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Seasonal variation in paediatric blood lead levels in Syracuse, NY, USA

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Venous blood lead values for 2,633 children aged 0–4 years in Syracuse, New York, collected between 1 April 1992 and 31 March 1993 were summarised by census tract for study of geographic variability. A demographic exposure model is presented showing housing stock and SES (socioeconomic status) parameters as the most significant predictor variables. A seasonal trend in blood lead levels was observed with late summer values about 40% higher than late winter values for census tracts with the highest geometric mean PbB levels. Seasonal variation is compared with a biokinetic uptake model to examine hypotheses about temporal variations in soil and dust lead exposure patterns.

Keywords: blood lead, seasonal variation, demographic correlates, biokinetic uptake

Introduction

Blood lead values in the general US population have declined significantly over the past decade. Results of the third National Health and Nutrition Examination Survey (NHANES III, 1988–91) show that among children aged 1–5 years, mean blood lead values have decreased from 13.7 μg dl^{-1} (NHANES II, 1976–80) to 3.2 μg dl^{-1} (Pirkle *et al.*, 1994). This can be taken as direct evidence that primary prevention of lead exposure is an effective public health measure. Air lead concentrations and dietary lead levels have both decreased in response to government regulatory action (NRC, 1993). However, Brody *et al.* (1994) estimate that as many as 1.7 million children, primarily from inner-city urban environments, have blood leads greater than the 10 μg dl^{-1} CDC health concern guideline. On a national basis, only 5.8% of children aged 1–5 years showed blood lead levels greater than 10 μg dl^{-1}; but in large central city populations, the figure was 21% without regard for race or ethnicity and more than 36% for the non-Hispanic Black population subgroup. A major conclusion drawn from the NHANES III, phase 1, study was that 'a concerted effort to identify the vulnerable risk groups will be vital to further reductions in lead exposure' (Brody *et al.*, 1994).

Such 'further reductions in lead exposure' will evolve from improved understanding of the urban childhood lead exposure dynamic. Many of the elements required for research efforts in this direction are already in place. Lead pollution of urban soils and dusts is a widespread contemporary phenomenon. While not exhaustively reviewed in this paper, evidence for it can be found in the works of Mielke *et al.* (1983), Duggin and Inskip (1985), Thornton *et al.*, (1985), Onyari *et al.* (1991), Mielke (1993) and Brinkmann (1994). The inadvertent ingestion of contaminated soils and dusts by children, through hand to mouth activity, has been shown to be a significant component of childhood lead exposure topology (Davies *et al.*, 1990; Body, 1991). Direct measures of hand lead can be related to soil and dust lead levels (Clark *et al.*, 1985; Brisco *et al.*, 1994). Finally, health authorities in many metropolitan regions of the USA operate blood lead monitoring programmes in order to identify children who may be at risk from lead poisoning. Some of these programmes have been gathering exposure measures for more than a decade, so a large number of exposure data exist. It might be argued that if the general air lead and dietary lead intakes decrease in importance, the relative significance of soil and dust lead ingestion pathways should increase. To the extent that lead exposure is dominated by ingestion of soils and dusts, this could lead to simpler formulation and improved predictive capability of exposure models. Databases with thousands of observations offer good potential for statistical models of relationships between blood lead level and such co-variates as lead concentration or loading in environmental media, age, sex, race and other geo-referenced demographic variables.

Unlike controlled studies, however, exposure measures (blood lead observations) from health

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authority monitoring programmes are more or less randomly distributed over time. Thus, temporal variations in the monitoring data need to be examined to ensure accurate interpretation of any exposure models which might be developed. Several investigators have reported seasonal variation in blood lead values (Hunter, 1977; Rabinowitz *et al.*, 1984; Mielke *et al.*, 1991; Blatt and Weinberger, 1993; Kimbrough *et al.*, 1994; Brody *et al.*, 1994), but few quantifications of the phenomenon have been described in detail. Studies by Robinowitz and Needleman (1982) and Hwang and Wang (1990) with neonates having blood lead levels in the 6–8 μg dl^{-1} range, both showed that infants born during summer months had blood leads about 1 μg dl^{-1} higher than infants born during winter months. The study by Hammond *et al.* (1983) showed similar results for new born infants, but by age 9 months when blood lead levels had risen to about 15 μg dl^{-1}, the amplitude of the seasonal variation was about 9 μg dl^{-1}. Baghurst *et al.* (1992), following the Port Pirie cohort, found that seasonal differences reached a maximum for children 15 months of age (22.1 μg dl^{-1} during the warmer season versus 19.9 μg dl^{-1} during the cooler season, for a test population of 607 children). At age 5 years (513 children remaining in the cohort), seasonal differences had declined; mean blood lead values were 14.7 μg dl^{-1} for the warmer season and 14.2 μg dl^{-1} for the cooler season. The present study examines the seasonal variation in about 2,600 blood lead values measured for children under 5 years of age in Syracuse, New York. Temporal trends in the data are stratified by age and geographic location.

