

INGESTION OF CHROMIUM(VI) IN DRINKING WATER BY HUMAN VOLUNTEERS: ABSORPTION, DISTRIBUTION, AND EXCRETION OF SINGLE AND REPEATED DOSES

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This study examines the magnitude of hexavalent chromium [Cr(VI)] absorption, distribution, and excretion following oral exposure to 5 and 10 mg Cr(VI)/L in drinking water administered as a single bolus dose (0.5 L swallowed in 2 min) or for 3 d at a dosage of 1 L/d (3 doses of 0.33 L each day, at 6-h intervals). Adult male volunteers ingested deionized water containing various concentrations of potassium chromate, and samples of urine, plasma, and red blood cells (RBCs) were collected and analyzed for total chromium throughout the studies. In the bolus dose studies, a fairly consistent pattern of urinary chromium excretion was observed, with an average half life of about 39 h. However, 4-d total urinary chromium excretion and peak concentrations in urine and blood varied considerably among the 5 volunteers. Studies of repeated exposure to smaller volumes ingested at a more gradual rate (i.e., 0.33 L over 5–15 min) showed similar urinary chromium excretion patterns but generally lower chromium uptake/excretion. Given that sustained elevations in RBC chromium levels provide a specific indication of chromium absorption in the hexavalent state, these data suggest that virtually all (>99.7%) of the ingested Cr(VI) at 5 and 10 mg Cr(VI)/L was reduced to Cr(III) before entering the bloodstream. The interindividual differences in total chromium uptake and excretion are plausibly explained by ingestion of appreciable doses on an empty stomach, which likely results in the formation of well-absorbed Cr(III) organic complexes in gastrointestinal tissues and possibly the blood. The lack of any clinical indications of toxicity in the volunteers and the patterns of blood uptake and urinary excretion of chromium are consistent with a predominant uptake of Cr(III) organic complexes [derived from Cr(VI)] that are excreted more slowly than inorganic forms of Cr(III). Therefore, it appears that the endogenous reducing agents within the upper gastrointestinal tract and the blood provide sufficient reducing potential to prevent any substantial systemic uptake of Cr(VI) following drinking-water exposures at 5–10 mg Cr(VI)/L. Based on these data, the chemical environment in the gastrointestinal tract and the blood is effective even under relative fasting conditions in reducing Cr(VI) to one or more forms of Cr(III).