HEALTH Realities®
INSTITUTE FOR HEALTH REALITIES
"When a man dies, he dies not of the disease he has at the moment, but of his whole life." ~ Anonymous
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Toxic Footprints

A series in four parts exploring chronic bodily environmental toxic contamination

Part I
Identifying, isolating and eliminating chronic bodily environmental toxic contamination

In the Western World, life expectancy has increased to 80 years. Nevertheless, at the Institute for Health Realities, we believe this represents only two-thirds of our designed lifespan. We also believe that chronic environmental toxic contamination plays a significant role in lifespan reduction by lowering our threshold for combating physical and emotional disease, infection and cancer, costing us billions annually.[1]

Environmental toxins are the product of both a natural phenomenon (volcanic activity) and of human interaction. According to the most recent accounting of mining and industrial production in the U.S., over seven billion pounds of toxic waste are added to our environment each year.* Heavy metals comprise the majority of this toxic waste and top the government's list of the most troublesome toxic agents.

To Our Friends of the Health Realities Journal,

Over the past 20 years, we've provided our readers with useful, cutting-edge health information that generally precedes standard-of-care by five to ten years. For example, in 1984, our journals on cholesterol predicted that many people would fall victim to the "one-size-fits-all" approach to recommended fat and cholesterol levels. And in 1994, we produced our now famous four-part series on the six subclinical defects that underlie all infections, cancers, toxicities, and diseases. The sixth of those defects, oxidative stress, is brought on by environmental toxic exposure. Hence, we're producing Toxic Footprints, a four-part journal series on chronic bodily environmental toxic contamination, following this outline:

Part I: An overview of chronic bodily environmental toxic contamination offers insight into detecting such toxicity through simple body chemistry studies.
Part II: The clinical implications of chronic toxic exposure are thoroughly explained with special attention given to the cholesterol paradigm that is popularly imposed.
Part III: We incorporate a forensic approach to observe the body's natural defenses, to detect when and where a toxin has breached those defenses, and to determine how much toxin is involved. Using this approach, the toxin can be safely and effectively eradicated.
Part IV: Iron status and the Toxic Free Iron Risk. Free iron is perhaps as big a health problem as iron deficiency and overload, causing infections, cancer, and disease. We explore these problems as well as mankind's most common genetic aberration.

From all of us at the Institute for Health Realities, I trust you'll find this Health Realities Journal series to be most informative and highly useful.

H.L. "Sam" Queen
Founder
Director of Research Development

Toxic Footprints

Among those listed, arsenic, cadmium, lead and mercury are particularly harmful, contaminating rivers, lakes, and the many effluents that reach our water supplies. Solvents, pesticides and other petrochemicals are equally problematic. But, above all, it is important to understand that we all experience some chronic, low-level exposure to these environmental toxins. Finding and eradicating these toxins is essential for attaining optimum health.

Identifying Toxic Footprints

Through our research and clinical application, we know that environmental toxins leave distinct chemical markers or "footprints" in the body's chemistries. Therefore, we can help healthcare professionals and their clientele identify the presence of environmental toxins in the blood and urine without the need for extraordinarily expansive (and needlessly expensive) testing. In addition, we have developed specific strategies for eradicating these toxins.

Using our Toxic Footprint protocol, doctors can routinely look for evidence of the major classes of environmental toxins in a comprehensive body chemistry profile. Most Toxic Footprints appear in the bloodstream upon initial exposure. After prolonged exposure, or when subsequent exposures occur, the footprints may shift or become more distinct. Regardless, it is possible to track Toxic Footprints in accordance with how the body is designed to process and eliminate each toxin or combination of toxins.

Isolating Toxic Footprints

Historically, doctors have used laboratory findings to help diagnose disease or to establish disease risk. Using this "disease model" as a guide, chemistry readings outside a predetermined reference range indicate disease or disease risk. Our research indicates that toxic exposure "moves" the target parameters relative to a personal baseline value. Detecting this movement away from the baseline reference indicates a Toxic Footprint. Hence, it is the change (or lack thereof, in the presence of other evidence) in a specific chemistry value (or values) that is of the utmost importance.

Most environmental toxins are fat-soluble, affecting us in one of three ways:

- Toxicologically (i.e. either nerve or brain disorders)
- Immunologically (i.e. allergies, autoantibodies, and altered viral or cancer-fighting abilities)
- Pathophysiologically (i.e. kidney or adrenal gland dysfunction)

Nerve and brain disorders, loss of mental acuity, immune problems (cancer, autoimmunity and food sensitivities), constipation and other dysfunctions involving the excretion of toxins are among the readily identifiable symptoms. However, common maladies like mental confusion, headache, neurological effects, fatigue, depression, repeat or chronic infections, and unexplained pain that emanates from a variety of joints, organs, and tissues may also indicate the presence of environmental toxins.