Methods

Data set description

Blood lead data for this pilot study were obtained from the Onondaga County Health Department Lead Poison Control Program monitoring effort for the period 1 April 1992 to 31 March 1993. The study was restricted to children under the age of 5 years living in or near the city of Syracuse, and from whom blood samples had been collected by venipuncture. While capillary samples were available in the data set, the venipuncture samples were felt to be more accurate, particularly at low blood lead (PbB) levels. No replicate blood lead determinations were included in the data, although about 12% of the children were tested more than once during the study period; the first occurrence for an individual blood lead record was that included in the data set. These selection criteria resulted in a sample population of 2633 observations.

The blood lead values showed a truncated log normal distribution. The Onondaga County Health Department (OCHD) accepted detection limit for lead in blood, during the study period, was 5 μg dl^{-1}; commonly, values greater than 0 but less than 5 were reported to OCHD at 5 μg dl^{-1}. For the summary results presented here, PbB values less than 6 μg dl^{-1} were estimated by extrapolation of the normal quantile plot after log transformation. Reported values of 5 μg dl^{-1} were replaced with 4 μg dl^{-1}, values of 4 or 3 μg dl^{-1}were replaced with 2 μg dl^{-1}, and reported values of 2 μg dl^{-1} were replaced with 1.6 μg dl^{-1}. The number of values so affected was 675; the resulting model distribution used is shown in Figure 1.

Data summary regression model and biokinetic uptake model procedures

All computations for data summary were performed with SAS. Demographic data from the 1990 Census were extracted from Summary Tape File 1A and merged, by census tract, with the OCHD blood lead monitoring data. SAS was used to normalise some of the 1990 Census Bureau summary data across census tracts and express the results as fractions (the fraction of housing stock in each census tract built prior to 1950, the fraction of census tract population within certain age groups *etc.*). The normalised data were used in PROC STEPWISE for initial development of a multiple linear regression model relating census tract geometric mean blood lead values to demographic variables. The normalised data were also imported into a GIS database for geographic distribution plots using ARC/INFO.

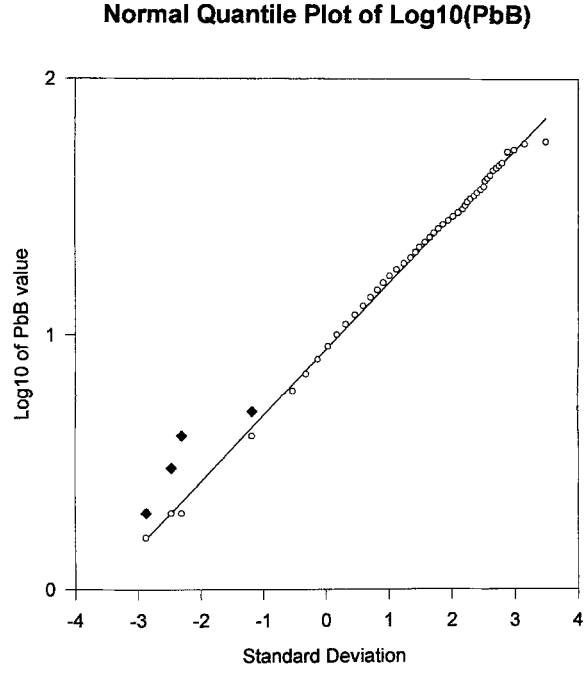

Figure 1 *Normal quantile plot for Syracuse test blood lead data set. Solid symbols represent reported values which were replaced to model the distribution as described in the text.*

An exploratory spatial analysis was conducted on blood level data aggregated into 1990 census tract groups. Each group was geo-coded by census tract centroid, extracted with ARC/INFO from the 1990 TIGER files. Weighted least squares fits to the experimental semi-variograms were achieved using Gerard Heuvelink's software package, which implements Cressie's procedure. Each analysis was based on approximately 50% of the 1431 possible pairs of inter-centroid distances.

Limited experiments were carried out with the age specific biokinetic uptake model for lead proposed by Leggett (1993) to see if temporal variations in PbB could be reproduced by simple seasonal alteration of soil and dust exposure patterns. The FORTRAN code of Leggett *et al.* (1993) was adapted for this purpose.

Results and discussion

The blood lead monitoring data used here represent samples from an over sampled population. They contain a geographic bias. Experience at the OCHD has shown that certain census tracts generally yield high blood lead values from tested children, and the Lead Poison Control Program screening efforts are somewhat disproportionately allocated to these high risk areas. As a result, we do not expect the blood lead values summarised here to reflect the general blood lead distribution in Syracuse. These limited data do not form an adequate basis for assessment of public health concerns; but they do apply to a real population of children and are worth examining in some detail. Thus, summary results apply to the 'tested population', for which the geometric mean (geometric standard deviation) PbB was 8.68 (1.87) μg dl^{-1}. Stratified by age group, as shown in Figure 2, the blood lead values show a familiar pattern of increasing to a maximum for 2 year old children, followed by a decline. When the results are summarised by the month of the year in which the test was performed, Figure 3, a distinct seasonal trend is evident. While differences between monthly geometric means are not significantly different, seasonal differences are significant at the $p=0.0001$ level. Geometric mean summer blood lead levels of 9.01 (1.82), for months 7–10, were 1.8 μg dl^{-1} higher than winter blood lead levels, for months 1–4, at 7.20 (1.74) μg dl^{-1}.

