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Influence of Social Factors on Lead Exposure and Child Development

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A brief overview of current views of child development is provided, with particular attention given to the role the child's physical and social environment plays in influencing the developmental process. Examples from the recent literature are used to illustrate how these factors can influence lead exposure and most importantly how they might interact with lead to ameliorate or exacerbate possible lead effects. An example is provided which demonstrates that failure to control adequately and to adjust the data statistically to correct for the influence of these factors can lead one erroneously to attribute cognitive and behavioral changes to lead. Finally, data from the Cincinnati Prospective Lead Study are presented to illustrate the application of structural equation modeling as a means for unraveling the complex web of sociodemographic, environmental and behavioral influences on childhood lead exposure. The latter analysis indicates that for children less than 24 months of age, lead-containing dust in the home and on the children's hands are important determinates of their blood lead levels. This relationship is influenced by the amount of maternal involvement with their child and other indices of interaction between the child and primary caregiver.

Principal Conceptual Models Used in Developmental Research

Although conceptual models of lead's effects on development are rarely explicitly stated, there are often implicit models which are reflected in a study. The majority of previous retrospective studies invoked what is best described as a main-effects model (see Fig. 1). In this model biological, social-environmental, and physical-environmental factors are viewed as exerting independent influences on development. Lead is viewed as having a deleterious effect on development regardless of the constitutional vulnerability or robustness of the child or the quality of the home environment in which the child develops. While this is undoubtedly true under conditions of very high lead exposure, it is less likely to be the case at lower levels of exposure. An example taken from recent research in the field of fetal malnutrition demonstrates the inaccuracy and inadequacy of such a main-effects model (*1,2*) (Table 1). This study consisted of the random assignment of fetally malnourished (low ponderal index) and control (normal ponderal index) infants, drawn from a low socioeconomic status (SES) black population, to environments differing in quality of support for intellectual development. They found that the detrimental effects of fetal malnutrition persisted in 30-month-old infants reared in a nonsupportive environment. Those high-risk infants receiving additional social stimulation exhibited intellectual performance considerably above those not receiving social-environmental intervention and performed at a level comparable to those infants not at risk due to fetal malnutrition. Thus, the developmental trajectory of infants subjected to various environmental insults is not determined solely by the nature of the insult. It is subject to modification by the child's subsequent interactions with his/her environment. Contrary to popular belief, a child's cognitive ability as reflected in performance on IQ tests is not a static biologic entity, but rather, it is highly labile and subject to the quality of the rearing environment.

Recognition of the limitations of the main effects model has led to the development of an interaction model (Fig. 1). This model posits that various outcomes arise as a result of an interaction between two or more independent variables, e.g., lead exposure and caretaking environment or lead exposure and nutritional status. This model allows for a diversity of possible outcomes. For example, effects arising from exposure to toxicants might be ameliorated or exacerbated by the quality of the rearing environment or the susceptibility of the child prior to exposure. Unfortunately, this model has seen only limited application in studies of environmental influences on child health and development. Furthermore, the covariates employed in the model require sophisticated choices. The limiting feature of the interactive model is that it assumes that the world of the developing child is static, i.e., that the biological, social, and environmental features remain fixed throughout the developmental process. Obviously, this is not the case.

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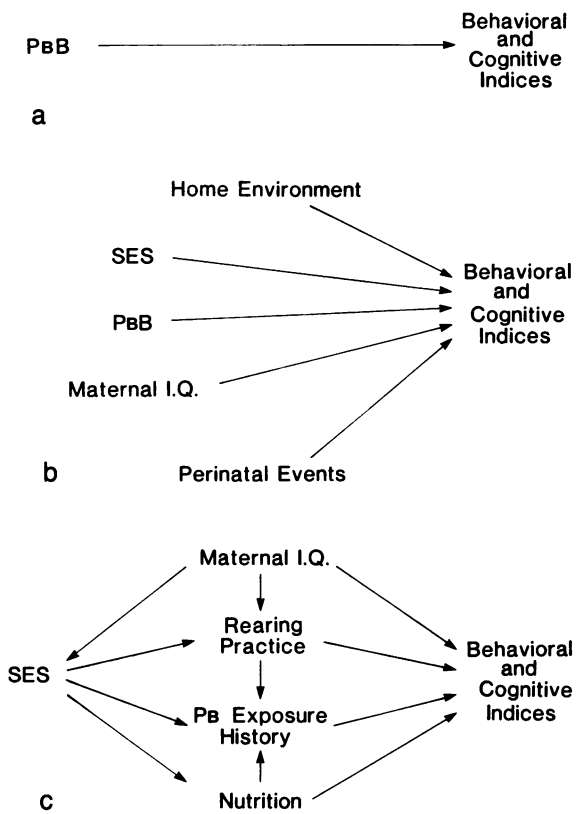

