

November 20, 1987

Doc. 149

School of Medicine

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RE: Ruffin -vs- Sawchyn, et al.

Dear Mr. Kalur:

I have read carefully the October 22, 1986 letter by Dr. H. L. Needleman to Mr. Mellino regarding Deneta Odoms' hospitalization, her treatment for lead poisoning, and the possibility that she is now suffering developmental deficits associated with lead poisoning. I have also read the medical records provided and the reports by Drs. Ownby, Rothner and Drotar and have further discussed her test performance with Dr. Drotar.

Dr. Needleman's concluding paragraph suggests that (a) the child presents severe deficits in cognition and abstract reasoning and (b) that these are consistent with "the residua of severe lead poisoning." These issues are addressed in that order.

Dr. Ownby's examination is faulted by his use of the 4th Edition of the Stanford-Binet Intelligence Scale. This recent revision of a widely respected early test has been severely criticized on the basis of unsatisfactory standardization. Few, if any, of the pediatric psychologists in my acquaintance use it in their assessment of children. Dr. Ownby's report is also insufficient in that he provides a score only for the one scale on which Deneta did poorly; three other scores are described as "average to low average." Data for other aspects of the examination are not provided. It's thus not possible for another psychologist to evaluate the results of the examination.

Dr. Drotar did provide more detailed information on his examination. These results can be viewed from two different perspectives. First, is Deneta's performance appreciably below the average of other children of her age? The answer is yes. It is reasonable to predict she will have difficulties with school achievements and, as Dr. Drotar noted, that she may need to be placed in a special academic program.

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Second, should she be considered to have severe deficits that might be linked to her history of lead poisoning? To answer this we pose the hypothetical question, how might this child perform if she had not had the episode of lead poisoning? We have information on some of the conditions that might be related to her ability level. She is black, she has sickle cell anemia, she has had iron deficiency anemia, her mother is deceased, and she was not enrolled in a preschool program. We note also that her verbal skills at the time of and shortly after her treatment were reasonably appropriate or even somewhat advanced for her age (2½ years) as shown in nurse's notes and other notes in the medical record. She was also daytime toilet trained, though she showed a not-surprising regression during treatment.

The most critical factor to consider in interpreting present test results is race. It is an unfortunate fact that black children in our society perform, on the average, appreciably more poorly on academic tasks and the intelligence tests that predict these tasks than do white children. A determination of whether this difference is due to heredity, cultural differences, the wording and content of the tests, lower self-expectations, anxiety in the presence of a white examiner, or a complex interaction among these, is not needed for this discussion. The norms used by Drs. Ownby and Drotar when they gave percentile figures are based on standardization groups that are primarily white. The deviation from peers suggested by the norms would be appreciably less if "peers" was defined as black children.

While the data from my own research were not collected for this purpose and may be somewhat biased, the extent of racial effects is still clear. These were children born at Cleveland Metropolitan General Hospital, a hospital that serves indigent people of both races. The test was the WPPSI, which is the test used by Dr. Drotar. The children were at age four years, ten months. The mean (average) IQ for 171 white children was 90.9; that for 87 black children was 82.6. Deneta's score is quite consistent with the mean score for the black children.

One might ask whether the verbal skills presented at a younger age, as the January 1985 note: "excellent verbal command for a toddler" would suggest that she should be at a higher level than is now seen. The evidence from research is that culturally related decreases in test scores become increasingly apparent in the later preschool years. For example, in the above mentioned data from my own study, scores decreased from 112 at one year to 102 at two years to 90 at three years. This finding is generally seen in studies of disadvantaged and/or black children.

Deneta was described as having iron deficiency anemia. There is evidence that iron deficiency anemia is associated with cognitive deficit. This may also be related to her performance.

Sickle cell trait does not appear to be related per se to developmental deficit. There is little in the literature on the effect of sickle cell anemia. Certainly the associated bouts of illnesses are not beneficial.

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Maternal death is another rare circumstance that may have adversely affected Deneta. There is little literature on the matter. It is interesting that the medical record noted that Deneta and her mother were very close and that the mother's behavior was loving and appropriate.

The lack of preschool education may have contributed to performance problems in Deneta's test results. Dr. Drotar noted that her areas of poorest performance involved organizing blocks, drawing, writing her name, recognizing letters and numbers and counting. If the home does not provide stimulation in this regard, preschool education may be compensatory. In my research, mentioned above, the 55 black children who had attended preschool had a mean IQ of 85.0; those that had not had a mean of 78.5. (Once again, the data are illustrative since the study was not designed to test this question.)

In view of the above noted considerations Deneta's performance in Dr. Drotar's examination is not remarkable.

The second issue bearing on the inference that Deneta suffers "severe deficits in cognition and abstract reasoning" as a result of lead poisoning depends on evidence relating such deficit to lead exposure. There is little question that high level lead exposure can lead to cognitive deficits. At the same time, there is little information about the amount of such deficit when other conditions are taken into account, nor is there much information about the circumstances that can influence whether a given child is affected. Most of the available research is old and was conducted without the care we now employ in terms of methodology. For example, most studies did not consider other conditions in the child's circumstances that are related both to lead exposure and to cognitive development. Thus an effect attributed to lead can actually be due, or due in part, to these other conditions. These conditions include parent intelligence, race, stimulation in the home and/or schools, nutrition, history of maternal substance use (drugs, alcohol, cigarettes) in pregnancy, other health problems, and so on. Without careful consideration of these and other methodological issues it is not possible to describe the extent to which cognitive deficit can be attributed to lead exposure for groups or for individuals.

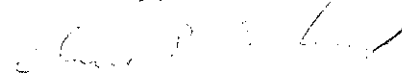
Current ongoing studies of low lead exposure are generally better with respect to methodology and measurement. Results of some of these are being interpreted as demonstrating an effect at low exposure levels. If these inferences are valid, one could argue by extrapolation that there would be a greater effect at the level of exposure suffered by Deneta. Unfortunately, even these studies suffer from some methodological limitations that leave a degree of uncertainty about the interpretation of the very small effects reported. My own study of blood lead sampled from birth through age three years and of cognitive development from age six months through age four years, ten months does not support a conclusion that low level lead exposure has a statistically significant effect on early child development.

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It is not possible to rule out absolutely the possibility that Deneta was adversely affected by lead poisoning. Nevertheless, the fact that her performance is not remarkable in view of other conditions and that the evidence in research reports is unclear, it is possible to say that if she has suffered deficits from lead exposure, these effects are quite small.

Sincerely,



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CBE/dhp