Eliminating Toxins

Our straightforward strategy for eliminating environmental toxins is based on the body's natural response to exposure in an attempt to defend itself. The body responds in one or more of the following ways:

- Handcuffing the toxin with protein or a fat/protein particle.
- Storing the toxin in some way.
- Conjugating it for removal through the intestines and kidneys.
- Converting the toxin to a water-soluble entity that can be excreted in the urine.
- Coping with the toxin immunologically, hormonally or via a lipid response.

Removal of the toxin and/or its conversion to a water-soluble entity requires the assistance of a process called biotransformation. Biotransformation occurs through activation of a three-phase enzyme "detoxification" system. A variety of enzymes that respond to different toxins[2] are activated in Phase I, with the cy-

▶Biotransformation[1]

Biotransformation is a three phase process by which the body rids itself of unwanted, potentially deleterious lipophilic toxins by metabolically converting these toxins to hydrophilic metabolites that are more readily excreted. Through biotransformation, the body is able to activate the arachidonic cascade in inflammation and to synthesize bile, prostaglandins and a variety of steroid hormones.

Phase I
Functionalization Reaction

In Phase I, activation of CYP450 and other critical enzymes catalyzes the hydroxylation (insertion of -0H + electrons) that converts a variety of potentially toxic, lipophilic substrates to a hydrophilic state that can be excreted through the urine.

Phase II
Conjugation

In Phase II, the water-soluble product of Phase I biotransformation (or toxins that may skip Phase I) are conjugated with a variety of agents, such as sulfates, glucuronic acid, and glutathione. These conjugated forms can be excreted through the urine or as a bile component.

Phase III
Cellular Conjugate Export

In Phase III, conjugates of glutathione and potentially harmful metabolites are excreted from cells via ATP-dependent export pumps. This mechanism prevents cellular accumulation of reactive hydrophilic metabolites of Phase I and II that (if not removed quickly) may interact with DNA or with proteins, resulting in cancer or auto-immune disease.

[1] Enzyme Systems that Metabolise Drugs and Other Xenobiotics: Current Toxicology Series, C. Ioannides, Ed., John Wiley & Sons, Ltd., 2002.

[1] Muir, T., and Zegarac, M., "Societal Costs of Exposure to Toxic Substances: Economic and Health Costs of Four Case Studies that are Candidate for Environmental Causation", *Environ Health Perspect* 109 Supple 6: 885—903, 2001.

* EPA (U.S. Environmental Protection Agency): Toxic Release Inventory (TRI). The full report is available on the Internet at http://www.epa.gov/opptintr/tri A slightly different view of the data is available through the National Library of Medicine at www.toxnet.nlm.nih.gov

tochrome P450 (CY P450) enzyme family[3] playing a major role. The greatest activity occurs in the liver, the blood brain barrier and the intestines, but may also occur in and around nerves and other cells. Phase II involves conjugation, where either glutathione, selenium, uric acid, or some other natural agent latches onto (conjugates) a heavy metal or other toxin to escort it out of the body. Potentially toxic molecules trapped in the cells from Phase I and II activity, are conjugated and removed during Phase III. In most cases, glutathione serves as the escort.

A modified response of the fat transport system is required to move the toxins to sites where the process of biotransformation can take place. Hence, a rise in serum cholesterol and triglycerides, or an exaggerated rise in HDL levels, may indicate the presence of a toxin. In this way, the body protects the nerves and brain from the toxin. However, in the process of altering the quantity and mix of blood fats to defend itself, a variety of unwanted health conditions may arise in the body. Heart and cardiovascular disease are very obvious examples.

Thus, the presence of environmental toxins may be the underlying cause of high cholesterol and triglycerides. Some who appear to have healthy blood lipid patterns (or unusually low blood lipid levels) may be at risk for greater reactivity to a toxin because they do not have the benefit of the fat transport system necessary to biotransformation. In the same vein, when the body responds immunologically to toxic exposure, other symptoms (including "allergic" hypersensitivity) may very well be present.

Understanding the many ways that the body defends itself against environmental toxins is key in developing appropriate strategies for neutralizing harmful effects. In fact, some people may need a higher fat diet to facilitate protection, removal, repair and recovery. As a by-product of this approach, we may identify better methods of addressing issues like high cholesterol and heart attack risk. Above all, avoiding typical cholesterol-lowering diets, drugs, and weight loss is highly beneficial when environmental toxins are present.

