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Including Recommended Dietary Intakes

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# CHROMIUM

## BACKGROUND

Chromium is involved in potentiating the action of insulin in vivo and in vitro (Mertz 1969, 1993, Mertz et al 1961) and several studies have shown beneficial effects of chromium on circulating glucose, insulin and lipids in humans, although not all studies were positive. These studies have been reviewed by Anderson (1997), Mertz (1993), Offenbacher et al (1997) and Stoecker (1996).

In man, chromium accumulates in liver, spleen, soft tissue and bone (Lim et al 1983). Research on chromium metabolism is limited by the lack of a good measure for establishing deficiency states in man. However, data from metabolic balance and urinary excretion studies suggest that only 0.4–2.5% of chromium is absorbed, the actual amount being determined by the environment of the gastrointestinal tract and ligands provided by foods (Clydesdale 1998).

Chromium is widely distributed through the food supply but the content within a given type of food can vary widely because of geochemical factors (Welch & Carey 1975).

Most ingested chromium is excreted unabsorbed in the faeces (Mertz 1969, Offenbacher et al 1986) whilst absorbed chromium is excreted mainly in the urine (Anderson et al 1983). Vitamin C appears to increase absorption (Davis et al 1995, Offenbacher 1994, Seaborn & Stoecker 1990). Animal experiments have shown that high phytate levels can reduce absorption (Chen et al 1973) although lower levels appear to have no effect (Keim et al 1987). There are no systematic data for humans. Animal experiments have shown that long-term consumption of some medicines can affect chromium absorption through affecting stomach acidity or gastrointestinal prostaglandins (Davis et al 1995, Kamath et al 1997). It has also been suggested that absorption may increase with chronic resistive exercise (Rubin et al 1998).

In man, diets very high in simple sugars (35% energy) have been shown to increase urinary chromium excretion (Kozlovsky et al 1986) which may be related to the insulinogenic actions of carbohydrates (Anderson et al 1990). Urinary excretion also appears to be increased by aerobic exercise (Anderson et al 1982, 1984, 1988).

Chromium deficiency is relatively rare but has been reported in patients on total parenteral nutrition (Brown et al 1986, Freund et al 1979, Jeejeebhoy et al 1977). It has been hypothesised that poor chromium status contributes to the incidence of impaired glucose tolerance and type II diabetes which has led to interest in a potential role for chromium supplements in type II diabetes. One Chinese study involved 180 subjects with type II diabetes being given placebo, 200 µg or 1,000 µg chromium as chromium picolinate for 4 months. The subjects showed decreased fasting and 2-hour insulins at two months at both supplement levels, with glycosylated haemoglobin and fasting and 2-hour glucose concentrations being lower in the higher supplement group only. The reduced glucose and insulin concentrations were maintained to 4 months and glycosylated haemoglobin in both dosage groups was also reduced (Anderson et al 1997).

Approaches to the estimation of chromium requirements have included balance studies (Bunker et al 1984, Offenbacher et al 1986), urinary chromium excretion (Anderson et al 1982, 1983, 1991, Anderson & Kozlovsky 1985, Paschal et al 1998), plasma chromium concentration (Anderson 1987, Veillon 1989) and blood glucose and insulin concentrations (Anderson et al 1991). However, none of these approaches has been found to be satisfactory (FNB:IOM 2001).

| 1 mmol chromium = 52 mg chromium |
|---|

## RECOMMENDATIONS BY LIFE STAGE AND GENDER

| ***Infants*** | **AI** | **Chromium** |
|---|---|---|
| **0–6 months** | **0.2 µg/day** | |
| **7–12 months** | **5.5 µg/day** | |

***Rationale*:** The AI for 0–6 months was calculated by multiplying the average intake of breast milk (0.78 L/day) by the average concentration of chromium in breast milk, and rounding. The figure for breast milk used was 0.25 µg/L based on the studies of Anderson et al (1993), Casey & Hambidge (1984), Casey et al (1985), Engelhardt et al (1990), and Mohamedshah et al (1998). The AI for 7–12 months was derived from consideration of the overall energy intake of infants of this age (3,530 kJ), the estimated contribution from breast milk (0.6 L/day at 0.25 µg/L = 0.15 µg chromium and 1,880 kJ), plus chromium from the amount of complementary foods needed to provide the additional 1,670 kJ using a chromium concentration of 3.2 µg/1,000 kJ (Anderson et al 1992). This gives a total chromium of 5.5 µg/day (0.15 µg from milk + 5.36 µg from foods).

