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Environmental Impact of Waste Incineration on Children

Using cell systems, scientists have found that the threshold for a toxic effect from cadmium is well within ambient levels, especially when there is synergy with other toxic agents. By using interdisciplinary findings from cell biology and looking simultaneously at biologic effects in both plants and humans, it is possible to implicate cadmium pollution from waste incineration in environmental stress effects, such as seeding in trees and learning and behavioral problems in children. A small homogeneous sample of white upper middle class prepubertal males was studied for effects of heavy metal exposure. Hair phosphorus and a history of exposure to heavy metals from dust, dirt, traffic, and cigarette smoke accounted for 38% of the variance in listening and achievement test scores. Cadmium inhibition (in genetically susceptible hosts) of pyrimidine-5'-nucleotidase is a reasonable explanation for excess seeding in trees and the changes in hair mineral content of children showing subtle neurotoxicity.

Key Words: Cadmium. Phosphorus. Hair analysis. Neuro-behavioral toxicity. waste incineration.

Cadmium is a stress agent in plants and animals.[1,2] Verbost has found that nanomolar levels of free cadmium inhibit the membrane calcium ATPase which increases intracellular calcium, a strong stimulus for stress responses.[3] Freedman has shown that cadmium binds to the steroid binding site of DNA with 50 times more affinity than zinc.[4] Dickerson has found that cadmium can greatly increase gene expression.[5] An emotional stress in rats produces a marked increase in liver metallothionein, a cadmium binding protein.[6] The available evidence from cell biology suggests that free cadmium not only causes stress but that other stresses release free cadmium and have effects mediated through cadmium. In animal studies done in fish, 12 ug/L cadmium was lethal to fish housed in groups in tanks while 1,600ug/L cadmium was tolerated by some fish when they were alone in the tanks (unpublished study, Robert Drummond, US EPA, Duluth, MN). Social stress appears to have a major impact on the toxicity of cadmium exposures.

Subtle learning and behavioral problems in children are attributed to a large number of influences including genetic susceptibility, age, gender, nutritional factors, and psychosocial stress. Excess seeding in trees is also looked upon as a non-specific stress response. It is possible that both these stress responses are associated with cadmium exposures, which at low dose cause toxic effects in genetically susceptible hosts with nutritional deficiencies and co-exposure to other toxic agents or stressors.

A significant source of air cadmium emissions in the United States is waste incineration.[7] Refuse derived fuel incineration is a major source of HCl emissions. Cadmium oxide and HCl produce $CdCl_2$, a covalent molecule that moves readily into biological systems.[8] The increase in emissions have diffuse and distant effects. They may be reflected in the findings of increased cadmium levels in moose and deer liver in northeastern US and Canada.[9]

Children appear to be accurate indicators of environmental exposures to heavy metals.[10] Recent studies suggest that the use of stimulant medication is increasing in children with learning and behavior problems, in spite of a major decrease in blood lead levels.[11] Reading achievement tests as well as I.Q. scores are correlated with slight but significantly elevated blood lead levels.[12] In spite of the marked drop in blood lead levels from the use of unleaded gasoline,[13] studies still show an effect from very small increases in blood lead,[14] well below the threshold for effects of lead in experimental studies.

Free cadmium increases blood lead levels by antagonizing zinc,[15] which increases lead absorption.[16] It also resorbs bone, where lead is stored, increasing blood lead levels.[17] It seems probable that some effects associated with blood lead elevations are actually due to cadmium and lead acting synergistically or even cadmium alone. Lockett and Leary[18] found that lead ingestion actually increased cadmium uptake in the brain. In cell culture systems cadmium is far more toxic that lead,[19] suggesting that cadmium may be responsible for neurotoxic effects associated with lead elevations in blood.

Currently, ambient cadmium air pollution is not being linked to any chronic human disease. The difficulty may lie in erroneous assumptions about cadmium and its effects. The key variable in cadmium toxicity is not the level of exposure. Rather, key variables are the route of exposure, nutritional factors, age, genetic factors, and the presence of other stressors or toxic agents that may react synergistically with cadmium.

