Vital Link

The professional journal of the Canadian Association of Naturopathic Doctors

Feature Articles

The Changing Faces of Lead

Volume 19, Issue 1

Spring 2012

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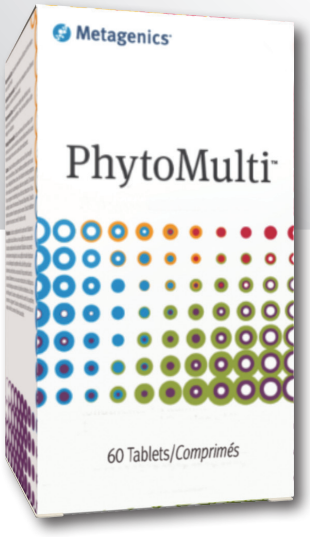

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- **5-HTP SAP** can be used to help treat mild to moderate depression and bipolar disorder;
- **5-HTP SAP** may be helpful to treat insomnia;
- **5-HTP SAP** is a treatment option for children suffering with night terrors;
- **5-HTP SAP** can be used to help reduce the pain associated with fibromyalgia.

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Vital Link

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The *Vital Link* is the professional journal of the Canadian Association of Naturopathic Doctors (CAND). It is published primarily for CAND members and features detailed reviews of specific causal factors: philosophical and research-based papers, clinical practice articles and case reviews, as well as international updates on the profession. The *Vital Link* has an outreach to other health care professions and promotes qualified naturopathic doctors to corporations, insurance companies and the Canadian government.

Forthcoming Themes

Summer 2012 Assessment and Diagnosis: A New Era

Fall 2012 Occupations and Health

Submissions
When writing for the *Vital Link*, keep in mind its broad readership and outreach to other professions. Your contribution to the *Vital Link* will benefit the naturopathic profession as a whole and provide you with personal professional exposure. Previously unpublished material is preferred. Please contact the managing editor for submission guidelines.

Circulation
The *Vital Link* is published three times per year and is distributed to over 2000 qualified Canadian NDs and students of CNME-accredited naturopathic programs in Canada and the U.S. The *Vital Link* is also distributed to the CAND's corporate members and in our media kit. The journal is available in print and e-formats, by paid subscription.

Advertising
Professional vendors providing NHPD-compliant products or other services to NDs are encouraged to advertise in the *Vital Link*. The CAND's advertising partners enjoy unequalled exposure to qualified Canadian naturopathic doctors.

Naturopathic Notes

Dr. Iva Lloyd, BScH, RPE, ND

The increasing impact of heavy metals and environmental toxins is forcing naturopathic doctors to re-examine how they practice. The causal factors of disease are not as clear cut as they used to be resulting in many conditions being resistant to standard treatments, whether conventional or naturopathic. Understanding health and disease is becoming increasingly complex.

This edition of the *Vital Link* examines the impact of lead exposure on health. It reviews the history of the issue of lead exposure and how the heavy metal has become a threat to health that can neither be ignored nor avoided. Dr. Marianne Trevorrow in her article "The pediatric lead labyrinth: a 2012 ND update" reviews the effects of lead on children and the assessment and treatment options available. Infants and children appear to be the population most affected by exposure to the metal. Lead crosses the placenta and affects development both *in utero* and in infancy.

Dr. Scott Clack's article "Lead in the elderly: normal aging or premature effects of a heavy metal?" reveals the long-term impact of lead exposure. His article very nicely walks a reader through key assessment questions that will assist in determining toxic load, the diagnostic considerations and the precautions associated with chelation in this population. Lead exposure has been linked to chronic diseases of almost every system including cardiovascular, musculoskeletal, endocrine, neurological and immune. Like many heavy metals it disrupts normal cellular function and hence its impact is pervasive and persistent. A compelling argument is made for the need to assess for heavy metals in the elderly.

In the article "The changing faces of lead-induced health problems" Dr. Walter Crinnion looks at the different sources of lead exposure over time and the battles whose outcomes exposed the detrimental effects of lead on health. Dr. Crinnion also examines the storage of lead in the body and the challenges in decreasing body burden.

Dr. LC Masur in his article, "*No lead is good lead*: Towards a lower threshold for the diagnosis of lead poisoning" provides a detailed review of the research on lead, including the different sources of lead, its toxic effects, the importance of lowering "acceptable" lead levels especially in children and the different diagnostic testing methods.

Dr. Lyn Patrick's paper rounds out the issue, reviewing the current literature on lead toxicity causes and effects.

Lead exposure at one time was classified as harmless, and its potential health risks were disregarded. I am disconcerted by the similarity between this situation to that of mercury and EMF exposure. Our primary challenge is an unwillingness of government and decision makers to learn from the past and embrace the precautionary principle as a way of living and of making decisions, especially with respect to health care.

Testing for lead and other heavy metals is becoming a requirement for each patient. Educating patients about the importance of heavy metal detoxification prior to conception needs to become an expected basic standard of care. Addressing a person's heavy metal body burden is not only part of preventative care but is becoming an integral part of successful naturopathic treatments.

I hope you enjoy these articles. We welcome your feedback.

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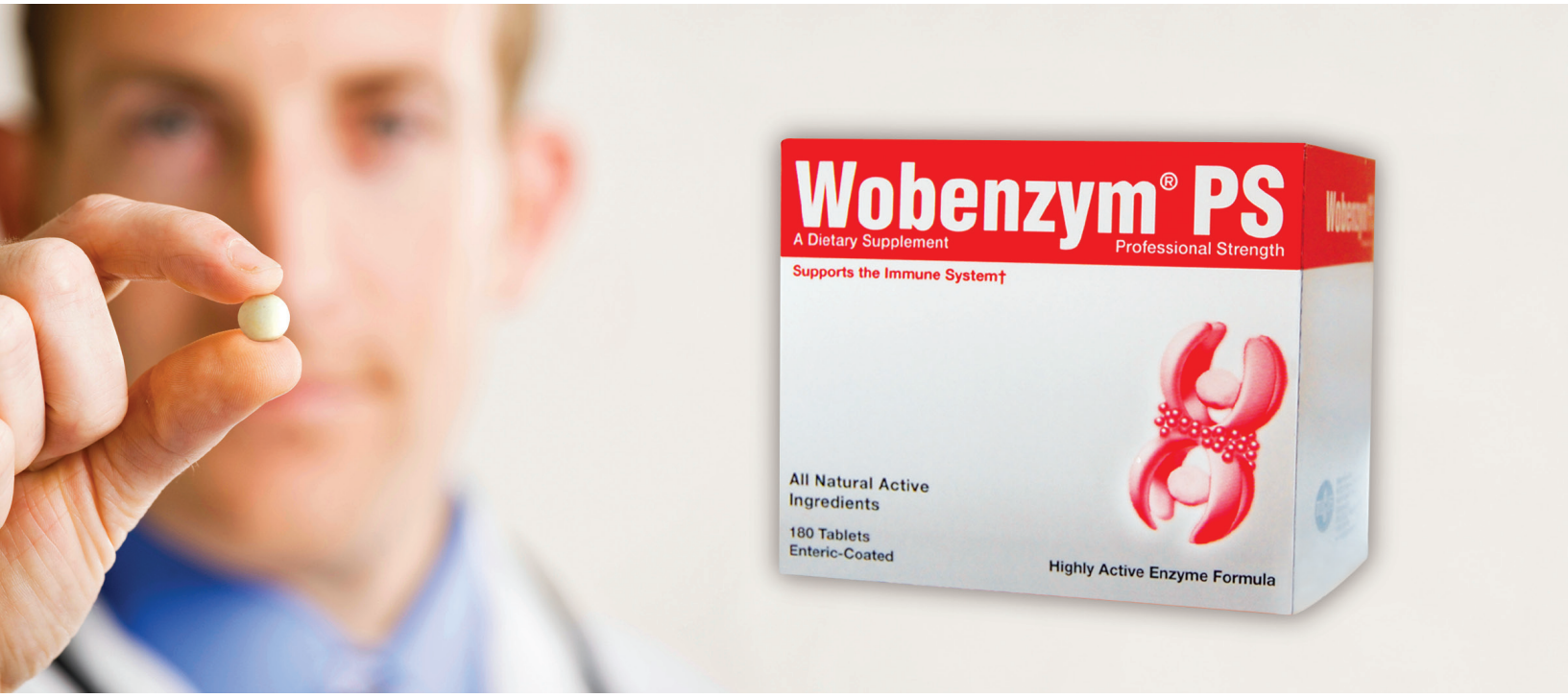

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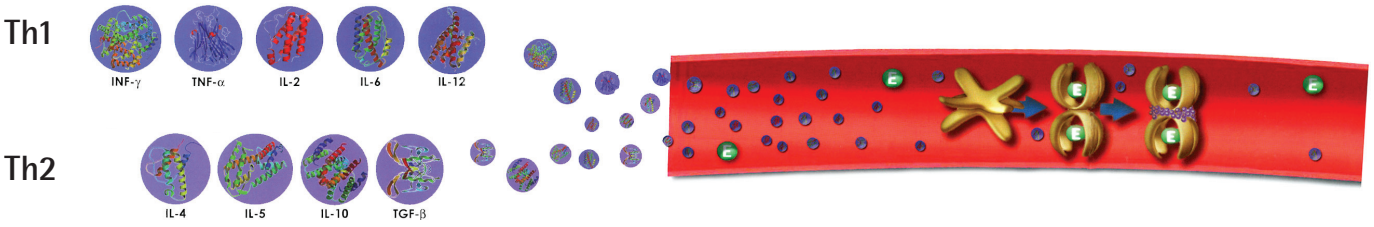

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Four Corners: Updates on the Profession

Canadian Association of Naturopathic Doctors (CAND) *www.cand.ca*

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This year marked the first time membership renewal was available online and it has been a great success with approximately 90% of NDs choosing to renew online. We are pleased to report that we have seen an increase over last year in new members reflecting support for the CAND's work on behalf of the profession nationally.

In late October the CAND held its Annual General Meeting in Vancouver, outlining our accomplishments during the past year, presenting our financial statements and electing three new board members. The CAND directors for 2011/2012 are: Dr. Jason Boxtart, ND, Chair (BC), Dr. Pat Wales, ND, Vice-Chair (AB), Dr. Lowell Greib, ND, Treasurer (ON), Dr. Meghan Walker, ND, Secretary (ON), Dr. Lois Hare, ND (NS), Dr. Parissa Bunin, ND (NB), Dr. Jilla Kahrobaei, ND (QC), Dr. Melanie Leppelmann, ND (MB), Dr. Amy Hiebert, ND (SK) and Dr. Tonia Winchester, ND (BC). A board planning session followed in November where the board reviewed progress on the CAND's strategic plan and set goals for the year. New committees struck were EMR and Data Collection to assist in gathering patient outcomes for use with government, insurance companies, corporations and allied health care professionals and associations, and the "Why" committee revisiting the reasons why people seek out an ND and how that might impact our marketing strategy.

Government relations will continue to be a key focus for the CAND as we continue our work on the NHPD Program Advisory Committee, engage a lobby firm to assist on issues around access and push for a greater recognition for the profession and the inclusion of NDs as practitioners in all health related federal legislation. Recently we partnered with Health Canada in creating and conducting a needs survey with NDs on adverse reaction reporting. The information collected will be used by Health Canada to develop education and training tools for health-care professionals.

The CAND supports regulation of the profession and is currently engaged in assisting a number of the provincial associations in their regulatory lobbying efforts and/or the development of updated regulations. An outline of the current regulatory status in Canada can be found under the "Regulation in provinces and Territories" tab at www.cand.ca/index.php?40

American Association of Naturopathic Physicians (AANP) *www.naturopathic.org*

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The American Association of Naturopathic Physicians is a mission driven organization. Inspired by our members' desires, the Board of Directors defines its priorities from which a staff created work plan is designed. Our work is focused in three areas: expanding consumer awareness of naturopathic medicine, expanding state and federal recognition of naturopathic medicine and providing the tools our members need to be successful in their practices. For us, success in each of these arenas requires we aspire to the highest standards of naturopathic medicine.

On January 23, 2012, the U.S. Department of Health and Human Services (HHS) issued a federal register notice defining "priority health professions" for the Indian Health Service Loan Repayment Program (IHS LRP), which awards up to $20,000 per year for the repayment of qualified student loans in exchange for an initial two-year service obligation to practice full-time at an Indian health program site. Naturopathic medicine was included in the list for the first time, representing the culmination of work by the AANP and the grassroots efforts of its student and physician members. HHS and the IHS have opened their doors to naturopathic doctors, and American Indians and Alaska Natives will now have access to naturopathic medicine. Eligibility for the IHS LRP is based on Tribal needs and requests for naturopathic medicine. To this end, the AANP and the Association of Accredited Naturopathic Medical Colleges are working together to increase demand for NDs.

More broadly, this victory paves the way for ND inclusion in all Federal loan repayment programs and cements the credentials of naturopathic medical education. It also will give the naturopathic community leverage in defining what integrative medicine and complementary and alternative medicine practitioners mean during Patient Protection and Affordable Care Act (PPACA) implementation.

Want to get involved in expanding federal recognition of NDs? Participate in the AANP's DC Federal Legislative Initiative (DC FLI). The DC FLI is being held May 5-7, 2012 in Washington DC. Also, please mark your calendars for the AANP's Annual Convention in Bellevue, WA, August 15-18, 2012.

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1. Dyerberg J, et al. Bioavailability of marine n-3 fatty acid formulations. *Prostaglandins Leukot Essent Fatty Acids* 2010 Sep;83(3):137–141.

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Council on Naturopathic Medical Education (CNME) *www.cnme.org*

The Council on Naturopathic Medical Education (CNME) accredits naturopathic doctoral (ND) programs in Canada and the U.S., and graduation from a CNME-accredited or pre-accredited ND program is a requirement for taking the NPLEX exam and becoming licensed or regulated as a practitioner. Currently, the CNME accredits two ND programs in Canada and four in the U.S., and also pre-accredits one program in the U.S. We invite practitioners who are potentially interested in becoming involved with the Council's important work to contact the Council.

There are a number of interesting developments happening in naturopathic medical education. For the first time, the CNME has approved the establishment of a branch campus by an existing accredited ND program. Starting in the fall of 2012, Bastyr University in Seattle, Washington, will be offering its ND program in San Diego, California.

For several years now, the Universidad del Turabo (UT) in the Commonwealth of Puerto Rico has been offering an ND program in Spanish. UT has submitted an "application for consideration" to the CNME — which is the first step in seeking CNME accreditation — and representatives from UT will appear before the Council at its meeting in May of 2012 to present information on the ND program and answer the Council's questions. Approval of an application for consideration does not confer any CNME accreditation status, but it does allow the university to move forward to the next step of the process: the submission of a self-study report for candidacy status (i.e., pre-accreditation). If UT is successful over time in seeking CNME accreditation for its ND program, this will be an important step in establishing naturopathic medicine as a recognized healthcare field in Latin America.

