The Violation of Childhood: Toxic Metals and Developmental Disabilities

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Abstract

Emphasizing that for any known toxic metal no safe dose level exists, this review describes existing evidence of the tetragenic effects of metal toxins on childhood development. Discussion of the currently available evidence of toxic effects focuses on cadmium, mercury, aluminum, arsenic, and, extensively, lead. The multicollinearity of metals is emphasized, and it is posited that metal interactions have a unique, negative, and synergistic relationship to children 's psychologic performance. The continuing lack of relevant and precise knowledge of the effects of low level exposure to toxic metals is noted.

In the early 1970s I was a teacher of children with learning and behavior disorders at Public School Number 12, an inner city elementary school situated in a chemical industrial neighborhood in south central Indianapolis. The neighborhood was composed of tiny shack-like houses (sources of peeling lead paint), blackened lots, and rutted roads, set amongst the giant buildings of the chemical plants. Directly across the street from the school was a chemical corporation making paint and pigments. By day and night the school and its neighborhood were clouded with the fumes that poured from the vents and smokestacks of the adjacent chemical plants. At night the sky was a brownish yellow.

During winter semester, Kenthia, a ten year old girl with borderline mental retardation and disordered behavior, fell into convulsions in the back of her classroom. Hospitalized, she was diagnosed as lead poisoned.

Soil samples taken from her family's residence turned up disturbing quantities of cadmium, arsenic, mercury and lead. Lead levels in the soil measured an astronomical 8,500 parts per million. Her two siblings, also diagnosed with mental retardation by school authorities, were subsequently determined to have highly elevated levels of lead in their blood.

Respiratory ailments, learning disabilities, sore throats, hyperactivity, and attentional deficits were common to the school children. While there was little medical knowledge as to what caused these conditions, circumstantial and coincidental evidence pointed to the neurotoxins in the air and embedded in the soil where Kenthia and her classmates played.

School and health authorities downplayed the environmental significance of the children's lead poisoning and its implication for the community's children. It was assumed there was a safe dose and background level for lead and other chemical pollutants. Since the poison in the soil was invisible, the poisoning incident was soon forgotten, and no one thought anything was wrong.

This paper grows out of the concern that the still current concept of a safe dose or no-effect level, per se, for lead and other metal toxins is unacceptable. These limits where they are set are based on the premise that there is a level of toxic concentration in human blood and tissue below which neurological and intellectual functioning, motor performance and behavior, and the normal bodily processes of growth and repair are all uninfluenced by the invading material. Such a notion is inconsistent with what is now known about children's neuropsychologic response to low levels of lead and other poisonous metals.

It is to be expected that regulators, lawyers, legislators, and the public want toxi-cologists to tell them what are safe levels (as opposed to hazardous levels). What is unexpected is the insouciance with which toxi-cologists have accepted the responsibility of establishing safe levels, that is, of proving that there are no effects at or below particular levels. Everyone accepts that it is for-

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mally impossible to prove the null hypothesis, but people say that for practical purposes there are levels that one can be "virtually certain" have no effects. But it is for practical reasons, in part, that the concept of an identifiable "no effect" level is unacceptable.

First, the minimum threshold for a real effect can not be determined satisfactorily. As Dews (1994) evinced, the minimum dose of a toxin below which there is no effect is the lower asymptote of the dose-effect curve. The slope of the sigmoid dose-effect curve becomes less and less as the asymptote of the "no effect" level is approached. The lower the slope, the greater is the effect of errors, random or nonrandom in the measurement of effect on the estimates of dose. At levels where the effect has become so low as to be virtually "no-effect," the precision with which the level can be measured is also so low that the estimate is useless for practical, as well as theoretical, purposes. Second, the nature of the human organism is such that it is capable of idiosyncratic response to ecological variation and distur-bancy of body chemistry. Rare occurrences are "lost" within risk calculations since the statistical methods underpinning such actuarial calculations are ill-equipped to deal with isolated cases.

The view that there is a background level in the environment which is safe is equally spurious. These are man-made toxins; if they are in the environment, then man has put them there. Air borne lead levels are more than two hundred times greater now than they were three thousand years ago. The amount of lead in our oceans has increased more than tenfold since ancient times. Sediment from the bottom of lakes in the United States contains an average of twenty times more lead now than it did just less than a hundred years ago. The average American has more than one hundred times as much lead in his or her blood as the average person did before smelting began. The lead content of bones is now five hundred to more than two thousand times higher (Patterson, 1965).

A plethora of studies conducted during the last two decades indicate the toxic effects of low levels of lead, cadmium, mercury, arsenic, and aluminum on development and have failed to determine no-effect thresholds. The evidence of these effects will be reviewed here in terms of (1) neuropsychologic performance of general populations, (2) mental retardations, (3) learning disabilities, and (4) behavior disorders.