FIGURE 1. Examples of hypothetical conceptual models relating presumed causal agents to developmental outcome: (*a*) simple main-effects models; (*b*) multiple main-effects model; (*c*) interaction model.

In an attempt to deal with this major limitation, Sameroff and Chandler (*3*) proposed a transactional model. This model is dynamic, in that it focuses attention on the role the child plays in shaping his/her environment. Attention is directed not toward a set of static traits which a child may exhibit at a particular stage of development, e.g., temperament, but rather toward the processes which produce the trait and are currently maintaining the trait, e.g., child-caretaker interactions. Other aspects and implications of the transactional model appear in papers by Pasamanick and Knoblock (*4*), Sameroff and Chandler (*3*), Sameroff and Seifer (*5*), and most recently Pearson and Dietrich (*6*). The last paper discusses in detail the impact of these models on study design, data analysis and interpretation, as well as intervention strategies with particular reference to environmental health issues. Also, a recent monograph (*7*) reports the results of a large-scale prospective study conducted and interpreted from the perspective of a transactional etiological model.

Table 1. Influence of fetal malnutrition and caregiving environment on intellectual development of infants.[a]

Pondral index	Infant stimulation	IQ at age 3 mo	18 mo	36 mo
Average	Yes	102	107	98 ± 14
	No	97	93	84 ± 10
Low	Yes	92	104	96 ± 11
	No	91	86	71 ± 10

[a] Data of Zeskind and Ramey (*2*).

Measurement of Critical Covariates

It has become apparent over the course of the last decade of research that there are several covariates that might interact with lead to influence its effects on developmental outcome or might covary with lead exposure in such a way as to obscure the relative independent effects of each. Increasingly, investigators have attempted to measure these covariates and control for their influences either via experimental design or subsequent data analysis.

Socioeconomic Status (SES)

There are two reasons for viewing SES as an important covariate in longitudinal studies of lead's impact on child development. First, it has been observed that moderate to high lead exposure is most prevalent in that segment of the population living in older deteriorating housing stock which contains high quantities of lead-based paint or in less desirable neighborhoods near industry or major highways which also contribute lead to the environment. Frequently, a correlation between SES and environmental lead exposure can be observed, since the majority of children living in these dwellings are from families of low SES. However, within a given study cohort there are numerous exceptions which weaken this association. For example, some of the most economically deprived families reside in modern public housing which contains very limited amounts of lead derived from lead-containing paints. At the same time, more affluent families are rehabilating some of the older, architecturally interesting dwellings which are known to contain considerable quantities of lead-based paint. This process might result in blood lead elevations in young occupants. Thus, caution is warranted if one assumes that SES is a strong predictor of lead exposure.

The second and more common reason for attending to SES is its presumed association with rearing practices and certain life events which can alter the course of a child development. Developmental researchers have shown that the social environment in which the child lives can have a profound influence on developmental processes. More specifically, cognitive abilities, as measured by standardized psychometric tests, have been shown to have a positive correlation with standardized measures of social rank. Therefore, most studies use some measure of SES as a covariate in an attempt to factor out this suspected social influence.

Unfortunately SES is only weakly correlated with the more proximate variable of interest, namely, the rear-

ing practices to which the child is subjected. The qualitative aspects of child–caretaker interactions have a far more profound influence on child development than, for example, the number of years of education completed by the head of the household (a typical component of the SES measure). Furthermore, it is possible—in fact, likely—that a wide range of child–caretaker interactions will be exhibited within a study sample with a very narrow SES range. Thus, in practice, SES is often a poor predictor for estimating either likelihood of exposure or quality of rearing environment.