| ***Children & adolescents*** | **AI** | **Chromium** |
|---|---|---|
| **All** | | |
| **1–3 yr** | **11 µg/day** | |
| **4–8 yr** | **15 µg/day** | |
| **Boys** | | |
| **9–13 yr** | **25 µg/day** | |
| **14–18 yr** | **35 µg/day** | |
| **Girls** | | |
| **9–13 yr** | **21 µg/day** | |
| **14–18 yr** | **25 µg/day** | |

***Rationale*:** As there are limited data to set an EAR, AIs were set for children. In the absence of any data, the children's AIs were derived from the adult AIs on a body weight basis.

| ***Adults*** | **AI** | **Chromium** |
|---|---|---|
| **Men** | | |
| **19–30 yr** | **35 µg/day** | |
| **31–50 yr** | **35 µg/day** | |
| **51–70 yr** | **35 µg/day** | |
| **>70 yr** | **35 µg/day** | |
| **Women** | | |
| **19–30 yr** | **25 µg/day** | |
| **31–50 yr** | **25 µg/day** | |
| **51–70 yr** | **25 µg/day** | |
| **>70 yr** | **25 µg/day** | |

***Rationale*:** As there are limited data to set an EAR, an AI was set for adults. As there are no national intake data or food composition data available either for Australia or New Zealand for chromium, data from the FNB:IOM review (2001) were used to derive the AIs. The US estimates were based on analytical studies of 22 well-balanced adult diets designed by US nutritionists (Anderson et al 1992).

These studies gave an average chromium concentration of 3.21 µg/1,000 kJ food (range 2–5.7 µg/1,000 kJ). As there is some evidence that dietary intake data may have a tendency to underestimate actual intake (Mertz et al 1991), the average concentration in food was applied to the highest median intakes of energy for a given age group within the adult men or women, using intake data from the Australian (ABS 1998) and New Zealand (MOH 1999) National Nutrition Surveys.

| ***Pregnancy*** | **AI** | **Chromium** |
|---|---|---|
| **14–18 yr** | **30 µg/day** | |
| **19–30 yr** | **30 µg/day** | |
| **31–50 yr** | **30 µg/day** | |

***Rationale*:** Because of lack of data to establish the additional needs in pregnancy, the AI was extrapolated from the AIs for adolescent girls and women on the basis of an average weight gain of 16 kg in pregnancy for pregnancies with good outcomes (Carmichael et al 1997).

| ***Lactation*** | **AI** | **Chromium** |
|---|---|---|
| **14–18 yr** | **45 µg/day** | |
| **19–30 yr** | **45 µg/day** | |
| **31–50 yr** | **45 µg/day** | |

***Rationale*:** The AI for lactation was estimated from the intake necessary to replace chromium secreted in milk plus the AI for women. The amount needed to be absorbed is 0.252 µg/L x 0.78 L/day (200 ng/day). With absorption at 1%, an additional 20 µg/day is needed.

## UPPER LEVEL OF INTAKE - CHROMIUM

**The ULs for chromium are unknown as there are insufficient data.**

A number of potential adverse effects of high chromium intakes in relation to renal failure, genotoxicity, carcinogenicity, hepatic dysfunction and reproductive function have been seen either in animal studies or in humans (Al-Hamood et al 1998, Bagchi et al 1997, Bataineh et al 1997, Cerulli et al 1988, Elbetieha & Al-Hamood 1997, Fristedt et al 1965, Kaufman et al 1970, Kusiak et al 1995, Loubieres et al 1999, Speetjens et al 1999, Stearns et al 1995, Wasser et al 1997). However, adequate human data on trivalent chromium are limited.

No adverse side effects were reported in a number of supplementation trials in which subjects received up to 1 mg chromium/day, mostly as picolinate, for several months (Flodin 1990, Hathcock 1997). These trials, however, were mainly studies of efficacy and not designed to find potential toxic effects. The limited data from all studies on subchronic, chronic and reproductive toxicity on soluble trivalent chromium salts do not give clear information on the dose-response relationship. Therefore, ULs cannot be derived.

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