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## Manganese Madness

By David Goodman, PhD

When Jonathon Ericson, environmental health scientist, began planning a two-day conference on toxic metals, his thoughts turned to the internal combustion engine. Day One he dedicated to lead of the tetraethyl variety added to silence engine knock. For fifty years, no-knock gasoline containing tetraethyl lead has spewed metallic wastes out of millions of tailpipes, tainting the soil and water—and the lungs and brains of little children.

For Day Two, Ericson planned to focus on MMT, an antiknock ingredient containing manganese. He issued invitations to speakers who could discuss the dangers of manganese to the brains of miners in Europe, Asia, South America and Australia who, after a few years on the job, run an increased risk for Parkinson's Disease. The manganese particles they inhale through the lungs then move to the brain with devastating effects.

Left with invitations for the final two conference speakers, Ericson turned away from internal combustion engines, tailpipes and toxic exhausts. Instead, he focussed on the dangers of a consumer product that has been on the market for about thirty years—the plastic bottle filled with soybean-based infant formula.

How a bottle of soy formula can be mentioned on the same program with a smoking exhaust pipe and brain damage in manganese miners is what kept the UC Irvine conference audience captivated. The soy story told by speakers Francis Crinella of the UC Irvine faculty and Trinh Tran from the UC Davis Department of Animal Studies unfolded like a Stephen King novel, complete with mindless villains, thickening plot and innocent victims too young to defend themselves.

Ericson's conference took place in September, 2000 at the University of California at Irvine. His two final speakers, Crinella and Tran, suggested that infants sucking on nipples of plastic bottles containing soy-based formula could absorb toxic amounts of manganese into their rapidly developing brains.

The melodrama—no, the tragedy—of toxic manganese in infant formula begins in 1980 when the Federal Government's Food and Nutrition Board established safe and acceptable values for manganese in adults, toddlers and infants. Permissible levels for the three age ranges were set at 2.5-3.0 mg/day in adults, 1.0 to 1.5 mg/day in toddlers and 0.5 to 1.0 mg/day in infants. The "safe" level for infants soon translated into soy formula products purchased by millions of mothers.

Despite government assurances, Phillip Collipp, a pediatric physician at Nassau County Medical Center in 1983 tested for the manganese in popular soy brands available locally, including Isomil, ProSoybee and Nursoy. They contained from 0.2 to 1.0 mg of manganese per quart of infant formula. Later that year, Bo Lönnerdal and Carl L. Keen, of the Department of Nutrition of UC Davis, tested baby formula taken from pharmacy shelves in eight countries. The manganese concentrations they found in soy formulas were higher, ranging from 0.4 to 2.2 mg; the mean value of 1.2 mg vastly exceeded the infinitesimal 0.005 mg found in human breast milk.

Nutritional scientists have reported how newborn babies absorb manganese from breast milk. Tiny amounts suckled daily a dozen times by the baby supply an adequate quantity of manganese to catalyze 50 biochemical reactions. The newborn's digestive system seems superbly attuned to absorb the scanty amounts of manganese it needs from its mother's milk.

However, soy formula, containing up to 200 times the manganese of breast milk, overloads the little body. The baby's immature liver cannot handle the load. With each swallow increasing the manganese content in its digestive track, what does the baby do to dispose of the excess? Bo Lönnerdal, a researcher from UC Davis, explained that in newborns, ingested manganese rises to high levels in the blood plasma and red blood cells and then permeates the liver, kidneys and other soft tissues of the body, including the brain. Only later, at the time of weaning, can the infant metabolize such large amounts of manganese.

Francis Crinella calculated that by eight months, an infant fed soy formula daily absorbs approximately 1.1 mg of manganese above metabolic need. "A significant amount, about 8 percent, is deposited in a brain region vulnerable to threat of manganese attack."