Child–Caretaker Assessments

Previous research has suggested that there might be an association between differences in the caregiving environment of children and the amounts of lead they ingest (*8–10*). On the other hand, several prospective studies (*7,11*) have demonstrated improved prediction of child IQ when additional maternal characteristics, such as social competence and behavior toward the child, were added to their developmental models. Thus the caretaking environment might influence both lead ingestion and developmental outcome either independently or interactively. While previous lead studies have attempted to control for the possible influences of SES, usually they have ignored the quality of the caretaking environment. This was due, in part to the lack of a widely accepted, standardized measurement instrument. This difficulty has been remedied by Caldwell and Bradley (*12*) with the development of the Home Observation for Measurement of the Environment (HOME) Inventory for assessing the type of caregiving environment. Bradley and Caldwell (*13,14*) demonstrated that two subscales of the HOME, "emotional and verbal responsivity of the mother" and "maternal involvement with the child" were strongly related to mental test performance at 36 and 54 months of age. More recently, Milar et al. (*10*) showed that these same subscales correlated significantly with increased lead burden in young children. Thus, a strong case can be made for the need to measure the caretaking environment and its influence on both lead exposure and development.

Parental Intelligence

It has been reported that there is a high concordance between maternal and child intelligence (*15*). Heber found that the majority of children with IQs in the retarded range had mothers with IQs less than 80. This raises the possibility that low IQ in lead-exposed children might be due in part to low maternal IQ. While this association is demonstrable when one considers the entire IQ range, it is likely to have less impact within the more restricted range of IQs observed in most lead studies. Furthermore, it is clear that maternal IQ influences child IQ not only via a heritability route but also via the child–caretaker route (see Fig. 1c). For example, it is known, in the case of adopted children, that the adopted child's IQ changes over time to more closely approximate that of the rearing mother (*16*). Thus, a mother's IQ can influence her interactions with her child and thereby influence development. It is important that both maternal IQ and caretaking style be measured and taken into account in any study of the influence of lead on development.

Retrospective Epidemiologic Studies

While several large-scale epidemiologic lead studies have recently been published (*17–19*) one study (*20*) will be used to illustrate the impact of the investigators' conceptual model of development and their use of covariates in the data analysis on the conclusion reached with respect to lead's effect on cognition as measured by a standardized IQ test. This is a follow-up study of 63 urban black school age children who had been studied 5 years previously (*21*). Two separate analyses of the data were provided. First, a simple between-group comparison was provided (Table 2). This is an example of a simple main-effect model, with no attempt to control for any of the critical covariates previously discussed. The initial impression is that moderate lead exposure (40–70 μg/dL during the preschool years) is responsible for a 12-point deficit in cognitive abilities as reflected in the General Cognitive Index. However, these investigators also measured several important covariates and provide a second analysis which shows the percent of total variance accounted for by each of the covariates alone, each of the lead measures alone and each of the lead measures after statistical adjustment for the covariates (Table 3). Among the control variables, parental IQ is shown to be quite important, accounting for 20.8% of the variability in the child's General Cognitive Index. Current school-age blood lead levels also appear to be associated with cognitive abilities (17.6%). However, when the lead effect is measured after removing the variability due to the control variables, the variance accounted for by lead drops to 7.7%.

This study (*20*) also provides us the opportunity to observe the impact of misclassification of subject's exposure level on study results (*21*). The subjects were initially recruited on the basis of blood lead levels in order to examine the influence of low to moderate lead exposure on cognitive performance. Average preschool blood lead levels were 33 ± 13 μg/dL. However, tooth lead analyses of the school-age children revealed that one child had a tooth lead level of 107 ppm (study cohort mean = 6 ± 6 ppm). His preschool PbB was 60 μg/dL and his school-age PbB was 43 μg/dL, both considered moderate exposure levels at the time of the study. His preschool General Cognitive Index Score (comparable to IQ) was 80, and his school-age General Cognitive Index Score was 48. Although the child was never clinically diagnosed as having lead poisoning, his data are consistent with an interpretation of marked decrease in intelligence secondary to a massive lead exposure (as