The test population showed a distinct geographic clustering of blood lead values, as shown by Figure 4. For ease of display, geometric mean PbB for three ranges are plotted; eight census tracts showed annual levels > 10 μg dl^{-1}, 12 had annual geometric means less than 6 μg dl^{-1}, and 32 tracts exhibited annual means between these limits. The remaining census tracts had fewer than 10 observations each and were excluded from the summary plot. The eight 'high PbB' census tracts accounted for 36% of the total observations in the data set; the six contiguous tracts southwest of the city centre contributed 32% of the PbB measurements in the test population. Clearly, this influences the geometric mean value for the tested population. If the geometric mean PbB is computed for each census tract, and then averaged, the mean for the tested population is reduced to 7.13 (1.66) μg dl^{-1}. Normalised to the 1990 Census Bureau demo-

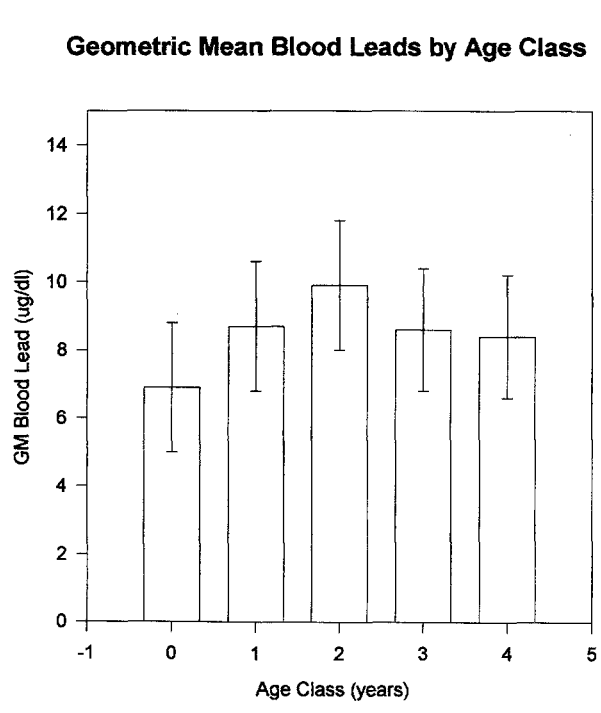

Figure 2 *Geometric mean blood lead values according to age group. Error bars represent one standard deviation.*

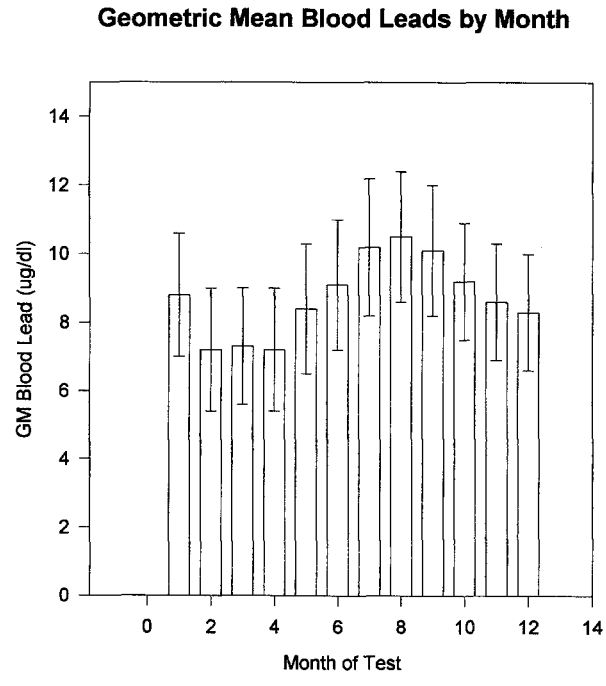

Figure 3 *Geometric mean blood lead values according to month of the year in which the blood sample was taken. Error bars represent one standard deviation.*