Heavy Metal Toxins

Generally, heavy metals have a density five times greater than water (specific gravity 1.0). Heavy metals such as arsenic, mercury, lead and cadmium are highly toxic, as well as persistent and pervasive. Given their persistent nature, heavy metals do not degrade as readily as other toxins. They merely change form. Also, due to their pervasiveness, toxic metals ultimatley affect us regardless of their environmental point of entry. Thus, we must do everything we can to discontinue any unnecessary heavy metal usage whatsoever, such as the use of mercury in "silver" dental fillings.

Heavy metals function as oxidant catalysts, producing oxidative stress from toxic free radicals in cell membranes, tissues and organs. This property, while harmful to humans, is advantageous to pesticide manufacturers. It is the heavy metal component in pesticides that attaches the poison to the lipoidal tissues of the target nerve and brain.

Heavy Metal Toxic Footprints

Each heavy metal leaves its own distinct Toxic Footprint in the blood and urine. Markers common to most heavy metals[4] include chronic inflammation, a measurable rise in Immunoglobulin A (IgA) and M (IgM) values or in serum cholesterol readings,[5] proteinuria,[6] low protein readings or difficulty with protein metabolism, and evidence of sulfhydryl binding. Thus, because heavy metal toxicity is so problematic, it is important to identify the Toxic Footprints associated with each.

Mercury

Mercury, perhaps the most toxic of all naturally occurring environmental toxins, has the potential to interfere with nearly every metabolic function. One way we can verify mercury toxicity is by pinpointing changes in the proteins that generally bind to it. For instance, mercury shows a special affinity for the sulfhydryl protein component of the many cysteine residues found on hemoglobin.[7] The act of binding to hemoglobin promotes an anaerobic condition by altering the shape of red blood cells, thereby thwarting them in their effort to travel smoothly through the microcirculation. Mercury also promotes oxidative injury to red blood cells, as evidenced by a reduction in serum G-6-PD and by enlargement (increased MCVreadings). Similarly, mercury binds to the sulfhydryl component of Immunoglobulin A (IgA) from mucosal linings and Immunoglobulin M (IgM) from B-lymphocytes, causing the serum IgA and IgM levels to rise in concert with exposure levels. Extensive exposure increases blood urea nitrogen (serum BUN) levels, decreases early morning serum cortisol and, ultimately, increases serum beta-2-microglobulin levels, indicating that either the brain's kidney (the choroid plexus) is saturated with mercury or that T-cell (T-lymphocyte) function has been lost. When metallic mercury is present, resistance to viruses, cancer[8] and autoimmune disease[9] is greatly diminished. Of importance to every doctor, OSHA now includes changes in the beta-2-

Nebert, D.W., and Russell, D.W., "Clinical Importance of the Cytochromes P450", *The Lancet* 360: 1155-62, October 12, 2002.

[3] Nelson, D.R., "Cytochrome P450 gene superfamily": Available at http://drnelson.utmem.edu/cytochromeP450.html.

[4] Queen, H.L., "Chronic Mercury Toxicity: New Hope Against An Endemic Disease", Queen and Company Health Communications, Colorado Springs, CO 1988.

[5] Bencko, et al., "Immunological Profiles in Workers Occupationally Exposed to Inorganic Mercury", *J Hygiene, Epidemiol, Microbiol, and Immunol* 34(1): 9-15, 1990.

[6] Picator, M., "Proteinuria in chronic cadmium poisoning", *Arch Environ Health* 12: 345-59, 1966.

[7] Klaassen, C.D., Ed., *Casarett and Doull's TOXICOLOGY: The Basic Science of Poisons*, Sixth Edition, McGraw-Hill Medical Publishing Division, New York, p.398, 2001.

[8] Park, S.H., et al., "Effects Of Occupational Metallic Mercury Vapour Exposure On Suppressor-Inducer (CD4+CD45RA+) T Lymphocytes And CD57+CD16+ Natural Killer Cells," *Int Arch Occup Environ Health* 73(8): 537-42, November 2000.

[9] Whitekus, M.J., et al., "Protection Against CD95-Mediated Apoptosis By Inorganic Mercury In Jurkat T Cells," *J Immunol* 162(12): 7162-70, June 15, 1999.

microglobulin reading to determine when a worker should be monitored more closely, removed from the workplace, or treated for heavy metal exposure.