Finally, the Council is in the process of revising its standards for approving naturopathic residency programs to make the standards more streamlined while still maintaining the rigor necessary to ensure a high quality educational experience. Currently, the CNME recognizes the following institutions as sponsors of CNME-approved residency programs: CCNM, SCNM, Bastyr University, and NCNM.

Association of Accredited Naturopathic Medical Colleges (AANMC) *www.aanmc.org* *@AANMC*

Indian Health Service and Naturopathic Doctors *Next Steps to Ensuring Underserved Populations have access to naturopathic medicine*

IHS' announcement that NDs are eligible to participate in the Indian Health Service Loan Repayment Program is just the first step to ensuring American Indians and Alaska Natives have access to naturopathic medicine. Each Tribe now has the ability to include naturopathic medicine as 'a priority health profession' and request NDs to support their health care needs. Unfortunately, few have been introduced to naturopathic medicine and it is incumbent upon us to educate stakeholders on how NDs can improve health status for this underserved population.

Approval of applications by licensed naturopathic physicians will rely on how the tribes rank their critical health care needs. Priority will be given to professionals who themselves are American Indian or Alaska Natives, as well as to "individuals recruited through the efforts of Indian Tribes or Tribal or Indian organizations." Applicants to the program *will only be accepted for positions advocated for by the tribes themselves,* necessitating the creation of strong professional relationships and cultural understanding of healthcare delivery on native lands. There are examples of successful relationships with tribes that independently hired NDs, and we have a great opportunity to replicate these programs. AANMC is working with AANP to gather additional information from inside the system identify potential partnerships with geographically and philosophically like-minded tribes. With patience and planning, we will create opportunities for graduates that will lead to broad-based utilization of naturopathic medicine, starting in this current award cycle.

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The Changing Faces of Lead-Induced Health Problems

Dr. Walter J. Crinnion, ND

Thanks to the pioneering work of Dr. Herbert Needleman, the role of lead in reducing the IQ of children was brought to light. While fighting for the health of children, Dr. Needleman came under attack from others who claimed that his research was bogus. Thankfully his work was vindicated and resulted in a widespread campaign to reduce children's exposure to lead. His work also resulted in lead being removed from interior household paint and gasoline in the late 1970s – two of the most common sources of lead exposure.

For decades, since Dr. Needleman got this information out, the most commonly understood health effect of lead was its ability to reduce IQ and learning ability in children. Common knowledge held that this was most predominate amongst inner-city children who were exposed to lead by chewing on the window sills of their homes. Yet, in the last decade studies have revealed that levels of lead below 10µg/DL cause a greater reduction in IQ in children than levels above 10µg/DL. Fortunately, the CDC very recently moved to reduce that level to 5µg/DL, but unfortunately this reduction may not be sufficient as newly published research shows that levels as low as 2 µg/DL can cause neurologic damage. Childhood lead exposures have also been associated with increased rates of attention deficit hyperactive disorder (ADHD).

Until 1978, lead was used in interior paint, when it was replaced by mercury (which may be an improvement, but is still a health risk). Lead has the ability to break down to a very fine particulate dust that stays in the air ducts of homes and gets embedded into the carpets and fabrics. Anyone living in a home that was built before 1978, is quite likely exposed to lead from the dust residing in their heating and air conditioning vents. Lead was also used in gasoline, meaning that soil in cities and areas close to roadways can have levels of lead far above safe limits. This is hazardous because this lead can then be tracked into the home on shoes and be blown in on dust and debris as well. Lead is used in paints because it makes brilliant colors and today is still used in ceramic paints, leading to cases of lead poisoning from plates, bowls and cups. This is especially true when these vessels are used to contain acidic foods and drinks. Lead is used in the vinyl making process and has been introduced into homes via vinyl mini-blinds, Christmas-tree lights and cables for computer joysticks. Other common sources of lead include metal-wicked, slow-burning candles which release lead into the home air, and some natural supplements Calcium supplements sourced from bone-meal rather than egg shells or oyster shells have been found to contain lead. Very high levels of lead have been repeatedly found in both traditional Chinese medicines and Ayurvedic herbal preparations.

While it is important to be aware of the most common external sources of lead exposure we must consider internal sources as well. While the half-life of lead in the blood is estimated to be approximately 35 days this can be misleading, as once lead enters the body, it typically stays in the body. Lead moves from the blood to other storage places within the body that are less easily measured. In fact, ninety-five percent of the lead in humans is stored in the bones, with the remaining 5% being housed in the soft-tissues of the body such as the brain, muscles, organs, etc. Bones are not static, and as they turn over they release calcium and lead into the blood stream. As a result lead levels go up in women who are pregnant, breastfeeding and who have osteoporosis (all times of enhanced bone turnover). In pregnant and breastfeeding women this is an added concern as it provides a source of lead exposure for the infant. In my practice, I had one woman whose lead burden was passed to her daughter and then to her grandson. Women with a high lead exposure earlier in life are also at higher risk for lead-induced health problems as they pass into menopause. As estrogen levels decline, there is an increase in bone turnover, and soft-tissue levels of lead rise.

Any exposure can result in a wide-range of lead-induced health problems primarily associated with the central nervous system and the cardiovascular system.

- Lead has demonstrated the ability to reduce the production of nitric oxide (NO) in the body, which is our natural vasodilator. This resulting drop in NO can manifest as an elevation in blood pressure, and should be considered in anyone with blood pressure that is resistant to standard blood pressure treatment. In fact, for each 1µg/DL increase of lead in the blood, homocysteine increases by 0.35nmol/L, which in and of itself has many adverse health effects. Elevated

lead levels have been associated with increased rates of stroke and heart attack and elevated lead levels can increase one's risk of dying from cardiovascular problems by a factor of 5.6! In fact, elevated bone lead levels (measured by x-ray) are associated with increased rates of all-cause mortality by a factor of 2.5. Since cardiovascular disease is one of the two most common causes of death it would be wise for NDs to test for lead in anyone with cardiovascular problems.

- Lead has been shown to reduce glutathione levels in the body. This, coupled with lead's ability to cause oxidative damage, greatly affects the tissues most sensitive to oxidative damage such as the central nervous system (including the eyes) and the cardiovascular system.

- Adults and children with elevated lead levels demonstrate reduced learning ability, and problems with memory, mood, coordination and balance.

- Lead has recently been associated with an increased risk of Parkinsonism, both in the medical literature and in my own practice. The vast majority of my Parkinsonian patients are post-menopausal osteoporotic women with high tissue levels of lead. These women lived for several decades while lead was in common use in gasoline and paint, and are now paying the price with their health.

- Individuals with elevated lead levels are over twice as likely to be depressed and over five times more likely to have panic disorder. Yet, lead testing is rarely found on any differential diagnostic algorithms for either of those problems.

- In addition, lead is known to cause microcytic anemia, fatigue, and kidney damage. When one looks at the increased oxidative damage from lead, along with its ability to reduce glutathione, it becomes evident why lead is a known mitochondrial poison. Its ability to damage the mitochondria is a major factor in reduced energy (fatigue) as well as the neurologic and cardiovascular issues. When the mitochondria are poisoned by lead or any other environmental toxicant, the cell will not receive the energy it requires, will not be able to do its function resulting in early cell death. Mitochondrial dysfunction has now been associated with many chronic illnesses including adult-onset diabetes and obesity and it is undoubtedly present in all of the patients with chronic fatigue syndrome, as well. The combination of reduced mitochondrial function and enhanced oxidative damage would obviously result in many of the signs and symptoms that we associate with aging.

The United States Centers for Disease Control is conducting ongoing studies to detect the common toxicants in US residents. Their studies, along with similar Canadian studies have shown that we all have lead in our blood and urine. However, urinary and blood lead are not reflective of one's total lead burden, but are mostly representative of current exposure (including that coming from bone). What is unknown is the level of lead burden that is commonly carried by all of us. But, because we all have some level of lead in us, when someone exhibits any of the multiple symptoms associated with lead, it might be reasonable to assess them for current lead exposure along with their body burden. This can be most easily accomplished by testing their first morning urine for lead content (along with the other heavy metals), and then again after the administration of a chelating agent (such as DMSA).

If an individual's health problems are being triggered by a body burden of lead and it is not appropriately diagnosed or addressed, then the symptoms will persist. Since all of us harbour lead and most people present with some of the common symptoms associated with its presence it is imperative that Naturopathic Doctors start thinking about this common toxicant more often.

About the Author

Walter J. Crinnion ND received his degree in Naturopathic Medicine from Bastyr University in Seattle, Washington in 1982 with their first graduating class. He then opened a family practice and began to specialize in allergies and in treating chronic health problems caused by environmental chemical overload. In 1985 he opened the most comprehensive cleansing facility in North America for the treatment of chemically poisoned individuals. He is a favorite and frequent lecturer at both Naturopathic and Allopathic (MD) medical conferences. He has published several articles in peer-reviewed journals on the topic of environmental overload. He has been on the board of directors of the American Association of Naturopathic Physicians and was the recipient of their first award for in-office research in 1999 and was awarded it a second time in 2002. He has been on the adjunct faculty of Bastyr University (Seattle, WA.), the National College of Naturopathic Medicine (Portland, OR.), and the University of Bridgeport School of Naturopathic Medicine (Bridgeport, CT.). He is a professor at the Southwest College of Naturopathic Medicine (Tempe, AZ.) and the chair of their Environmental Medicine Department. In 2001 he appeared three times with Barbara Walters on ABC's "The View". His first book: *Clean, Green and Lean* was published by Wiley and Sons in 2009.

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"No Lead is Good Lead" – Towards a lower threshold for the diagnosis of lead poisoning

LC Masur, MD

Regulatory bodies continue to grapple with the concept of what constitutes lead toxicity and are moving slowly towards the position held by many health practitioners that "no lead is good lead". The action level ("intervention level") commonly accepted as the threshold for the diagnosis of lead toxicity ("poisoning") has dropped from a blood lead level (BLL) of 15 μg/dL (0.724 μmol/L) in 1991[1] to the current value of 10 μg/dL (0.483 μmol/L)[2].

At the current action/intervention level, about 250,000 children in the United States are thought to have lead poisoning and, by extrapolation on the basis of population, about 25,000 Canadian children also may have lead poisoning[3].

A November 2010 publication from the Centers for Disease Control and Prevention (CDC) implied that a new, much lower, threshold BLL of 5 μg/dL (0.241 μmol/L) might be more appropriate, especially in pregnant women "where there is good evidence that maternal lead exposure during pregnancy can cause fetal lead exposure and can adversely affect both maternal and child health".[4] More recently, a January 2012 Blood Lead Work Group report[5] recommended that a BLL of 5 μg/dL (0.241 μmol/L) be adopted as the new threshold to identify children with elevated blood lead levels.

Lead – Background and Historical Summary

Lead (Pb) is a heavy, soft, white/bluish-gray metal which occurs naturally but rarely in the pure form, often being found in ores with zinc, silver, copper and other elements. It is released into the environment by natural events such as soil erosion and volcanic or thermal eruptions. Because of its many useful properties, such as resistance to corrosion and malleability, as well as the fact that it can be readily alloyed with other metals, lead has many industrial and commercial applications. Most of the high lead levels found currently in the environment come from anthropogenic activities such as mining, and from manufacturing processes that produce lead, lead alloys or lead compounds. Environmental levels of lead have increased more that 1000-fold over the past three hundred years due to human activity, most of this occurring in the fifty years leading up to 2000 and mostly related to the use of leaded gasoline.[6] In the United States (USA), the Environmental Protection Agency (EPA) banned the use of leaded gasoline as a fuel for highway transportation after December 31, 1995.

Common Sources of Lead

The most common uses and anthropogenic sources of exposure to lead and its alloys are in batteries and ammunition. Other common uses are in pipes, caulking compounds, weights, cable covers, radiation shielding, and as pigments in paints, dyes and glazes. Lead can be released into the environment in the combustion of coal, petroleum products and the incineration of trash, as well as released into drinking water distributed through pipes connected with lead-based soldering compounds.

Less Common Sources of Lead

Less common sources of lead include leaded gasoline, which is still available for use as fuel in small aircraft and by off-road vehicles (over the past 30 years leaded gasoline has been phased out as a highway transportation fuel in most countries of the world). Individuals in occupations such as lead smelting and refining industries, brass/bronze foundries, the manufacture of rubber products, the plastics industries, soldering, steel welding and cutting operations, battery manufacturing, lead compound manufacturing, construction and demolition industries, or who are firearms aficionados (lead bullets), fishers (lead weights) or who have hobbies such as oil painting and stained glass making where lead is used may be at higher risk for lead exposure.

Lead from past anthropogenic activities is still an ongoing issue. Lead contaminates soil along highways (from leaded gasoline exhaust), is found in soils in orchards and farm fields (from lead in pesticides), in dusts inside older buildings (from the chipping and wearing of lead-based paint), and in water (due to run-off from landfills containing waste from the manufacture of ammunition and batteries and from tailings from lead ore mines). Ingestion of alcohol produced from illegal stills and inhalation of tobacco smoke may be a source of lead. Inexpensive jewelry may contain high levels of lead and thus may be a source of exposure and potential transdermal absorption of this element. Hair darkening agents may contain lead acetate (prohibited in Canada but not in the USA) which is readily absorbed transdermally. Some non-

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western cosmetics such as surma and kohl may contain lead. Rasa Shastra ayurvedic medicines may contain lead and other potentially toxic metals.[7]

Lead from contaminated soil and water can build up in plants and the animals that consume them, and, if they are part of the human food supply, can be a potential, although less common, source of lead exposure.

Extensive information on the sources of lead, as well as other reference materials, is available on the ATSDR web site.[6]

Summary of Toxic Effects of Lead

There are many factors that can influence whether adverse health effects due to lead exposure will or will not occur. The dose, the duration of the exposure, the route of administration (oral ingestion, inhalation, dermal contact, etc.) and how much of the dose was absorbed are many of the factors that need to be considered. One must also take into account exposure to other chemicals that occurred concurrently[5] and any additive or multiplicative effects which may make matters worse, the age and gender, dietary habits, intake of certain nutrients, family traits, lifestyle and the general state of health.