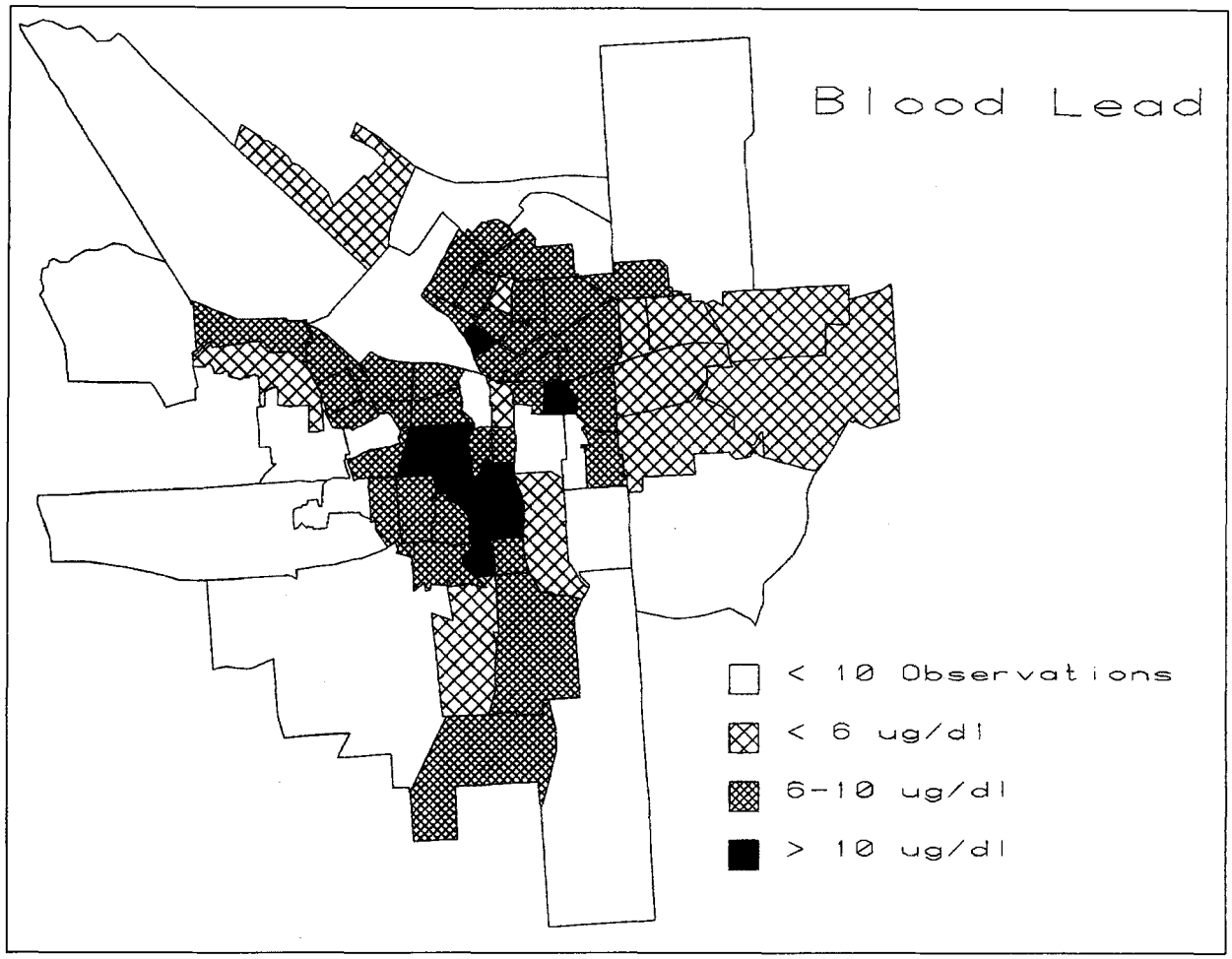

Figure 4 *Geometric mean blood lead values stratified by census tract and divided into 'low', 'intermediate', and 'high' annual values.*

graphic data, the screening rate for this data set averaged 15% across all census tracts, but ranged from a high value of 40% for some tracts to less than 2% in others. If the census tract mean values are further weighted by screening rate, the geometric mean blood lead for all 0–4 year old children in the Syracuse metropolitan region is estimated at 5.7 μg dl^{-1}. Confirmation of this estimate awaits the analysis of larger, less biased data sets.

In terms of preliminary spatial analysis, elevated blood lead level data (> 10 μg dl^{-1}) analysed included measures for summer, winter, the entire year, greater than the average and empirical probability of exceeding the threshold level. In all cases, the raw data suggested a Gaussian semi-variogram model with a localised spatial field (less than or equal to 0.5 km radius, range of 0.1 km). The spatial field for elevated PbB appears to be strongly structured, but very localised. This scale of autocorrelation is about the size of the inner-city census tracts in Syracuse. Thus, to a first approximation, blood lead level data aggregated at the census tract level can be considered as independent from each other. A more extensive aggregate spatial analysis needs to be performed at a much finer scale of geographic resolution.

The combined effects of age, season, and geographic location on geometric mean paediatric blood lead levels are shown in Table 1. Census tract stratification was by 'low', 'intermediate' and 'high' locations as indicated in Figure 4. The age groupings divided the 1 and 2 year old children into separate classes on the assumption that they exhibit the most frequent 'hand-to-mouth' activity, and defined a third class for the 0, 3, and 4 year old children. Season was divided into Winter (W = months 1–4), Summer (S = months 7–10) and Spring/Autumn (SA = months 5–6 and 11–12). The numbers in the parentheses are the numbers of children in each subgroup. These data show that not only do the 2 year old children exhibit the highest blood lead values, but also the greatest geographic differences and seasonal variation. The amplitude of seasonal variation depends upon age, in agreement with the observations of Hammond *et al.* (1983) and Baghurst *et al.* (1992) and, in Syracuse, it depends upon geographic location.