Table 2. Means, standard deviation, and *t*-tests of McCarthy Scale data for low and moderate school-age lead levels.[a]

Scale	Lead levels: Low (10–30 μg/dL)	Lead levels: Moderate (40–70 μg/dL)	*t*-Test	*p*
McCarthy Scale				
General cognitive index	93.9 ± 13.8	81.6 ± 14.2	3.48	0.001
Verbal	48.0 ± 7.6	40.6 ± 8.9	3.57	0.001
Perceptual-performance	43.2 ± 10.7	39.7 ± 10.0	1.35	0.18
Quantitative	43.2 ± 8.1	38.5 ± 6.7	3.47	0.016
Memory	43.9 ± 6.6	38.9 ± 9.0	2.53	0.014
Motor	48.7 ± 9.8	45.7 ± 11.2	1.15	0.25

[a] Data of Ernhart et al. (*20*).

reflected by the extreme tooth lead levels). If this one case in 63 is dropped from the data set (since it does not fit the criteria of "low-to-moderate lead exposure"), there is no longer any evidence of a relationship between lead and intelligence. This example, illustrates the dangers in attempting to use single or infrequent blood lead determinations to index a child's total lead exposure history, as well as highlighting a problem common to all retrospective lead studies, i.e., an inadequate knowledge of prior lead exposure.

When reviewing all of the recently published retrospective studies of the effects of lead on child development, one can see that concern about the potentially harmful effects of lead is warranted. However, there is as yet no substantial body of data to support the contention that lead produces behavioral disorders such as hyperactivity. Furthermore, after appropriate control for critical covariates the variance in child IQ attributable to lead is small (less than 5%) and often statistically insignificant. It is unlikely that there is a causal link between low level lead exposure and child IQ. Rather, recent studies suggest that apparent links are most likely due to inadequate measurement and statistical control for rearing environment and other major covariates. If a relationship does exist it is most likely to be observed in high-risk groups, e.g., the socially disadvantaged, children reared in nonsupportive environments, and/or children subjected to other stressful life events.

Obviously, in order to understand fully the impact of lead exposure on child development, it will be necessary to sort through a very complex web of social, environmental and behavioral interrelationships. To assist in this task, some investigators have recently turned to longitudinal studies. These prospective studies have the potential for furthering our understanding of the complex interplay between environmental toxicants, psychosocial factors and developmental outcome. However, new levels of analysis, capable of dealing with the complexity of intercorrelations and longitudinal, repeat measurements will be needed. The following section briefly illustrates the application of structural equation modelling to explore the relationships between environmental sources, rearing environment, dust lead, hand lead, and blood lead levels.

Dust Lead, Hand Lead and Blood Lead Relationships

Subjects. The data presented in this report were derived from a subset of children participating in a large prospective study of childhood lead exposure and its effects on child development. The sample was restricted to those children for whom we currently have complete data with respect to blood lead levels, measures of lead in the home environment, measures of the quality of caretaking, and characterization of the quality of the

Table 3. Variance of McCarthy Scale scores associated with lead and control variable.[a]

Predictor variable	Descriptive statistics	McCarthy Scale: General cognitive	Verbal	Perceptual performance	Quantitative	Memory	Motor
Control variables							
Sex		1.1	2.8	1.0	0.0	0.0	8.4*
Parent IQ	(84 ± 16)	20.8*	20.2*	9.8*	7.1*	11.4*	1.9
Parent education	(11 ± 2)	5.2	0.0	2.7	2.8	0.1	2.0
Lead measures, ignoring control variables							
Preschool lead	(33 ± 13)	6.3*	12.0*	1.2	0.0	0.3	10.0
School-age lead	(27 ± 7)	17.6*	18.3*	9.5*	3.6	8.5*	9.6*
FEP	(27 ± 20)	8.4*	10.6*	1.4	0.2	0.6	7.0*
Dentine lead	(6 ± 6)	3.1	8.9	0.0	1.1	0.2	5.8
Lead measures, control variance removed							
Preschool lead		1.4	4.7	0.0	0.4	1.7	4.7
School-age lead		7.7*	8.0*	4.3	1.0	3.4	7.4*
(with outlier removed)		2.6	—	—	—	—	—
FEP		4.5	5.7*	0.3	0.0	0.1	3.1
Dentine lead		0.0	1.2	0.8	3.5	2.4	1.2

[a] Data of Ernhart et al. (*20*).
* $p < 0.05$.

housing occupied by the subjects during the study interval. Data from approximately 45 children were available for analysis. Further description of the full study cohort has been reported elsewhere (*22*).

