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Air Lead: Relation to Lead in Blood of Black School Children Deficient in Glucose-6-Phosphate Dehydrogenase

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pendicularly by its thicker end into the other moldavite (at about one third of its length). The other moldavite is flat, oval, and olive green. Measured from the junction, the drop is 20 mm long and 11 mm wide at its maximum width. The oval, flat moldavite is 38 mm long, 27 mm wide, and 8 mm thick. Both parts of double moldavites differ only slightly in their indexes of refraction, the drop having an index of refraction at 20°C for sodium light, $n^{20°}_{\lambda Na}$ of 1.4928, and the oval specimen having an n value of 1.4920. The specific gravity of the whole double moldavite is 2.356. In a benzene immersion under the stereoscopic microscope the two moldavites can be distinctly seen to be thrust into each other. The junction of the two moldavites, examined between crossed Polaroids, is anisotropic with relatively higher interference colors of the first order; the other parts of the joined moldavites display a common anisotropy known in moldavites (*1*). The surficial sculpture is rather coarse in the basic piece and somewhat finer in the plunged drop. The schlieren and elongated vesicles (bubbles) in both parts of the double moldavite are perpendicular to each other (see Fig. 1c). This unique specimen is deposited in P. Horský's private collection of moldavites in České Budějovice, Czechoslovakia.

The double moldavite from Slavče (Fig. 1b) weighs 17.65 g. It is a flat, oval moldavite with a pronounced surficial sculpture and is bottle green in color. A piece of different translucency and color adjoins it on its side (compare Fig. 1, b and d). Even if the more translucent piece is regarded as an extreme case of inhomogeneity of the moldavite matter, we are nevertheless of the opinion that the differing piece is a remnant of another moldavite which struck against the flat, oval moldavite at a time when both were still plastic. After cooling and solidification, the greater part of the moldavite to which this piece belonged was broken off (probably during infall to the earth's surface). Both moldavites forming the double specimen differ not only in color but also in surface sculpture, that of the larger piece being much coarser. This moldavite, which is somewhat bent, measures 11 by 26 by 40 mm, and the adjoining part of the second moldavite is 21 mm long and 3 mm thick. A microscopic study shows that the second moldavite is thrust about 2.5 mm into the basic piece. The moderately arcuate boundary of the junction of the pieces does not show any sculpture. Both pieces are also distinguished by their indexes of refraction. For the basic moldavite n is 1.4899, and for the adjacent moldavite fragment it is 1.5013 to 1.5016. The fragment exhibits a higher optical anisotropy (see Fig. 1d). The specific gravity of the whole specimen is 2.341. This double moldavite is in the private collection of moldavites of M. Kos in České Budějovice. It resembles to some extent the two-color moldavite from Lipí (*2*), in which the boundary of the junction of the two differently colored parts is also sharp.

These two finds of double moldavites furnish evidence for an inhomogeneous swarm of moldavites closely preceding their fall. The possibility of a collision was, however, considerably limited because the moldavites fell in the same direction with an equal velocity. We have examined 25,000 pieces of moldavites, but only the two specimens reported here show evidence that there were collisions during the flight and that during this phase of flight the moldavites were probably in a plastic state with somewhat different viscosities corresponding to their size and chemical composition. It is the first time that such moldavites were discovered.

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3. We thank P. Horský and M. Kos from České Budějovice, who found the double moldavites, for the loan of the specimens for study.

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Air Lead: Relation to Lead in Blood of Black School Children Deficient in Glucose-6-Phosphate Dehydrogenase

Abstract. *Forty-four black children at two elementary schools within 0.7 mile of a battery plant had significantly higher* ($P < .001$) *concentrations of lead in their bloods (34.1 ± 9.7, micrograms per 100 milliliters, mean ± standard deviation) than 122 students (26.3 ± 7.1) at seven schools 1 to 3 miles distant; 5 months later there was a comparable difference between red cell lead values (54.1 ± 18.5 versus 37.4 ± 12.6). Among the blacks, those deficient in glucose-6-phosphate dehydrogenase had a higher* ($P < .005$) *concentration of lead in the blood after correction for anemia (32.9 ± 9.7) than the nondeficient (25.7 ± 8.8), and a higher concentration in the red cells (47.3 ± 14.7 as compared to 35.6 ± 15.8,* $P < .001$); *the enzyme effect was independent of geographic location.*

The epidemiologic correlation of atmospheric exposure to lead and blood concentration of lead in the community (*1*) raises the question of individual or genetic factors contributing to an increased concentration of blood lead. We report here an increase in the blood lead of black school children living in Omaha within 1 mile of an emission source, with an independent increase in those children deficient in glucose-6-phosphate dehydrogenase (G-6-PD).