The assumption has been made that food is the most important source of exposure to cadmium, aside from cigarette smoke in the general population.[7] Although levels of cadmium in air are quite low at this time, 0.0002-0.0006 ug/m^3,[20] there is no barrier to absorption from air. In animal studies cadmium is taken up rapidly from the atmosphere[21] while absorption orally is quite low unless there are nutritional deficiencies.[15]

Chronic low dose exposure of young plants and animals to cadmium results in multiple alterations of nutritional factors. These effects are distinct from those caused by lindane, for instance.[22] They are also distinct from those of other trace metals.[23] While other metals do not enter cells easily unless the cell energy supply is injured, cadmium enters cells rapidly during the log phase of growth.[24] It alters the cell membrane increasing the permeability of the cell to other divalent ions.[25]

In both plants and animals, genetic factors play an important role in toxic effects from exposures. There are great individual differences even within a species and among in-bred laboratory animals. Since the induction of metal binding proteins confers some protection against cadmium toxic effects, level of accumulation of cadmium is not generally associated with a toxic effect. Both genetic and nutritional factors affect the biologic effects of cadmium in a cell.[15]

In a dentine study there was a correlation between dentine lead but no correlation between dentine cadmium and neurobehavioral problems in children.[26] The authors concluded that there was no evidence that cadmium was exerting a toxic effect on the children. Since free cadmium ions are responsible for a toxic effect and the majority of cadmium is bound and detoxified, associating cadmium tissue levels with pathological effects could be the wrong approach. In a recent study relating blood cadmium to hypertension, a large SD was found in the hypertensive group, preventing the elevation noted from achieving statistical significance.[27]

Another important factor is the presence of other agents. Cadmium reacts synergistically with a number of other toxicants such as chloroform[28] and lindane,[22] causing toxic effects when they are together at doses that are not toxic when they are given singly. It is important to recognize that these synergistic effects of cadmium with multiple other agents are distinctive. Metals have complex effects and no two are alike. Cadmium by altering cell metabolism has a profound effect on the toxic organics. This is particularly important since in studies of environmental pollution one is always dealing with mixtures of chemicals. Children exposed to waste incineration are also drinking chloroform in drinking water. They are exposed to toxic organics applied to lawns and farm land.

Stress is prevalent and there are many kinds of stress agents: physical, chemical, biological, and psychosocial. The effects of stress are to increase variability. Stress is a chaos factor. A small change can produce a large effect.[29] Since all these diverse inciting agents induce biochemical events that are clearly linked to cadmium, it is reasonable to consider the hypothesis that cadmium may actually be the link between stresses of various kinds and toxic effects.

To investigate the relationship between environmental exposure to cadmium from waste incineration and neurotoxicity in children, it is necessary to look for an increase in toxicity in a diffuse area. Rather than look for a dose response effect, one must look for increased toxicity in those showing indicators of stress. The effect of low dose cadmium toxicity is an increase in variability. An influence on multiple nutrient minerals needs to be sought. Since cadmium influences plants as well, the finding of toxic effects on plants is additional support for the hypothesis that cadmium air pollution is having a toxic effect on children.

To find evidence of heavy metal induced neurotoxicity, manifested by lowered achievement test scores, a hair analysis study was undertaken in a group of upper middle class boys. Although the intent was not to study the impact of waste incineration on children, the most significant source of air cadmium in the children studied was waste incineration.

Hair analysis has been used in several studies of children with learning disabilities.[30,31,32] Questions have been raised about the validity of hair analysis and it has never been used as a standard of lead exposure in lead studies. However, in a study by Rabinowitz a clear relationship was found between parenteral lead exposure, blood lead and hair lead level.[33]

In Barrett's study,[34] which is widely cited as evidence of the inaccuracy of hair analysis done by commercial laboratories, the hair was not taken from the inch close to the scalp thus invalidating the study. In a more recent article, Klevay[35] concludes that hair cadmium and lead measurement are satisfactorily accurate, precise and free from contamination for research studies. Murray recommends hair analysis for assessment of industrial cadmium exposure.[36]

Thatcher[37] has used hair analysis to show that low hair calcium and elevated hair lead and low hair zinc and elevated hair cadmium are associated with tests of cognitive dysfunction. Pihl[32] and Stellern[30] found both elevated hair lead and cadmium levels in children with learning disabilities.

METHOD

Sample: The 20 third grade teachers in an upper middle class suburban community public school system were asked to rate the two most and the two least distractible children of those children in their class for whom consents were obtained. Sixty three boys were evaluated. A total Connor's score was obtained as a measure of non-specific behavioral deviation, a manifestation of stress.