Cadmium and Lead

Some individuals are genetically predisposed to cope with lead and cadmium toxicity through natural resistance and storage mechanisms.[10] However, high exposure to cadmium affects the kidneys, causing dysfunction in the renal proximal tubules and bringing about proteinuria and/or a rise in urine beta-2-microglobulin similar to that of mercury. Thus, low serum albumin, coupled with measurable urine albumin, and/or elevated urine or serum beta-2-microglobulin levels are reliable cadmium markers.[11] With prolonged exposure, cadmium is taken up by bone, sometimes causing a highly painful condition known as itai itai ("ouch, ouch") disease. A urine neutral endopeptidase is a classic footprint of cadmium.[12]

Lead, on the other hand, interferes with the process that the phagocytic monocytes use to signal T-cells of the presence of cancer cells, hindering cancer defenses.[13] In addition, lead adversely affects nucleotide metabolism and hemoglobin quality (causing fatigue with reduced mental status[14]) and competes with calcium uptake and ionic calcium function, thereby promoting arterial calcification and high blood pressure. Consequently, tests for acid stress, free calcium excess, connective tissue breakdown and functional deficiencies in zinc and calcium mark the presence of lead. The porphyrin fingerprint" test, performed by almost all County and State Health Departments, confirms the presence of lead.

Copper And Iron

Copper (specific gravity 8.92), a trace mineral, and iron (specific gravity 7.86) are heavy metals by definition, yet highly beneficial to human health when properly bound to protein. Both are required to help prevent anemia and both play roles in energy production and transfer. Left in a free or unbound state, however, either can be as toxic as any other heavy metal. The body uses transferrin (iron-binding protein) and cerruloplasmin (copper-binding protein) as "safety escorts." Therefore, evidence of disruption in this binding process represents a Toxic Footprint for the free, unbound metal.

		Inflammatory Response	Eosinophil	Basophil	IgA	IgE	IgG	IgM	Serum Beta-2 Microglobulin	Urine Beta-2 Mocroglobulin	BUN	Fasting 8a.m. Cortisol	Protein	Albumin
Heavy Metals		↑	↑	–	↑	–	–	↑	↑	↑	↑↓	↓	↑	↓
	Mercury	↑	↑	↑	↑+	–	–	↑+	↑	↑+	↑↓+	↓+	↑	↓
Solvents		↑	–	–	–	–	–	–	–	–	–	–	–	↓
Pesticides		↑	↑	↑	–	–	–	↑	– ↑	–	–	–	↑	↓
Petro-chemicals	Latex	↑	↑+	↑+	–	↑+	–	–	–	–	–	–	–	–
	Perfluoro-carbons	–	–	–	–	–	–	–	–	–	–	–	↓	↓
	Other	↑	↑	–	–	–	↑	–	–	–	–	↑	↑	↓

Table 1a. Toxic Footprint (Chemistry) Map
A guide to the assessment of the environmental exposure that may influence a client's health or blood picture. *The areas marked with a red plus (+) represent the most reliable indicators for exposure to a particular class of toxin.*

Petrochemicals

Petrochemicals are environmental toxir originating from fossil fuels. Example include petroleum, natural gas and coal, along with oxidative byproducts, chemis-

[10] Bjorkman, L., Vahter, M., and Pedersen, N.L., "Both The Environment And Genes Are Important For Concentrations Of Cadmium And Lead In Blood," *Environ Health Perspect* 108(8): 719-22, August 2000.

[11] Noonan, C.W., et al., "Effects Of Exposure To Low Levels Of Environmental Cadmium On Renal Biomarkers," *Environ Health Perspect* 110(2): 151-5, February 2002.

[12] Nortier, J., et al., "Urinary Neutral Endopeptidase In Workers Exposed To Cadmium: Interaction With Cigarette Smoking," *Occup Environ Med* 54(6): 432-6, June 1997.

[13] Fischbein, A., et al., "Phenotypic Aberrations Of CD3+ And CD4+ Cells And Functional Impairments Of Lymphocytes At Low-Level Occupational Exposure To Lead," *Clin Immunol Immunopathol* 66(2): 163-8, Feb. 1993.

[14] Kim, Y., et al., "Evaluation Of Lead Exposure In Workers At Secondary Lead Smelters In South Korea: With Focus On Activity Of Erythrocyte Pyrimidine 5-Nucleotidase (P5N)," *Sci Total Environ* 286(1-3): 181-9, March 8, 2002.

try products and chemistry byproducts. While most petrochemicals are burned directly as fuels, experimentation with certain unburned fuels has given birth to new technologies. Much of what occupies our modern technical world has come from such research - computers, fabrics, automobiles, building materials, food additives, household cleaners, formaldehydes, pharmaceuticals, etc., plus a wide range of products made from latex, rubber, and plastic. Therefore, petrochemical toxicity poses a significant threat to human health.