Ambient lead was monitored in central Omaha (*2*) from May through November 1970, with simultaneous studies of the blood lead of inner city black school children to evaluate the possible role of anemia and of G-6-PD deficiency. Air was sampled by means of high volume collectors at five sites, three sites being in proximity to the schools studied and to three known lead emission sources (Fig. 1). Samples were taken at an elevation of 15 feet (4.5 m), and all data are reported as the average of 24-hour samples collected three times weekly (*3*).

In April 1970, 3,400 school children (of over 10,000 volunteers) from both suburban and inner city Omaha were tested for G-6-PD activity by the fluorescent spot technique of Beutler (*4*). Presumptive deficiency was found in 14.5 percent of black males and 2.4 percent of black females, aged 5 to 19 years. Testing was then done in May (at nine inner city schools) of 166 of these black school children for concentration of lead in the blood (*5*), G-6-PD activity (*6*) (44 percent deficient, 18 percent intermediate) with complete blood count and reticulocytes in 79 children. Lead concentrations were corrected for anemia to the mean hematocrit (Hct) of 38 percent by the formula: blood lead concentration (μg/100 ml) $\times$ [38/Hct (%)].

Because of the known effect of both

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lead and G-6-PD deficiency on function of the red blood cell (RBC) membrane (*7*), direct assay of the RBC lead, proposed by many as a more valid index of biotoxicity than whole blood lead (*8*), was done by the same methods (*5*) in November on 79 of the previously studied children plus 29 additional students at these schools; red cell indices and haptoglobins were also determined (*9*). Of the November group, 45 percent were deficient in enzyme; 29 percent had intermediate values.

In May we found that 44 black children at elementary schools 1 and 2, 0.2 and 0.7 mile from battery plant A, had a blood lead concentration (mean ± S.D.), uncorrected for anemia, of 34.1 ± 9.7 μg/100 ml—significantly higher ($P < .001$) than that of 122 black students (26.3 ± 7.1 μg/100 ml), at the five other elementary schools and at the two junior-senior high schools. The hemoglobin of the 29 children tested at schools 1 and 2 was lower than that of the 79 at schools 3 to 9 (12.4 ± 0.9 as compared to 13.1 ± 1.4 g/ml, $P < .005$), but this was primarily age related and independent of location and did not correlate with corrected or uncorrected blood lead. After standardization to the mean hematocrit of 38 percent in the 79 children with complete blood counts (Table 1), the blood lead concentrations at schools 1 and 2 were still higher than at schools 3 to 9.

After standardization for hematocrit, as shown in Table 1, the 32 students deficient in G-6-PD (0 to 4 μ*M* reduction of nicotinamide adenine dinucleotide phosphate per minute per 10^{11} red cells) had higher concentrations of blood lead than the 29 nondeficient (> 15 μ*M*) even though blood lead concentration in the enzyme-deficient group did not differ significantly before correction (29.1 ± 8.1 as compared to 27.3 ± 9.0 μg/100 ml), venous hematocrit (37.1 ± 3.6 as compared to 38.3 ± 4.5 percent), hemoglobin (12.7 ± 1.1 as compared to 13.2 ± 1.4 g/100 ml), mean red cell volume (84.2 ± 5.1 as compared to 82.8 ± 4.9 μm³), serum haptoglobins (51.9 ± 36.4 as compared to 68.6 ± 38.6 mg/100 ml), or reticulocytes.

The geographic distribution for the 108 students tested in November was similar to that of the blood lead in May (Table 1); the 29 students at schools 1 and 2 again had significantly higher concentrations of lead in their red blood cells than the 79 at schools 3 to 9.

The RBC lead of the enzyme-deficient children, 47.3 ± 14.7 μg/100 ml, was higher ($P < .001$) than the 35.6 ± 15.8 μg/100 ml of the nondeficient. A difference was noted in both geographic locations although it was again not significant by the *t*-test for small number of students tested in the high lead area of schools 1 and 2. Two-way analysis of variance, by Doolittle's procedure for cells of unequal size (*10*) showed no significant interaction between variances related to geographic location and enzyme deficiency. The multiple correlation coefficients failed to show any significant correlation of RBC lead, blood lead, or standardized blood lead with any of the hematologic parameters.