Neurology textbooks identify manganese as a neurotoxic metal. In 1837, an English physician noted that some workers in a manganese mill appeared lethargic and their faces unexpressive. By the turn of the century, the disease of "manganism" had been described in medical journals. The disease struck miners exposed to toxic dust, and appeared to cause emotional lability, irrationality, hallucinations and impulsivity. Chronic exposure produced more severe symptoms, including muscular weakness, difficulty in walking, tremor, immobile facial expression, and speech disturbances—symptoms reminiscent of Parkinson's Disease. Sufferers of the Parkinson's-like neurological disease secondary to chronic poisoning accumulate large amounts of manganese in a circumscribed region of the brain.

The primary site of manganese toxicity regardless of the route of exposure—by mouth, inhalation or injection by intravenous tube—in humans, monkeys, rabbits and rats, is a mass of nervous tissue buried deep within the cerebral hemispheres. This is the basal ganglia, part of the extrapyramidal system controlling body movement. The neuronal damage caused by the manganese tends to be more extensive in young, immature animals than in adults.

Six years ago, tragic incidents in two London hospitals alerted the medical community to the vulnerability of sick babies to manganese attack. Suffering liver disease, the babies received nutrient solutions containing small amounts of manganese through intravenous tube feeding. Although the manganese

concentration was no greater than that in soy formula, and considered safe by government standards, it caused brain damage after feeding periods lasting a few months to two years. Of 57 babies receiving "safe" amounts of manganese, two fell ill with movement disorders and six suffered damage to their basal ganglia.

John Donaldson, toxicologist and speaker on Day Two at the UC Irvine conference, described how manganese could cause a biochemical lesion in the basal ganglia. He reported how manganese overload can step up the brain's electric charge, increase its virulence tenfold, and attack vulnerable dopaminergic neurons.

Arvid Carlsson, last year's Nobel Prize winner in medicine, has shown that damage to these basal ganglia dopamine cells is symptomatic of Parkinson's Disease. At the conference, Donaldson warned that when "incredible" amounts of manganese are fed to infant mammals, the metal is capable of "running amok" in the basal ganglia dopamine nerve cells. "After chronic early exposure, they can be brain-damaged later in life," he said.

When Francis M. Crinella, Clinical Professor of Pediatrics at the University of California at Irvine, spoke, he described the effects of manganese overload in adolescents. His research had detected relatively high levels of manganese in the scalp hair of hyperactive children compared to matched controls. This replicated earlier studies by UC Irvine psychiatrist Louis Gottschalk, who detected elevated manganese in scalp hair of youths detained for felony crimes and incarcerated in four Southern California prisons. These findings, wholly unexpected, persuaded Crinella to launch inquiry into the most likely source of manganese in the hair, then to ask whether this had anything to do with hyperactivity in children, a syndrome attributed to a disturbance in the basal ganglia. To Crinella, the low levels of manganese in California soil, air and water meant the primary intake had to be through diet. Since adolescents are able to metabolize at least 97 percent of manganese ingested, exposure had to occur earlier in life, possibly during infancy. This hypothesis was first stated by Collipp in 1983 who had tested hair samples of babies fed soy-based infant formula and found them high in manganese. Crinella speculated that soy infant formula might provide one explanation for the current epidemic of adolescent violence sweeping the nation.

Crinella contacted his colleague Bo Lönnerdal at UC Davis to take a further look at the effects of manganese on the brain, particularly its toxicity to dopamine neurons in the basal ganglia. Lönnerdal and a graduate student Trinh Tran tested for behavioral and brain disorders in rat pups. For 18 days, four groups of rat pups suckled on the mother's breast and received by micropipette an additional dose of manganese salt dissolved in water. The doses corresponded to the amounts of manganese found in rat breast milk (0.05 mg) and several brands of soy-based infant formula (0.25 mg and 0.50 mg) found on pharmacy shelves today. The control group received just sugar water (0.0 mg). After 18 days of controlled feeding, the rat pups were returned to their cages and left undisturbed until 50 days of age. Then through Day 64 they were given behavior tests for evidence of disability. The animals given high amounts of manganese did less well on maze and shock avoidance than those given lesser amounts.