In the absence of a substantial database for the geographic distribution of environmental lead levels in Syracuse, it is not yet possible to examine the detailed relationship between PbB and lead in soils and house dusts. However, blood lead levels

Table 1 *Geometric mean blood leads stratified by census tract group, age, and season. Geographic stratification is: Low CT's = PbB < 6 μg dl^{-1}, Med CT's = PbB 6–10 μg dl^{-1}, Hi CT's = PbB > 10 μg dl^{-1}. Seasonal stratification is: w = January – April; S = July – October; SA = May, June, November, December. Units are μg dl^{-1} (n = population tested).*

		Season		
	Age	W	SA	S
Low CT's	0, 3, 4	5.1 (15)	5.6 (112)	5.7 (20)
	1	5.0 (8)	5.8 (81)	5.7 (31)
	2	5.9 (17)	5.9 (73)	6.1 (26)
Medium CT's	0, 3, 4	6.2 (58)	6.2 (320)	8.2 (100)
	1	7.2 (37)	8.2 (300)	9.4 (81)
	2	8.6 (45)	9.2 (285)	10.2 (82)
High CT's	0, 3, 4	7.1 (20)	6.8 (185)	10.0 (59)
	1	9.8 (25)	10.9 (200)	12.5 (60)
	2	10.4 (31)	14.1 (267)	14.3 (95)

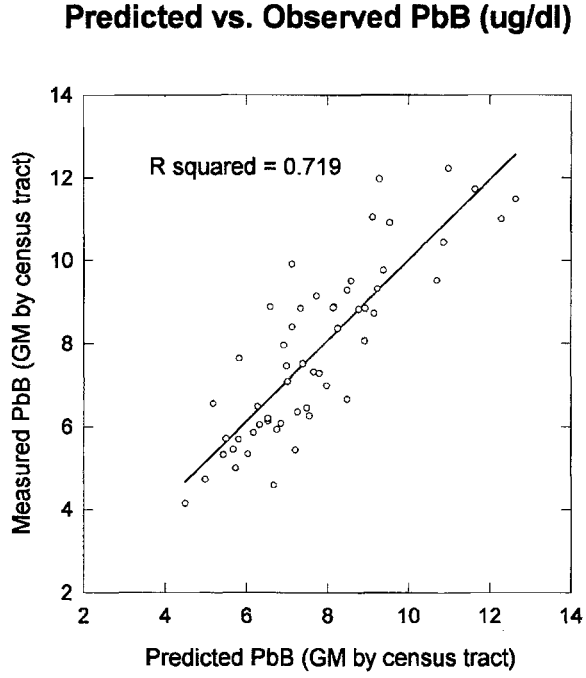

Figure 5 *Comparison of observed and predicted geometric mean blood lead values for 54 census tracts. See text for regression equation.*

do show a correlative relationship to demographic variables associated with housing stock. As a preliminary form of exposure model, an unweighted multiple linear regression model was developed using the geometric mean census tract PbB as the dependent variable and 26 possible demographic parameters (age, sex, race, fraction of rented housing, value and age of property *etc.*) extracted from the 1990 Census summary data as independent variables. In attempting to obtain the maximum correlation coefficient with SAS Proc Stepwise, Mallow's guideline comparing the C_p statistic with the number of variables to be included in the optimum model indicated that five variables should be used. The independent regressors were: the lower quartile (VLQ) and the median (VMED) housing unit value in dollars; the upper quartile of rent paid (RUQ) in dollars/month; and the fraction of housing in each census tract built before 1949 (OLD), and that built between 1949 and 1985 (MED). The equation for this regression model is:

$$\begin{aligned} \mathrm{PbB} = &-4.97 - 0.000277(\mathrm{VLQ}) \\ &+ 0.000135(\mathrm{VMED}) \\ &+ 0.01269(\mathrm{RUQ}) + 14.234(\mathrm{OLD}) \\ &+ 10.905(\mathrm{MED}) \end{aligned} \quad (1)$$

The adjusted r^2 value for the correlation is 0.7144.

A plot of the observed census tract geometric mean blood lead levels *versus* those predicted by the 'demographic' exposure model above is shown in Figure 5. The significance of the relationship expressed in Equation 1 lies not in its potential for prediction, but rather in the qualitative aspects of childhood lead exposure patterns which it suggests. If we imagine the value of the rent contract as an indicator of socio-economic status and property value to be a surrogate for housing condition, and consider the geographic distribution of those parameters, Equation 1 indicates that blood lead levels are at least partially a function of where people can afford to live.

