array}$$

Parameter estimates for men were derived for the Albany Study using the asymptomatic level (L_E) for each individual and from the average air lead and blood lead values for the women in the "7-City Study" of Tepper and Levin¹ to produce a model for U.S. women.

The model predicts the average blood lead concentration for U.S. women in several ambient air lead concentrations to be:

<u>Mean Air Lead ($\mu\text{g}/\text{m}^3$)</u>	<u>Blood Lead ($\mu\text{g}/100 \text{ ml}$)</u>
0	15.75
1	16.41
2	17.05
3	17.68
4	18.31
5	18.92

The Goldsmith-Hexter² model and the model of Knelson, et al,³ are reviewed; the assumptions in both models are considered unrealistic since both models are undefined at zero air lead concentration and both models imply that blood lead concentration has no upper bound.

²Coulston, F., Golberg, L., Griffin, T., and Russell, C. Albany Medical College. Final Report, June 1972, February 1973.

¹Tepper, L. B. and Levin, L. S. College of Medicine, University of Cincinnati. Final Report, 1972.

²Goldsmith, J. R. and Hexter, A. C. Science, 158:1967.

³Knelson, J. H., Johnson, R. J., Coulston, F., Golberg, L., and Griffin, T. Proceedings of the International Symposium on the Environmental Health Aspects of Lead. 1972.