Petrochemical exposure can stimulate hypersensitivity reactions, with symptoms ranging from a simple rash or allergy to deadly anaphylaxis. Lethal anaphylactic reactions can generally be attributed to latex, plastics and rubber.

		Total Serum Cholesterol	LDL	HDL	Triglyceride	Chol/HDL Ratio	Platelet Count	Platelet Aggregatioln	SGOT	SGPT	GGTP	Total Bilirubin	MCV	G-6-PD	Acetylcholin-esterase
Heavy Metals		↑	↑	↓	–	≥4.0 ↑	–	↑	–	–	–	↑	↑	↓	–
	Mercury	↑	↑	–	–	–	–	↑	–	–	↑	↑+	↑+	↓+	–
Solvents		↑	↑	↕	↑+	↑	–	↑	↕	↕	↕	–	–	–	–
Pesticides		–	–	↑+	↑	≤2.9 ↓+	↓	↑	↕	↕	↕	↑	↑	↓	↓–
Petro-chemicals	Latex	–	–	–	–	–	↕	↑	–	–	–	–	–	–	–
	Perfluoro-carbons	↓	↓	↓+	↓	↓	–	–	↑	↑	↕	↕	–	–	–
	Other	↑	↑	↓	↓	↑	–	–	↑	↑	↕	↑	–	–	–

Table 1b. Toxic Footprint (Chemistry) Map
A continuing guide to the assessment of the environmental exposure that may influence a client's health or blood picture.

Latex

Latex is the formed, yet pliable, end-product of a special emulsion comprised of a colloidal, particle-filled liquid suspended within another liquid. Core latex particles may be derived from a variety of sources, including rubber or plastic, or found in the milky emulsions of certain plants, such as the milk thistle. As an environmental toxin, latex can bring about either a hypersensitivity reaction upon contact with human skin or an immune activation in response to particle-filled gases from rubber and many plastics. Latex allergy is most commonly diagnosed in individuals who are exposed to latex frequently. Those employed in the health care or rubber industries, as well as children with spina bifida and other congenital diseases requiring multiple surgeries are at risk. Latex exposure increases immunoglobulin E (IgE) levels and raises histamine and heparin-rich cell levels, including the basophilic and eosinophilic white blood cells and the mast cells of connective tissues. Hence, an elevated IgE, coupled with a high basophil or eosinophil count, marks latex toxicity. When these readings are accompanied by a hypersensitivity reaction, a significant risk of life-threatening anaphylaxis exists. Anaphylaxis can occur independently of a rise in IgE (or any other immunoglobulin), as long as histamine-rich cells are present. Nevertheless, any rise in IgE that accompanies the rise in histamine-rich white cells presents the greater health risk.

Anaphylaxis risk can be assessed in a similar manner. Characteristically, the risk for anaphylaxis begins with exposure to latex or some other petrochemical that elevates IgE along with the basophil or eosinophil white blood cell count. When this scenario is accompanied by an excess of unbound calcium and followed by a second exposure to a trigger antigen, anaphylaxis can occur. The most common trigger antigens include bee sting, peanuts, and a variety of prescription drugs. In dentistry, the drug Naproxen, an anti-inflammatory analgesic agent given after tooth extraction,[15] has a long history of serving as an antigen. Neuromuscular blocker drugs[16], as

[15] Cistero, A., et al., "Coronary Artery Spasm And Acute Myocardial Infarction (Heart Attack) In Naproxen-Associated Anaphylactic Reaction," *Allergy* 47(5): 576-8, October 1992.

[16] Assem, E.S., "In Vivo And In Vitro Tests In Anaphylactic Reactions To Anesthetic Agents," *Agents & Actions* 33(1-2): 208-11, May 1991.

well as certain antibiotics and opioid anesthetics, are examples of high-risk medical antigens. Medical and dental procedures that require the use of these drugs should be avoided if any Toxic Footprints for anaphylaxis risk are present.

Plastics and Rubber

Plastics (mostly hydrocarbons) and rubber (the pliable end-product of the latex-like emulsion of rubber trees) tend to "outgas" to some extent with hydrocarbon-based plastics being the most active. Plastic polymer "breakdowns" entering the food chain may cause food and chemical sensitivities. Rubber also has the capacity to elicit a hypersensitivity reaction similar to latex or plastic. Therefore, the markers for plastic and rubber toxicity are very similar (Tables 1a & 1b).