Blood Lead Determinations. Blood samples were obtained by venipuncture. All analyses were performed in duplicate. Lead was measured by anodic stripping voltammetry (ASV) with the use of an ESA Model 3010A instrument. Further details of analytic procedure and proficiency have been reported (*22,23*).

Environmental Sampling and Analysis. Environmental sampling was carried out when the children were 7 and 19 months of age. If the child changed residences during the study interval an additional assessment was carried out in the new residence.

Interior surface dust was collected by three sweeps of a defined area by using a 2-L/min vacuum pump (personal air sampler). Recovery studies have shown that this procedure yields a reliable and high mean recovery rate (84% ± 4%) from a variety of surface types (*24*). Lead was recovered from the surface of the child's hands by repeated wipings of both hands with a total of six wet wipes (Walgreens Brand Wet Wipes). Recovery studies were undertaken to examine the influence of towel type and number of wipes on recovery of known amounts of lead applied to subjects hands. The procedure chosen yielded a mean recovery rate of 84 ± 7% (*24*).

Housing Evaluations. All dwellings occupied by study participants were evaluated with respect to age of dwelling (19th Century, 20th Century-pre WWII, or 20th Century-post WWII), condition of dwelling (satisfactory, deteriorating, or dilapidated), and type of dwelling (public housing, rehabilitated housing, or nonpublic/nonrehabilitated). A more detailed description of procedures, operational definitions and housing stock occupied by study participants has been reported (*25*). Two observers independently evaluated each residence. Interobserver reliability exceeded 90% on all items. Previous preliminary analyses have shown high correlations between this qualitative evaluation of housing, interior lead levels, and child's blood lead level (*26*).

Social Measures. The Home Observation for Measurement of the Environment (HOME) (12) was used at 6, 12, and 24 months to quantitate various aspects of the child's rearing environment. Two trained observers attended and independently scored each home visit. Interobserver agreement exceeded 95% on all items.

Determination of the socioeconomic status (SES) of the families was made through use of the Hollingshead Four-Factor Scale (*27*). This scale was administered at 3 months and 15 months.

Criteria for Selection of Variables. Our central hypothesis predicted that lead in house dust (PbD) influenced blood lead (PbB), with hand-to-mouth activity being a significant route of exposure. Thus hand lead (PbH), PbD, and PbB were included in all analyses. Quality of rearing environment, socioeconomic status of the parents, and quality of housing were predicted to have important influences on this route of exposure and thus were included. Many other variables obviously can influence PbB and could have been incorporated into these analyses. However, these analyses were viewed as exploratory. Furthermore, an equally important objective was to examine various data analytic procedures on a relatively well defined, simple data set. Therefore, other predictors and modifiers of lead exposure were excluded from consideration at this time.

Data Analysis

Prior to data analysis all lead measures, i.e., blood lead, dust lead, and hand lead values were subjected to

Table 4. Simple correlations between environmental or social variables and child's blood lead level.[a]

Variable		Child's age					
		9 mo	12 mo	15 mo	18 mo	21 mo	24 mo
N		41	43	47	47	44	42
Hand lead	*r*	0.44	0.53	0.44	0.51	0.35	0.48
Dust lead	*r*	0.44	0.67	0.54	0.51	0.40	0.43
SES	*r*	−0.30	−0.32	−0.30	−0.37	−0.30	—
HOME (total score)	*r*	−0.33	−0.46	−0.30	—	—	—
Maternal involvement	*r*	—	−0.37	−0.29	−0.23	—	—
Organization of environment	*r*	−0.41	−0.31	—	—	—	—
Provision of play materials	*r*	—	−0.37	−0.34	−0.29	—	—
Housing:							
Type							
Public	*r*	—	—	−0.31	−0.34	−0.43	−0.33
Rehabilitated	*r*	—	—	—	—	—	—
Condition							
Satisfactory	*r*	−0.48	−0.50	−0.39	−0.39	—	—
Deteriorating	*r*	0.42	0.42	0.39	0.38	—	—
Age							
19th Century	*r*	0.34	0.37	0.46	0.42	0.56	0.54
20th Century	*r*	—	—	—	—	—	—

[a] All reported correlations are significant at $p < 0.05$.

