population groups in the sample would lead to lower blood-lead values near the end of the survey.

If, in spite of this concern about the adequacy of evaluating time trends within the NHANES II study, the data are utilized to investigate time trends, then the data must be looked at on a multivariate analysis basis. The statistical technique of multivariate correlation can be used to evaluate the contribution of the various variables from the NHANES data that are likely to affect blood-lead levels. Analysis of the data shows that variables significant in relation to blood-lead concentration are sex, race, age, degree of urbanization, income level, region of the U.S., quarter of the year, and time. It is necessary to consider these variables as discrete variables in an analysis because they are not continuous with time. These variables are taken directly from the NHANES II data. Lead usage data for this period is reported by the EPA and is included in the analysis as a continuous variable with the quarterly average usage for the USA.

Table I-5 shows the coefficients for these variables. For example, the coefficient of -3.47 for a male discrete variable base, and a female discrete variable means that females show an average blood lead concentration of 3.47 μg Pb/100 ml less than males with all other variables held constant. Blacks have a mean blood lead of 3.26 μg/dL higher than whites, while children 0-6 years of age have a mean blood lead of 3.71 μg/dL higher than 7 to 18-year-old youth, and 1.89 μg/dL higher than 19 to 74-year-old adults. If we look at degree of urbanization, the coefficients indicate that there is not a significant blood-lead difference between those persons living in highly urbanized areas of 1 million plus population (NHANES urbanization categories 1 and 2) and those persons living in urbanized areas of less than 250,000 to 1 million (NHANES urbanization categories 3 and 4). The average blood-lead difference between residents of 1 million plus population groups and populations with 2,500 to 25,000 plus (NHANES urbanization categories 5, 6 and 7) population outside urbanized areas is 0.55 μg/dL. The average blood lead for residents of rural areas (NHANES urbanization category 8) is 1.0 μg/dL less than for areas with more than 1 million plus population. It is well known that the difference in exposure to automotive lead emissions, lead usage in gasoline, or lead in air, is many times higher in urbanized areas than in rural areas. However, the difference in lead in blood between residents of highly urbanized areas and rural areas is only 1 μg/dL, an insignificant difference.

If discrete multivariate analyses are also carried out for individual years, the results shown in Table I-6 are obtained. The results continue to show the differences due to sex, race, income level, and age. However, for the four years, there are no consistent differences between the highly urbanized populations and rural populations. The difference in lead usage in gasoline between highly urbanized areas and rural areas is much greater than the difference in total lead usage between 1976 (beginning of the NHANES II survey) and 1980 (end of the NHANES II survey).. Yet the urban-rural difference in lead in blood is only 1 μg/dL, while the difference obtained from

TABLE I-5

Coefficients from Multivariant Correlation of Blood Lead Concentration Data

Discrete Variable Base	Discrete Variable	Coefficient Blood Pb, μg/100 ml	Standard Deviation
Male	Female	-3.47	0.12
White	Black	3.26	0.19
Region 1	Region 2	-0.18*	0.23
Region 1	Region 3	-1.52	0.26
Region 1	Region 4	0.40**	0.30
Population 1 MM +	Population < 250 M to 1 MM	0.09*	0.18
Population 1 MM +	Population 2.5 M to 25 M+	-0.55	0.20
Population 1 MM +	Rural	-1.00	0.18
Age 0-6 years	Age 7-18 years	-3.71	0.18
Age 0-6 years	Age 19+ years	-1.89	0.14
Income under $6,000	$6,000 to $15,000	-0.91	0.16
Income under $6,000	$15,000 plus	-1.72	0.17
Quarter 1	Quarter 2	0.08*	0.20
Quarter 1	Quarter 3	-0.17*	0.26
Quarter 1	Quarter 4	0.58**	0.27
1976	1977	-1.79	0.28
1976	1978	-1.48	0.28
1976	1979	-3.47	0.42
1976	1980	-3.33	0.77
Continuous Variable			
Pb usage, 10^9 g/quarter		0.07922	0.026

* Not statistically significant.
** Marginal statistical significance.

