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LEARNING DISABILITIES ASSOCIATION OF CANADA
TROUBLES D'APPRENTISSAGE—ASSOCIATION CANADIENNE

Maison Kildare House, 323 Chapel, Suite 200, Ottawa, Ontario K1N 7Z2 (613) 238-5721

November 17, 1986

Dr. F. Kenneth Hare, Chairman
The Royal Society of Canada
Commission on Lead in the Environment
241 Jarvis Street
Toronto, Ontario
M5B 2C3

Dear Dr. Hare:

Re: Final Report of the Royal Society of Canada. Commission on Lead in the Environment, Sept. 1986

The Learning Disabilities Association of Canada appreciates this opportunity to comment upon the Final Report of the Commission.

Our work during the past five years on the lead issue has been directed to the protection of children's health and development. We appreciate the Commission's acknowledgement of our work in the report. Our comments will address the health aspects of the report primarily. We have some general comments and then some more specific comments.

General Comments

In our 1982 brief to Environment Canada on lead, we stated the following:

> "Two overlooked concepts in the consideration of safety levels for toxic substances have been those of gradation of effect, and neurotoxicity. In the first instance there has been an erroneous assumption that as long as there were no overt clinical symptoms - no harm was being done...secondly that the brain is subject to biological influences on its development and function, (including behaviour and learning)".

It would seem that neither of these concepts has found fertile ground in the Commission's report. We realize that the heart of any value judgement in regulatory action is perceived risk to health. However, we do not believe that perception of risk excludes known effects due to lead which are not visible, or clinically obvious.

We would agree with the dissenting opinion of the US scientists on the final report, and support the position that the objective evidence of impairment of heme synthesis, vitamin D and calcium metabolism, increases in ALAD, and the other plethora of organ-system and biochemical effects seen at levels common to urban children, **are indeed health effects.** Despite the Commission's observation that frank lead poisoning is rare in Canada we would say that these are indications of the some degree of "lead poisoning".

Experimental Animal Literature

We found the omission of any reference to the experimental literature amazing. The field of toxicology has its basis here; and there is a rich international scientific literature on lead. Attached to our June 1985 brief to the Commission was a major review contained in Neurobiology of the Trace Elements by Dr. Ted Petit of the University of Toronto. This review included sections on the behavioural effects of lead on animals and children; mechanisms of lead entry and storage in the brain; CNS absorption; neurochemistry of lead exposure; EEG effects; synaptic changes and transmission; subcellular mechanisms; myelin, vascular, and neuronal changes. This chapter contained 229 references but none of the experimental work was cited by the Commission.

Dr. Deborah Rice's important work using primates at Health and Welfare is world-class, and should be accepted on its merit. We disagree with the Commission's rebuttal paper to the dissenting position of the US scientists, that the lead levels used in the feed to produce blood-levels comparable to children have affected the results. As Dr. Rice explained to the Commission, this level was necessary because the feed bound the lead and much was excreted.

The experimental animal literature supports and buttresses the clinical studies. For example, Dr. Ellen Silbergeld and others have found that the precursor aminolevulinic acid can interfere with neurotransmission using GABA - a major inhibitory transmitter. **Disinhibition** may underly many of the behavioural effects observed in the low-lead "asymptomatic" children. Such changes start occuring at about 10 ug/dl. Many other pieces fit from a neurochemical and neuroanatomical standpoint. The brain areas which accumulate lead, such as the limbic system, are involved in

behavioural reactivity, emotionality, motivation, and learning and memory. Animal studies have shown that lead can alter cognitive and associative behaviours.

Other pieces that fit and add up to an overwhelming case against lead are the EEG studies. Otto's longitudinal study supports the clinical findings of auditory-language effects, and there appeared to be no level below which this effect was not operating in his five-year follow-up study, (1985).

Actually it would be prudent to ensure that no child would have a blood lead level above 8-10 ug/dL.

Neuropsychological Effects

Page 223 of the report states:

> "It has emerged that there are grounds for supposing that lead has real neurobehavioural effects on children. The effects shown by the tests are, however, small...and tend to be swamped by confounding factors related in particular to socio-economic status".

Both Needleman and Rutter have noted that such "small" (4-6 I.Q. points) differences demonstrated in several studies produce a serious increase (four-fold) in the population of children scoring below 80. Also a shift in the mean of that magnitude results in depriving one child in ten of achieving superior function (above 130). This single effect represents a serious societal and economic drain on a nation at a time when technology requires an intelligent and productive population.

It is noteworthy that the Scholastic Achievement Test Scores (SAT) in the United States had fallen in an unbroken line from the academic years beginning 1963 to 1980 - when they started to rise again. During 1976 and 1980 US blood-level fell by 37%, paralleling the fall in leaded gasoline sales and levels.

Secondly, the implications that such effects are "small" and therefore of little consequence is extremely cavalier in our opinion. As parents and professionals concerned with young people with such "invisible handicaps" we know how subtle deficits can be devistating to them in their educational pursuits, and in other aspects of their lives, possibly disrupting their futures. We would not claim that lead is

the sole cause of learning disabilities, however, in some cases, it could be the chief cause and the factor that tips the scales for many other children who will never achieve full potential. Many other neurobehavioural effects of lead such as on attention, auditory-language, and impaired classroom behaviour would be as serious to the child as the IQ differences.

