

GENERAL CONCEPTS

Across all demographic strata in industrialized nations, element deficiencies are recognized as being involved in the pathogenesis of many health conditions, including heart disease, hypertension³ and cancer.⁴ ¹² Certain populations demonstrate a relatively high incidence of elemental deficiencies, including pregnant women,¹³⁻¹⁵ children and adolescents,^{16,17-19} the elderly,²⁰ and those who are immunocompromised.²¹⁻²³ However, in the United States the recommended dietary allowance (RDA) (currently designated DRI*) is not met by diet alone in most population groups for magnesium, calcium and potassium.^{24, 25} Element deficiencies during the perinatal period can contribute to behavioral, immunological and biochemical abnormalities that can last in adulthood.²⁶ Essential element deficiencies during embryonic development can cause mental retardation²⁷ and may contribute to the severity of genetic abnormalities.²⁶ The causes of such widespread element

deficiencies vary with each element, but may be categorized as those which decrease supply and those that increase demand. Figure 3.1 illustrates how the overall contributions of various factors combine to dictate the progression toward iron deficiency or repletion. Supply is lowered by depleted soil,²⁸ toxic element interference,²⁹ poor food choices²⁵ and compromised digestion, including iatrogenic-induced gastric acid reduction.^{30, 31} Most of these factors affecting demand also generally impact other essential elements.

The term “mineral” that has been used in many discussions of the nutritionally essential elements actually refers to natural compounds formed through geological processes. Since the nutritionally essential chemical elements covered in this chapter occur in the human body in forms quite distinct from those found in the crust of the earth, the more general term “element” will be used. The same may be said of the toxic elements. Thus, the terminology “nutrient and toxic elements” in clinical assessments of blood, urine, hair

* While the term dietary reference intake (DRI) has been adopted for discussions of intake levels, the older term, recommended dietary allowance (RDA), will be used instead because of its continued widespread use by clinicians and laypeople. The term tolerable upper limit (UL) is used when discussing current recommended upper intake levels.

TABLE 3-2 — SUMMARY OF TOXIC METAL ASSESSMENTS AND PROTECTIVE MEASURES

Metal	Toxicity Symptoms	Body Burden Assessment	Biochemical Marker	Protective Measures ^{1, 2}	Mechanism
Aluminum	Abnormal speech, myoclonic jerks, osteomalacia, progressive encephalopathy, Alzheimer's disease, Parkinson's disease	Whole blood, serum, hair, urine	Total urinary porphyrins	Phosphorous Calcium	Lowers intestinal absorption
Arsenic	Peripheral arteriosclerosis (“blackfoot disease”), “rice-water” stools, proteinuria, hyperkeratosis, “milk and roses” hyperpigmentation, garlic breath odor, stomatitis	Whole blood, urine, hair, nails	Urinary uroporphyrin, coproporphyrin I/III, hexacarboxyporphyrin	<i>Emblica officinalis</i> , selenium, NAC, glutathione	Competes for binding sites
Cadmium	Femoral pain, lumbago, osteopenia, renal dysfunction, hypertension, vascular disease	Whole blood	Coproporphyrin I	Zinc, antioxidants	Competes for binding sites
Lead	Microcytic hypochromic anemia, renal dysfunction, hypertension, anorexia, muscle discomfort, constipation, metallic taste, low IQ (children)	Whole blood, urine, hair	Urinary coproporphyrins, (sometimes I), zinc protoporphyrin, ALA	Calcium, ascorbate, alpha lipoic acid, iron adequacy	Lowers intestinal absorption
Mercury	Mental symptoms (erethism, insomnia, fatigue, poor short-term memory), tremor, stomatitis, gingivitis, GI and renal disturbances, decreased immunity	Whole blood, urine, hair	Urinary pentacarboxyporphyrin, coproporphyrin III, precoproporphyrin	Selenium, NAC	Protects against cellular toxic effects