The main target for the adverse health effects of lead is the nervous system. It may cause weakness in the wrists, the distal joints of the hands, the ankles and distal joints of the feet. Increased lead levels may cause increased blood pressure especially in middle-aged or older adults. Lead may be associated with the development of anemia. In cases of severe toxicity it can damage the brain and kidneys in both adults and children and may lead to death. High levels may be associated with miscarriage, premature delivery and low birth weight and in males may cause decreased production of sperm. According to the International Agency for Research on Cancer (IARC), lead is not known to be a definite carcinogen in humans, but is thought by the EPA to be a "probable" carcinogen. Extensive information on the toxic effects of lead, as well as other reference materials, is available on the ATSDR web site.[6]

Evidence in Support of Lowering the Blood Lead Level (BLL) Indicative of Toxicity Especially in Children

Children are more vulnerable to lead toxicity/poisoning than are adults; this is the case from conception, in utero and all through infancy, childhood and even the teen-aged years. Non-adults are also more sensitive to the adverse health effects of lead than are adults and "no safe blood level in children has been established".[6] The adverse health effects of lead are mostly dependent on the dose and the duration of exposure. Higher doses may result in anemia, kidney damage, colic, muscle weakness, brain damage and even death. Lower doses may be reflected in abnormal blood cells (e.g. basophilic stippling) as well as in developmental and behavioral

abnormalities. At even lower levels, lead still can have detrimental effects on mental development, IQ and physical growth.

Conversion of Blood Lead Levels in µg/dL to Syteme International (SI) Units

Blood Lead Levels (BLL)

1.0 µg/dL = 0.04826 µmol/L
5 µg/dL = 0.241 µmol/L
10 µg/dL = 0.483 µmol/L
15 µg/dL = 0.724 µmol/L
20 µg/dL = 0.965 µmol/L
25 µg/dL = 1.206 µmol/L
30 µg/dL = 1.448 µmol/L
35 µg/dL = 1.689 µmol/L
40 µg/dL = 1.930 µmol/L
45 µg/dL = 2.172 µmol/L
50 µg/dL = 2.413 µmol/L

It is well known that lead can severely affect the cognitive development and behavior of young children.[9,10] For children less than 6 years of age the Center for Disease Control (CDC) has defined an elevated blood lead level (i.e. "lead poisoning") as ≥10 µg/dL (≥ 0.483 µmol/L); a blood lead level (BLL) above 15 µg/dL (≥ 0.724 µmol/L) requires specific treatment actions; with a BLL above 30 µg/dL (≥ 1.448 µmol/L) there is an even more serious concern and further interventions are required; and with a BLL above 70 µg/dL (≥ 2.378 µmol/L) there is increased probability of seizures, profound disability and even death.

In addition to the concerns about "lead poisoning", there is considerable evidence for subtle effects at even lower blood lead levels.[11] A 2007 article in *Pediatrics* – "Interpreting and managing blood lead levels of less than 10 µg/dL in children and reducing childhood exposure to lead: recommendations of the Centers for Disease Control and Prevention Advisory Committee on Childhood Lead Poisoning Prevention"[12] – is a very helpful resource.

As the BLL goes up, the potential for serious health and development problems increases. Children under the age of about 36 months are at the greatest risk for increased lead exposure, absorption and consequent adverse health effects as most of their waking hours are spent at or near floor/ground level resulting in increased access and exposure to lead hazards. Additionally, the developing nervous systems of these young children are more susceptible to the adverse effects of lead.

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Some evidence in support of lowering the blood level indicative of toxicity in children:

To reiterate: "no safe blood level in children has been established".[6]

In addition to the concerns about "lead poisoning", there is considerable evidence for subtle effects at even lower blood lead levels (BLL).[11]

Lead has long been known to alter the hematological system by inhibiting the activities of several enzymes involved in heme biosynthesis:[6]

- δ-aminolevulinic acid dehydratase (ALAD) is particularly sensitive to the action of lead. Inhibition of ALAD activity occurs over a wide range of BLLs beginning at <10 µg/dL (0.483 µmol/L);
- The anemia induced by lead is primarily the result of both inhibition of heme synthesis and shortening of erythrocyte lifespan;
- Lead also can induce inappropriate production of the hormone erythropoietin leading to inadequate maturation of red cell progenitors, which can contribute to the anemia.

A recent study in children 8–10 years of age suggested that lead accelerates skeletal maturation, which might predispose to osteoporosis in later life. Lead also has been associated with increased occurrence of dental caries in children and periodontal bone loss, which is consistent with delayed mineralization in teeth observed in studies in animals. Current mean BLLs in these cohorts were <5 µg/dL (< 0.241 µmol/L).[6]

Studies of older populations with current mean BBLs <10 µg/dL (0.483 µmol/L) have reported associations between BLL and/or bone lead and poorer performance in neurobehavioral tests.[6]

One of the major concerns regarding lead toxicity is the cognitive and neurobehavioral deficits that are observed in children exposed to lead[6]:

- Prospective studies have provided the greatest amount of information. Analyses of these and other studies suggest that an IQ decline of 1–5 points is associated with an increase in BLL of 10 µg/dL (0.482 µmol/L);
- Of special interest and concern are the results of recent studies that have reported neurobehavioral deficits in children associated with BLLs <10 µg/dL (0.483 µmol/L) and an apparent lack of threshold down to even the lowest BLLs recorded in these studies.

A preponderance of the evidence indicates that lead exposure is associated with decrements in cognitive function:[6]

- Meta-analyses conducted on cross-sectional studies or a combination of cross-sectional and prospective studies suggest that an IQ decline of 1–5 points is associated with an increase in BLL of 10 µg/dL (0.483 µmol/L) and, most importantly, no threshold for the effects of lead on IQ has been identified;
- These and other studies have shown that the slope of the lead effects on cognitive variables is steeper (the effect is greater) at lower than at higher BLLs (i.e. not a linear dose-response relationship);
- Collectively, the results of the pooled analysis, and of additional studies, provide suggestive evidence of lead effects on cognitive function in children at BLLs <10 µg/dL (0.483 µmol/L) and, possibly as low as 5 µg/dL (0.241 µmol/L).

Each IQ point raises worker's productivity by 1.76–2.38%, and that the economic benefit for each year's cohort of 3.8 million 2-year-old children ranges from $US110 to $US319 billion[6].

Using an environmentally attributable fraction model, it was estimated that the present value of economic losses in the United States attributable to lead exposure in amounts to $43.4 billion per year in each annual birth cohort. More recently, one study estimated that mild mental retardation and cardiovascular outcomes resulting from exposure to lead amounts to almost 1% of the global burden of disease, with the highest burden in developing regions.[6]

In a multi-center study of 780 children, chelation therapy lowered blood lead by a mean of 4.5 µg/dL (0.202 µmol/L) during the 6 months after initiation of treatment, but it did not improve scores on tests of cognition, behavior, or neuropsychological function in children with BLL below 45 µg/dL (2.172 µmol/L).[6]

- Re-analysis of these data showed that improvement in test scores was associated with greater falls in BLL only in the placebo group;
- A further evaluation of this cohort showed that chelation therapy lowered blood lead, but produced no benefits in cognitive, behavioral, or neuromotor end points.

The conclusion reached by the investigators in this series of studies was that chelation therapy is not indicated in children with moderate blood lead levels.

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Use of Laboratory Testing for Lead

Testing for acute toxicity / "poisoning"

Whole blood analysis is the world standard test for the assessment of excessive recent exposure to, and absorption of, toxic metals (i.e. "poisoning") and is the test used to assess blood lead levels (BLL).

Testing for exposure to and absorption of metals

Urine metal analysis assesses recent metal exposure, absorption and subsequent excretion of metals such as lead in the urine.

Whole blood analysis, as noted above, is the world standard test for the assessment of metal toxicity ("poisoning"). It also provides limited quantification of net retention / body burden of metals over the past 3 to 4 months (approximately the lifespan of the cellular components of blood). A further limitation is that quantifying the burden of metals in blood does not assess the burden of metals sequestered elsewhere in the body (e.g. the burden of lead carried by mineralizing tissues such as bone and teeth).

Fecal metal analysis is an excellent way to assess the degree of exposure and excretion of orally ingested metals (e.g. in diet, pharmaceuticals and supplements) and may be useful to some extent in monitoring biliary excretion of metals.

Hair analysis for metals assesses recent exposure, absorption and subsequent excretion via the hair root over the past 2 to 4 months. The limitation of hair analysis is that testing cannot control for possible external contamination of hair by metals and other elements that may be present in the environment.

Testing for chronic retention (body burden) of metals

Urine metal analysis, as noted above, assesses recent metal exposure, absorption and subsequent excretion of metals in the urine. When this analysis is performed subsequent to provocation by oral or intravenous chelating or metal-complexing agents, such as:

- Meso-2,3-dimercaptosuccinic acid (Succimer; DMSA),
- 2,3-Dimercapto-1-propanesulfonic acid (Dimaval; DMPS), or
- Ethylene diamine tetraacetic acid (Calcium Disodium Versonate; $Ca\text{-}Na_2\text{-}EDTA$),

it can provide an excellent qualitative assessment of net retention / body burden of toxic metals, including lead.

Whole blood testing, as noted above, does provide an assessment of recent exposure to, and absorption of, metals as well as a partial, and transient at best, estimate of net retention/body burden of metals over the past 3 to 4 months.

Assessment of the Body Burden (Chronic Retention) of Lead and Other Metals

Detailed protocols, including dosage calculations, for the mobilization and assessment of body burden of lead and other metals are available.[13,14]

Patient preparation prior to provocative testing

Because administration of provoking agents can potentiate a large efflux of metals to the kidneys, and because some metals may be nephrotoxic, it is prudent to assess renal function (glomerular filtration rate; GFR) prior to provocation. The creatinine clearance test is the gold standard for this assessment and utilizes both serum and accurately timed urinary levels of creatinine in the GFR calculation. The serum creatinine level and the commonly reported estimated GFR (eGFR) alone may not be adequate for the assessment of glomerular function in some patients.

Children are not just "little adults" and provoking agents should be dosed and used especially cautiously in the pediatric population. Oral DMSA and intravenous EDTA do have regulatory approval in the USA for the treatment of lead poisoning, but DMPS has no such approval. The use of provoking agents is at the discretion, and within the judgment, experience and expertise, of the administering clinician.

Some additional notes on preparation prior to provocation:

- All suspected sources of exposure to the metal(s) of interest should be avoided before testing urine for metals.
- Nonessential medications and/or dietary supplements should be discontinued for a minimum of 24 hours, and perhaps even up to 4 to 5 days prior to, and during, specimen collection.
- Women should not collect urine samples for this test while menstruating due to the possibility of metals contained within the cellular components of blood leading to spurious results.
- Patients should be well hydrated before beginning testing (e.g. adults should consume 1.5 to 2 L of water over the course of the day prior to provocation) and, if an oral agent (e.g. DMSA, DMPS) will be utilized, should fast for at least 8 hours (e.g. overnight) prior to administration and sample collection.
- Provoking agents should not be administered during pregnancy or lactation as they can cross the placenta and are excreted in breast milk.
- Only containers that are provided by the laboratory with the test kits should be utilized for urine collection and transportation as non-standard containers may be contaminated with metals and their use could result in inaccurate test results.

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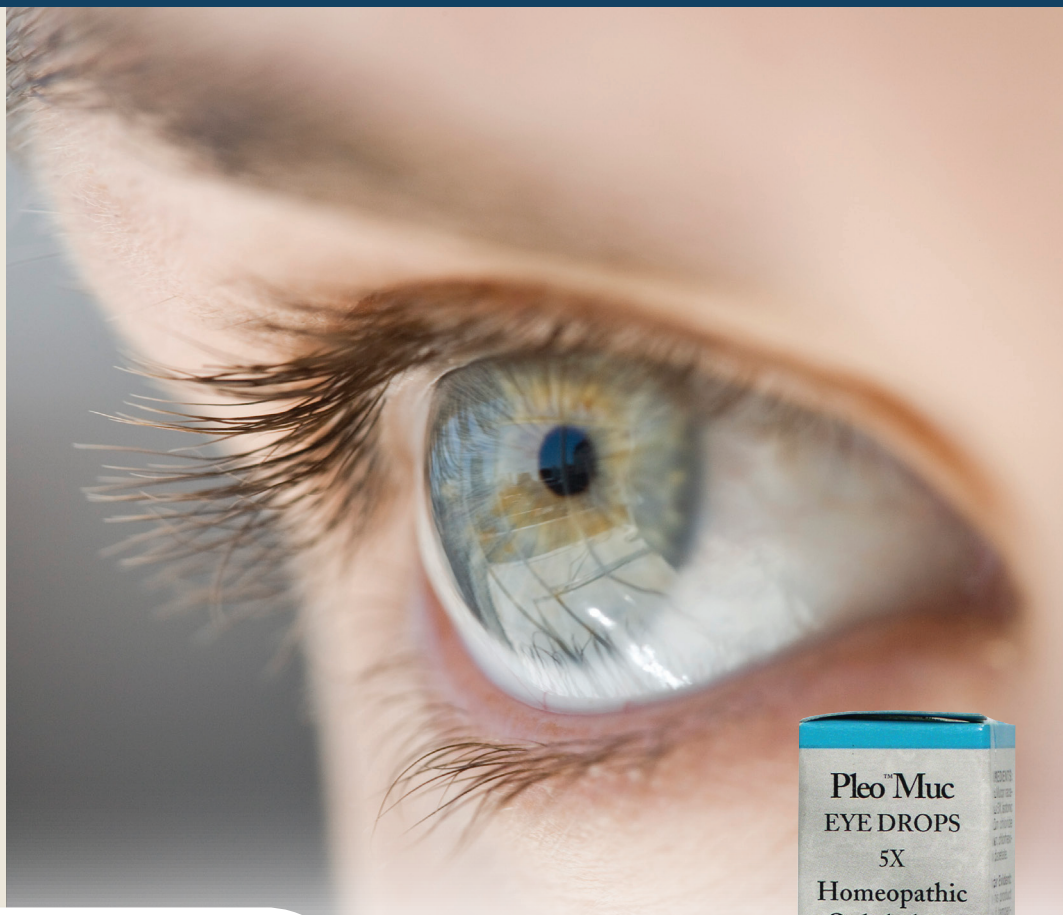

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Pre-provocative urine testing

Acute and/or ongoing metal exposure can cause transient increases in urinary metal excretion and thus lead to higher than expected urine metal test results. A pre-provocative urine test is important to evaluate whether or not acute or ongoing metal exposure has occurred.

Post-provocative urine testing

Provoking agents (e.g. DMSA, DMPS, $Ca\text{-}Na_2$-EDTA) are administered after first emptying the bladder. Subsequent urine production is collected for a specified period (usually 6 hours).

