There is a growing appreciation for the effects that exposure to chemicals and heavy metals have on the body, in particular the brain and nervous system. Some heavy metals including lead, aluminium and mercury can cross the blood brain barrier and accumulate in brain tissue. Heavy metals can also block certain nutrients, by competing for particular binding sites on enzymes and other proteins.

**Mercury**

Mercury is one heavy metal, of which acute toxicity produces significant mental and emotional dysfunction. Low grade chronic exposure, not uncommon in some environments, may also contribute to mental and behavioural disturbances and developmental conditions.

Mercury is found in the environment as elemental mercury, inorganic mercury or organic mercury (ethyl-, methyl-, alkyl-, or phenylmercury). In our environment it is present in thermometers, thermostats, dental amalgams, paint, cosmetic products, laxatives, diuretics and antiseptics. Organic mercury is the most toxic and most frequent form of mercury exposure, found in fish, poultry, pesticides and insecticides. Up to 80% of inhaled elemental mercury and up to 100% of methylmercury is absorbed and can cross the blood brain barrier.

Mercury can bind to thiol groups in most proteins including enzymes, glutathione and structural proteins and can produce its toxic effects by denaturing biological proteins, inhibiting enzymes and interrupting membrane transport. Mercury can accumulate in the kidneys and liver, however, the majority of its toxicity effects and its primary target is the central nervous system, where it can impede the uptake and release of certain neurotransmitters and contribute to demyelination, autonomic dysfunction and abnormal neuronal migration. Symptoms of toxicity include peripheral neuropathy, cerebellar ataxia, akathisia, spasticity, memory loss, dementia, constricted vision, impaired hearing and depression.

Mercury generates highly toxic hydroxyl radicals, causing oxidative damage, which may be one of the first detrimental effects of mercury toxicity. Inhibition of Na/K ATPase can result in astrocytic swelling and destruction. It can also interrupt excitatory amino acid pathways in the CNS, leading to neurotoxic accumulation of serotonin, aspartate and glutamate. This may contribute to excitotoxic effects and further oxidative damage. Some studies indicate a potential role for mercury toxicity in the development of Alzheimer’s disease. Experimental studies have found that even the smallest amounts of mercury were able to cause nerve cell changes typically seen in Alzheimer’s disease.

Studies also show that adult exposure to low levels of mercury, such as through high fish consumption may cause observable deficits in neurobehavioural performance measures.

In a New Zealand study examining a group of 465 patients diagnosed with chronic mercury toxicity, 32.3% exhibited severe fatigue, 88.8% memory loss and 27.5% depression. Removal of amalgam mercury fillings combined with appropriate treatment resulted in a significant reduction of symptoms to levels reported by healthy subjects.

**The Developing Brain**

Both the adult and foetal brains are susceptible to the effects of mercury toxicity, although the developing nervous system is more vulnerable to injury than the adult nervous system. Features of prenatal mercury toxicity include reduction in CNS mitotic activity, interference of neuron migration and damage to astrocytes. Methylmercury can
cross not only the blood brain barrier but also the placenta. Most of our knowledge of mercury as neurotoxic and the cause of developmental disorders comes from the results of past accidental exposure to high levels of mercury, namely in Japan and Iraq.\textsuperscript{9, 10} Prenatal poisoning with high levels of methylmercury have been shown to cause mental retardation and cerebral palsy.\textsuperscript{11}

In more recent years, exposure to a mercury containing compound, thimerosal, found in some vaccines, has been strongly linked to increased incidence of autism.\textsuperscript{12, 13} It has been found that autistic children had a higher mercury exposure during pregnancy due to maternal dental amalgam and thimerosal-containing immunoglobulin shots. It was also hypothesized that children with autism have a decreased detoxification capacity due to genetic polymorphism, contributing to the accumulation of mercury and therefore detrimental effects of the metal.\textsuperscript{14}

Studies from New Zealand, the Faroe Islands and the Seychelles have determined that bench mark levels of 4 – 25ppm of mercury in maternal hair may carry a risk to the infant.\textsuperscript{15} It has also been suggested that latent or delayed adverse effects might be emerging at exposure above 10-12ppm as children mature and that the association between prenatal mercury exposure and child development may be complex and require further study.\textsuperscript{16}

Hair mineral analysis, and relevant chelation, may prove an important and valuable avenue of therapy for pre-conception health.

**Chelation, detoxification and excretion of mercury**

Once absorbed, mercury has a low excretion rate. When released from cells, mercury complexes with glutathione in the liver and secreted in bile as a cysteine-mercury or glutathione-mercury complex. Reduced glutathione levels have been shown to be lowered in several cells on exposure to all forms of mercury. Mercury also generates highly toxic hydroxyl radicals, further depleting stores of the antioxidant glutathione.\textsuperscript{17} Successful methods of increasing glutathione include supplementation of glutathione precursor nutrients, particularly glutamine and cysteine.

Cysteine is also a key element of metallothionein. Metallothioneins scavenge sulfhydryl reactive metals, including mercury and cadmium. Cysteine therefore is an important nutrient to assist both the production of glutathione and metallothionein and protect against the effects of heavy metal exposure. High doses of cysteine have been associated with increased transport of mercury-cysteine complexes across the BBB, into the brain. It is therefore suggested that supplementing low doses of cysteine, together with other amino acids is more beneficial. Therefore, combining a chelating and detoxification product with whey protein may provide a comprehensive treatment regime to support mercury detoxification.\textsuperscript{18}

Selenium may protect against mercury toxicity, both by binding mercury and through antioxidant properties to eliminate reactive oxygen species induced by mercury.
References