The audience now turned their attention to the next paper, by Francis Crinella,

on levels of basal ganglia dopamine. Crinella's data were clear-cut, unmistakable and replete with implications. Rats given 0.05 mg of manganese daily for 18 days, the amount comparable to the manganese in breast milk, did as well as the control group given no manganese. Rats given supplemental manganese in the dose five times higher, or 0.25 mg, suffered a 48 percent decline in levels of basal ganglia dopamine. The rats dosed daily with the highest amount, 0.50 mg, had a staggering 63 percent plunge in dopamine.

When asked the meaning of these dramatic findings, Crinella answered that many labs previously had reported the toxic effects of manganese. The basal ganglia frequently were the target for neurotoxic effects. Dramatic declines in dopamine due to manganese overload had been reported before. He also described the lingering threat of toxic alterations in brain cells weeks after manganese is discontinued.

The value of Crinella's data and that of Trinh Tran was that they provided a link between a moderate manganese exposure during early infancy, dopamine neurotoxicity and the possibility of cognitive disorders in later life.

"The brain undergoes a tremendous proliferation of neurons, dendrites and synapses during the first months of life. Some neurons will be pruned during childhood for maximum information efficiency," said Crinella. "The brain is especially vulnerable in early life precisely because such rampant growth is taking place, and at that time intrusions by potentially toxic substances like manganese perturbing the emerging neural organization can exert long-term effects. Manganese ingested during a period of rapid brain growth and deposited in the critical basal ganglia region may affect behavior during puberty when powerful stresses are unleashed on the dopamine neurons and altered behavioral patterns appear." According to Crinella, these altered behavioral patterns during late childhood and early adolescence may be diagnosed as hyperactivity with attention deficit disorder.

Or perhaps as a "manganese toxicity syndrome." Crinella's presentation provoked much discussion. Is the manganese ingested in soy formula at infancy a source for behavioral disorders later on? Bo Lönnerdal and Carl Keen were impressed by the findings but warned against premature generalization. Young rats appear more susceptible than human babies to manganese toxicity. They absorb 80-85 percent of the manganese they ingest, while the figures for human infants at six months old are closer to 35 percent. It is in providing the worst-case scenario of what can happen to human infants fed manganese that the rodent research may prove most instructive.

A dissenting opinion about soy dangers came from John Lasekan, a pediatric nutritionist at Ross Products Division of Abbott Laboratories. His published research claims that manganese is a trace metal absolutely essential for life and that premature and low birth weight infants may be at risk for developing a deficiency in manganese. He claims that the soy-based formulas support normal growth and normal plasma biochemistry, comparable to infants fed human milk during at least two months of life. Mardi Mountford, spokesman for the International Formula Council adds: "There are no reports of manganese toxicity in healthy infants fed soy formula. Parents can be assured that infant soy formulas are safe and nutritious feeding options for their infants."

Yet some remain unconvinced. "It's overwhelming," says Everett "Red" Hodges,

founder of the Violence Research Foundation, citing the evidence supporting Crinella's hypothesis that infants ingesting soy-based infant formula at the levels available in commercial products 15 years ago might be at risk. "Criminals aged sixteen and seventeen years old today, some of whom were born to poor mothers in 1983 and 1984, could have received from the government soy formula with enough manganese to disrupt growing brains, and this may be why these adolescents have difficulty restraining aggressive impulses today."

Stanley Van Den Noort, a neurology professor and former Dean of the UC Irvine College of Medicine, agrees with Hodges and Crinella. "I think the data presented at the conference are convincing that manganese is a neurotoxin. Newborn infants exposed to high levels of manganese may be predisposed to neurological problems. We should exercise strong caution in the use of soy-based formula around the world."