TABLE I-6

Coefficients from Multivariant Correlation

of Blood Lead Concentration Data by Year

Discrete Variable Base	Discrete Variable	1976 Coefficient Blood Pb, µg/100 ml	1976 Standard Deviation	1977 Coefficient Blood Pb, µg/100 ml	1977 Standard Deviation	1978 Coefficient Blood Pb, µg/100 ml	1978 Standard Deviation	1979 Coefficient Blood Pb, µg/100 ml	1979 Standard Deviation
Male	Female	-3.39	0.28	-3.74	0.24	-3.45	0.22	-3.45	0.19
White	Black	4.19	0.41	2.59	0.39	3.25	0.41	2.36	0.32
Region 1	Region 2	-	-	-	-	-0.59*	1.27	-1.57	0.41
Region 1	Region 3	-	-	0.27*	0.47	-	-	-2.50	0.47
Region 1	Region 4	-	-	-	-	-	-	-3.10	0.64
Population 1 MM +	Population <250 M to 1 MM	1.53	0.55	-0.26*	0.44	-0.64	0.14	-0.13*	0.17
Population 1 MM +	Population 2.5 M to 25 M+	0.05*	0.56	0.76	0.47	0.01*	0.14	-0.45	0.14
Population 1 MM +	Rural	-0.19*	0.47	-1.49	0.43	-0.10*	0.12	-0.16**	0.11
Age 0-6 years	Age 7-18 years	-4.65	0.42	-3.50	0.37	-3.62	0.36	-2.88	0.31
Age 0-6 years	Age 19+ years	-3.13	0.33	-1.66	0.29	-1.69	0.28	-1.11	0.24
Income under $6,000	$6,000 to $15,000	-1.60	0.36	-0.54	0.33	-0.92	0.33	-0.71	0.27
Income under $6,000	$15,000 plus	-2.25	0.40	-1.37	0.35	-2.15	0.34	-1.32	0.27
Quarter 1	Quarter 2	-0.56*	0.69	2.15	0.62	0.53*	0.38	-0.65	0.57
Quarter 1	Quarter 3	-0.59*	0.72	2.73	0.55	1.26*	1.31	-2.14	0.57
Quarter 1	Quarter 4	-0.25*	0.66	1.74	0.42	1.53	0.39	-2.50	0.42

* Not statiscally significant.

** Marginal statistical significance.

the NHANES data for 1976 and 1980 was approximately 5.5 to 6 μg/dL. Therefore, it seems impossible that this degree of change in blood lead expressed from the NHANES data is due to auto exhaust lead. Additionally, the change in air-lead concentration over the period 1977 to 1980, based on the average of all EPA non-point source sampling sites, was less than 0.5 $\mu g/m^3$. These data are fully described in Section III. Based on an air-lead to blood-lead ratio even as high as 1:2, the resulting decrease of lead in blood should not exceed 1 μg/dL. It seems improbable that the 6 μg/dL decline in blood lead could be due to decline in lead usage during 1976-1980 period.

The acceptance of the explanation for the decline in blood-lead values in the NHANES data as being due to gasoline lead ignores the following:

1. Lead in adult canned food has been reduced by approximately 35% over the period 1974 to 1979. This decrease resulted from improvements in the production, cleaning up of canmaking lines, and improved lead-soldering techniques.

2. Lead in infant foods has been reduced even more than in adult foods over a similar time period. For example, lead in infant formula was reduced from 0.055 ppm in 1976-77 to 0.02 ppm in 1979-80. Similar decreases were made in infant juices, solid foods, and evaporated milk. (Paper by C. F. Jelinek, "Levels of Lead in U.S. Food Supply," Presented at Analytical Methodology for Lead in Foods Symposium, Association of Official Analytical Chemists Meeting, October 19, 1981, Washington, D.C.)