$\log_e$ transformation to obtain approximate normality of their distributions. Analyses were conducted with the use of the CORR procedure (for Pearson product-moment correlation coefficients) and the first stage of the SYSREG procedure (to obtain ordinary least-square regressions) which are part of the SAS statistical package (*28*). The structural equations model was estimated via the third stage of SYSREG (*29*). A backward elimination of covariates was employed to evolve a final model from the initial, overparameterized structural model. Criteria for elimination of exogenous (predictor) variables included, in order: initial elimination of paths with beta weights not in the predicted direction, further elimination of paths (beta weights) which did not attain statistical significance ($p < 0.05$), and final elimination of paths which did not add to the internal consistency of the model across ages (*30*).

Results

Simple Correlations

Table 4 summarizes the correlation between PbB and various predictor variables. Correlations tended to be moderately strong and relatively consistent across ages. As predicted, PbD and PbH were significantly correlated with PbB. Likewise, presumed mediating variables such as the total HOME score and three of six HOME subscales were significantly correlated with PbB at various ages. Quality of housing and SES were also found to be associated with PbB. This level of analysis does not estimate, nor control for, the high degree of correlation among these predictor variables.

Multiple Regression

A commonly employed technique to deal with these intercorrelations would involve an ordinary least squares multiple regression analysis for each age cohort. All possible predictor variables are usually entered simultaneously. The next step would involve a backward elimination procedure wherein nonsignificant covariates would be sequentially dropped from successive analyses until a final reduced model was attained. This type of analysis can be used to maximize the explained variance with the most parsimonious set of predictors. However, it obscures the underlying structural relations among variables. Furthermore, if separate unweighted regressions were performed independently for each of the three dependent variables, PbD, PbH and PbB, the estimated beta weights would be biased, since the simultaneous nature of the system of equations would not be taken into account.

Structural Equations

Figure 2 summarizes the qualitative aspects of this analysis. Figure 2*a* shows the hypothesized relationships. Figure 2*b* shows all the relationships that were tested in the course of the analysis, while Figure 2*c* shows the results of the analysis. Figure 2*d* is discussed in the discussion section. The analysis supported the hypothesized path from housing quality (House) to PbD to PbH to PbB. A second pathway (not mediated by PbH and not predicted) was also detected between PbD and PbB (see Fig. 1*c*). As predicted, one subscale of the HOME (Maternal Involvement with Child) was found to be a significant modifier of PbH. However, unexpectedly, two subscales, Variety in Daily Stimulation and Emotional and Verbal Responsivity of Mother, were found to be associated with PbD. SES was also unexpectedly found to have a small but significant association with PbB.

Quantitative aspects of the final systems model for 18-month-olds are depicted in Figure 3. The values in parentheses are the y-intercept in $\log_e$ units. The values next to the arrows are the beta weights associated with each particular pathway. The arrows in this diagram or "flowgraph" imply causal ordering of events. However, the analysis does not test directionality of correlated relationships, nor "prove" causality, which is not a statistical issue but rather a logical inference (*31*).

Table 5 summarizes the results of the six structural equations models developed for each of the six age cohorts. The reported values are path or beta coefficients in the case of the exogenous variables and y-intercepts in the case of the three endogenous variables, PbD, PbH, and PbB. Coefficients were found to be remarkably stable across ages both with respect to their sign or direction of correlation and magnitude of the coefficient. Likewise, the weighted R^2 for the system models were relatively stable, ranging from 0.44 to 0.59.