This is not a new hypothesis; it could be formulated from the results of a number of previous investigations. The geographic clustering of elevated blood leads has been shown in Newark, NJ (Guthe *et al.* 1992) and in Philadelphia, PA (M. Dakins, personal communication). Age and condition of housing stock are known to influence measures of lead exposure (Brunekreef *et al.* 1983; Chisolm *et al.* 1985; Clark *et al.* 1985; Binns *et al.* 1994). While direct causal relationships remain elusive, the influence of socio-economic factors on PbB levels has been demonstrated by the investigations of Bornschein *et al.* (1985) and Lanphear *et al.* (1994). All these observations, together with the results of the present study, suggest that exposure measures from monitoring programmes could be combined with geo-referenced data for lead in environmental media and merged with socio-demographic data to create childhood lead exposure models.

Seasonal variation in exposure patterns may be an important consideration in the development of such exposure models. Hunter (1977) has argued that blood lead levels may be related to vitamin D metabolism; but his data also show a relationship between seasonal variation in air lead and blood lead levels. More recently, Rosen *et al.* (1980) and

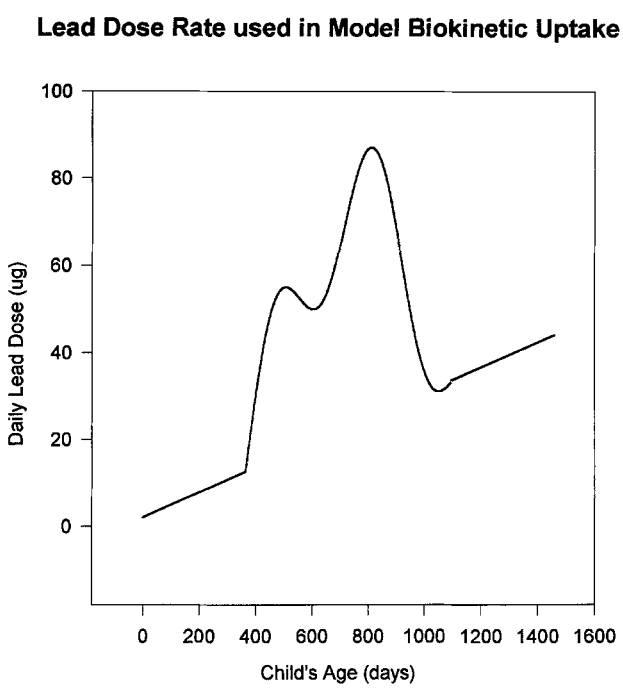

Figure 6 *Pattern of total daily lead intake (μg day^{-1}) used in biokinetic uptake model of seasonal variation in blood lead values.*

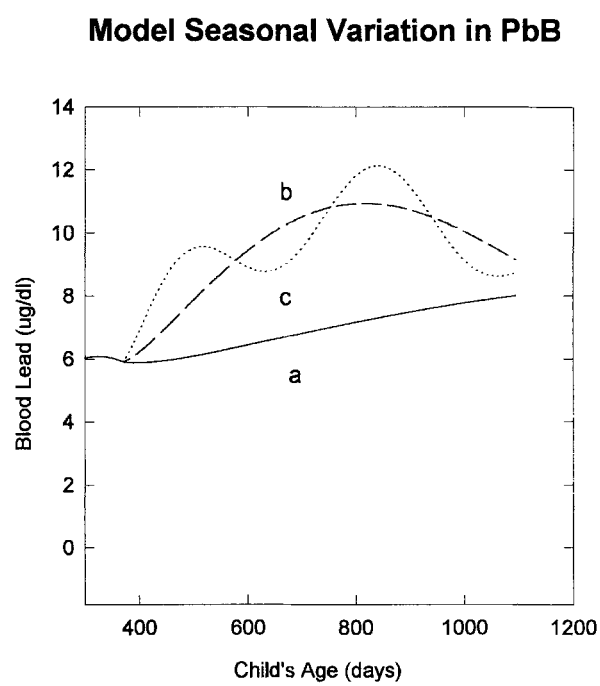

Figure 7 *Predicted blood lead values using Leggett's biokinetic uptake model. Curve (c) results from the dose pattern illustrated in Figure 6. See text for explanation of other curves.*

Mahaffey *et al.* (1982) have shown that serum concentrations of 1,25-dihydroxy vitamin D do not fluctuate on a seasonal basis, although the precursors do. In Syracuse, it seems possible that climatic variations might have a significant effect on exposure pathways. During 4–5 months of the year, not only is the ground frozen but snow cover limits contact with lead-contaminated soils. In effect, a potential exposure source is 'switched on and off' in a seasonal cycle. We employed Leggett's age-specific biokinetic uptake model to explore what sorts of lead exposure patterns could result in the observed temporal variation in blood lead levels.

