INTRODUCTION

Trace elements often have a bimodal or even a trimodal effect. Severe deficiency of certain trace metals can result in death or severe crippling of an animal or in birth defects of the newborn. The next level of intake is the nutritional level where chronic deficiency over a lifetime may cause major diseases such as cancer and heart disease. The next level of intake is the toxic level which may result in severe crippling or death of the animal. The primary area of concern in this chapter will be the nutritional intake level and the deficiency level and their relationship to animal or human disease. The trace elements described in this chapter are: chromium; cobalt; copper; fluorine; iodine; iron; manganese; molybdenum; nickel; silicon; selenium; tin; vanadium; and zinc.

CHROMIUM

Glucose Metabolism

The most prominent feature of chromium deficiency in rats (Schwarz and Mertz, 1961) and other animals (Davidson and Blackwell, 1968) is impairment of glucose tolerance. After a few weeks on a torula yeast diet, glucose removal rates declined almost 50%. The decline can be reversed quickly by one oral dose of 20 μg or an intravenous dose of 0.25 μg/100 g body weight of trivalent chromium (Mertz et al., 1961; Mertz et al., 1965). Chromium deficiency in a more severe degree leads to a syndrome like mild diabetes mellitus, including glycosuria and fasting hyperglycemia.
Since 1966, evidence for a human nutritional requirement for chromium has been looked at in patients with an impairment of glucose tolerance including "maturity onset" diabetes, middle-aged and elderly subjects with impaired glucose tolerance, and infants with protein-calorie malnutrition. In the first therapeutic trial (Glinsmann and Mertz, 1966) four of six maturity onset diabetics improved after administration of 180 to 1000 µg of chromium as CrCl₃ for periods of 7 to 13 weeks. In another study (Sherman et al., 1968) treated 10 adult diabetics with 150 µg daily for 16 weeks and observed no improvement of glucose tolerance. In another experiment 4 of 12 diabetics treated with 1 mg chromium each day for six months had improved glucose tolerance, but 16 treated for a shorter time period with 150 µg chromium daily did not improve (Schroeder, 1968). In 10 elderly patients (over 70) treated up to four months with 150 µg of chromium, glucose tolerance was restored to normal in four (Levin et al., 1968). Half of a group of middle-aged subjects treated with 150 µg daily for 6 months had a marked improvement (Hopkins and Price, 1968). Benjanuvatra and Bennien (1975) have measured hair chromium analyses for 28 subjects with adult-onset diabetes mellitus and 28 nondiabetic control subjects from Bangkok, Thailand. Hair chromium concentrations were significantly lower in the diabetic than in the control group.

Davidson and Burt (1973) have suggested that the "diabetogenic" effect of pregnancy is related to a significantly lower concentration of plasma chromium of pregnant women. The "diabetogenic" effect of pregnancy is characterized by impairment of peripheral glucose metabolism, decreased glucose tolerance, and an exaggerated insulin excretion in response to glucose challenge.

Morgan (1972) has measured liver chromium content from post-mortem examinations in elderly patients with diabetes, with ischemic heart disease, with hypertensive cardiovascular disease, and without disease. The results in µg/g were controls 12.7; arteriosclerotic 9.96; hypertensive 10.2; and diabetics 8.59. The diabetic group was significantly decreased in regard to the control population (p = 0.05). Serum chromium has been related (Newman et al., 1978) to angiographically determined coronary artery disease. Human aortas sampled from 17 adults where there is little advanced atheromatous plaque formation contain higher concentrations of chromium than do aortas from 15 adults in which atheromatosis is prevalent.

In a study on a patient with long term total parenteral nutrition Jeejeebhoy et al., (1977) have reported that a chromium deficiency in man, causes: (1) glucose intolerance, (2) inability to utilize glucose for energy, (3) neuropathy with normal insulin concentrations, (4) high free fatty acid concentrations and low respiratory quotient and, (5) abnormalities of nitrogen metabolism.