Sample's exposure to past and current lead: The children were all born in the period 1978-1979. Ohio EPA records show the highest monthly averages for air lead in 1978 and 1979 occurred in April and May and were 1.0 ug Pb/m^3. Air lead levels are currently under 0.4ug Pb/m^3 in central Ohio. One third of the children grew up in old homes in which leaded paint was used. The majority of upper middle class white children of this age had blood lead levels under 6ug/dL while the national mean was 15ug/dL.[16]

Sample's exposure to past and current cadmium: There were only two children living with continuing passive smoke exposure. Cadmium is volatile and gets into the air from transpiration from vegetation,[38] traffic, fossil fuel combustion, industrial sources and waste incineration.[7] There are no industrial emitter sites or limited access highways in or directly bordering the suburb. The hair was collected before there was much exposure to conditions conducive to transpiration from vegetation.[38]

Local air levels of cadmium using conventional air filters were 0.0044ug/m^3 in 1981 and 0.0006ug/m^3 in 1986 according to the National Aerometric Databank (Personal Communication, Ohio EPA). This drop follows a drop in total suspended particles that in turn follows the decrease in lead emissions.[13] Lead additives are considered the source of cadmium in auto emissions.[39] The drop in air cadmium may reflect this drop in emissions. However, cadmium plated metal parts and vulcanized tires are also sources of cadmium pollution from traffic.[7] Studies in wetlands receiving run-off from highways are still showing 12 ppm cadmium in bacteria that are cadmium accumulators. The levels are less than .5ppm where there are no obvious sources of pollution (personal communication Robert Pfister, Ohio State University).

A refuse derived fuel, large capacity (1,700 tons per day), municipal waste incineration plant 5 miles away has been operating since late 1983. These plants produce significant air pollution with a variety of toxic substances.[8] The cadmium emission from the plant, based on one stack sampling, is 3g/hr (personal communication Ohio EPA). Using the average cadmium content of garbage, 12 ug/g, and the average retention rate of cadmium using electrostatic precipitators of 70%,[7] the estimated cadmium emission rate is 256 g/hr. This amounts to 2 tons/year.

In view of the discrepancies between air measurements of cadmium and probable emissions, it is necessary to turn to biological monitors. Trees accumulate cadmium and suffer toxic effects.[40,41] In six tree species the cadmium content of leaves varied from none-detectable to 0.58ppm on Oct. 15, 1987. Any level greater than 0.3ppm is attributable to enrichment from human activities.[7] Cadmium levels in lichens in tree bark by busy streets were 8ppm and cadmium levels in house dust were 8 ppm in samples from homes with no smokers, collected by the author for analysis at a research laboratory. There was no gradient with increasing distance from the incineration plant, which is similar to studies using fruiting bodies of molds.[42] House dust appears to be a significant source of human exposure to cadmium whose source is environmental.

The children had been exposed for two years to air pollution from the RDF plant when they took the achievement tests and they had been exposed for three years when they had the hair analysis.
Socio-Economic and Geographical Characteristics: All 63 families were contacted by phone and agreed to participate in the study. At a home visit written consent for the study was obtained. Based on the home value and parental occupation/income, 85% of the families who agreed to participate were considered upper middle class. This population was remarkably free of psychosocial confounding factors that could influence school achievement. Since it was a volunteer group, there may have been an increased participation by parents with health concerns about their children.
Past Environmental Exposure History: Parents answered questions regarding increased environmental exposure to sources of heavy metals in the first four years of life, when body zinc stores are lowest and there is the greatest uptake of heavy metals. This was obtained by the investigator prior to all other data collection and scoring of the Connor's scale. The interview was used to obtain information from parents regarding environmental exposure to dust and dirt between the ages of one and four. The age of the house, degree of dust, and distance from a busy street were ascertained using a five point scale with 0 for no exposure and 4 for heavy exposure. Information about the child's play in dirt, eating dirt, and putting fingers in mouth during waking hours was also obtained. The number of smokers living in the home was noted. There were seven questions with a maximum score possible of 28.

Achievement Tests: The investigator was provided with the results of an achievement test called the ERB (Educational Records Bureau Comprehensive Testing Program) administered 12 months prior to the study. This test battery consists of one hour tests given over a five day period. It assesses listening, writing, math, reading, and word analysis skills. ERB scores were available on 51 children. IQ scores were not obtained but all the children had IQ scores above 80 and the mean IQ in the grade was 113 on the Otis Lennon.