Whether or not the manganese in soy formula today, with an average value of 0.16 mg per quart (0.15 mg per liter), poses an acute danger may be secondary to the issue of why more and more mothers in the United States imagine they have given birth to a baby soy bean instead of a human child. "Why else feed so many newborn infants soy 'milk'?" asks Naomi Baumslag, Clinical Professor of Pediatrics at Georgetown University Medical College and President of the Woman's International Public Health Network. For years Baumslag has waged a campaign against the medical profession's cavalier attitude towards soy infant formula. "Only 50 percent of newborns today suckle at the mother's breast, even once. After six months, the number has fallen to only one mother in five. Often, mothers for the sake of convenience plunk soy bottles into the infant's mouth. Sales of soy formula have doubled during the past ten years." Baumslag states, "There is great deal of scientific evidence that soy formula can be damaging to newborns, quite aside from the manganese." Soy "milk" can be dangerous for what it has and does not have. A spoonful of soy formula lacks many nutritional, immune and developmental factors. The spoonful may be deficient in linoleic and oleic essential fatty acids, DHA-brain growth factor, epidermal growth factor, lactoferrin, casomorphin, and immune factors like IgA, neutrophils, macrophages, T-cells, B-cells and interferon that mother's milk provides to defend her baby. The spoonful of soy "milk" unfortunately, does contain phytates, protease factors, soy lectins, enormous amounts of phytoproteins, and genistein, a moderately potent estrogen-mimic in humans. She asks, "Why deprive the newborn infants of perfectly good breast milk—nutritionally superior food in every way for the baby—and feed them soy beans?"

The powers in government and corporations have not reacted to these voices raised against the potential dangers of manganese in soy infant formula. The government can hardly be unaware of the simple logic: (1) Excess manganese is toxic. (2) Babies absorb excess manganese. (3) Excess manganese is toxic to babies. Carl L. Keen believes that the original administrative problem was that the government established teenage requirements for manganese, then extrapolated backwards to determine a level they believed to be safe and acceptable for toddlers and newborns. The problem of infant exposure to excessive manganese identified 15 years ago still persists, but what can scientists like Drs. Keen and Crinella do about it?

Sitting at his desk in the Social Ecology building, Jonathon Ericson pondered

how he could bring the soy infant formula problem to the public's attention. Why not, he thought, provide the answer at the end of the two-day conference? Day One would fill the audience's mind with indisputable evidence that a lead compound of the tetraethyl variety, from inception as a gasoline additive in the 1920s until its removal from fuel in the 1980s, was causing brain damage in children around the world. Day Two would extend the warning to manganese, both in the antiknock compound MMT and as a contaminant in baby formula. What he did was invite two government policy makers, Robert Presley and Phillip Lee, to discuss what society must do today to resolve the soy formula crisis.

Chairing the panel was Senator Robert Presley, California State Secretary of the Adult & Juvenile Corrections Agency, responsible for 170,000 incarcerated felons. Presley thanked Jon Ericson for providing him with the challenge. His solution was to recommend increased funding for studies of brain development. When asked why this was important, he said, "Somewhere in the soy formula story may lie the answer to a lot of crime." Phillip R. Lee, Former U.S. Undersecretary of Health and Human Services, now Senior Advisor to the Institute for Health Policies, took a moment to applaud independent research. Then he offered his advice: "The MRI scan detected brain damage in the sick babies in London. In the U.S., we might identify sensitive populations of newborns, then launch longitudinal studies combining the scans and behavioral testing to find out what infant feeding has to do with aberrant behaviors occurring during late childhood years."

Two conclusions emerge from the conference. First, the need to educate the public about the potential dangers posed by the soy formula now fed to 750,000 infants per year. Second, to accelerate studies on the effects of toxic metals on the brain and on human behavior.

How the unfolding melodrama will end, nobody knows. Since the September, 2000 conference, scientists are stepping up their efforts to pinpoint the manganese syndrome. They are investigating the effects on calcium and iron deficiency in pregnant rat dams, known to enhance uptake of manganese in the infant. Second, they are going to look more carefully at the effects of manganese excess in infant primates.

Meanwhile, manganese levels in soy formula remain high. One soy-based product on the shelf today provides up to 0.72 mg manganese daily. And soy products for infants sold in foreign countries can be even higher.

In 1983, Phillip Collipp offered the following advice to the formula industry: "Reduce manganese in infant formula to the levels found in human milk." So far, the industry has not responded.

*[David Goodman](#), PhD is a neuroscientist and journalist whose popular writings feature information on healthy brain development and its enemies.*

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