Fluorocarbons and Perfluorochemicals

Fluorocarbons are stable petrochemicals that can be packaged and used for several years. Lubricants, refrigerants, fire extinguishing agents, aerosols, and flame and heat resistance substances are prime examples. Perfluorochemicals, on the other hand, are mostly inert chemical substances with a high oxygen-carrying capacity. Perfluorochemical acids and esters can be emulsified to form a surfactant and transfused into people to temporarily raise their oxygen carrying capacity. Whether exposed intentionally through use of a medication, or acquired from environmental contamination, perfluorochemicals have a particularly depressing effect on total serum cholesterol and triglycerides.[17] Therefore, elevated IgE readings and eosinophilic and basophilic white blood cell levels,[18] coupled with a drop in the serum lipids (total serum cholesterol[19], LDL, HDL, and serum triglycerides)[20] are reliable markers.

Solvents

Solvents and pesticides, representing the largest petrochemical class, pose the greatest threat to health. Tars and toxic waxes or oils exemplify this group. Each year, millions of gallons of oil-based solvents are used industrially, contaminating the environment either through evaporation or by direct entry into landfills, waterways and foods. Like any other petrochemical, solvents tend to degrade over time. However, they may be transformed into even more toxic substances in the process. Vinyl chloride, for instance, may degrade into the more toxic trichloroethane, a noted cancer-causing agent.

Hydrocarbon-based solvents tend to leave little or no residue as they vaporize, explaining, in part, their popularity in the cleaning industry. Fumes from solvents may be inhaled or absorbed through the skin. Either way, being fat soluble, organic solvents have an especially strong affinity for the lipoidal tissues of the nerve and brain. Consequently, the body must readily process and eliminate these solvent toxins or risk serious nerve, brain, or neurological damage. An elevation in serum triglycerides[21] is a classic marker. Any failure of the body to respond by elevating serum triglyceride[22], possibly due to genetics, malnutrition, or some previous exposure to a lipid-depressing petrochemical, may prove fatal.

Alcohol

Alcohol, although not a petrochemical, is a typical solvent. As it is fat-soluble, it targets the lipoidal tissues of the nerves and brain. To minimize its toxic effect, the body responds by converting "excess" alcohol into fatty acids that can be transported as serum triglyceride,[23] stored as a component of adipose tissue, or transported to the mitochondrial furnaces attached to protein. Hence, elevated levels of total serum cholesterol and triglycerides[24] serve as clear markers.

As with other fat-soluble toxins, chronic and heavy alcohol usage (≥ 280g. per week) tends to damage the liver. Beginning with a gradual rise in the GGT enzyme (indicating difficulty in using proteins), an error develops in synthesizing transferrin, the iron-binding protein. In due time, the serum level of free (unbound) iron elevates with a corresponding decrease in total iron binding capacity (TIBC). This highly toxic unbound iron inflames the liver, causing liver enzyme levels to rise. Thus, cirrhosis of the liver from chronic alcohol ingestion occurs not only from the direct effect of the alcohol, but from the secondary effect of toxic free iron.

Combining heavy alcohol usage with mercury or arsenic exposure reduces the body's ability to cope. Needing more protein to neutralize the toxicity of the arsenic and mercury, the presence of these heavy metals inactivates the protease enzymes required by the liver to synthesize protective albumin and functional transferrin.[25] With the alcohol transport mechanism hindered, the liver, nerves and brain are susceptible to low alcohol toxicity levels.

Toluene and Xylene

In a 1989 study sanctioned by the U.S. Senate Subcommittee on Health, the EPA found that the amount of airborne neurotoxins released into the atmosphere by industry had increased ten-fold since 1985. At the top of the list was toluene, which is widely used in the print industry as a solvent for ink and in the automobile industry as a paint solvent[26]. It is closely