We are not aware of extensive applications of the Leggett model beyond the tests given by the author (Leggett, 1993) in the development work. When operated with the EPA LEAD99D default conditions (setting bioavailability for all media to 50%) it gives results for blood lead predictions about twice as high as the EPA model. That is, for children in the 0–5 year old age group, the Leggett model lead absorption fraction must be in the range of 20–25% in order to give comparable results. In our initial implementation, we established an exposure regime to simulate that of the Birmingham cohort (Davies *et al.*, 1990). Water and dietary lead intake was increased in a linear fashion from 2 to 44 μg day^{-1} over the period from birth to 4 years of age (23 μg day^{-1} at age 2 years). Soil and dust ingestion was taken as a sine function increasing from 0 μg day^{-1} at age 1 to a maximum of 42 μg day^{-1} at age 2 years and decreasing again to 0 μg day^{-1} at age 3 years – one half of a 4 year cycle. Under these conditions, the predicted blood lead value for 2 year old children reached 11.6 μg dl^{-1} (compared to 11.7 μg dl^{-1} observed) when the age-specific lead absorption fraction in the model was 50% at age 100 days, 25% at age 1 year, and 16% at age 5 years.

As a model seasonal exposure pattern for Syracuse, we used the age-specific lead absorption fractions and the dietary input described above. Soil and dust ingestion was a sine function of period 4 years, starting at age 1 year, reaching a maximum of 50 μg Pb day^{-1} at age 2 years, and declining again to 0 at age 3 years. Superimposed on this was an additional sine function input with an amplitude of 30 μg Pb day^{-1}, a frequency of 1 year, and applied for the age period 1 to 3 years. The dose pattern for this model is shown in Figure 6. The resulting blood lead value predictions from the Leggett model operated in this fashion are shown in Figure 7; curve (a) shows the predicted blood lead values from the background dietary inputs, curve (b) indicates the predicted PbB levels from diet plus soil and dust ingestion increasing smoothly to a peak value of 65 μg Pb day^{-1} at age 2 years, and curve (c) illustrates the model blood lead predictions for the seasonally modulated inputs described by Figure 6. These predictions are, of course, model specific; much further investigation is necessary in order to address their potential accuracy. However, the qualitative results are clear; seasonal variation in blood lead levels similar to those observed for children in the 'high blood lead' census tracts can be produced by seasonal variations in soil and dust ingestion patterns.

Conclusions

Blood lead levels in Syracuse, New York show substantial temporal and spatial distribution structure. Maximum variations were observed for children in the 1–2 year age group, and temporal variations were greater in geographic areas where geometric mean blood lead values were higher. After geographic stratification, PbB values were significantly correlated with housing stock variables; particularly with the age of the residential property. Preliminary application of a biokinetic uptake model suggests that temporal variations in blood lead values could be explained by seasonal patterns of exposure to lead-contaminated soils and dusts. Together these observations suggest the feasibility of using blood lead screening programme data in combination with geo-referenced measures of lead in environmental media in order to develop models of the childhood lead exposure dynamic.

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References

Baghurst, P.A., Tong, SL., McMichael, A.J., Robertson, E.F., Wigg, N.R. and Vimpani, G.V. 1992. Determinants of blood lead concentrations to age 5 years in a birth cohort study of children living in the lead smelting city of Port Pirie and surrounding areas. *Archives of Environmental Health*, **47**, 203–210.

Binns, H.J., LeBailly, S.A., Poncher, J., Kinsella, T.R. and Saunders, S.E. 1994. Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. *Pediatrics*, **93**, 164–171.

Blatt, S.D. and Weinberger, H.L. 1993. Prevalence of lead exposure in a clinic using 1991 Centers for Disease Control and Prevention recommendations. *American Journal of Diseases in Children*, **147**, 761–763.

Body, P.E., Inglis, G., Dolan, P.R., and Mulcahy, D.E. 1991. Environmental lead: a review. *Critical Reviews of Environmental Control*, **20**, 299–309.

Bornschein, R.L., Succop, P., Dietrich, K.N., Clark, C.S., Que Hee, S., and Hammond, P.R. 1985. The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. *Environment Research*, **38**, 108–118.

Brinkmann, R. 1994. Lead pollution in soils adjacent to homes in Tampa, Florida. *Environmental Geochemistry and Health*, **16**, 59–64.

Brisco, M., Pierre, K., and Mielke, H.W. 1994. Hand wipes as a measure of lead exposure: differences between inner-city and non-inner-city locations. Xavier University of Louisiana, New Orleans, LA.

Brody, D.J., Pirkle, J.L., Kramer, R.A., Flegal, K.M., Matte, T.D., Gunter, E.W. and Paschal, D.C. 1994. Blood lead levels in the US population, phase 1 of the third national health and nutrition examination survey (NHANES III, 1988-1991). *Journal of the American Medical Association*, **272**, 277–283.

Brunekreff, B., Noy, D, Biersteker, K. and Boleij, J. 1983. Blood lead levels of Dutch city children and their relationship to lead in the environment. *Journal of the Air Pollution Control Association*, **33**, 872–876.

Chisolm, J.J., Mellits, E.D., and Quaskey, S.A. 1985. The relationship between the level of lead absorption in children and the age, type, and condition of housing. *Environment Research*, **38**, 31–45.