The known seasonal variations in blood lead invalidate the regression coefficient of the uncorrected blood lead of children sampled in May, with the hematocrit and their RBC lead in November; but on the assumption that there is a comparable seasonal difference, the regression coefficient for the nondeficient (blood lead μg/100 ml = 7.32 + 0.3685 RBC lead μg/100 ml +

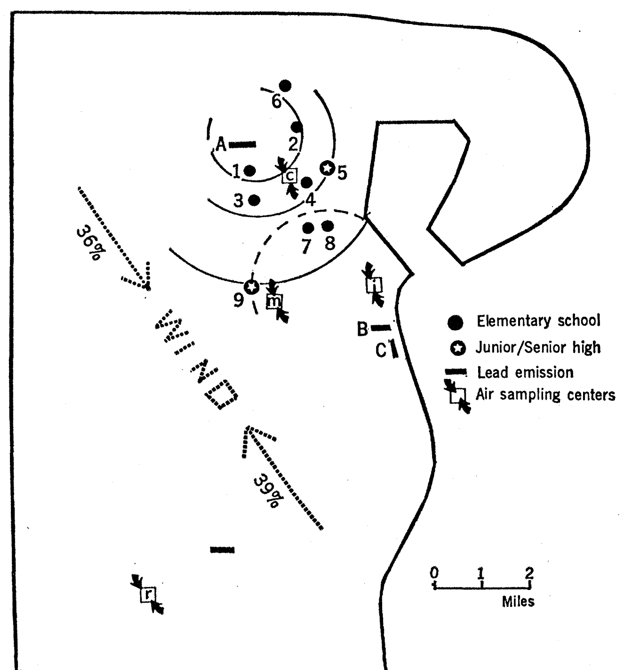

Fig. 1. Outline map of Omaha showing relation of schools 1 to 9 to emission sources A (battery plant), B (lead refinery), and C (battery plant). Air lead (μg/m³, mean ± standard deviation) of monthly composite, May through November 1970, was 1.69 ± 0.28 at commercial site *c*, 2.81 ± 1.04 at industrial site *i*, 1.48 ± 0.35 at mixed commercial - residential site *m*, and 0.79 ± 0.13 at residential site *r*. The prevailing winds and percentage of time observed annually are indicated by the large arrows.

Table 1. The blood lead (May) was corrected to a standard hematocrit of 38 percent, and the red cell lead (November) is expressed as micrograms per 100 ml, mean ± standard deviation. The samples consisted of black school children, aged 5 to 19. The RBC lead group includes 29 children not tested for whole blood lead. Both assays were made with heparinized blood that had been refrigerated for 48 to 72 hours. The G-6-PD activity in micromoles of nicotinamide adenine dinucleotide phosphate reduced per minute per 10^{11} red cells. Student's *t*-test for significant difference of lead concentrations for enzyme deficiency (G-6-PD, 0 to 4 as compared to > 15) and location—schools 1 and 2 within 1 mile of a lead emission source as compared to schools 3 to 9 at 1 to 4 miles. NS, not significant.

G-6-PD	Schools			*t*-Test for location 1 to 2 vs. 3 to 9 (*P*)
	1 and 2	3 to 9	Total	
		Blood lead [μg/100 ml × 38/Hct (%)]		
0–4	38.2 ± 12.8 (9)	30.9 ± 7.1 (23)	32.9 ± 9.7 (32)	< .1
4.1–15	38.3 ± 10.4 (7)	27.2 ± 8.3 (11)	31.5 ± 10.7 (18)	< .05
> 15	27.0 ± 11.2 (5)	25.4 ± 8.2 (24)	25.7 ± 8.8 (29)	NS
Total	35.6 ± 12.6 (21)	27.9 ± 8.2 (58)	29.9 ± 10.2 (79)	< .025
t-Test for enzyme	NS	$P < .025$	$P < .005$	
		RBC lead (μg/100 ml)		
0–4	57.0 ± 15.5 (16)	42.4 ± 11.5 (32)	47.3 ± 14.7 (48)	< .001
4.1–15	52.0 ± 16.4 (17)	36.1 ± 13.1 (15)	41.1 ± 16.2 (32)	< .05
> 15	48.8 ± 25.5 (6)	33.1 ± 11.6 (32)	35.6 ± 15.8 (38)	< .025
Total	54.1 ± 18.5 (29)	37.4 ± 12.6 (79)	41.9 ± 16.2 (108)	< .001
t-Test for enzyme	NS	$P < .005$	$P < .001$	