Discussion

Complex interrelationships among antecedent and outcome variables may be described at various levels of sophistication. The simple correlations may be computed or a series of multiple regressions performed, one for each outcome variable. The technique of calculating multiple regressions with a fully parameterized model is often referred to as "path analysis." However, it is only in this situation of full parameterization that this technique results in consistent estimates of the the parameters (beta or path coefficients). A better and more flexible approach, i.e., structural equations, recognizes the possibility that the equation errors in each of the outcome variables may be correlated, resulting in inconsistent and biased estimates of the effects of the "exogenous" (antecedent or predetermined) variables on the "endogenous" (outcome or dependent) variables. This approach leads to weighting the parameter estimates by the inverse of the covariance among the errors (*29*), known in the statistical literature as a "generalized" least-squares (GLS) solution. Path analysis and simple multiple regression are examples of "unweighted or ordinary" least-squares or OLS solutions.

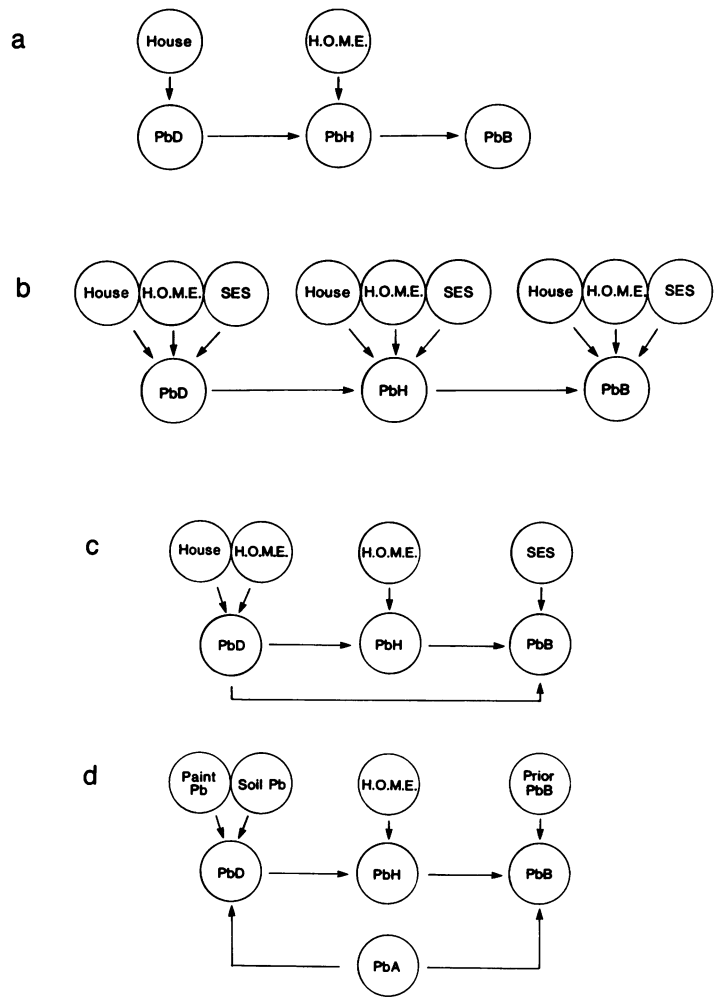

FIGURE 2. Flow graphs on path diagrams depicting causal ordering of relationships: (*a*) hypothesized relationships; (*b*) relationships tested; (*c*) relationships found to be statistically significant; (*1d*) model to be tested in next series of analyses. HOUSE = house type; HOME = Home Observation for Measurement of the Environment; PbD = surface dust in μg/g; PbH = lead from handwipes in μg; PbB = blood lead in μg/dL; SES = socieconomic status; PbA = air lead in μg/m^3).

The PbD to PbH to PbB path was estimated, beginning with all exogenous variables being allowed to affect all three endogenous variables. By backward elimination, a final model with the same exogenous variables included for each (single) equation at each age was estimated. This "systems regression" utilized Zellner's GLS procedure. To judge the significance of each effect, an alpha level of 0.05 was chosen. The stability of the model, in the age range investigated, was also a guide to retention/elimination of predetermined variables and served as a further check on the convergence to the final model (in the presence of so much confounding). For these small sample sizes, a stability guide is perhaps to be recommended over a correction to the alpha level to prevent the power of the hypothesis tests from diminishing to minimal levels.