When interpreting post-provocation results, it is important to realize that some mild, post-provocation elevation of common metals (e.g., aluminum, arsenic, lead, and mercury) is to be expected, depending upon which provoking agent is used. Affinities of the specific provocative agents for the metals being tested, dosage of provocative agents and urine collection period must be taken into consideration when assessing the significance of the post-provocation results. There is wide variation amongst patients (genetic individuality, nutritional status, total body burden, exposure history, etc.) so individual tolerance to toxic metals also will vary. A patient's history and symptoms always should be considered when determining whether or not the levels of excreted elements might be possible causative or contributory factors. There are no scientifically validated reference range data available for post-provocation urine metal mobilization testing, and therefore it is left to the judgment of the practitioner whether or not any post-provocation result is clinically significant.

The importance of combined pre- and post-provocative testing

Acute and/or ongoing exposure to one or more metals is an important factor both in the analysis of pre-provocative ("pre") urine test results and in the assessment of post-provocative ("post") findings. For example, a normal "pre" and an elevated "post" level of metal(s) might support a diagnosis of chronic/net metal retention (body burden) while an elevated "pre" and an elevated "post" could suggest a mixture of both acute/on-going exposure as well as chronic metal retention (body burden). A high "post" without an accompanying "pre" makes it impossible to differentiate between chronic/net retention (body burden), acute/ongoing exposure or even some combination of the two.

Summary

Exposure to lead has a multitude of potentially adverse health consequences, especially during fetal development and early childhood. Public health agencies are beginning to recognize that "no lead is good lead" and are moving toward a lower threshold level for the diagnosis of lead toxicity, especially in children and governments are taking action to reduce exposure to lead. Objective testing is available to assess exposure, absorption, toxicity/poisoning and body burden/net retention of lead and many other potentially toxic metals.

About the Author

Dr Chuck Masur is a graduate of The University of Calgary Faculty of Medicine and received his MD there in 1979. Chuck completed his Family Medicine and subsequent Community Health Sciences residencies at The U of C in 1981 and 1995, respectively. Dr Masur practiced Family Medicine in rural and urban settings in Alberta for nearly 30 years before joining Doctor's Data, Inc (DDI) in 2008 in the role of Scientific Support Physician.

Besides his years of practice in Alberta, Dr Masur also held positions as Deputy Provincial Health Consultant at Alberta Health and as Assistant Medical Officer of Health for the City of Calgary. He has published works in several fields including laboratory medicine, public health, complementary and alternative medicine, pharmacology and therapeutics, and strategic planning.

Chuck has been very active in the international health and community development fields as well. He has managed over 40 community-based development projects in countries around the world and has served in volunteer capacities in Central and South America, the Middle East, South Asia and Russia.

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Eleutherococcus senticosus	Root	1:4	37.5 mg
Ganoderma lucidum	Fruiting body	1:3	50 mg
Ligustrum lucidum	Fruit	1:4	25 mg
Schisandra chinensis	Fruit	1:4	25 mg
Atractylodes macrocephala	Rhizome	1:4	25 mg
Glycyrrhiza glabra	Root and stolon	1:5	10 mg

Medicinal Ingredient	Plant Part	Amount Per Cap (mg)	Quantity Crude Equivalent (mg per ml)
Deep Immune Vegicaps (200 mg capsule, 5:1 extracts)			
Astragalus membranaceus	Root	40 mg	200 mg
Codonopsis pilosula	Root	30 mg	150 mg
Eleutherococcus senticosus	Root	30 mg	150 mg
Ganoderma lucidum	Fruiting body	30 mg	150 mg
Ligustrum lucidum	Fruit	20 mg	100 mg
Schisandra chinensis	Fruit	20 mg	100 mg
Atractylodes macrocephala	Rhizome	20 mg	100 mg
Glycyrrhiza glabra	Root and stolon	10 mg	50 mg

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Environmental Defence has conducted testing for lead and other heavy metals in make-up, and we found that some lip products tested had lead levels even higher than Canadian guidelines allow. For our report *Heavy Metal Hazard: The Health Risks of Hidden Heavy Metals in Face Makeup* (2011), Environmental Defence conducted testing on 49 makeup products from women's makeup bags, including lipstick, eyeshadow, blush and foundation. Heavy metals such as lead, beryllium, arsenic, nickel, thallium and selenium were found in all but one of the products we tested.

Lead enters makeup as a contaminant; it is not allowed as an intentional ingredient. But recent FDA (U.S. Food and Drug Administration) test results show a great disparity in lead levels in lipstick, demonstrating that it is technically possible to keep the amount of this toxin much lower than is currently permitted in Canada. And there is a difference between what is permitted, and what is safe.

Helping your patients protect themselves

In 2010, Environmental Defence launched the Just Beautiful campaign, to confront the pervasive problem of toxins in cosmetics. The campaign works to educate consumers on safer alternatives, to conduct product testing and research, to conduct outreach to the businesses that make healthy alternatives, and to encourage Health Canada to make improvements to Canadian Cosmetics Regulations, so that personal care products will be safer for Canadians.

On the Environmental Defence blog, Toxic Program Manager Maggie MacDonald has posted information about the *Heavy Metal Hazard* report, and a link to the results of the FDA study, both of which list the amount of lead found in each product, and the name of each product tested. Tests have revealed that price is not an indicator of how safe a product is. In fact, the FDA's tests found that one of the cheapest lipsticks was the least contaminated. Visit environmentaldefence.ca/blog to learn more, and share the article with your patients, so they can see if the products they use have high levels of lead and heavy metals.

Unlike lead, which is an impurity, we also recommend that you avoid the Toxic Ten ingredients that can be found listed on cosmetic labels. The list includes parabens and phthalates, hormone disrupting chemicals and suspected carcinogens. For a full list, visit justbeautiful.ca to download our Toxic Ten pocket shopping guide, and share the guide with your patients, to help them make safer choices.

At justbeautiful.ca your patients can find tools to protect themselves through learning about the toxins in cosmetics. But, more importantly, they can also help to protect their friends and families by joining the call for stricter controls on what ingredients are allowed in cosmetics in Canada, by signing and sharing our petition, and telling loved ones about the nasty stuff lurking in our makeup bags.

CONTEST:
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The Impact of Food Intolerances and Lead on Cognitive Function

Dr. Iva Lloyd, ND

For many children the factors contributing to an inability to focus or concentrate, and other cognitive concerns, are vast including food additives and colourings, food intolerances, environmental toxins, heavy meals, stress, lack of physical exercise and lack of time outside[2]. Many of these factors interact making it difficult to determine which ones are having the greatest impact. In reality, assessment and treatment involves identifying and addressing all factors.

This case reviews the assessment of a nine year old boy, 'Ryan' who was brought in for a naturopathic assessment to address concerns of focus, concentration and general challenges at school. Ryan was a "C" student who frequently got into trouble because he was easily distracted. He seldom got his school work done on time which resulted in a lot of homework that he struggled to complete. According to the testing done in Ryan's school he had ADD.

Ryan was very sociable and well-behaved. He came from a very supportive family and there didn't appear to any family stressors accounting for his behaviour or symptoms. He was polite, a good sleeper, got along with his brother and was otherwise quite healthy. His favorite activity was video games. The only other notable findings were encopresis (fecal incontinence) and peeling of the skin on his fingers, which he continuously picked. Both of these symptoms are commonly found in those with ADD/ADHD.[1] His encopresis occurred about 2 to 3 times a week and seemed unaware or unconcerned by these episodes, even when they were brought to his attention. There was no history of constipation, the episodes did not appear be connected to stress or external stressors and he was active on a daily basis. His skin (especially on his fingers) tended to peel and he would pick the skin when that happened. He was a pretty good eater, yet his diet did consist of a lot of pizza, Kraft dinner, perogies, cheese and crackers.

On physical exam it was noted that while his thumbs were both peeling at the tips, there was no erythema or inflammation; his lower lip was swollen and erythematous; there were no remarkable finding in the abdomen, lung or heart exam and other than the thumb his skin was unremarkable. Using a traditional Chinese medical assessment, his spleen/stomach pulse was rapid and his tongue was pink, moist, and narrow at the tip.

Food intolerances and environmental toxins are both known causes of ADD/ADHD.[2] During the first visit an IgG food panel was done and he was prescribed fish oil (EBI brand) at 1 capsule BID, probiotic (Multistrain by CytoMatrix) at 1 capsule QD and L-glutamine – 1000 mg BID. His mom was asked to keep a food diary and there was a discussion about ideal portions of the different food groups and the need to avoid food additives and colourings. In the 2nd visit, one month later, there were no significant changes noted at school, with the encopresis or with his skin. Ryan had been taking his supplements yet there had been no dietary changes. The IgG food panel revealed a high intolerance to all dairy, eggs and wheat; and a moderate intolerance to mushrooms, pecans, citrus and coffee.

In the 3rd visit two months later there had been some improvement (parents reported about 30%) in Ryan's ability to focus and concentrate and that he was bringing less homework home. The teacher remarked that he seemed more settled. There was no change in the oncopresis or with his skin. Due to the link between heavy metals, especially lead, and cognitive decline in children,[3] a heavy metal post-provocation test was done using 200mg of DMSA.

The provocation heavy metal test showed high lead (11 ug/g), high mercury (5.8 ug/g) and high normal cadmium (0.5 ug/g). Ryan was prescribed Biochelate (an EDTA chelator) at 10 gtt BID, Zinc-Copper (CytoMatrix) at 1 QD, OsteoSAP (NFH) at I BID, Kidney Support (Integra) at 1 BID, Chlorophyll at 1 dropper QD and DMSA (100 mg) – two once a day for three consecutive days every other week. He remained on his fish oil, probiotic and L-glutamine.

The next visit was scheduled for four months later. During this visit his parents reported that Ryan was doing much better on all fronts. His school work had dramatically improved and he was staying on the treatment plan. A subsequent post-provocation heavy metal challenge test, plus essential elements test was performed (see chart). It revealed even higher lead (19 ug/g) and mercury levels (8 ug/g); as well as, imbalances in a number of nutrients. The protocol was continued. The OsteoSAP was changed to BID and the Kidney Support was changed to TID. Five months later the provocation test was repeated. The lead level had dropped to 3.4 ug/g and the mercury to 1.2 ug/g. Ryan was still doing much better at school and hadn't had any bowel incontinence in over four months. His hands had also improved and even with the periodic "cheating" with food there were no flare-ups in the hands at all.

The decision was to keep Ryan on his nutrient supplements,

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yet stop the chelators (Biochelate and DMSA) for one year and then to retest the heavy metal levels. One year later there were no complaints about Ryan's health. He was continuing to excel at school and he was still following the dietary recommendations 80% of the time. A pre- and post-provocation heavy metal test was performed with essential elements. What was found was that the lead level had increased to 9.1 ug/g and the mercury to 4.2 ug/g. The chelators have been resumed and his parents have been strongly encouraged to have their water tested for signs of heavy metals. They have been provided with a handout on sources of mercury and lead to determine whether or not there is ongoing exposure contributing to the levels going back up after one year of detoxification.

As a practitioner it was a pleasure to see the parent's dedication to their son's health long after the initial concerns had been addressed. They truly understood the concept of preventative medicine and the long-term risks associated with heavy metal exposure. They are committed to yearly tests to ensure that the heavy metal levels do not increase and to address any nutrient deficiencies that may be indicated.

Ryan showed tremendous improvement both physically and cognitively. There were no changes from a family or school point-of-view. The changes were due to addressing food intolerances and heavy metal burden. There was just over two months between addressing food intolerances and starting on the chelation of heavy metals. This was sufficient time to see a noticeable difference between the two approaches. Although the removal of food intolerances improved focus and concentration, it wasn't until the heavy metal chelation was introduced that Ryan started to excel at school, his grades improved, his confidence improved and he found school easier. The last provocative test still show that the lead levels are higher than recommended which means that our work is not completed, but it has been a great start.

Urine Toxic Metals

POTENTIALLY TOXIC METALS

METALS	RESULT µg/g creat	REFERENCE RANGE	WITHIN REFERENCE RANGE / ELEVATED / VERY ELEVATED
Aluminum	21	< 60	
Antimony	0.5	< 0.5	
Arsenic	46	< 117	
Barium	3.6	< 7	
Beryllium	< dl	< 0.6	
Bismuth	0.2	< 20	
Cadmium	< dl	< 0.5	
Cesium	11	< 12	
Gadolinium	< dl	< 0.4	
Lead	19	< 5	
Mercury	8	< 5	
Nickel	4.2	< 15	
Palladium	< dl	< 0.3	
Platinum	< dl	< 1	
Tellurium	< dl	< 0.3	
Thallium	0.3	< 0.8	
Thorium	< dl	< 0.05	
Tin	5.3	< 15	
Titanium	N/A	< 15	
Tungsten	0.2	< 0.6	
Uranium	< dl	< 0.04	

URINE CREATININE

	RESULT mg/dL	REFERENCE RANGE	2SD LOW 1SD LOW MEAN 1SD HIGH 2SD HIGH
Creatinine	12.1	25- 180	

SPECIMEN DATA

Comments:
Date Collected:
Date Received: 10/19/2010
Date Completed: 10/20/2010
Method: ICP-MS
pH upon receipt: Acceptable
<dl: less than detection limit
Provoking Agent: DMSA DMPS
Collection Period: Random
Volume:
Provocation:

Urine Essential Elements

ESSENTIAL ELEMENTS

ELEMENTS	RESULT mEq/mg creat	REFERENCE RANGE	PERCENTILE 2.5th 16th 50th 84th 97.5th
Sodium	170	43.5- 348	
Potassium	110	26- 180	
	µg/mg creat		
Phosphorus	310	350- 1700	
Calcium	41	30- 250	
Magnesium	74	20- 300	
Zinc	0.66	0.15- 2.5	
Copper	0.16	0.012- 0.12	
Sulfur	930	308- 1980	
Manganese	0.004	0.0005- 0.02	
Molybdenum	0.044	0.02- 0.25	
Boron	1.1	0.8- 8.4	
Chromium	0.006	0.0005- 0.01	
Lithium	0.023	0.01- 0.25	
Selenium	0.074	0.04- 0.35	
Strontium	0.088	0.06- 0.48	
Vanadium	0.001	0.0002- 0.004	
			68th 95th
Cobalt	< dl	< 0.007	
Iron	< dl	< 2	

URINE CREATININE

	RESULT mg/dL	REFERENCE RANGE	2SD LOW 1SD LOW MEAN 1SD HIGH 2SD HIGH
Creatinine	12.1	25- 180	

SPECIMEN DATA

Comments:
Date Collected:
Date Received: 10/19/2010
Date Completed: 10/20/2010
Method: ISE; Na, K Spectrophotometry; P ICP-MS; B, Ca, Cr, Co, Cu, Fe, Mg, Mn, Mo, Se, Sr, S, V, Zn Creatinine by Jaffe Method
pH Upon Receipt: Acceptable
<dl: less than detection limit
Provoking Agent: DMSA DMPS
Collection Period: Random
Volume:
Provocation:

About the Author

Dr. Iva Lloyd BScH, RPE, ND is the founder of Naturopathic Foundations Health Clinic a multi-disciplinary clinic in Markham, Ontario that focuses on the naturopathic and energetic aspects of assessment and treatment. She teaches periodically at the Canadian College of Naturopathic Medicine and she is past-Chair of the Canadian Association of Naturopathic Doctors (CAND).