3. A large effective media educational program has been carried out to alert urban parents about the lead-paint problem. This program has been effective in reducing, but not eliminating, the number of young children found with elevated blood-lead levels.

4. There have been other extensive programs to reduce exposures to lead from ceramic ware, plumbing, and many other consumer products.

In conclusion, the claim that the reduction in blood-lead levels over the period 1976 to 1980 can be attributed primarily to decreased usage of lead in gasoline cannot be substantiated.

D. Significance of Vehicle Lead Emissions in Dust, Dirt, and Soil

Several years ago, opponents of the use of lead in gasoline speculated that inhalation of airborne lead represented a major contribution to total lead intake. As solid scientific data developed, it became obvious that direct inhalation of airborne lead at levels to which the public is generally exposed results in a relatively small contribution to total lead intake. When it became apparent that direct inhalation of airborne lead was not a major contributor to lead intake, the emphasis shifted to the speculation that airborne lead falling out into dust and dirt or onto food and water and consequently being ingested represented a significant source of lead intake. While there has been considerable speculation about the contribution of lead fallout, few scientific data have been generated to quantify this contribution.

Pathways by which lead from fallout could contribute to total lead intake include hand-to-mouth activity, especially in young children, and entry into the food chain by direct contamination of food products or uptake by edible plants. To determine the significance of increased lead in dust and soil, Barltrop[1] studied populations in villages with varying amounts of lead in soil. Blood-lead levels in children and their mothers living in rural areas with minimal airborne lead but high levels in soil and dust were reported. The results are shown in Table I-7 for both the child and mother.

TABLE I-7

Soil Lead - Blood Lead Relationship[1]

Soil Lead, ppm	Blood Lead, μg/100 ml	
	Children	Mothers
<1,000	20.7	14.1
1,000-10,000	23.8	18.7
>10,000	29.0	14.8

These results indicate that lead in soil and dust does not influence the adult (mother); however, approximately 1,000 ppm of lead in soil resulted in an increase in the blood-lead level of children of less than 1 μg/100 ml.

Ter Haar and Aronow[2] carried out a study using Pb^{210}, which is a decay product of naturally-occurring radon gas, as a tracer or marker for ingested dust or soil. The levels of lead and the tracer in normal children and children hospitalized because of suspected excessive lead intake were compared. The data, summarized in Table I-8, show that in the children hospitalized because of suspected excessive lead intake there was no evidence of overt ingestion of dust and dirt as indicated by approximately the same

level of Pb^{210}, the tracer for dust and dirt intake, in feces in both groups. However, the output of stable lead in feces was much higher in the group with suspected excessive lead intake, indicating that some lead source rather than dust and dirt was responsible for the excessive lead intake, most likely lead in paint. These findings are consistent with the findings of Barltrop.[1]

TABLE I-8

Lead and Lead-210 in Feces[2]

	Lead		Lead-210	
	μg/g	μg/day	pCi/g	pCi/day
Normal Children				
Average	3.8	79	0.034	0.680
Range	1.2-8.5	22-174	0.016-0.120	0.252-2.185
Median	3.3	76	0.027	0.583
"Lead" Children				
Average	111	1781	0.043	0.540
Range	3.9-1640	8-29,030	0.018-0.102	0.072-1.615
Median	18.0	218	0.037	0.298

The Ter Haar and Aronow findings are confirmed by the more recent work by Yaffe, Flessel et al. Dr. Flessel has given us permission to submit to EPA the prepublication copy of their paper entitled "Identification of Lead Sources in California Children Using Stable Isotope Ratio Technique," attached as an appendix. They found, by the isotopic ratio technique, that the moderately elevated blood-leads of the children studied were due to lead paint sources rather than vehicle lead emissions. One case study carefully examined lead sources in ten children of 3-15 years of age, living together in dilapidated housing close to a busy freeway. Eight children had blood-lead levels of 28-43 μg/dL. Another case study examined sources of lead in two-year-old twins with blood lead levels of 25-43 μg/dL, living in a modest but well-maintained inner-city duplex apartment. Measurements of lead isotopic ratios in gasoline, aerosol, freeway dust, interior paint, indoor dust, exterior paint, and soil along the house, backyard and curb were made. The investigators concluded that the source of lead causing the elevated blood-lead levels in these children was exterior lead-based paints that had contaminated the soil in the yard. This is one of the most exhaustive attempts to identify sources of lead causing low to moderate elevations in blood-lead levels in children.