[17] Gilliland, F.D., and Mandel, J.S., op. cit. p. 560-568.
[18] Leung, P.S., et. al., "Evaluation Of Possible Histamine Release From Human Peripheral Blood Cells Using An Enzyme Immunoassay (HRT) With Components Of Intravenous Catheters," *Allergie et Immunologie* 25(8): 346-53, October 1993.
[19] Olsen, G.W., Burris, J.M., Mandel, J.H., and Zobel, L.R., "Serum Perfluoroctane Sulfonate And Hepatic And Lipid Clinical Chemistry Tests In Fluorochemical Production Employees," *J Occup Environ Med* 41(9): 799-806, September 1999.
[20] Gilliland, F.D., and Mandel, J.S., "Serum Perfluorooctanoic Acid And Hepatic Enzymes, Lipoproteins, And Cholesterol: A Study Of Occupationally Exposed Men," *Am J Ind Med* 29(5): 560-8, May 1996.
[21] Queen, H.L. "Sam," "Triglycerides: Pieces Of The Cholesterol Puzzle," *Health Talk*, 11(1): 1-8, Jan. 1992.
[22] Queen, H.L. "Sam," "Reversing Chemical- Or Neurotoxic-Induced Damage To The Brain and Nervous System: Nerve And Brain Repair," *Health Talk* 9(2): 9-16, September 1990.
[23] Soderberg, B.L., and Laposata, M., "Fatty Acid Ethyl Esters: Markers Of Ethanol Intake," *Am Clin Lab* 20(8): 18-20, September 2001.
[24] Ezenwaka, C.E., Premanand, N., and Orrett, F.A., "Studies On Plasma Lipids In Industrial Workers In Central Trinidad And Tobago," *J Natl Med Assoc* 92(8): 375-81, August 2000.
[25] Queen, H.L., "Sam," "Protease Enzymes: Part I; Laying The Groundwork For Clinical Use," *Health Realities Journal* 18(1): 1-8, Fall 2000.
[26] Baelum, J., et al., "Response Of Solvent-Exposed Printers And Unexposed Controls To Six Hour Toluene Exposure," *Scand J Work Environ Health* 11: 271-80, 1985.

followed by xylene (used to help mold plastics and materials used for making dentures). Even low-level toxic contamination from these solvents elevates triglyceride levels.

Dioxin

Dioxin is used not only as an herbicide (i.e., Agent Orange), but as a solvent in the manufacturing process for "white" paper products and toiletries. Again, elevated serum triglycerides provide the most notable footprint.

Carbon Disulfide (CS_2)

This highly flammable and poisonous solvent is used in the adhesive industry and as a preservative for edible fruit. Certain industry workers and those who ingest overly contaminated foods may exhibit a rise in total serum cholesterol, higher LDL levels, diminished HDL levels and elevated serum triglyceride measurements. In addition, long-term exposure to carbon disulfide may be marked[27] by higher serum triglyceride levels (related directly to blood CS_2) and elevated serum lead levels (with higher blood pressure measurements). There is also an increased risk for ischemic heart disease that corresponds to relevant lipid changes.[28]

Pesticides

For centuries, sulfur was used to control insects.[29] As the world's population increased, scientists responded to a demand for increased food production and the need for better hygiene with quicker-acting "pest-control agents." These synthetic chemicals are highly toxic. Presently, 2.6 billion pounds of pesticides are added to the environment each year.[30] Pesticide exposure adversely affects all ages. In the U.S., routine pesticide monitoring carried out by various state and federal agencies has shown that exposure often begins in the womb.[31] By age 10, virtually every person living in America exhibits a measurable level of pesticide toxicity.[32]

According to the World Health Organization, Third World inhabitants suffer from pesticide exposure the most, due, in part, to inadequate education about proper pesticide handling, overuse, and misuse.[33] As a result, over three million pesticide poisoning cases occur worldwide every year. Moreover, in adulthood, the risk for breast and prostate cancer in the more industrialized areas of the world tends to rise in concert with pesticide tissue levels.[34,35] Worldwide, an estimated 220,000 die annually of pesticide poisoning and its consequences.

Pesticide Footprints

Pesticides are generally transported through the body attached to HDL. While they may be "complexed" with non-esterified fatty acids (NEFAs), albumin, triglycerides, and LDL, rising HDL levels serve as clear markers. Pesticides cause an exceptional rise in HDL relative to total serum cholesterol,[36] as well as extraordinarily low cholesterol/HDL (≤ 2.9) ratios. When pesticides act as solvents, all serum lipids rise as well. More severe neurological symptoms occur when total serum cholesterol levels are low (≤ 160 mg/dl) and HDL levels fail to rise in response to pesticide exposure.

Pesticides attached to HDL can be eradicated by aiding the body's natural defense mechanisms. As with most toxins, the body attempts to cope by:

- Transporting pesticides to the mitochondria where they are burned along with fats.
- Transporting them to the liver for biotransformation, creating a water soluble entity that can be excreted in the urine.
- Depositing them directly into the catabolic pool of the liver from the bloodstream and eliminating them through the intestines along with bile.

Because pesticide toxicity is so prevalent, it is very important to recognize specific footprints of the more commonly used pesticides.

