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J. R. Allen, Primate Center
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D. E. Green, Enzyme Institute

November 21, 1972

Dr. Carol Angle
Dept. of Pediatrics
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University of Nebraska
Omaha, Nebraska 68105

Dear Dr. Angle:

I very much appreciate your sending me a reprint of your paper:

Air Lead: Relation to Lead
in Blood of Black School
Children Deficient in
Glucose-6-Phosphate Dehydrogenase,
Science 177:520 1972

Several of us here are interested in the effects of ingested lead on pregnant females and neonates. We are, therefore, desirous of obtaining as much relevant data as we can on this subject before we begin our investigations. Your findings on a possible causal relationship between blood lead (esp. RBC lead) and G-6-PD is a very good lead for us.

Perhaps you would be kind enough to answer some questions on the G-6-PD-RBC lead relationship.

1. P. 521, paragraph beginning "after standardization for hematocrit etc." I am not clear on the significance of the clause, "even though blood lead concentrations in the enzyme-deficient group did not differ significantly before correction etc."
2. Is your reason for placing school #6 in the 3-9 group because of meteorological conditions, or is it due to some other factor(s)?
3. What is the health status of the most severely G-6-PD depressed children? Are the children in schools 1 and 2 chronically sicker than those in school 3-9?
4. Referring to Table 1, RBC lead figures.
 - a. Are the following differences significant $p < .05$?
 - (1) 57.0 ± 15.5 and 48.8 ± 25.5 ug/100 ml.
 - (2) 57.0 ± 15.5 and 42.4 ± 11.5 ug/100 ml.
 - (3) 42.4 ± 11.5 and 33.1 ± 11.6 ug/100 ml.
 - (4) 48.8 ± 25.5 and 33.1 ± 11.6 ug/100 ml.

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5. Would you expect the same or similar relationship between G-6-PD and RBC lead to exist among white children?
6. What is the health status of the 43 adult male garbagemen and the 100 lead workers, especially the latter because of the high RBC-lead concentration? Would the 128.7 ± 41.5 ug Pb/100 ml RBC exceed 80 ug Pb/100 gm blood?
7. What are your findings from ALAD determinations on the G-6-PD deficient children, ref. 14?
8. Have you found any differences in the ingested lead between children in schools 1 and 2, and 3-9?
9. Have you reported the air lead levels at the various schools yet?
10. What air lead concentrations were the lead workers exposed to?
11. What is your source of "reports of acute hemolytic crises in enzyme-deficient subjects . . . with only moderate increases in blood lead"? .

Your responding to these questions would be most appreciated and very helpful.

Sincerely,

J. Wesley Clayton, Jr., Director
Center for Environmental Toxicology

JWC:js