General Populations

Lead is probably the most widely researched of all environmental tetragenic agents. Interest in the effect of lead on health has quite a long history. It is recognized as an environmental hazard in Victorian times and referred to in Roman times. Focused research interest on the link between environmental lead and children's development is of much more recent origin.

Needleman and Gatsonis (1990) conducted a meta-analysis of 12 modern studies of childhood exposures to lead in relation to intellectual quotient. There were seven blood and five tooth studies from various countries with sample sizes ranging from 75 to 724. The joint P values for the blood lead studies were < .0001 for two methods of statistical analysis, while in the tooth lead studies they were < .0005 and < .004. The hypothesis that lead impairs children's IQ at low dose was strongly supported by the quantitative review, and the effect was robust to the impact of any single study. Baghurst et al. (1992) in a longitudinal study of 494 Australian children living near a lead smelter observed an inverse association between blood lead concentrations and intellectual abilities at the ages of two, four, and seven years. Other toxic metals and their synergistic interactions have been investigated in relation to IQ. Marlowe (1988) in a multi-metal study (N = 58) reported a strong link between exposure to mercury and the metal-metal interaction of lead with cadmium and intellectual deficit in rural Wyoming children. Hair mercury levels accounted for eight percent of the variance in the children's psychometric performance, while the interaction of hair lead and hair cadmium levels accounted for an additional 12 percent. Thatcher, Lester, McAlaster, and Horst (1982) reported a significant negative relationship between hair lead and hair cadmium levels and psychometric test scores in 149 children in rural Maryland public

schools. The results of these IQ studies accord well with the view that the "no effect" threshold exists throughout the whole range of metal burdens regarded as normal in children.

General population studies have also demonstrated that academic achievement, classroom behavior, and motor performance are sensitive to low levels of lead and other metal pollutants. Part of Needleman et al.'s 1979 Boston study examined the relationships between teachers' ratings of first and second grade children on an informal 11-item classroom behavior scale and children's dentine lead levels (N = 2,146). The relationship of negative reports increased in a dose related fashion for all 11 items. Bellinger, Needleman, Hargrave, and Nichols (1981) conducted a three year follow-up study and measured on- and off-task behavior of a sample of these children. Utilizing applied behavior analysis, the results showed hat high lead children displayed more off-task behavior in eight behavioral categories when compared with middle lead subjects and low lead subjects. A ten year follow-up study of these children demonstrated that the effects of low level lead exposure persist (Needleman and Allred, 1990). When these subjects were reexamined it was found that subjects with dentine lead levels greater than 20 ppm in 1978 had markedly high risk of dropping out of school, lower class standing, greater absenteeism, and impairment of fine motor skills as compared to those subjects who had dentine lead levels less than 10 ppm in 1978.

Lansdown, Yule, Urbanowicz, and Millar (1983) in a replication of Needleman's 1979 study suggested a dose response relationship between teacher reported deviant behavior and increased blood lead levels in 166 London children who lived near lead works. Hyperactivity and conduct problems were also found to be significantly related to children's blood lead levels independent of age. Children in the top 50% of the lead distribution scored significantly lower on reading and spelling achievement. Tuthill (1992) utilizing the same measure of classroom behavior used in the 1979 Needleman and 1983 Lansdown studies discovered a striking and highly linear dose response relationship between increasing hair lead concentrations and negative teacher ratings in 277 first graders in western Massachusetts.

Winneke et al. (1982) reported negative associations between 317 German children's dentine lead levels and mother and teacher ratings of attention, endurance, and organization. Maracek et al. (1983) showed that increasing lead levels in Philadelphia's inner city black children were associated with deficits in visual-motor functioning and perceptual integration, right-left orientation, and verbal abstraction. The results suggest that levels of lead exposure common to inner-city children are sufficient to produce neuropsychologic impairment.

Charles Moon and colleagues' study of 69 Wyoming school children (1985) found that the combination of lead, arsenic, cadmium, mercury, and aluminum hair concentrations and their synergistic interactions accounted for 23 percent of the variation of test scores for reading, spelling and visual-motor performance. Marlowe et al. (1985) showed that hair lead values accounted for 10 percent of the variation of 80 Wyoming children's teacher-rated nonadaptive behavior scores, and the interactions of lead with arsenic and lead with cadmium accounted for an additional seven percent. Inverse relationships between hair aluminum values and children's fine and gross motor performance have also been reported (Marlowe, 1992).