"Or related to social-economic status" (p. 223)

The UK commission on lead (1983, page 57) acknowledged "It is clear that social factors can have a marked effect on lead uptake". As Lin-Fu and others have pointed out, frequently the multiple handicaps of poverty may mask the effects of lead on development - while compounding those effects. The Commission casts doubt on the neurobehavioural effects seen in "asymptomatic" children by suggesting these outcomes could have been due to socio-economic factors. We disagree strongly with this hypothesis, moreover we consider that studies have sometimes overcontrolled for these factors. Dr. James Pirkle of the Centers for Diseases Control has stated, "It is clearly inappropriate to control for "social factors" without accounting for the fact that lead levels are related to "social factors"." What is seen as an independent variable could be a dependent variable. For example, the nutritional status of poor urban children exposed to lead may explain their metabolic responses to lead, this becomes a vicious cycle and an unfortunate additional burden on the health and development of these children. Again, there are other Type II errors, committed in the design and analysis of some studies, (rejecting valid associations as spurious), addressed by Needleman in his paper to the International Workshop on the Effects of Lead Exposure on Neurobehavioural Development, in Edinburgh, September 1986.

Prenatal Lead Exposure

As pointed out by Rosen et al. in their critique, there has been a number of large studies of the effects of prenatal lead on various outcome measures. The Bellinger, Needleman data were subjected to every statistical model available, and the results held. Both this and the Cincinnati study (Krafft SN:9/13/86) showed effects at 8-10ug/dL. The Cincinatti study showed that cord levels this low were linked to lower birth weight, an effect reputed to be as

striking as the link seen between maternal smoking and decreased birth weight. They reputed adverse effects on the child's early neurological development.

We found the Commission's position on these studies to be particularly disturbing (page 204). The inference that such effects are not serious because they may not be permanent also flies in the face of previous studies of lead's effects.

The Commission's position on the studies of prenatal effects as reported on page 204 would seem to trivialize these findings. We find particularly distasteful the implication that such effects are not serious because they MAY not be permanent - an opinion which counters most longitudinal research. Again the suggestion that such deficits might be ameliorated by infant stimulation also begs the reals question. The additional statement attributed to Yule and Rutter that "Lowered IQ appears to operate only within socially disadvantaged children" is one with which many would disagree. However, why is this considered "An important point"? Surely socially disadvantaged children deserve to be protected from any and all gratuitous neurological harm which comes from lead.

It would seem to us that every possible hypothesis and argument has been brought forth to discredit the health research, to implicate every possible alternative for the adverse development effects - except lead. Surely if doubt exists, one gives the benefit of such doubt to children, and not to lead. Even Rutter concluded, that "It would be safer and "scientifically more appropriate" to act as if the hypothesis were true", (Science 25 November, 1983).

MMT

In the absence of adequate studies showing MMT is safe for use with the three-way catalyst, we feel that MMT should not be given the passing grade it received from the Commission. It was banned in the United States for that very reason, and we see no indication that the same effect would not be possible here. This could result in far more serious emission increases than misfuelling, because of the numbers of cars with faulty emission controls that would result.

The Executive Report of the World Health Organization states that "inhalation of manganese produced central nervous system effects, while oral administration produced fewer effects". Concerning general population exposure, the WHO document noted that urban air concentrations may increase due to the use of manganese compounds as petro additives. They recommended "that an epidemiological survey should be conducted in communities exposed to annual mean concentrations of manganese in air exceeding 1ug/m3." Shukla et al. reported that manganese administered to young rats affected two transmitters, dopamine and norepinephrine and their precursors. The authors cited manganese fuel additives as a source of manganese hazard. We should have some indication of curbside levels of manganese for public-health purposes.

Blood-lead Levels

The Commission notes (xxxix) that Canadians seem to be better off than many other countries. However, it is inappropriate to compare 1984 Canadian levels with 1976-1980 US levels from the NHANES survey, when these were dropping. The most recent Hispanic HANES (1985) indicated that US children had blood-lead levels averaging 10ug/dL. This is slightly lower than the 10.4 average for Ontario children reported in the 1984 study. Again the Ontario Blood-Lead Survey states that it would be expected that Canadian and US children might have similar blood levels. The US move on lead in gasoline in 1985 was to drive lead levels down to the lowest possible level in children.

Conclusion

The Learning Disabilities Association of Canada agrees wholeheartedly with the Commission's first recommendation:

> Public Health and environmental policy should be to reduce lead to its lowest possible level.

We recommend that:

1. the Commission match its recommendations to the above statement. In particular, we would welcome an amendment to recommendation 5. We understand from Dr. Hare's statements at the press conference and briefing that 1993 was not the Commission's date to remove lead from gasoline. We would support a move by the

Commission to advise the federal government that measures to reduce lead to the US level be accomplished by 1990, as suggested in their Interim Report.

2. the allowable lead content of consumer paints be reduced from the existing level of 0.5% or 5000 ppm to the US level of 0.06% or 600 ppm. This was a recommendation of the UK Commission on Lead (1983).

3. provincial lead screening programs for children under six living in urban areas, or close to lead sources, be instituted.

4. soil testing programs for city playgrounds, and those near highways be instituted, with measures to make them safe for children.

5. new regulations to eliminate lead solder from cans containing food and beverages, with quick action in canned milk used in formulas especially in remote regions.

6. the Commission review all its recommendations and conclusions especially those concerning workplace exposures of pregnant women to lead, to reflect the ever-increasing body of scientific knowledge that says that lead is unsafe at any level in the human body.

Dr. Needleman has said "As a psychiatrist and a pediatrician I would state that the acceptable level of impairment of a child's intellect and of behavior is zero. This seems self-evident, and should not have to be defended."

We concur.

Respectfully submitted by,

Barbara McElgunn
Research and Liaison Officer (Health)