The results of this systems analysis should be viewed as preliminary, since the analysis was exploratory in

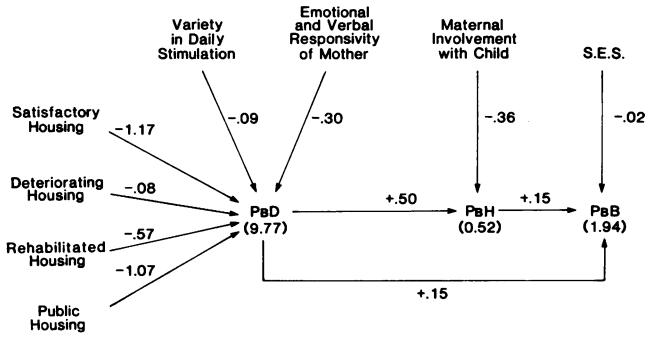

FIGURE 3. System model for 18-month-olds indicating the intercept for endogenous variables and unstandardized weights. Numbers in parentheses are *y*-intercept values in $\log_e$ units. PbB = 1.94 − 0.02 (SES) + 0.15 (PbD) + 0.15 (PbH), where PbD = 9.77 − 1.07 (Public) − 0.57 (Rehab.) − 1.17 (Satis.) and PbH = 0.52 − 0.36 (Matern. Invol.) + 0.50 (PbD).

Table 5. Intercepts and unstandardized beta weights for all systems models.

Variable	Age					
	9 mo	12 mo	15 mo	18 mo	21 mo	24 mo
N	41	43	47	47	44	42
Intercepts						
PbD	10.23	10.48	10.07	9.77	10.86	10.34
PbH	0.31	0.12	0.39	0.52	2.41	2.67
PbB	1.71	1.59	2.01	1.94	2.63	1.70
Effects on PbD						
Public housing	−1.26	−1.32	−1.23	−1.07	−0.78	−0.66*
Rehabilitated housing	−0.50	−0.32	−0.64	−0.57	−0.11	−0.07*
Satisfactory housing	−1.50	−1.48	−1.03	−1.17	−2.44	−2.45
Deteriorated housing	−0.15	−0.27	−0.02*	−0.08	−1.56	−1.77
Variety in daily stimulation	−0.15	−0.14	−0.24	−0.30	−0.29	−0.27*
Verbal responsivity of mother	−0.16	−0.19	−0.14	−0.09	−0.10	−0.06*
Effects on PbH						
Maternal involvement with child	−0.32	−0.30	−0.25	−0.36	−0.42	−0.47
PbD	0.49	0.49	0.42	0.50	0.27	0.28
Effects in PbB						
SES	−0.02*	−0.02	−0.02	−0.02	−0.02	−0.01*
19th Century housing	0.04*	−0.03*	0.17*	0.09*	0.25	0.22
20th Century, Pre-WW II	0.06*	−0.05*	0.09*	0.03*	0.01	0.10
PbD	0.13	0.19	0.15	0.15	0.06*	0.13*
PbH	0.13	0.13	0.14	0.15	0.11*	0.23
Weighted r^2	0.59	0.59	0.52	0.57	0.44	0.44

* Parameters were not statistically significant.

nature. A second confirmatory analysis on a different subset of children drawn from the same cohort will be conducted when more data become available. Nonetheless, the analysis indicates some interesting relationships and suggests directions for future analyses. First, the suspected relationship among PbD, PbH, and PbB has been confirmed in yet another cohort of children (*32–34*). Furthermore, the analysis indicates the important interplay between environmental sources of lead and social factors in the determination of hand lead and blood lead levels in very young children. While others (*10,35*) have shown a relationship between HOME scores and blood lead levels, this study indicates that this relationship is mediated, at least in part by hand lead. A further discussion and interpretation of results as well as a more detailed description of this analytic approach has been previously reported (*30*).

This analysis has demonstrated that structural equation modeling offers a useful approach to unraveling the complex interactions present in a data set of this kind. It also is of heuristic value in ongoing exploratory data analysis and hypothesis generation. It is likely to be of similar value in many other areas of environmental research and warrants the particular attention of those dealing with complex epidemiologic issues.

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