There are numerous other studies that support the conclusion that fallout lead from vehicle lead emissions is not a significant contributor to

ingested lead. For instance, studies of the relationship between traffic density and/or proximity to highways and blood-lead levels usually show that blood-lead levels are within the normal range. If there are statistically significant differences, the differences are 1-2 μg/dL and of no physiological significance. A study carried out by the California State Department of Health[3] determined blood-lead levels in children living and attending school adjacent to and removed from the San Diego Freeway, near Los Angeles. The school adjacent to the San Diego Freeway carrying 200,000 cars per day was less than 200 feet from the Freeway, while the other two schools were 2400 and 2800 feet from the Freeway. All blood-lead values were less than 30 μg/dL, and proximity of neither school nor residence to the San Diego Freeway had an appreciable effect on lead in blood (Tables I-9 and I-10).

The blood-lead levels of Dallas, Texas residents living near traffic densities ranging from less than 1,000 cars per day to 30,000 cars per day were compared in order to determine whether there was an influence of traffic on lead absorption.[4] The blood-lead levels ranged from 7 to 33 μg/dL for children and from 4 to 21 μg/dL for adults. Air-lead levels increased with traffic density from 0.5 to 1.9 $\mu g/m^3$. However, there was no relationship between traffic density and blood-lead levels of either children or adults. The results of these studies are typical of other studies showing the lack of relationship of blood-lead levels to traffic density.

Dr. Houk, in discussing the NHANES II data at the April 15 Lead Hearings, showed that the decrease in blood-lead over the four-year period was approximately the same for the age groups of 6 months to 5 years, 6 to 17 years, and 18 to 74 years. If vehicle lead emissions make a significant contribution via the ingestion route for children, then one would expect a much greater decrease in blood-lead for the 6 months-to-6 years age group than occurred in the other age groups. The fact that this did not occur supports the conclusion that vehicle lead emissions do not contribute significantly to blood-lead via the ingestion route of dust, dirt, and soil.

References for Section I-D

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3. State of California, Department of Health: Report on Study of Blood Lead Concentration in Culver City School Children, Report to Board of Education by Dr. Margaret Deane, November 1, 1977.

4. Johnson, D. E., Prevost, R. J., Tillery, J. B., Kimball, K. T., and Hosenfeld, J. M.: Epidemiologic Study of the Effects of Automobile Traffic on Blood Lead Levels. EPA-600/1-78-055, Environmental Health Effects Research Document, August 1978.

TABLE I-9

Mean Blood-Lead Levels by Proximity of Home to Traffic[3]

Proximity of Home	May 1977 Survey		January 1978 Survey	
	No. of Children	Blood Lead, µg/dL	No. of Children	Blood Lead, µg/dL
El Marino Children				
<300 ft from traffic	34	14.5	26	14.8
>300 ft from traffic	42	14.0	41	17.2
Farragut Children				
<300 ft from traffic	11	13.1	21	15.0
>300 ft from traffic	14	13.9	22	15.8
Howe Children				
<300 ft from traffic	-	-	13	15.8
>300 ft from traffic	-	-	31	16.2

TABLE I-10

Mean Blood-Lead Levels by School[3]

School	Distance From San Diego Freeway	May 1977 Survey		January 1978 Survey	
		No. of Children	Mean Blood Lead, µg/dL	No. of Children	Mean Blood Lead, µg/dL
El Marino	<200 feet	76	14.2	67	16.3
Farragut	2,800 feet	25	13.5	43	15.4
Howe	2,400 feet	-	-	44	16.1