Dr. Lloyd is the editor in chief of the *Vital Link*, and sits on various other editorial boards. She has written many articles on health related topics for *Energy Currents, International Energy*, for the *Healthy Living* magazine and for *Naturopathic Doctor News and Review*, as well as other journals. She has been featured in *Chateleine, Glow* and other magazines.

She is the author of four books, *Building a Successful Naturopathic Practice, Messages From The Body – a guide to the energetics of health, The Energetics of Health, a naturopathic assessment* and *The History of Naturopathic Medicine, a Canadian perspective*.

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The Pediatric Lead Labyrinth: a 2012 ND Update

Dr. Marianne Trevorrow, ND, MA

In clinical naturopathic practice, there are few subjects as complicated as discussing lead or other metal toxicity in young children. This can be particularly difficult when these patients present with developmental or cognitive problems such as ADHD, autism, learning, sensory or mood disorders. Polarized views on the topics of an accepted or 'safe' level of lead in the body as well as screening for and removal of lead seem to be everywhere. It is definitely a subject that divides CAM providers (including ourselves) with the conventional MD community.

More than once, when discussing patient history, current diet, supplements and or medications during a first office call, I've had parents ask me directly if their child's autism, or ADHD, or anxiety is caused by heavy metals such as lead. And while the simple answer could often be "yes, quite possibly", the causal relationship between lead toxicity and symptomatic presentation is less direct. While testing gives us information to guide our clinical thinking, there is also the thorny issue of 'what kind of testing' and 'is this information that we want to deal with at this point in the case?' There is also a larger issue, in that our discussion of lead as a toxic body contaminant can lead to discussions of numerous other toxicants, and sometimes even a sense that we are fighting a losing battle against our own environment.

In the developed world, the primary ways that children can be exposed to lead are through lead-based paints and ceramics, lead particles in dust or soil, and aerosol exposures such as tobacco smoke or industrial pollution.[1] To a large degree, these sources have been lowered or eliminated in North America and Europe since the late 1970s due to governmental regulation and as a result, blood levels in the general pediatric population have been declining steadily since then.[2] Still, lead remains one of the most significant environmental toxicants affecting many children living in urban environments in both the developed and developing worlds, particularly children living in conditions of low socioeconomic status (SES).[1,3] In the developing world, the continued presence of lead in paints, ceramic glazes, and plumbing, as well as lead from industrial pollution and leaded gasoline put children at additional risk.[1] Finally, poor nutrient intake, particularly of the essential minerals iron, calcium, and zinc, also increases susceptibility to the toxic effects of lead.[4]

At the same time, as evidence continues to accumulate linking lead exposure to long lasting neurotoxic effects in children, the Center for Disease Control (CDC) and World Health Organization (WHO) have lowered threshold levels for lead toxicity in blood.[5,6] The most recent threshold levels for elevated blood lead were set in the 1990s at 10µg/dl (0.483 µmol/L). Since that point, however, several studies have been published showing that intellectual impairments, attention deficits, and behaviour problems can occur in children with blood lead levels considerably less than 10µg/dl[7-9] and at levels as low as 5µg/dl or less.[10] In the words of one prominent American childhood environmental health researcher "there is, at present, no detectable threshold for the adverse effects of lead exposure on cognitive development or academic abilities".[7]

Compared to adults, there are several factors that make infants and children more susceptible to the neurotoxic effects of lead. One is their increased intake of food and liquids in proportion to their smaller body weight. Another appears to be the timing of pre- and post-natal lead exposures. Several studies have shown that maternal and fetal blood lead levels are equivalent, indicating that there is no tissue barrier between lead circulating in the blood of a pregnant women and her developing fetus.[2,11] In addition, GI absorption of lead may be enhanced during pregnancy (along with iron and calcium), and lead may be freed from maternal bone stores in response to increased requirements for calcium during fetal bone formation. Lead can also be passed into breast milk.[12] All of this is of concern in children born to women with ongoing exposures and high body burdens of lead from such sources as construction, manufacturing, painting, or medical radiography. Other causes of increased exposure include long term smoking or illegal drug use before and during pregnancy as well as poor maternal intake of calcium, iron and zinc.[4,13] In the case of adopted children, pre-natal history itself may be unavailable or unreliable in determining whether exposures have occurred or to what extent.

Lead appears to impact important periods of brain and nervous system development in the 3rd trimester of pregnancy and early infancy, according to several experimental studies.[11,14,15] As outlined in Table 1, lead exerts its toxic effects on the developing

nervous system through its ability to substitute for calcium and zinc in metabolic processes, including its ability to cross the blood brain barrier.[16] By inducing mitochondrial release of calcium, for example, lead will not only damage the mitochondria themselves (inhibiting synaptic transmission), but may also directly initiate apoptosis in brain cells.[17] Other important effects involve the disruption of heme biosynthesis, abnormal myelin formation and indirect neurotoxic effects via the generation of reactive oxidative species (ROS) and resultant oxidative stress. Through lipid peroxidation, lead also causes an inhibition of several neurotransmitters, including acetylcholine and dopamine.[18] A 2003 review by environmental neuroscientists Lidsky and Schneider cited considerable experimental and animal studies to propose that lead acted as a 'neurodevelopmental toxicant' by interfering with both hard wiring and differentiation of cells in the developing CNS, as well as directly interfering with neurotransmission.[18] The authors argue that the effects involving glutaminergic and dopaminergic transmission, in particular, form the basis for observed impairments in intelligence, attention, and memory in children.

Table 1 – Mechanisms for lead toxicity in the Central Nervous System (CNS)

- Competition with and substitution for calcium
- Disruption of calcium homeostasis
- Stimulation of release of calcium from mitochondria
- Direct damage to mitochondria/mitochondrial membranes
- Inhibition of anti-oxidant enzymes (i.e. superoxide dismutase)
- Alteration of lipid metabolism
- Substitution for zinc/iron in metabolic processes including heme formation
- Accumulation in brain by astrocytes
- Sequestration and mobilization of lead from bone stores
- Long half life in the brain (2 years)

Modified from Lidksy and Schneider[18]

As a naturopathic physician in clinical practice, however, trying to understand how and where to apply these experimental and population studies can be challenging. For example, while numerous studies have suggested links between lead and ADHD and/or behaviour problems,[19,20] there is no consistent lead 'signature' or neuropsychological syndrome equivalent to better-defined conditions such as fetal alcohol syndrome or pre-natal cocaine exposure .[21] Clinically, my personal experience is to suspect lead problems in my hyperactive, anxious or emotionally 'brittle' kids, particularly when coupled with environmental exposures. That being said, I frequently also find that elevated lead levels rarely show up without elevated levels of one or more of the other heavy metal toxicants such as cadmium, aluminum, arsenic, or mercury. Additionally, further urine testing may also find neurotoxic solvents derived from pesticides, household cleaners, or industrial pollutants, suggesting that the patient may have problems with metabolism and excretion as much as with the exposures per se.

When reviewing test results, another factor to consider is whether heavy metal exposures may have caused clinically significant effects. Not all children with high body burdens of lead, after all, go on to have ADHD, autism, mood disorders or lowered intelligence. While careful clinical history can often elicit the possibility of exposure to lead (or other heavy metals), it can be difficult to predict which children may have the impaired cellular or GI/hepatic detoxification pathways associated with elevated body levels of metals, placing them at increased risk for developmental or behavioural disorders.[22] Most often, by the time a child clinically presents with symptoms of developmental or intellectual delay, a significant amount of exposure to lead (or other metals) has already occurred.

In the case of autism spectrum or related disorders, elevated lead is often found in conjunction with significant gastrointestinal problems (including inflammation, malabsorption, and chronic motility issues), leaving the naturopathic doctor in a difficult situation of trying to figure out how to prioritize treatments in an environment where parents are exhausted, discouraged, and often emotionally and financially stressed. Additionally, while the standard oral chelating agent dimercapto-succinic acid (DMSA) is considered to be safe in children,[23] these GI tract disturbances also increase the likelihood of significant undesirable and adverse behavioural effects, including increased hyperactivity, insomnia, tantrums or stimming*, which may cause parents to discontinue therapy.[24]

*** Stimming:** shorthand for self-stimulatory behaviours commonly seen in children with autism spectrums disorders. Common stims include hand flapping or repeatedly spinning objects. These behaviours are also found in many children with hyperactive-type ADHD.

For ND clinicians, there is also the issue of how to properly discuss testing for lead in blood versus urine and hair samples with parents. I would suspect that most NDs (myself among them) have had the sinking experience of sitting parents down to discuss a positive urine or hair test for metals only to be met with the response of 'our doctor (MD) told us that the blood test was fine and there's no science behind urine/hair tests.' The frustration I often have on hearing this lies in the fact there is still no generally accepted biomarker for lead accumulation within the environmental health community, and that there is considerable controversy over whether blood lead (the marker most often used in scientific papers) gives an accurate picture of lead accumulation in tissues. A recent review of lead biomarker testing in the National Institute of Health (NIH) publication *Revue of Environmental Health*, reinforced this point while arguing that the selection of appropriate biomarker testing was 'critically important for management of patients with potential lead toxicity'.[25]

Blood lead, as stated above, is still the standard biomarker for screening and diagnostic purposes, as it is believed to correspond well to soft tissue lead levels, including mobilized bone lead. [26] Although blood lead primarily represents recent exposure, it may also reflect past exposures in many children, as bone stores are continually mobilized into the bloodstream during periods of significant growth.[26, 27] Urine lead originates from plasma lead that has been filtered though the kidneys. When amounts are adjusted to account for kidney glomerular filtration rates (measured using creatinine clearance ratios), it has shown good correspondence with blood levels, although larger studies confirming this have yet to be carried out.[26] The addition of provoking agents such as DMSA or IV Calcium disodium edetate (EDTA) promote the mobilization and excretion of lead from blood and soft tissues, giving a good indication of longer-term body burden of lead, particularly when compared with pre-provocation testing.[28]

Hair is an attractive option for lead testing in very young children, or children who may be resistant to venipuncture in that it is non-invasive, and can also assess recent lead exposures.[25,26] The main problem with hair, however, is that it is very difficult to control for possible external sources of contamination such as environmental pollutants. That being said, a recent Indian/German study found that hair and DMSA challenge testing were supportive of each other in finding long term exposure to lead and other metals.[29]

Whichever biomarkers are used to establish the presence of lead in pediatric patients, there are a number of intervention strategies that can help infants and children lessen or eliminate its toxic effects in the body. First and foremost is primary prevention by removal of environmental sources of lead, including remediating exposure to lead sources in house dust, water, soil, ceramics and consumer products. With significantly elevated blood lead levels (over 45µg/dl or 2.15µmol/L) or levels between 25µg/dl and 45µg/dl where remediation has not been successful, the CDC and American Association of Pediatrics (AAP) call for the use of IV edetate calcium disodium edetate (CaNa2EDTA) and/or oral DMSA to increase urinary excretion of lead, in addition to more aggressive environmental interventions including dust and /or soil abatement, residential paint remediation, and the use of HEPA indoor filtration.[30]

Investigational trials of oral DMSA chelation in children with blood lead levels below 45µg/dl, however, have been equivocal. Since 2000, two large trials in general pediatric populations showed that while blood lead levels decreased after several months of chelation, scores on neuropsychological tests of behaviour and cognition showed either little or no improvement.[31,32] On the other hand, a more recent (2009) clinical trial of oral DMSA in children with autism spectrum disorders and heavy metal toxicity showed that treatment improved autism symptoms, (including cognitive function), with no significant adverse effects on liver enzymes, WBCs, or GI symptoms.[23,33]

Like many NDs who work extensively with children with environmental health problems, I have several documented cases of dramatic improvements with oral DMSA chelation for children with lead toxicity and autism, ADHD, cognitive problems, or related conditions. There is also now some clinical evidence from DMSA/autism trials to suggest possible cognitive benefits of oral DMSA chelation. Still, as responsible clinicians, we need to address parental expectations around chelation. We need to be clear that on balance, the evidence is mixed for lead chelation with oral DMSA when initial blood levels are lower than 45µg/dl (which will be most of the kids we see) if the goal/purpose is to improve cognitive and behavioural symptoms. It is also not a quick process; significant improvements in cognitive function and behaviours can take six months or more to achieve as lead is released from bone stores into soft tissues where it can be accessed by DMSA.

Despite these potential benefits, oral chelation is also not without risk. In young children, a major concern with DMSA therapy has been maintaining adequate levels of essential minerals such as calcium, magnesium, zinc, and potassium. This is of particular importance to children under the age of five, who have proportionally increased demands for these minerals to facilitate growth and development, (including CNS development), and smaller body mass. To investigate this concern, the 2009 autism/DMSA study did look at urinary mineral levels during DMSA treatment rounds (10mg/kg three times a day for three days with an 11-day break between rounds). They found significant daily losses of chromium and potassium in subjects' urine, but that zinc, calcium and magnesium levels did not change.[23] Interestingly, these urinary losses of potassium did not result in any lowering of blood potassium levels, indicating that electrolyte balance was maintained. However, given the myriad functions of potassium in the body — including its critical role in cardiac function — concerns remain. At this point, testing mineral levels periodically is essential for DMSA chelation protocols in children, as is additional supplementation with calcium, iron, zinc and trace minerals, including chromium and potassium. Further studies should also help to clarify whether their finding that the most essential minerals were spared utilizing the 3day/11day regime is generally true for other pediatric populations. Most importantly, while the autism/DMSA trial did not find any adverse effects on hepatic or renal function in subjects undergoing oral chelation, it is unclear whether this can be generalized to children younger than their minimum age of three years.