The effects of maternal metal levels and related foetal metal levels have been less well documented than studies concerned with educational attainment and behavior of the developing child. Nevertheless, as Waldron (1980) noted, "What may be of importance to the development of neuropsychiatric abnormalities is the degree to which children are exposed at the time of maximum brain development, which includes exposure in utero." Lead as Barltrop (1969) pointed out is readily transferred across the human placenta and can be found in foetal tissue. Wibberly et al. (1977) reported pronounced mean elevations of lead in placenta from still and malformed births. Bryce-Smith, Deshpande, Hughes, and Waldron (1977) found that the mean lead and cadmium levels in the bones of still and malformed births was five to ten times greater than normal. Moreover, Moore, Meredith, and Goldberg

(1977) showed that early post natal blood lead elevations were significantly associated with an increased risk of mental retardation subsequently. Two prospective studies have found that the lead concentration in the umbilical cord blood is predictive of developmental progress in childhood (Bellinger et al., 1987; Dietrich et al., 1987). Marsh et al. (1987) examined 81 infant-mother pairs and demonstrated a dose-response relationship between hair mercury concentrations during gestation and the frequency of psychomotor retardation, seizures, and neurological signs in the children. Lewis, Worobey, Ramsay, and McCormak (1992) determined lead, cadmium, mercury, cobalt, chromium, nickel, and silver in amniotic fluid taken from 92 pregnant women undergoing amniocentesis. A prenatal toxic risk score based on the levels of the seven metals was derived which was negatively related to the intellectual development and health status of the subjects' children at 34 months of age. While lead as an environmental teratogen has been recognized for many years, the above summarized studies suggest that in utero exposure to lower levels of lead and other metals than are still considered safe by some governments are linked to congenital abnormalities and retardation.

Mental Retardations

Five hair analysis studies have examined toxic metals in populations of mentally retarded individuals without assigned etiologies. Wunderlich, Cameron, and Loop (1980) reported increased lead and cadmium levels in mentally retarded children and youth (N = 217) when compared to laboratory norms, while Phil, Drak, and Vrana (1980) found increased lead levels in 31 mentally retarded youth when compared to a 22 member control group. Marlowe, Errera, and Jacobs (1983) observed significantly higher lead and cadmium levels in mentally retarded children in rural Tennessee (N = 64) versus a comparison group (N = 71). Based on laboratory norms, 44 percent of the retarded children were elevated in lead compared to eight percent of the nonretarded children. Marlowe, Medeiros, Errera, and Medeiros (1987) determined toxic elements in a mentally retarded "etiology unknown" group (N= 60) and in a mentally retarded Prader-Willi syndrome group (N = 19). The mentally retarded "etiology unknown" group was significantly higher in hair concentrations of cadmium, aluminum, and nickel, and their mean hair cadmium level was above the laboratory's normally tolerated limit. Additionally, 20 percent of the mentally retarded "etiology unknown" group demonstrated elevated hair lead concentrations compared to none in the Prader-Willi syndrome sample. Hui-min, Guo, and Zhu-li (1990) reported significantly higher cadmium levels in Chinese children with mild mental retardation (N = 85) when compared to a control group (N = 415). The authors posited that if the cadmium content in the hair of male children is higher than 0.239 ug/g, and high than 0.180 ug/g in the female, mental retardation can be diagnosed. The sensitivity and specificity of the test was greater than 90%; false positive and false negative rates were lower than 10%.

In an earlier blood lead study a mentally retarded sample with "probably etiology" showed no significant difference in blood lead concentrations from those of a normal control sample, while a group of mentally retarded children "etiology unknown" had statistically raised blood lead concentrations (David, McGann, Hoffman, Sverd, and Clark, 1976).

Learning Disabilities

Rimland and Larson (1983) presented a brief summary of 10 research studies conducted from 1975 to 1981 on the relationship between hair element levels and learning disabilities. Sample sizes ranged from 40 to 1,400. Of the nine studies comparing metal concentrations between learning disabled populations and controls, five reported significantly higher lead and/or cadmium concentrations in learning disabled populations, and they were somewhat higher in lead, cadmium, and/or aluminum than controls in the remaining four studies. Subsequently, Marlowe, Errera, Cossairt, and Welch (1984) reported significantly higher hair lead and hair aluminum levels in a 9 to 12 year old group of learning disabled children (N = 25) when compared to a control group (N = 22). Discriminant function analysis revealed by using lead, calcium, silicon, aluminum, vanadium, mercury, and zinc lev-

els, subjects could be correctly classified as normal controls or learning disabled with 91.7% and 76.1% accuracy. In 1987 Marlowe examined hair element levels in a population of underachieving Northern Arapahoe children on the Wind River Reservation in central Wyoming. The Arapahoe children (N = 64) evinced significantly higher lead and arsenic levels than a non-Indian comparison group (N = 56), and discriminant function analysis revealed subjects could be correctly classified as Indian or non-Indian with 100% accuracy using measures of metals and nutrient minerals in the hair. Increased aluminum levels correlated significantly with teacher ratings of deviant behavior in the Arapahoe children.