Hair Sampling: All hair samples were obtained between late March and late April of 1987. A few children didn't want to have their hair cut and some had such short hair that a sufficient sample could not be obtained. Fifty hair samples were obtained and two duplicate samples were sent to determine the accuracy of the laboratory. The hair kits were delivered to each home and the parent was instructed in how to obtain the hair sample. The hair was washed with shampoo, rinsed carefully, air or blown dried, and cut from the area behind the ears in multiple small sections close to the scalp and no more than one inch away from the scalp. This procedure removes 98% of exogenous cadmium and does not remove endogenous cadmium.[43] The hair was weighed on a cardboard included in the kit, so that a 0.5 gram sample of hair could be sent for analysis.

Hair Analysis: The hair analyses were performed by Doctor's Data Inc., (West Chicago, Il), by a method described in full by John Errera.[30] Induction coupled plasma emission argon spectrometry is used for metal analysis.[44] Two separate samples were obtained for two children and analyzed independently. In both sets of hair, values for Ca, Mg, Zn, Mn, P, and Cd were very consistent.

Hair Minerals Used in Data Analysis: Lead and cadmium were of chief concern. Arsenic and mercury were not included because the values were usually low and the duplicate samples were variable. Calcium, magnesium, and zinc were of interest because they may provide protection against heavy metal toxicity. The hair phosphorus level was included in data analysis because of my observation that hair phosphorus was frequently low in children with attention deficit disorder. This correlation was subsequently studied and found significantly associated with learning and behavioral problems.[45] Manganese was entered because of its association with neuro-toxicity. Chromium was included because it has been noted to be very low in some children with learning and behavioral problems. Because cadmium binds to cobalt in vitamin B-12, hair cobalt was considered of interest.

Statistical Methods: The data was analyzed using Pearson correlations and multiple regression.

RESULTS

Descriptive statistics are shown in Table 1. Of note is the marked skew in the distribution of hair cadmium. The mean hair cadmium was identical to that found in an adult control group of another study

done in Philadelphia.[36]

Complete data was compiled for 43 children. Of the fifty children who had hair analyses, seven children lacked ERB scores because they were not in the district one year ago.

Neurobehavioral Correlations: There was consistent correlation between the exposure history and hair phosphorus with the neuro-behavioral measures (Table 2). The exposure history score was positively correlated with the Connors Total Score and inversely correlated with the achievement scores. This relationship was significant for each subtest score except math. It was highly significant for the mean ERB ($p = .007$). There were no significant relationships between other hair mineral elements and the behavioral measures. Hair phosphorus was significantly correlated with the ERB listening test ($r = .505$) and the mean ERB ($r = .501$). Cadmium, Calcium, Magnesium, Zinc, Manganese, and Chromium had no significant correlations. Hair lead ($r = -.37$, $p = .007$) and hair cobalt ($r = -.30$, $p = .03$) were inversely correlated with hair phosphorus.

Finally, a multiple regression analysis of mean ERB, exposure history score, and hair phosphorus, controlling for age, ($R^2 = .38$, $F(3,39) = 8.098$, $p = .003$) indicated that exposure history was significantly related to a decrease in mean ERB ($B = -1.53$, $F(1,39) = 6.38$, $p = .016$) and hair phosphorus was positively related to mean ERB ($B = .43$, $F(1,39) = 8.88$, $p = .0049$).

To explore further the relationship of hair phosphorus with the other parameters, the data was divided by hair phosphorus and the parameters were re-analyzed by a t-test. In the low hair phosphorus group the SD of the hair minerals were significantly larger than in the high hair phosphorus group (Table 3).

DISCUSSION

A sample of children with and without signs of behavioral stress noted by their teachers was studied using hair analysis and an objective test of listening and achievement. Hair phosphorus, which had been noted previously to be associated with learning and behavioral problems in children,[45] was inversely correlated with indicators of toxic influences on brain function associated with elevated blood and dentine lead levels in previous studies.[12,26,46] The children with increased stress, as measured by behavioral deviation on the Connor's test, and low hair phosphorus, had increased variability of hair mineral content, double the hair cadmium and lead, and lower listening and achievement test scores.

Hair lead and hair cobalt were negatively correlated with hair phosphorus. Since lead exposure was decreasing and cadmium exposure was increasing, it is probable that the minimal elevation in hair lead reflected cadmium enhancement of lead absorption. Cadmium can also be implicated in the association of hair cobalt with hair phosphorus. In previous lead studies, there has been no attention given to co-cadmium exposure except in hair analysis studies where cadmium elevations are generally found associated with lead elevations.[30,31,32] In this study, the levels of lead are so low, that it is not biologically plausible to associate the effects directly to lead and the children were clearly exposed to a significant source of air cadmium.