Clark, C.S., Bornschein, R.L., Succop, P., Que Hee, S.S., Hammond, P.B. and Peace, B. 1985. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environment Research*, **38**, 46–53.

Davies, D.J.A., Thornton, E., Watt, J.M., Culbard, E.B., Harvey, P.G., Delves, H.T., Sherlock, J.C., Smart, G.A., Thomas, J.F.A. and Quinn, M.J. 1990. Lead intake and blood lead in two-year-old U.K. urban children. *Science of the Total Environment*, **90**, 13–29.

Duggin, M.J. and Inskip, M.J. 1985. Childhood exposure to lead in surface dust and soil: a community health problem. *Public Health Review*, **13**, 1–54.

Guthe, W.G., Tucker, R.K., Murphy, E.A., England, R., Stevenson, E. and Luckhardt, J.C. 1992. Reassessment of lead exposure in New Jersey using GIS technology. *Environment Research*, **59**, 318-325.

Hammond, P.R., Bornschein, R.L., Succop, P.A. and Clark, C.S. 1983. Profiles of lead burdens in early childhood, a longitudinal study in a high risk environment. *International Conference on Heavy Metals in the Environment*, Heidelberg, Vol. 1, pp 262–268. CEP Consultants.

Hunter, J.M. 1977. The summer disease; an integrative model of the seasonality aspects of childhood lead poisoning. *Journal of the Society of Science and Medicine*, **11**, 691–703.

Hwang, YH. and Wang, JD. 1990. Temporal fluctuation of the lead level in the cord blood of neonates in Taipei. *Archives of Environmental Health*, **45**, 42–45.

Kimbrough, R.D., LeVois, M. and Webb, D.R. 1994. Management of children with slightly elevated blood lead levels. *Pediatrics*, **93**, 188–191.

Lanphear, B.P., Weitzman, M., Tanner, M., Clarkson, T. and Winter, N.L. 1994. *The relation of lead-contaminated house dust and blood lead levels among urban children.* University of Rochester, Rochester, New York.

Leggett, R.W. 1993. An age-specific kinetic model of lead metabolism in humans. *Environmental Health Perspectives*, **101**, 598–616.

Leggett, R.W., Eckerman, K.F., and Williams, L.R. 1993. An elementary method for implementing complex biokinetic models. *Health Physics*, **64**, 260–271.

Mahaffey, K.R., Rosen, J.F., Chisney, A.W., Peeler, J.T., Smith, C.M., and DeLuca, H.F. 1982. Association between age, blood lead concentration, and serum 1,25 dihydroxy cholecalciferol levels in children. *American Journal of Clinical Nutrition*, **35**, 1327–1331.

Mielke, H.W., Anderson, J.C., Berry, K.J., Mielke, P.W., Chaney, R.L. and Leech, M. 1983. Lead concentrations in inner-city coils as a factor in the child

lead problem. *American Journal Public Health*, **73**, 1366–1369.

Mielke, H.W., Adams, J.E., Huff. B., Pepersack, J., Reagan, P.L., Stoppel, D. and Mielke, P.W. 1991. Dust control as a means of reducing inner-city childhood Pb exposure. *Trace Substances in Environmental Health*, **25**, 121–128.

Mielke, H.W. 1993. Lead dust contaminated U.S.A. communities: comparison of Louisiana and Minnesota. *Applied Geochemistry* **Suppl Issue 2**, 257–261.

National Research Council 1993. *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations*. National Academy Press, Washington, DC.

Pirkle, J.L., Brody, D.J., Gunter, E.W., Kramer, R.A., Paschal, D.C., Flegal, K.M. and Matte, T.D. 1994. The Decline in blood lead levels in the United States, the national health and nutrition examination surveys (NHANES). *Journal of the American Medical Association*, **272**, 284–291.

Onyari, J.M., Wandiga, S.O., Njenga, G.K. and Nyatebe, J.O. 1991. Lead contamination in street soils of Nairobi City and Mombasa Island, Kenya. *Bulletin of Environmental Contamination and Toxicology*, **46**, 782–789.

Rosen, J.F., Chisney, A.W., Hamshar, A.J., DeLuca, H.F. and Mahaffey, K.R. 1980. Reduction in 1,25-dihydroxy vitamin D in children with increased lead absorption. *New England Journal of Medicine*, **302**, 1128–1131.

Thornton, I., Davies, D.J.A., Watt, J.M. and Quinn, M.J. 1990. Lead exposure in young children from dust and soil in the United Kingdom. *Environmental Health Perspective*, **89**, 55–60.

Rabinowitz, M.B., and Needleman, H.L. 1982. Temporal trends in the lead concentrations of umbilical cord blood. *Science*, **216**, 1429–1431.

Rabinowitz, M.B., Needleman, H.L., Burley, M., Finch, H. and Rees, J. 1984. Lead in umbilical blood, indoor air, tap water, and gasoline in Boston. *Archives of Environmental Health*, **39**, 299–301.

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