The few dose response studies in the field of learning disabilities have also suggested the deleterious effects of metals on psychological measures. Moore (1975) found increased hair lead levels correlated with decreased hand/eye coordination and reaction time in learning disabled males. Stellern, Marlowe, and Errera (1983) linked increased hair lead and hair cadmium levels to decrements in visual perception in children with learning problems. Minder, Das-Smaal, Brand, and Orlebeke (1994) showed that learning disabled children with relatively high concentrations of lead in their hair reacted significantly slower in reaction time and were less flexible in changing their focus of attention.

Behavior Disorders

Beginning in 1980 there have been seven studies of the relationship between toxic metals and behavior disorders in children and youth. All seven studies have observed increased toxic metal levels in behaviorally disordered populations. Hansen, Christen-sen, and Tart (1980) reported significantly higher hair lead concentrations in 20 hyperactive children drawn from a children's psychiatric hospital in Risshov, Denmark, when compared to a control group. Schauss (1981) reported increased toxic element hair levels of lead, cadmium, aluminum, mercury, and/ or arsenic and elevated copper levels in a sample of 81 behaviorally disordered children in western Washington. Kracke (1982) found significantly higher hair lead levels in 40 Kansas emotionally disturbed children when compared to controls. Marlowe and Errera (1982) observed significantly higher hair lead concentrations in rural Tennessee children with severe behavior problems than in a comparison group, and hair lead concentrations were associated with teacher ratings of nonadaptive behavior. Raloff (1983) cites an Illinois study which compared hair elements in 24 delinquent violent male children with those of 24 nonviolent male siblings. All 24 violent children were elevated in lead and cadmium as well as iron and calcium, whereas none of the nonviolent siblings exhibited these patterns. Marlowe, Errera, Ballowe, and Jacobs (1983) reported significantly higher hair concentrations of lead and aluminum in 22 emotionally disturbed Wyoming children when compared to 25 controls, and the disturbed children's aluminum, cadmium, and arsenic levels correlated with teacher ratings of nonadaptive behavior. Marlowe, Bliss, and Schneider (1992) compared hair element levels in a group of emotionally disturbed youth with violent behavior (N = 23), a group of nonviolent emotionally disturbed children (N = 47), and a control group (N = 170). The violent disturbed group exhibited significantly higher levels of lead, cadmium, and mercury than the other two groups, and the nonviolent disturbed group was significantly higher than controls in lead and cadmium.

Conclusions

I have spent some time dealing with the effects to children of toxic metals. These are common elements, widely produced, widely used and distributed through the entire ecosphere. In high doses their effects to man are well established, but their low level effects in asymptomatic children are still not fully understood. In contrast to lead, there has been little inquiry into the possible deleterious effects of chronic low level exposure to cadmium, aluminum, mercury, and/ or arsenic. What is certainly the case is their potential for impairment is greater than was once supposed at much lower levels of exposure than are currently considered "safe."

Although there are important differences between metals and their uptake and distribution by the body and their toxic effects on metabolism, there are important similarities between the incidence and acute effects of

metals. Metals often occur together in the same ore bodies, and their concentrations in hair, amniotic fluid, and blood often show strong positive correlations. The overt symptoms of lead poisoning are also similar to the acute effects of other metals, e.g., deficits in visual and auditory functions, impaired fine muscle control and coordination, emotional lability, and behavioral reactivity. As such, it is possible that some of the deleterious low-level effects attributed to lead in correlational studies may instead by due to other metals and/or their interactions. Studies investigating the effect of a single metal cannot be causal in nature since they have not removed the effects of other metals present in the given source.

At present there is little concern and no composite risk standards for exposure to combinations of metals. This descriptive review suggests product vectors of metals have a unique, negative, and synergistic relationship to cognitive alterations. The total metal toxicity of the child must be considered.

Metal induced cognitive alterations may signal the early stages of an ongoing toxic process that will become more disabling with age. Needleman and Allred's 1990 ten year follow up study of "high" dentine lead first graders supports such a phenomena. A metal that produces subtle cognitive alterations in many children may produce a learning disability or mental retardation in those who are especially susceptible because of genetic, perinatal, or other factors, e.g., malnutrition. Under-nutrition can influence susceptibility to metal toxicity. Deficiencies in calcium, iron, zinc, and phosphorus are all known to increase such susceptibility, and the large majority of studies reviewed here have not examined nutritional correlates. It may be that the ratios of nutrient minerals to metals may be more important than the absolute values of the metals.

In sum, operational concepts such as "no-effect thresholds" represent at best the overt manifestation of a value system which balances benefit (however defined) against detriment. Continued lack of relevant and precise knowledge of the effects of chronic low level exposure to toxic metals is at variance with the aims of a free human society. To be constrained in one's ability to achieve to the
full of one's potential is to be less than free.

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