Organophosphates

Organophosphates, first developed as nerve gas in WWI, are among the most toxic of the synthetic pesticides. Organophosphates primarily target the central nervous system, depressing acetylcholinesterase activity. First, nerve transmissions controlling muscle contraction and relaxation occur as pulses. Each pulse begins with a rise in acetylcholine. Then, as soon as the signal to contract is transmitted, acetylcholine must be neutralized to make way for the next pulsed signal. Acetylcholinesterase is the enzyme that effects this neutralization. In destroying acetylcholinesterase, all muscle control is lost.

Organophosphates, similar to other fat-soluble hydrocarbons, may function not only as pesticides, but also as solvents. Exposure is marked by elevated triglycerides, acetylcholinesterase loss and changes

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in GGTP, SGOT and SGPT levels.[37] Initially, these enzyme levels decline as zinc and other enzyme-dependent co-factors, including vitamin B complex and protease enzymes, are exhausted. As inflammation becomes more severe or chronic, GGTP, SGOT and SGPT readings typically rise.

Warfarin (Coumadin)

Warfarin is a pesticide that is a popular medicine as well. Commonly known as Coumadin, the term Warfarin is derived from The Wisconsin Alumni Research Foundation that conducted research and provided funding for developing the medicine. Coumadin is a popular anticoagulant used to treat the thrombotic (clot-promoting) aspect of human heart disease. Employment of a pesticide in treating heart disease is based on the age-old adage, "dosage doth make the poison". But when doctors forget that the medicine is first a pesticide, toxic reaction can exacerbate the condition (thrombosis) that it is intended to treat. For example, in the absence of adequate albumin levels, administering Coumadin may initiate a protective lipid response similar to any other pesticide, thereby raising the risk for thrombosis. Hence, Coumadin's risk/benefit ratio improves greatly when the blood contains healthy levels of albumin and non-esterified fatty acids (NEFA, or free fatty acids). This NEFA/albumin complex readily transports fatty acids where they are needed, safely transporting Coumadin, by extending the NEFA/albumin complex to form a NEFA/albumin/Coumadin complex.[38]

In a situation of compound exposure, where a Coumadin user may become exposed to, say, a solvent, the risk of thrombosis is elevated. The NEFA/albumin complex is diverted to defend against the solvent toxicity, leaving the individual exposed to the toxic pesticidal component of the medicine. Therefore, Coumadin users should be screened for solvent toxicity and warned about exposure.

It is important to look for Toxic Footprints before prescribing a pesticide-based anticoagulant. High serum triglyceride levels (≥150 mg/dl) indicate a tendency towards anaerobic metabolism. Combined with blood sugar readings between 96-107 mg/dl.[39], too little NEFA may be available to properly transport a pesticidal medicine.

Paraquat

Paraquat, a highly toxic herbicide and insecticide, is used on roadsides as a defoliant and on foods as a pesticide. It is therefore relatively prolific. Paraquat significantly interferes with protein metabolism by disrupting the workings of a critical enzyme needed to breakdown type IV collagen protein in a healthy way.[40] This interference may help explain why paraquat poisoning is suspected as a causal factor for scleroderma (systemic sclerosis).

Acute paraquat poisoning, characterized by an immediate rise in urine paraquat, edema and hemorrhage, leads to a fatal respiratory system failure. In contrast, chronic paraquat exposure is marked by low urinary excretion and a 2.7-fold rise in lipid peroxidation. The latter is followed by pulmonary fibrosis (interference with normal protein metabolism), oxidative stress, chronic inflammation, and connective tissue breakdown. Therefore, high HDL and serum protein levels are typical toxic footprints.

Conclusion

Chronic environmental toxic exposure poses a measurable threat to our health by diminishing natural defenses against emotional and physical disease, including infection, cancer and degenerative conditions. Healthcare professionals are reasonably well trained to recognize acute toxic poisoning. Unfortunately, many in the medical community attempt to evaluate the effects of chronic toxic exposure using the same criteria, which does not work. Therefore, very few doctors fully appreciate the role that chronic toxicity plays as a causal factor for ill health.

Through our research and clinical application, we have identified the Toxic Footprints left by environmental toxins in the body's chemistries. Using our Toxic Footprint protocol, doctors can routinely test for the presence of environmental toxins in a comprehensive body chemistry profile. By narrowing the search for offending agents, doctors are better able to provide pointed and effective answers to difficult health challenges.

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Writing and Editing: H.L. "Sam" Queen, M.A., C.C.N., C.N.S.

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