Supporting data that the effect was due to cadmium was sought in plant toxicity. The trees in a diffuse area around the plant showed a variety of stress responses. The most notable was a marked increase in seeding that antedated a severe drought. Using data from lead studies and animal studies, it is possible to implicate cadmium toxic effects in the seeding response.

Pyrimidine-5'-nucleotidase, an enzyme system well studied in the normoblast which controls the cell energy supply, is inhibited by lead.[47,48,49] A similar enzyme on the cell membrane, pyrimidine-5'-nucleosidase, is inhibited twice as much by cadmium compared to lead.[50] Trees do not take up lead but they do take up cadmium. Using the knowledge that inhibition of pyrimidine-5'-nucleotidase leads to accumulation of cytidine triphosphate, the precursor of cyclic cytidine 3',5' monophosphate, which is a blastogenic agent in animals,[51] it is reasonable to hypothesize that the marked increased seeding that was noted in the same year the children took the ERB test was a biological marker of a toxic effect of cadmium on pyrimidine-5'-nucleotidase in plants. This would strongly suggest that a concurrent effect of cadmium on pyrimidine-5'-nucleotidase, mimicking the effect associated with elevated blood lead levels, is responsible for lowering hair phosphorus in children showing the neurotoxic effects of decreased listening skill and reading comprehension.

In experimental studies with low dose chronic exposure to cadmium, ATP levels in cells fall.[15] Because hair phosphorus is not correlated with dietary phosphorus, blood phosphorus, or body phosphorus it has not been considered important.[52] No previous investigator has considered the possibility that hair phosphorus could serve as a marker for a toxic effect of free cadmium on cell energy. In studies using NMR, Koop found that the first effects of cadmium on rat myocardium was an increase in inorganic phosphorus and a fall in intracellular pH.[53] It is not unreasonable to suspect that hair phosphorus is influenced by free cadmium effects on hair cells. In additional studies, this author has found that hair phosphorus can be normal and elevated in children showing learning and behavioral problems due to problems other than attention deficit.[54]

Hair analysis has been discounted because the hair mineral content does not correlate with organ levels at autopsy and there is a great deal of variability.[55] Cadmium strongly influences cell minerals. Free cadmium, which is the same diameter as calcium and sodium, affects the activity of calmodulin, cyclic-AMP, protein kinase, cell membrane permeability, and has important effects on neuro-endocrine function.[56,57,58] The more variability found, the more free cadmium may be present. In this behavior cadmium acts like a chaotic factor which is described by James Gleick in his book Chaos.[29]

The toxic effects on trees and children occur through multiple metabolic effects of cadmium in conjunction with stresses of various kinds and exposures to other toxic agents. These effects are unassociated with accumulation, and occurring in those individuals with genetic susceptibility to heavy metal effects.[59] Moreover, cadmium increases the tissue levels of other toxic agents through its alterations of cell minerals.[22] Gittelman found that children with learning disabilities had higher blood lead levels than siblings with the same exposure.[60] This observation can be explained by a genetic susceptibility to cadmium which by blocking zinc increases lead absorption.

Although cadmium appears to be implicated in this study, can one be sure that the waste incineration plant is the source of the exposure? The author has noted increased seeding in trees in urban, suburban, and rural settings across the country. In areas of Europe where leaded gasoline is still being sold, this was less prominent. Since traffic is also a source of cadmium emissions, it is reasonable to ask whether the waste incineration was responsible for exposing sensitive children and trees to increased cadmium. A confounding factor could be a reduction of lead antagonism of free cadmium in air which occurred during the time period studied. Cook found that lead prevented deaths in animals exposed to a stress hormone and endotoxin, whereas all animals died when exposed to cadmium, a stress hormone, and endotoxin.[61]

Since waste incineration is such an important source of cadmium, it is probably wise to assume that it plays a role. Moreover, using US Department of Justice records of methylphenidate (ritalin) purchased by pharmacies in Ohio, the author found that within 75 miles of the waste incineration plant there was the largest increase in ritalin purchased in the state, although there has been continued increases since 1983 throughout the state. Methylphenidate is commonly used for attentional problems which are found in children with low listening skills. Children with low hair phosphorus had low listening skills in this study.

There are obvious limitations in studies of effects of environmental pollution. Studies of cadmium are particularly difficult since one can not use dose response or body burden to detect a toxic effect. Although this was a very small study, the effects found are in agreement with much larger studies correlating elevations in blood lead level with neurotoxicity in children. In this study, however, there was a clear exposure to an important source of cadmium and a toxic effect was found in children showing stress responses, a factor that appears to bring out cadmium toxic

effects.