Beyond primary prevention, remediation and chelation, there are also a considerable number of nutritional strategies that can assist in lowering the toxic effects of lead in young children. These may be particularly suitable for children under the age of three, or children who are poor candidates for DMSA therapy. Some nutrients, such as calcium, zinc, and iron inhibit GI absorption of lead. With regards to calcium, extensive literature has shown that diets low in calcium increase lead absorption and toxicity

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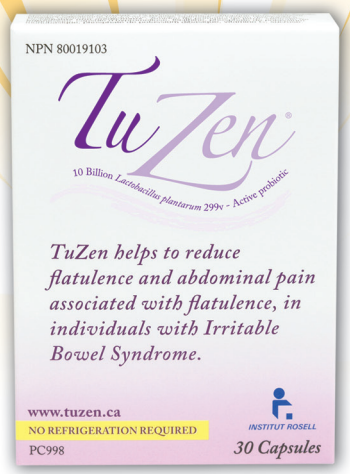

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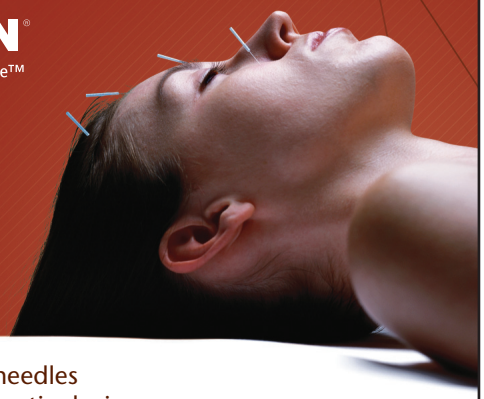

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and that conversely, high intake of dietary calcium decreases GI lead absorption.[34,35] Since a great deal of lead's toxicity in the body arises from its ability to substitute for calcium in cellular processes, determining calcium intake through diet and considering supplementation to dietary reference intake (DRI) guidelines of 500mg/day for 1-3 year-old and 800mg/day for 4-8 year-old children may be a logical first step for any child at risk of lead toxicity, particularly those on restricted diets.[36]

Similarly, iron and zinc are also believed to compete with the absorption of lead via a common iron-lead metal transporter called DMT1.[37] In conditions of iron deficiency, DMT1 is believed to be upregulated, increasing the absorption of lead as well as iron and zinc. Several studies have found that children with iron deficiency have higher blood lead levels and that iron repletion can decrease blood lead levels. [3,38,39] Fortunately, iron status is easily obtained through standard blood tests including CBC and ferritin levels (indicating iron stores) and supplements can be readily obtained (or compounded) to replete iron levels. In the case of zinc, we may still lack effective assessment tools for determining zinc status,[40] however, evaluation of intake and considering adequate supplementation in a multi-mineral supplement with copper would also be helpful in many at-risk children.

Antioxidant nutrients such as selenium and vitamins C, E and B6, work to scavenge free radicals and prevent lipid peroxidation caused by lead.[41] Vitamin C is also believed to act as a mild chelator through its ability to increase the urinary excretion of lead,[42] and decreases the toxic effects of lead on heme synthesis. [43] All of these nutrients are present in high quantities of fresh fruits and vegetables, and easily available through food-based natural health products such as greens or berry supplements.

Individual nutrient effects are summarized in the table 2 below:

Iron

- diets low in iron increase lead absorption
- lead can substitute for iron in heme biosynthesis
- lead inhibits delta-aminolevulinic acid dehydratase (δ-ALAD), enzyme in heme biosynthesis
- iron supplementation (if deficient) reduces lead GI absorption

Calcium

- diets low in calcium increase lead absorption
- lead is deposited in bone during calcium deficiency and can be remobilized during periods of growth, pregnancy, breastfeeding
- lead disrupts calcium balance and metabolism in tissues/CNS
- calcium supplementation reduces lead GI absorption

Zinc

- diets low in zinc increase lead absorption
- lead can substitute for zinc in several zinc-mediated metabolic processes.
- zinc supplementation prevents lead-induced inhibition of δ-ALAD enzyme.
- co-factor of anti-oxdidant enzyme SOD

Vitamin C

- reduces body retention of lead
- increases urinary excretion of lead
- reduces lead induced lipid peroxidation

Selenium

- may beneficially influence absorption, tissue distribution and metabolism of lead
- enhances availability of glutathione
- neutralizes lead-induced ROS
- partially reduces nephotoxicity/neurotoxicity of lead

Vitamin E/B6/β-carotene

- prevents lipid peroxidation caused by lead.
- supplementation increases activity of anti-oxidant enzymes
- enhances biosynthesis of glutathione in response to presence of lead.

Adapted from Ahamed M, Siddiqui MKJ[43]

Figure 2 – Nutrients known to affect lead toxicity/absorption in the body

To review, the selection of dietary and nutrient based therapies should be based on the individuality of the patient's case and take into account such factors such as age, presenting symptoms, and delivery method. I would also argue that as much as possible, food-based strategies to increase nutrients should be considered first in young children, with supplementation used as an adjunct where either clear deficiency exists, or the child has self-selected a low nutrient dense diet and is resistant to strategies aimed at increasing the range of foods he or she is willing to eat.

Conclusion

Since the last century, public health strategies to lower children's environmental exposures to lead in the developed world have succeeded, to a large extent, in reducing the average body levels of this metal. Even within this larger picture, individual children may still have significant exposure histories. These can arise from sources as varied as paint chips or dust from older buildings, contaminated soils or industrial pollution, older plumbing fixtures or lead glazes on ceramics. Additionally, children exposed pre- or post-natally to lead from smoking, drug use, or occupational lead may be less commonly thought of as 'at-risk', but may still have significant lead levels upon testing. In the case of many children with autism spectrum disorders, exposures themselves may be minimal, but tissue lead may still accumulate due to genetic difficulties or inborn errors with cellular metabolism and GI excretion. It is possible that this may also be the case for children with ADHD and mood disorders. While these latter conditions have not been as well studied in terms of susceptibility to toxic effects, the data on autism is suggestive that it should be an area for future investigation. Hopefully, further research will help answer the question of whether many of these behavioural disorders of childhood could also benefit from screening and treatment of lead toxicity.

New information is also emerging to validate the use of less invasive screening methods, such as the provoked urine challenge and hair testing, methods that NDs have been using for years in children. While chelation therapy remains controversial in young children when lead levels are considered 'mildly elevated' by conventional standards, recent clinical trials of DMSA in an autistic population with elevated heavy metals provided good evidence that DMSA can impact behaviour and cognitive functioning in a positive way. These studies demonstrated that the three days on /11 days off oral DMSA chelation protocol that many NDs follow is actually quite safe, even for children as young as three years of age.

Still, many parents may not be willing to consider the additional costs or demands required by oral chelation therapy, especially when functional benefits may take months to appear. For these families, as well as for children who may have multiple health challenges, or those younger than three years at presentation, there is increasing evidence that nutrients such as iron, calcium, zinc and vitamin C, may help decrease many of the toxic effects of lead in the body, if not themselves promote the metabolism and excretion of lead in a more gradual way. Finally, a more global concern arises that in the coming century, lead may become increasingly a toxin of children of lower SES or of the developing world. For

this reasons, as global citizens ourselves — as well as naturopathic physicians — we need to become conscious of the ways in which our consumer choices may increase the presence of lead in anyone's backyard.

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About the Author

Dr. Marianne Trevorrow, ND, MA, holds degrees from McGill University, the University of Chicago, and Bastyr University, where she completed her ND in 2006. After a year of post-graduate work in Nutrition and Anti-Aging Medicine at Bastyr, she moved to Victoria, BC, where she has been private practice since 2007. She is Board certified in Chelation Therapy and licensed to prescribe. Alongside her primary care practice, her clinical focus is on environmental and developmental disorders in children, including ADHD, autism, Down's syndrome, asthma, allergies, and other chronic conditions of childhood. She is affiliated with Autism Canada and the Autism Research Institute, and is a frequent author, panellist and speaker on women's health and pediatric environmental health and autism. She is also a director of the BCNA, a Board Examiner for NPLEX, and a peer reviewer for several CAM biomedical journals. Her email is drmarianne@inspired-health.ca

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Lead in the Elderly: normal aging or premature effects of a heavy metal?

Dr. Scott Clack, BSc, ND

Lead toxicity is considered by many people to be a problem of the past. Canadians believe three of the most common sources of lead intoxication from the environment have been removed: lead-based paints, lead solder in food cans, and leaded gas. What Canadians might not know is that lead is still lurking where it can impact health. Young children and the elderly are the most vulnerable. This paper will inform the reader about the potential for health problems from lead intoxication in Canadian seniors.

How do we know that our patients might have lead toxicity? It is known that seniors, aged 60 to 79 years of age, have the highest BLLs in the population with the exception of children under five.[1] If they lived, or have lived most of their lives in urban settings, their risk in increased. Symptoms of lead toxicity (from bioaccumulation) in seniors are not well documented unless the BLL test shows a detectable level of lead. Symptoms of lead poisoning include: hypertension; decline in mental function; memory loss; mood disorder; nausea and vomiting; muscular weakness or loss of coordination; pain, numbness or paresthesia; inability to concentrate; and impotence.[2] Lead toxicity should also be considered in any elderly patient with hypertension, diabetes, obesity, kidney disease, and neurodegenerative disorders (e.g., Parkinson's disease).[3]

Neurodegeneration in aging populations is a growing burden to health care systems. Studies show chronic exposure to lead might upset brain metabolism of N-Acetyl-aspartate (NAA) and choline (Cho) in basal ganglia, occipital and frontal lobe.[4] NAA and Cho are negatively correlated with blood and bone lead, leading to neuronal and axon damage or loss.[4] The impact on frontal and occipital lobes matches memory and visual attributes that decline in the elderly. A second study using MRI and magnetic resonance spectroscopy (MRS) showed diminished hippocampal volume in lead-exposed workers that could be evident in short-term memory failure.[5] Finally a 2009 study in Circulation concluded that bone lead is associated with a Hazard Ratio (HR) of 2.52 for all-cause mortality and 5.63 for cardiovascular mortality.[6]

Knowledge of early signs and symptoms are equally important for NDs in family practice to identify the possible effects of low levels of lead toxicity. Common clinical signs include diffuse muscle weakness; general fatigue; myalgia; arthritis/joint pain; loss of appetite; unusual taste in the mouth; headaches; insomnia; irritability and/or personality changes; diminished libido; tremulousness; and unexplained weight loss.

The process of diagnosing lead toxicity involves a complex mix of symptomology and diagnostic testing. Initial steps begin with a very detailed health and personal history. Care must be taken to query patients about their residence history, occupations, hobbies, and lifestyle. For example, if you fail to learn that they grew up near Trail, B.C. or Belledune, N.B., you will not factor in that they were exposed to lead from local smelting operations. Other facts to gather include:

- Smoking history: length of smoking, amount; consider second-hand exposure
- Occupational: working in/at lead smelters, or battery manufacturing; plumbing or trades work involving lead solder; printing/thermographic printing; car mechanics (working with brake linings and/or wheel balancing); electronic or optical manufacturing; explosives use or manufacturing (e.g., mining, ammunition manufacturing, or military/police workers); gardening; woodworking or tradeswork using epoxy or varnish; manufacturing with flame retardants (e.g., infants' clothing); ceramics or glass-blowing; exterminators (pesticides, rodenticides); painters; diesel truck drivers
- Hobbies/interests: gardening; automobiles; woodworking; stained glass; jewellery; pottery; fishing (use of lead weights); militia enthusiasts who have lead-based soldier displays; firearm enthusiasts.
- Dietary intake: grain based foods are considered to be the greatest source of lead; this includes ingestion of cereals, breads, pastas, crackers. Another factor is the purchase of exotic or foreign foods (versus locally grown) from countries who have not adopted more stringent standards about environmental lead or manufacture of food tins. Foreign spices and herbs are considered to be key food sources of lead. This also applies to herbal and patent medicines.

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- Lifestyle – living in homes with pre-1978 paint; renovations of pre-1978 homes; use of glazed pottery for cooking or food storage; use of leaded glass containers (glasses, carafes); reduced exercise or periods of extended convalescence contributing to osteoporosis and release of lead from bone; alcohol consumption.
- Health status: patients with postural sway, neuromotor impairment; sensory nerve impairment; or neurologic symptoms; hypertension; chronic kidney disease; osteoporosis or osteopenia; cataracts; cognitive decline or degenerative dementia.

Blood lead level (BLL) has been the primary biomarker for surveillance of lead toxicity from employment exposure and is more consistent with recent environmental contamination.[7] A 2007 paper prepared by an expert panel of the Association of Occupational and Environmental Clinics suggested that people with BLL of 10-19 µg/dL should reduce their exposure.[10] People with BLL 20-39 µg/dL should be removed from their source of lead exposure, while those with levels above 50 µg/dL should be considered for chelation therapy depending on their signs and symptoms in addition to removal from the source.[10] Higher BLLs are also associated with psychiatric symptoms (e.g., depression, anger).[8] If a senior lives in older housing with lead inputs from past lead-based paint and lead in plumbing, this exposure could be detected by BLL.[10]

Bone lead is measured using K-shell x-ray fluorescence (KXRF) of the tibia or patella.[8,13] This measurement reflects cumulative lead levels, and has greater association with cognitive function.[9] This can apply to current occupational or past exposure to lead. Cumulative lead levels reach 95% in adults.[10] "A combination of decreased "external" lead and normal rates of bone resorption can result in bone constituting the predominant source of circulating lead in elderly individuals or in individuals with a history of long-duration high exposure and low current exposure".[11]

Tests used by naturopathic doctors to assess for lead burden might include one or a combination of the following: hair analysis, provocative challenge testing, and/or RBC toxic elements analysis. Hair analysis is the most cost-effective test for Canadian patients, and recent studies show its usefulness.[12] Schamberger indicated in his study that when sample washing was performed properly by a lab, variance between tests was reduced.[14] He further stated that hair analysis has been helpful in forensic investigations on the composer Ludwig von Beethoven and several ex-US presidents. He concluded that several elements when found in hair including lead, did show "relationships between body burden, dosage, and exposure or toxicity". Afridi et al showed that scalp hair samples of smoking compared to non-smoking diabetic patients had higher levels of lead, cadmium and arsenic, concluding that toxic metals may contribute to the development of diabetes.[15]

Provocative challenge tests are used to show that a body burden of heavy metals does exist.[16] To conduct a provocation test, a baseline

urine sample should be collected: 4 or 24 hour urine collections are reported in the literature.[16] The patient then takes a dose of dimercaptosuccinic acid (DMSA) and collects urine samples for 4 or 24 hours.[16] In the Hoet study, urine and blood lead levels increased steeply after a 1 g dose of DMSA.[16] Hoet concluded that DMSA offers diagnostic value when blood lead is greater than 30 µg/dL. A 2009 study of 149 males (87 being lead exposed workers) used 10 mg/kg of DMSA in pre/post provocative challenge tests and compared the lead levels to blood samples. The study concluded, "the lead mobilization test is a more reliable diagnostic test than BLL for assessment of toxicologically active fraction of total lead body burden and imminent health risks in occupational workers".[17]

Once heavy metal toxicity is confirmed through patient history and lab testing, effective treatment is the next critical step to helping the patient feel better and alleviate their symptoms. It is important that treatment have two phases: preventing new accumulation of the metals, while removing the burden of metals that have become stored. Preventing new accumulation would include safety precautions during work or hobbies where exposure exists, or ceasing the activity altogether (not always an option in an individual's economic circumstances). Water filtration and air purification at home (and work, if possible) are important choices, as well as simple steps such as not wearing shoes inside the home that have also been worn outside.