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0.502 Hct; estimated standard error, 5.91; $r = 0.757$) differs from the deficient (blood lead = 9.25 + 0.3518 RBC lead + 0.197 Hct; estimated standard error, 8.10; $r = 0.448$). On comparison of these two regression coefficients for any given blood lead and hematocrit, the enzyme-deficient group has a consistently higher red cell lead concentration than the nondeficient group (*11*).

The uncorrected mean blood lead, 28.4 μg/100 ml, of the 166 urban black students contrasts with the maximal mean value for urban women of 20.5 μg/100 ml reported by the Seven City Study of 1971 (*12*). Blood lead in the Omaha school children was not related to age, sex, or history of smoking, suggesting a masking effect by other phenomena. Lack of direct correlation between blood lead and air lead at the sampling sites does not invalidate the circumstantial evidence of proximity to a lead emission source since effective concentrations may relate more to particle size and localized climatic conditions than to prevailing winds (*13*).

The mean red cell lead of the 108 urban black school children (41.9 ± 16.2 μg/100 ml) was lower ($P < .001$) than that of 43 adult male garbagemen, ages 20 to 40, with variable exposure to exhaust fumes (53.5 ± 12.9 μg/100 ml) and of 100 lead workers (128.7 ± 41.5 μg/100 ml), but higher ($P < .001$) than that of 32 freshmen nursing and medical students predominantly from rural Nebraska (24.25 ± 7.61 μg/100 ml). In all of these groups, correlation of RBC lead with RBC δ-aminolevulinate dehydratase was comparable to that reported for whole blood lead (*14*).

A possible interaction of G-6-PD deficiency and lead toxicity is supported by (i) reports of acute hemolytic crises in enzyme-deficient subjects (specific type unknown) with only moderate increase in blood lead, by decrease in reduced glutathione in lead workers, and by in vitro studies of progressive decrease of G-6-PD in rats treated with low doses of lead (*15*). In our study, hemolytic effects of lead were found in neither the enzyme-deficient nor nondeficient children.

What was found is that G-6-PD deficient individuals have an apparent increase in blood lead associated with relatively higher concentrations of lead in the RBC and lower in the serum. All subjects were black, and the A^- variant of G-6-PD is the most likely; the effect of specific enzyme types including G-6-PD deficiency among white persons has yet to be studied. The significance of red cell binding is still speculative. It may be protective, as suggested by Goyer (*16*) and by clinical observations of the severe neurotoxicity of lead in anemic children, or it may be an index of biotoxicity, at least for the red cell, as supported by its correlation with δ-aminolevulinate dehydratase (*14*).

If validated, the apparent increase in blood lead in G-6-PD deficient blacks is of potential significance to the 12 percent of black males and 1.4 percent of black females with this genetic variant, most of whom live in inner city areas of high ambient lead (*16*).

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3 May 1972

Protein Absorption by the Intestine of the Fetal Rat in Utero

Abstract. *Horseradish peroxidase (molecular weight, about 40,000) injected into the amniotic sacs in pregnant rats has been identified ultrastructurally, 6 to 18 hours later, within the fetal intestine in the absorptive cells and the underlying vascular endothelium. This indicates that macromolecular protein within amniotic fluid swallowed by the fetus can be absorbed and transported by fetal intestine, and may indicate that physiological compounds can be transported by this enteric route to contribute to fetal development.*

Although the mammalian gastrointestinal tract is not believed to function to any significant degree before birth (*1*), there is evidence that the intestine in the fetus in certain species possesses an absorptive capacity (*2–4*). The passage of macromolecules through the absorptive cells of the intestine has not been demonstrated in the fetus, however, in contrast to the many reports documenting this event in the newborn animal (*5, 6*). We present here ultrastructural evidence of intestinal uptake and transport, in the fetus of the rat, of horseradish peroxidase [a cytochemically demonstrable protein (molecular

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