CONCLUSIONS AND NEED FOR FURTHER STUDIES

Because it is possible to block cadmium's bioavailability, it is vitally important to detect toxic effects of cadmium pollution. The most important source of air cadmium is considered waste incineration, but it is likely that traffic emissions and other sources are also important. The environmental stresses noted in this decade including global warming, acid rain damage to the environment, and forest damage are not currently linked to cadmium.[62.63] Ironically, lead, a far less toxic metal, is linked to human disease;[46,64,65] and aluminum, a far less toxic metal, is linked to ecological injuries.[66] In both cases, it is imperative to consider the possibility that the elevation of these metals occurs in response to a cadmium toxic effect.

By linking a toxic effect attributed to lead in humans with a non-specific toxic effect in plants consistent with a cadmium toxic effect, it is possible to implicate low level ambient cadmium toxicity in both toxic events. By identifying the causal link between human and environmental problems with cadmium toxicity, cost-effective strategies to deal with these problems will be discovered. Using available studies, it is clear that cadmium needs to be carefully evaluated in combination with toxic organics. Environmental pollution is always mixed.

Further studies need to carefully measure cadmium emissions, especially from such important sources as waste incinerators. Volatile species of cadmium are not being looked for in standard measurements of air cadmium.[67] With rising levels of cadmium in the environment evident from increased emission sources and increased measured levels in moose and deer liver, yet falling levels by air filters, the measurement problems need to be addressed. Biological monitoring of cadmium levels in house dust and lichens appear much more effective than conventional air filters.

In cell culture, the effects of lead on cadmium toxicity need to be studied to find out if the falling lead levels in air are actually increasing cadmium toxic effects. Lead functions like zinc in cells and zinc deficiency greatly increases cadmium toxic effects. The effects of inhaled lead, which is not toxic to cells, may be quite different from ingested lead which appears to increase cadmium absorption.

Cadmium is ubiquitous in all environments. Measures of accumulation may fail to yield significant p values, but, using the chaos model, increases can be suspected by finding increases in the SD of cadmium. Parameters affected by cadmium toxicity such as hair minerals can be measured to delineate effects of free cadmium on hair cells. Since blood lead is also affected by free cadmium, it is necessary to measure cadmium exposure and look for cadmium toxic effects, decreasing cell energy and altering cell minerals, to differentiate a cadmium toxic effect from a lead toxic effect in the large on-going studies of lead toxicity.

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CONCLUSIONS AND NEED FOR FURTHER STUDIES

1. Most important source of air cadmium is waste incineration.

2. Traffic emissions and other soources are also important.

3. Elevations of blood lead in human disease and aluminum in ecologic injuries can occur in response to a cadmium toxic effect.

4. Biological monitoring of cadmium levels in house dust and lichens is more effective than conventional air filters.

5. Effects of lead on cadmium toxicity need to be studied in cell culture. Falling lead levels in air appear to be increasing cadmium toxic effects.

6. It is necessary to measure cadmium exposure, look for cadmium toxic effects augmented by stress, decrease in cell energy and altered cell minerals to differentiate a cadmium toxic effect from a lead toxic effect.

Linking Environmental Exposure to cadmium from Waste Incineration

with Neurotoxicity

1. Finding of toxicity in a diffuse area, i.e. increase in ritalin distribution in central Ohio since the operation of the incineration plant.

2. Increase in toxicity in those showing indicators of stress.

3. Increase in variability in response to a stress factor.

4. Co-toxicity in plants, particularly trees.

Environmental Impact of Waste Incineration on Children

by **Sandra M. Stewart-Pinkham, M.D.**

1. Threshold for a toxic effect of ambient cadmium in synergy with other environmental chemicals is likely based on interdisciplinary findings from cell biology.

2. Cadmium is a stress agent capable of inducing seeding in trees and causing learning and behavioral problems in children.

3. Cadmium inhibition of pyrimidine-5'-nucleotidase in genetically susceptible hosts is a reasonable explanation for excess seeding in trees, mediated through cyclic-CMP

4. Low hair phosphorus asssociated with decreased listening and achievement test scores mimics correlations of blood lead with the same signs of neurotoxicity.

5. These findings are associated with increased cytidine triphopshate in RBC's from inhibition of pyrimidine-5'-nucleotidase, an enzyme ihibited by cadmium in a dose dependent manner.