Chelation therapy provides the standard of care for toxicity/poisoning caused by lead or other heavy metals at levels greater than 40 µg/dL. Treatment protocols can be contentious: intravenous versus oral; risk of redistributing the metals to other parts of the body where they can do more harm; deciding when to discontinue treatment. There is also the question of using chelation for subclinical toxic lead burden. Finally there is the added complication that formal training in chelation has been introduced in the naturopathic curriculum in recent years, and is often not in the scope of practice for NDs in many jurisdictions in North America. Potential for side-effects and harm to the patient requires any practitioner to possess proficient knowledge of these medicines before recommending them, and should consider referring their patients to qualified practitioners. The risks of chelation in the elderly include: [19, 20]

1. mineral depletion and potential health problems related to mineral deficiencies (e.g., skin lesions may develop after EDTA depletes zinc)
2. nephrotoxicity in a dose-related manner when using EDTA
3. transient increase in hepatic transaminase activity when using DMSA
4. severe mucocutaneous reaction – DMSA
5. redistribution of lead from bone into soft tissues such as the brain, kidney and liver, potentially causing more harm to those organs (see discussion below on combining EDTA and DMSA in therapy).

The optimal approach for lead toxicity appears to be a combination therapy using Disodium ethylenediaminetetraacetate (CaNa2EDTA) and DMSA. CaNa2EDTA has long been the preferred treatment for plumbism since the 1950s.[22] EDTA chelation causes significant diuresis of zinc and other minerals in addition to symptomatic side-effects, requiring monitoring and replenishment of minerals.[18,19] The advantage of EDTA is its ability to remove lead from trabecular bone which is the main repository of any lead burden.[19] 2,3 dimercaprol (BAL) is a second chelator that has been favoured for treatment of lead and/or arsenic poisoning.[21] In this study, the authors stated that DMSA is safe and effective for lead toxicity but has an inability to remove it from intracellular sites because of its lipophobic nature.[21] DMSA has been found to be effective in cases of acute lead toxicity.[22] When administered at a dose of 30 mg/kg body weight (BW) to 17 adults with BLLs of > or = 50 µg/dL over 35 courses, DMSA significantly reduced blood lead concentrations and increased urine lead excretion.[22] Results showed more than 50% of patients reported improvements in headaches, lethargy and constipation within the first two days of chelation. Compared to other chelators, DMSA spares most key minerals (zinc, copper, iron and calcium) compared to other agents and is known to have minimal adverse side effects. Crinnion concluded that combination protocols using CaNa2EDTA (EDTA) followed by DMSA "might be warranted".[19] EDTA removes lead from trabecular bone causing increased deposition in soft tissues. If the EDTA treatment is soon followed by DMSA treatment, an increased level of lead is removed from the soft tissue.[19]

Several natural medicines and treatments are recommended, both for treatment of lead burden and preventing new accumulation. The key is to optimize and/or support glutathione (GSH) levels. GSH concentrations in blood have been shown to be reduced in studies of lead-exposed workers and children.[24] Lead also binds to enzymes such as delta-aminolevulenic acid dehydrogenase (ALAD) and glutathione reductase, impairing their function and further exposing people to the negative effects of free-radicals.[24] Antioxidants as well as supplements that support methylation and trans-sulfation are effective adjuncts to chelation, sometimes reducing their side effects.[24] The top choices for antioxidant therapy include vitamin C, N-acetylcysteine, alpha lipoic acid, vitamin E, methionine, zinc, taurine and pyridoxine.[24] This report concludes that some of these supplements have limited chelating action, and that their synergism with pharmaceutical chelating medicines is the most important factor.[18]

Other natural supplements and treatments have been shown in limited research to be beneficial for patients with lead toxicity. Milk thistle (silymarin) is touted for its hepatoprotective properties:[25] it promotes protein synthesis, regenerates liver tissue, controls inflammation, increases glucuronidation, and blocks glutathione depletion. Garlic was compared to d-penicillamine (another chelator) in a study of 117 battery workers.[26] At a dose of 1200 µg allicin three times daily, the garlic supplemented group received similar benefits in symptom relief compared to d-penicillamine and it was concluded that "garlic can be recommended for the treatment of mild-to-moderate lead poisoning". Two studies show activity that increases and sustains sweating, such as exercise or infrared sauna therapy, can be effective modes of excreting many toxic elements.[27,30] Anecdotal knowledge of supplements like chlorella and cilantro may direct NDs to include them in their heavy metal detox protocols.

Two other adjunctive treatments evaluated in animal studies bear mention. Curcumin was shown in one study to have a major role in preventing lead-induced neurotoxicity, helping retain spatial reference memory.[28] Given the known pro-oxidant effect of heavy metals on nitric oxide, another study showed the benefit of L-Arginine plus DMSA for lead induced hypertension when compared to either treatment alone.[29]

In conclusion, lead toxicity is still present in our population in spite of efforts to reduce it. It is important that naturopathic doctors, as primary care practitioners, consider this aetiology in their differential diagnoses of all their patients, especially children and the elderly. NDs should familiarize themselves with the symptomology of lead toxicity and use available testing to rule it out. Many naturopathic medicines, focusing on antioxidant therapy, are useful for these cases. Preferred treatment for reducing lead burden includes chelation therapy, offered by trained NDs or through referral to qualified practitioners.

About the Author

Scott Clack graduated in 1997 from Bastyr University. In activities with the naturopathic profession, Dr. Clack was a Director of the BCNA from 1998 through 1999, as well as being appointed to the BDDT-N between 2002 and 2004. He was selected Naturopathic Doctor of the Year by the Ontario Association of Naturopathic Doctors in 2005. He currently practices from Touchstone Naturopathic Centre in Mississauga, ON with a focused interest in the treatment of neurodevelopmental and neurodegenerative disorders, as well as allergies.

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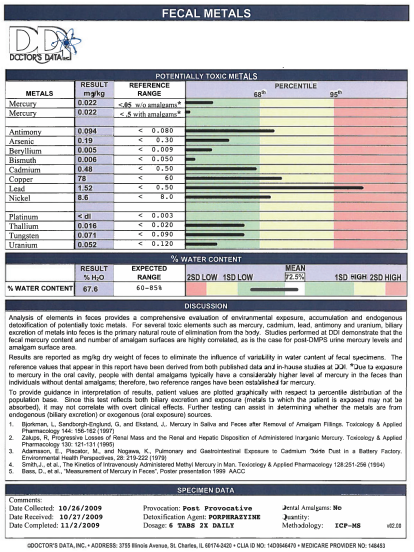

FECAL METALS

DDI DOCTOR'S DATA LLC

POTENTIALLY TOXIC METALS

METALS	RESULT mg/kg	REFERENCE RANGE	PERCENTILE 68th 95th
Mercury	0.022	<.05 w/o amalgams *	
Mercury	0.022	<.5 with amalgams *	
Antimony	0.094	< 0.080	
Arsenic	0.19	< 0.30	
Beryllium	0.005	< 0.009	
Bismuth	0.006	< 0.050	
Cadmium	0.48	< 0.50	
Copper	78	< 60	
Lead	1.52	< 0.50	
Nickel	8.6	< 8.0	
Platinum	< dl	< 0.003	
Thallium	0.016	< 0.020	
Tungsten	0.071	< 0.090	
Uranium	0.052	< 0.120	

% WATER CONTENT

	RESULT % H_2O	EXPECTED RANGE	2SD LOW	1SD LOW	MEAN 72.5%	1SD HIGH	2SD HIGH
% WATER CONTENT	67.6	60-85%					

DISCUSSION

Analysis of elements in feces provides a comprehensive evaluation of environmental exposure, accumulation and endogenous detoxification of potentially toxic metals. For several toxic elements such as mercury, cadmium, lead, antimony and uranium, biliary excretion of metals into feces is the primary natural route of elimination from the body. Studies performed at DDI demonstrate that the fecal mercury content and number of amalgam surfaces are highly correlated, as is the case for post-DMPS urine mercury levels and amalgam surface area.

Results are reported as mg/kg dry weight of feces to eliminate the influence of variability in water content of fecal specimens. The reference values that appear in this report have been derived from both published data and in-house studies at DDI. *Due to exposure to mercury in the oral cavity, people with dental amalgams typically have a considerably higher level of mercury in the feces than individuals without dental amalgams; therefore, two reference ranges have been established for mercury.

To provide guidance in interpretation of results, patient values are plotted graphically with respect to percentile distribution of the population base. Since this test reflects both biliary excretion and exposure (metals to which the patient is exposed may not be absorbed), it may not correlate with overt clinical effects. Further testing can assist in determining whether the metals are from endogenous (biliary excretion) or exogenous (oral exposure) sources.

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SPECIMEN DATA

Comments:
Date Collected: 10/26/2009
Date Received: 10/27/2009
Date Completed: 11/2/2009
Provocation: Post Provocative
Detoxification Agent: PORPHYRAZYME
Dosage: 6 TABS 2X DAILY
Dental Amalgams: No
Quantity:
Methodology: ICP-MS

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Lead Toxicity Causes and Effects

Dr. Lyn Patrick, ND

"It is impossible to live in a lead-free world. Lead is ubiquitous in the environment. It is in the air, water, and soil, in short it is unavoidable. However, compared to the amount of lead a person would ingest from eating and drinking ordinary foods, the amount expected from the use of cosmetics would be extremely small" - The Cosmetics Toiletries and Fragrance Association, the trade association for the manufacturers of personal care products that includes Maybelline and L'Oreal, whose lipsticks measured by the FDA contained lead above 7.0 ppm, 70 times the allowable limit in candy.[1]

The subject of lead toxicity is the most widely researched and extensively published example of disease causing environmental toxin exposure in the world. Despite our advanced understanding and remarkable progress in eliminating major environmental exposures (leaded gasoline, lead paint) in the developed world, there are still approximately 454,000 school-aged children in the United States that have blood lead levels over 10 µg/dL, placing them at significant risk for lifetime cognitive deficits as a result.[2] There is also strong evidence supporting the fact that lead body burden contributes to cardiovascular disease risk for as much as 35 percent of the adult population.[3] Recognizing the potential signs and symptoms of chronic lead exposure in patients and the conditions that may be related to its exposure and accumulation is a necessary skill in practicing the naturopathic principle of *tolle causam*.

Historical Examples of Lead Toxicity

Lead toxicity is not just a phenomenon of post-industrial society. In Rome, as early as 20-AD, historical accounts of gout, colic, and "dropsy" were reported in the general population, as were sterility, infertility, and stillbirth among the aristocracy (who had the highest lead intake) – all of which have been attributed to both acute and chronic lead toxicity.[4] Much like today, lead was everywhere in ancient Rome: in face powders, rouges, and mascaras; as a pigment in paint, a spermicide for informal birth control; a condiment popular for seasoning food; a wine preservative perfect for stopping fermentation or sweetening poorly produced wines; and was considered an irreplaceable metal in the production of pewter cups, plates, pitchers, pots and pans. As well, lead plumbing ("plumbing" originates from the Latin word for lead: plumbum) was a common technology for delivering water supplies in households.[5] Even though the Romans knew lead caused illnesses in the general population and early deaths in the slaves that mined the metal, they could not quell their insatiable need for lead, and as a result, it has been argued, the Roman Empire fell.[4]

By the twentieth century, the United States was the world's leading producer and consumer of refined lead, in 1980: consuming 1.3 million tons of lead per year and accounting for approximately 40% of the world's supply (10 times more, per person, than the exposure in ancient Rome).[5]

In 2003, the municipal water supply of Washington DC was contaminated due to leaching from the inside of lead delivery pipes. This leaching resulted from of the addition of chloramine as a disinfectant, which acted as a corrosive and allowed extensive amounts of lead particulate to leave the lining of the pipes and enter the water supply. In 2004, excessive levels of lead were detected in up to 68 percent of residential tap water samples, at levels as high as 48,000 ppb. The legal limit is 15 ppb for the highest 10 percent of homes sampled.[6] A later report published in 2009 in *Environmental Science and Technology*, found that 42,000 children who were *in utero* or under two years of age during the period of contamination were considered to be at risk for health and behavioral problems as a direct result of drinking lead-contaminated water.[7] In 2010, the Center for Disease Control reported that 15,000 homes in the Washington, D.C. area might still have water supplies with dangerous levels of lead.[8]

There had been no historical precedent in U.S. history for this kind of municipality-wide lead contamination event. As a result of the *Washington Post* initiating an investigation, Environmental Protection Agency (EPA) documents were revealed detailing widespread problems with inaccurate reporting of lead levels at water agencies across the United States. This eventually led to federal congressional investigations and changes in Environmental Protection Agency policies.[9]

Natural disasters like hurricanes and the demolition and reconstruction of buildings that follow these disasters also

contribute to lead exposure. The events following Hurricane Katrina in August of 2005, and the massive reconstruction and renovation effort resulted in significant lead contamination of house dust and soil. The majority of homes in New Orleans were built prior to 1950 and 83 percent contained documented lead hazards, primarily due to lead-based paint.[10] A post-Katrina investigation in 2007 found that soil lead levels had increased significantly since 2000 with more than 60 percent of households having at least one elevated lead sample either inside or outside of the home.[10] Nearly half the homes with bare soil in the yard had elevated soil lead, and 27 percent of those homes had soil lead exceeding 1,200 ppm, three times the Housing Urban Development/U.S. Environmental Protection Agency standard.

