

NEUROPSYCHOLOGICAL EFFECTS OF LEAD
SEPARATING SCIENCE FROM SPECULATION

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Introduction:

The scientific method, which most of us learned in high school, is a specific technique used by scientists to generate scientific information. To oversimplify, the scientific method has two major components, Hypothesis Generation, and Hypothesis Testing.

Typically, a hypothesis is generated when a scientist *notices* some apparent relationship between events, or when the results of experimentation have suggested additional or even unrelated areas for discovery. After formulating his hypothesis, the scientist goes about the process of *hypothesis testing*, which involves using acceptable experimental paradigms and techniques including statistical analysis, to determine whether the hypothesis can be supported. Notably, scientists do not speak of *proving* a hypothesis. Rather they used techniques of statistical analysis to assist in determining whether the results of a particular experiment are more or less likely to reflect a true relationship.

Once a relationship has been supported by a particular experiment. The scientific community then begins the process of *replication*, or doing other similar experiments to see if the original result can duplicated. Over time, and with repeated experiments, evidence may mount and scientists will feel increasingly comfortable in endorsing a hypothesis. Often however, additional and repeated experiments will yield findings which are directly contradictory. In such cases the evidence for or against a particular finding is

said to be *equivocal* and opinions which represent only one side in such a controversy constitute speculation rather than science.

In recent years, a methodology called meta analysis has been developed to enable us to combine the results of studies and thereby draw somewhat stronger conclusions. It is the conclusion of those meta analyses, combined with findings which appear to be repeatedly replicated which represent what we truly know about the neuropsychological effects of lead.

II. The Allegations:

In typical lead paint suit, the plaintiff's allege that exposure to lead has resulted in some or all of the following neurocognitive deficits:

- Lowered IQ (including mental retardation)
- Learning Disabilities
- Language Disabilities
- Visual Motor Problems
- Attention and Executive System Dysfunction
- Visual problem solving dysfunctions

More recently, behavioral problems including delinquent and criminal behavior have begun to make an appearance as being causally related to lead exposure.

III. The Evidence

Of all the neurocognitive and behavioral findings attributed to lead exposure at non acute levels, the only one which passes the test of repeated replication and meta analysis is the finding that in groups of children, the groups with higher lead levels tended to have IQ scores that were from 1-4 points lower than those with lower lead levels. These findings have been remarkably consistent over the last five years and are amply described in the meta analyses by Joel Schwartz and Pocock and Smith.

In the Pocock and Smith study, the authors concluded that “a typical doubling of body lead burden (from 10-20 mg/dl...) blood lead... is associated with a mean deficit in full scale IQ of 1-2 points. Similarly Schwartz concluded that “an increase in blood lead from 10-20 mg/dl was associated with a decrease of 2.6 IQ points in the meta analysis.

Curiously Schwartz also found that in disadvantaged populations, the effect was smaller (1.85 IQ points). He suggests that in these populations, other factors may have “already impaired the pathways most sensitive to lead.” It is important to note that this level of change is within the normal expected intradividual variation in IQ testing and is of absolutely no functional consequence in an individual child.

A number of authors and experts have seized upon these findings as concluding that, for each 10 mg rise in blood lead over 10 mg, there is a corresponding decrease in IQ of about 2 points. This however represents a misreading of the studies. Indeed Schwartz argues that the findings are not a simple linear dose response relationship, but rather reflect a logarithmic relationship which may be U or Wave shaped. He further states that his findings suggest that “the effect of each incremental increase in lead exposure diminishes.”

Findings related to other alleged injuries are far more equivocal. While a number of studies have indicated that children with higher exposure levels demonstrate more significant problems with attention. Others have not found this effect. Indeed, even David Bellinger who is perhaps one of the most well known and well respected authors in the field has remarked that “there is, however little support for the hypothesis that low level lead exposure increases a child’s risk of attention deficit hyperactivity disorder.”

Indeed, the literature in the area of attention deficit disorder notes the presence of strong familial relationships which lead a number of researchers to conclude that a large number of children with ADHD have acquired this on an inherited basis. A form of ADHD is also associated with CNS insults of prematurity, head trauma, and early neurological disease. In a majority of children, the hypothesized cause is varied and most are at this point considered to be idiopathic.

The literature provides little solid support for the hypothesis that lead is causally related to learning disabilities. Leading researchers in the field again point to genetic/familial

factors, brain insults associated with prematurity, early neurological illness and head trauma as the major known etiologies. Most cases however are described as idiopathic.

The literature does not make a causal connection between lead exposure and language disorders. Again researchers point to a strong genetic/familial component, brain insults of prematurity, head trauma, etc. as the major known etiologies. Again most cases are idiopathic. Some authors have reported lowered verbal Iqs in lead exposed children, but an equal number do not find lowered verbal IQ.

A number of authors have noted a relationship between lead exposure and some aspects of visual motor functioning, and other between lead exposure and performance IQ. Again, consistency is lacking and an equal number of respectable studies do not find a lowering of performance IQ.

Three recent studies attempt to derive a causal connection between lead exposure and delinquent or criminal behavior. (Needleman 1996; Burns 1999; Nevin 1999). The Needleman and Burns studies suffer from problems regarding the nature of the data collected (Maternal reports, self reports teacher reports on a standard checklist) and from the lack of control of important confounding variables such as use of corporal

p[punishment, parental history of behavioral problems , substance abuse, or criminal activity; domestic violence, etc., all of which are well known to generate behavioral difficulties in children. The children were not evaluated for concurrent psychiatric illness, emotional disorders, or special education placement which are also known to be associated with in an increase in .behavioral problems. The final one of these studies is not p[psychological in nature and was not completed by a psychologist (Nevin, 2000). In this study Mr. Nevin simply overlays rates of criminal behavior, and unwed pregnancy, on exposure to lead from gasoline and paint. He does not account for any confounding factors, and this kind of study does not rise to the level of scientific evidence,

A FINAL WORD:

In applying the results of scientific research to a particular area of study, we need to be ever mindful of what constitutes *proof* or *evidence*. Correlation does not imply causality and not every study is applicable to a particular set of circumstances. For example, in order for a study to be directly relevant to a particular matter, it must use the same measurements and refer to individuals who fit the same categories. It should be noted that much of the literature regarding lead level consists of measurements of lead in teeth or bones. These measurements cannot be converted to blood lead level, and studies which use these measurements, while they may be useful in drawing some general conclusions about lead and neuropsychology *cannot be applied as proofs in circumstances where the plaintiff's lead burden is measured in blood*. This is even more obvious when one

considers that tooth or bone lead levels represent *prior* exposure, while EBLs measure *current* exposure. There is no methodology which enables us to draw any conclusions regarding the timing or duration of exposure except to note that the exposure obviously had to occur at some point prior to the measurement.

While it may be tempting to draw broad, sweeping conclusions from isolated findings, scientific methodology requires that proof be demonstrated by consistency and accretion of relevant evidence. Indeed even Bellinger admits that “Consistency in specific findings would provide greater reassurance that attributing a causal role to lead is justified.”

The scientific method does not allow us to make assumptions or inferences, but rather requires that we base our conclusions upon acceptable scientific proofs derived from methodologically acceptable scientific experimentation and study.