Although lead-based paint has been banned for household or commercial building use in the US since 1978, and in Canada, voluntarily since 1991, it remains a major contributor to household lead-containing dust. Particulate from lead-based paint still accounts for the majority of lead found in soil surrounding residences and soil lead is correlated with elevated blood lead levels in children.[11] In a study of children with lead levels ≥ 20 µg/dL in New York, 14 percent of elevated levels were attributed to home renovation with the owners or tenants performing 66 percent of that work.[12] Despite lead based paints having been banned for household and commercial use, lead is still legally allowed to be used in "industrial paint" for bridges and traffic lines. In the U.S., 900,000 bridges are currently painted with lead-containing paint, contributing to dust-borne lead.[13]

Common Sources of Lead

In addition to drinking water and paint, lead exposure can occur from a number of other sources. Inorganic lead can be found in paint, dust, welding fluxes, children's toys, jewelry, and lunchboxes, imported dishware with lead-based glazes, crystal-leaded glass, and candles with lead-containing wicks.[14] Vinyl mini-blinds manufactured outside the United States prior to1996 have been shown to be a significant contributor to childhood lead poisoning. In a 1996 North Carolina survey, 75 percent of childhood lead poisoning cases in a six-month period included lead dust from vinyl mini-blinds as a contributing factor.[15]

Inorganic lead exposure can also occur from lead dust exposure at indoor firing ranges and the ingestion of imported wine or wild game that has been killed with lead bullets as well as residing near a lead smelter.[16] Herbal products from India, China, and other parts of Asia may also be potential sources of significant lead exposure. Certain Ayurvedic herbal products manufactured in South Asia have been found to be contaminated with lead ranging from 5-37,000 µg/g.[17] Since 1978, 55 cases of acute lead toxicity have been directly related to the ingestion of Ayurvedic medicines in the United States and other countries. [18,19]

Cosmetics are another common source of lead exposure. Recently, the FDA released a report that included levels of lead in commercial lipstick as high as 7.19 ppm.[1] The FDA does not regulate lead in cosmetics; however, due to risk for lead toxicity from lead-contaminated food, the FDA issued guidance for the allowable level of lead in candy at .1 ppm.[20]

Organic lead, known as tetraethyl or akyl lead, was found in all commercial gasoline in the U.S. until 1978 when the allowable amount of lead was reduced to 0.05 grams per gallon. EPA estimated that between the years of 1985 and 1992, the even lower standards of .01 grams per gallon resulted in almost one million fewer incidences of blood lead levels exceeding 25µg/dL, the level then established by the Centers for Disease Control as a measure of elevated blood lead levels.[5] As with household and commercial paint, lead was completely eliminated from commercial gasoline for cars in the U.S. in 1978. Using the data available from the blood lead level reductions in the 1970s and 1980s resulting from the removal of leaded gasoline, studies have found a significant correlation with declines in violent crime in the 1990s, paralleling these blood lead reductions.

However, fuel containing lead is still legally used in the U.S. for small piston-engine aircraft, and since is well absorbed through the skin, excessive exposure to this fuel is considered to have significant occupational hazards. Lead induced central nervous system toxicity manifests as bradycardia, fatigue, anxiety, hallucination, hypotension, intoxication, hyperreflexia, nausea, anorexia, and pallor.[21] Organic lead, however, does not cause the classic basophilic stipling used to detect inorganic lead poisoning, even when blood lead levels are elevated to 50 µg/dL.

Lead Absorption and Metabolism

Lead exposure occurs mainly through the respiratory and gastrointestinal (GI) tracts. Gastrointestinal absorption is inversely associated with dietary intake of iron, calcium, magnesium, and dietary fat.[23,24] Infants are the population most at risk for excessive lead exposure as they can absorb up to 50 percent of lead ingested from food, water, contaminated dust, or soil, compared to adults who absorb only 10-15 percent.[25] Once absorbed, 99 percent of circulating lead is bound to erythrocytes for approximately 30-35 days (only one percent of absorbed lead is found in plasma and serum) and is dispersed into the soft tissues over the following 4-6 weeks. Due to the short half-life of lead (35 days) in the bloodstream, blood lead levels cannot be used to diagnose or rule out evidence of exposure that occurred more than six weeks prior to testing.[26]

Autopsy studies show that one third of soft tissue lead is stored in the liver, followed by kidney cortex and medulla, pancreas, ovary, spleen, prostate, adrenal gland, brain, fat, testis, heart, and skeletal muscle. Distribution patterns of lead stored in soft tissue do not appear to change over a lifespan, despite a fairly high turnover rate.[27]

The majority (80-95%) of lead retained in adults is stored in bone while in children approximately 70 percent is stored in bone, with the remainder housed in the soft tissues of the renal cortex, liver, and central nervous system.[28] In the bone, lead can be stored in two separate compartments. The first is the exchangeable pool at the bone surface of either cortical or trabecular bone and the second is the non-exchangeable pool deeper in cortical bone. Lead is constantly being liberated from storage depots in bone and approximately 40-70 percent of blood lead in adults comes from the exchangeable pools of bone lead.[29]

Lead can readily enter the plasma from the storage depots in the bone surface, but can only leave the non-exchangeable pool in the cortical bone and move to the surface of the bone during times of active resorption as in pregnancy, lactation, and menopausal bone loss. One of the more challenging clinical scenarios is the presentation of lead toxicity in a patient who is actively turning over bone, as occurs in menopause, when chelation will only serve to pull lead from an ever replenishing source. Hyperthyroidism, cisplatin chemotherapy, postmenopausal osteoporosis, and periods of immobility will also increase bone mobilization of lead and result in elevations of blood lead.[30-33]

Lead crosses the placenta, is stored in fetal tissue, and is measurable in neonatal blood with levels that will be similar to that of the mother. Lead is measurable in human milk with concentrations similar to that of the mother's plasma, not whole blood, so levels are usually similarly low.[34, 35]

Inorganic lead is excreted unchanged, primarily in the urine and feces as well as secretion in the bile, gastric fluid, and saliva. Lead can also be excreted through the nails and sweat; two studies have shown significant losses of lead in the sweat of study subjects undergoing sauna therapy, compared to the urine.[36,37]

Measuring Lead Exposure – Proper Detection

Whole blood lead levels correlate well in epidemiologic studies with risk for cardiovascular mortality and are considered the current standard for assessing lead toxicity. They do not, however, reflect total body burden as measured by bone stores and thus cannot be used to assess total body lead levels. [38] Cortical bone lead, currently measured by x-ray fluoroscopy (XRF), is considered the most sensitive biomarker for cumulative lead exposure.[39] Unfortunately, because XRF is only available at academic research centers, it is not widely used. A standardized EDTA mobilization testing using CaNaEDTA is also considered a sensitive tool for assessing body lead burden in adults. An elevated body lead burden is defined as the presence of 80-600 µg urine lead in a 72-hour collection after infusion of 1 g calcium disodium EDTA. [40,41] Studies using 1 or 2 g intravenous CaNaEDTA followed by a 6 to 72-hour urine collection, beginning at the initiation of intravenous administration, have also been shown to reliably reflect the potentially toxic fraction of lead body burden.[40,42] EDTA-chelatable lead levels, using the 80-600 µg urine reference, have been correlated with progressive renal insufficiency, peripheral nerve damage, and neurobehavioral symptoms.[43,44]

There is agreement among the Centers for Disease Control (CDC), the Agency for Toxic Substances and Disease Registry, and the Environmental Protection Agency that there is no toxic threshold for lead. This means that there is no measurable level of lead in the body below which harm does not occur.[45] The American Association of Pediatrics (AAP) does not recommend chelation treatment of elevated blood levels unless the whole blood lead is above 25 µg/dL, even though there is significant evidence that cognitive damage occurs even below the current action level of 10 µg/dL.[46] Low-level pediatric toxicity symptoms that occur below 10 µg/dL include lowered IQ, hyperactivity, delinquency, subclinical hearing and balance disturbances, increased dental caries, numerous neurobehavioral problems and cognitive defects.[2,47-50] Researchers in the field of pediatric lead toxicity have argued that the actionable level of blood lead should be reduced to 2.0 µg/dL based on data showing that neurotoxic effects of lead are measurable at levels as low as 2.5 µg/dL.[51] In January of 2012, the Advisory Committee on Childhood Lead Poisoning Prevention of the Centers for Disease Control and Prevention recommended that the level of concern for blood lead be lowered to 5 µg/dL and that children with blood lead above 5 µg/dL be followed and assessed for environmental exposure. The Committee also recommend that they been screened for calcium, vitamin C, and iron sufficiency and treated if they are iron deficient.[52]

Currently, the AAP and the CDC recommend that families of children with blood lead levels at or above 10 µg/dL should have the source of lead exposure identified and removed (remediation) or that the family should be removed from the home if remediation is not possible. Intervention with a chelating agent such as DMSA is recommended with blood lead levels of 25-45 µg/dL, but only if environmental remediation has not been sufficient to normalize levels.[46] Although continuous EDTA therapy has been used extensively in acute lead toxicity in children, it is recommended that children who have a blood lead level of 45 µg/dL or higher not receive a provocative chelation test but be referred for appropriate evaluation and chelation therapy due to the potential for exacerbation of encephalopathy.[46]

Low-level lead poisoning is a serious problem in the U.S. with the CDC estimating that 454,000 school-aged children have blood lead levels over 10 µg/dL. This includes a significant number of children of all racial backgrounds: 25.6 percent of 1-5 year-olds had blood lead levels over 5 µg/dL.

Current Symptoms and Conditions Related To Lead Toxicity

A thorough review of signs and symptoms of acute pediatric lead toxicity is beyond the limits of this article. The reader is referred to the review by the authoritative source on this subject.[54]

Signs of chronic exposure in adults include memory loss, cognitive difficulty, insomnia, depression, lassitude, nausea, abdominal pain, loss of coordination, and headaches. As blood lead levels rise, anemia results as the endpoint of lead's main effect of blocking heme production.[54,55] On visual inspection, a gray line along the gum, with bluish-black edging to the teeth may be another indication of chronic lead poisoning.[56]

Early symptoms of neurotoxicity due to lead exposure in both adults and children include decreased attention span, memory loss, irritability, headache, and low-level cognitive impairment. As childhood exposure continues, impulsiveness, inability to follow directions, lassitude, lowered IQ, and poor attentiveness are seen related to blood lead levels of 10-35 µg/dL.[54,55]

The most commonly documented neurological symptom of lead exposure in adults is peripheral neuropathy, typically involving extensor muscle groups. There is minimal sensory involvement, and if radial or peroneal nerves are involved, and the lead toxicity is advanced, the neuropathy will be exhibited as wrist or foot drop.[56]

Acute renal failure has been identified in lead toxicity resulting from occupational exposure. Research has shown that chronic renal disease, as well, occurs in an environment of elevated total body lead (as assessed by XRF). Positive EDTA-mobilized lead tests in a sub-population of the Normative Aging Study showed renal insufficiency was related to evidence of lead body burden, even though the group had normal blood lead levels of 4.9 ± 2.6 µg/dL.[57]

These and other studies of renal insufficiency indicate that the current reference range for blood lead does not necessarily correlate with low lead body burdens and that renal damage can occur in what the Center for Disease Control and Prevention currently considers a safe blood lead level of under 25 µg/dL for an adult.[58-61]

Cardiovascular disease is another area in which blood lead levels within the normal reference range may reflect significant risk for vascular pathology. In pre- and postmenopausal women, those in the highest quartile of blood lead levels (mean 6.3 µg/dL) had a 3.4-fold increased risk for diastolic hypertension, compared with the lowest quartile of blood lead (mean 1.0 µg/dL).[62] In the postmenopausal cohort, those in the highest quartile of blood lead had an 8.1-fold increased risk for diastolic hypertension.

In 2006, *Circulation*, the journal of the American Heart Association, published an editorial entitled "Low-Level Environmental Exposure to Lead Unmasked as Silent Killer".[63] The editorial was referring to an article in that edition of the journal in which the authors examined a database of 13,946 participants in the 3rd National Health and Nutrition Examination Survey (NHANES) who all had a blood lead level equal to or less than 10 µg/dL and had died of acute cardiovascular events.[64] They looked at risk for cardiovascular mortality from acute myocardial infarction and stroke based on blood lead level and adjusted for a significant list of confounders including body mass index, smoking status, alcohol intake, diabetes, C-reactive protein, total cholesterol, physical activity, low income, high education, urban residence, post- menopausal status, hypertension, diabetes, calculated glomerular filtration rate, and urban versus rural residence. The participants in the study who had a blood lead level above 3.8 µg/dL were 2.5 times more likely to die of a stroke and almost 2 times more likely to die of an acute myocardial infarction than those with a blood lead level under 1.9 µg/dL.

The physicians who authored the editorial, researchers in the field of lead exposure and toxicity, stated that blood lead concentrations as low as 2.07 µg/dL "likely represent a public health hazard".[63] According to the data collected in the NHANES 1999 to 2000 survey (designed to represent the overall U.S. population) 38% of U.S. adults have a blood lead level above this threshold of 2.07 µg/dL.[64]

Fertility

Occupational exposure to lead has been linked to an increased frequency of stillbirths, miscarriages, spontaneous abortion, reductions in sperm count and motility, decreased fertility, and reduced libido.[65]

Women who have lead-exposed male partners have higher rates of miscarriage and their children, who have either parent employed in an occupation with high lead exposure, have increased rates of congenital epilepsy and cardiovascular disease.[66,67]

Ocular Conditions

Since the CNS is the highly affected by lead exposure, there is a link between the development of cataracts and exposure to lead. Those individuals with high bone lead have a 2.5 times increased risk for cataract.[68]

About the Author

Lyn Patrick, ND graduated from Bastyr University with a doctoral degree in naturopathic medicine in 1984 and was in private practice specializing in environmental medicine in Arizona and Colorado. She currently lectures internationally on the subjects of metal toxicity, environmental medicine and chronic hepatitis C and is a faculty for Dr. Walter Crinnion's Postgraduate Course in Environmental Medicine.

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What more could you ask of a base remedy?

Rescue 3 & Rescue + {soon to be Relief 3 & Relief +}

2 core remedies for Ig & emotional responses:
- neutralize reactions to allergens,
- reduce healing crisis,
- normalize Histamine, Serotonin & ACC thereby calming Ig responses.

Balance reactions to:

Histamine; Puffiness, tearing, soreness, itchy.

Serotonin; Un-well/nausea feeling upon contact with an allergen.

Acetyl Choline Chloride (ACC); brain fog, reduced mental clarity & function. (ACC is the most predominate neurotransmitter in the brain, when it becomes unbalanced people have a hard time thinking straight or being able to concentrate. It's the main neurotransmitter for brain allergies.)

They are great on their own to calm any reactional crisis or as a base in any protocol where reactions are a potential.

Rescue + goes beyond addressing the reactional response, it also offers emotional support with the addition of the 5 English flowers in its formula.

Rescue 3 (Reactions)	+	5 Flowers (Emotions)	=	Rescue + (Reactions + Emotions)

Protocol suggestions:

Balancing Ig: 5 drops TID

In Crisis/Panic: 5-10 drops every 15 minutes as needed. Rescue + is better for panic.

Background remedy in detox or where a healing crisis is possible: 3-5 drops with other remedies.

All ***Vibrant Health*** remedies (now called ***Viatrexx*** for international purposes) use multiple low to high level homaccord dilutions (sample range: 6X or 4C + 73K + 60M), this will address physiological, intra & extra cellular, metabolic and emotional levels. In this way all the core functional and causal dimensions are addressed.

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