was greater in the exposed animals than in the controls (23 µg/g).

5.4 Reproductive and developmental toxicity

Sperm morphology and motility analyses, testis and epididymis weight determination, and estrous cycle characterization were performed in rats and mice as part of a subchronic dietary study (Hébert et al., 1993). No significant differences from control values were found for any of the following reproductive parameters: testis, epididymis, and cauda epididymis weights, spermatid count, spermatid number per testis or per gram testis, spermatozoal motility and concentration, estrous cycle length, or relative length of time spent in the various estrous stages. A reproductive NOAEL of 4000 mg of copper sulfate pentahydrate per kg of diet (66 and 68 mg of copper per kg of body weight per day for male and female rats, respectively) was established for these parameters in this study.

There is some evidence that copper is a developmental toxicant. Embryotoxicity and teratogenicity were reported in hamsters when dams were injected intraperitoneally with copper citrate (DiCarlo, 1980) or intravenously with copper sulfate or copper citrate (Ferm & Hanlon, 1974). Such effects were noted at single injection doses as low as 2.13 mg of copper per kg of body weight for copper sulfate and 0.25-1.5 mg of copper per kg of body weight for copper citrate. When mice (7-22 females per group) were fed diets supplemented with 0, 500, 1000, 1500, 2000, 3000, or 4000 mg of copper sulfate per kg of diet for 1 month, fetal mortality and decreased litter size were observed in the 2000-4000 mg/kg of diet groups. Various skeletal and soft-tissue malformations were seen in 2-8% of the surviving fetuses from the two highest dose groups (Lecyk, 1980). The low concentrations of supplemental copper (500 and 1000 mg/kg of diet) had a beneficial effect on development.

5.5 Mutagenicity and related end-points

Copper sulfate has been reported to induce reverse mutations in *Escherichia coli* (Demerec et al., 1951) but not in TA98, TA100, TA1535, or TA1537 strains of *Salmonella typhimurium* in the presence or absence of microsomal activation (Moriya et al., 1983; Wong, 1988). Negative microbial mutation results have been reported for copper sulfate and copper chloride in *Saccharomyces cerevisiae* (Singh, 1983) and *Bacillus subtilis* (Nishioka, 1975; Kanematsu et al., 1980; Matsui, 1980).

Copper chloride or copper acetate concentrations of 20-150 mmol/litre induced errors in viral DNA synthesis from poly(c) templates (Sirover & Loeb, 1976), and copper sulfate concentrations of 1 mmol/litre, but not 0.03-0.3 mmol/litre, caused DNA strand breakage in rat hepatocytes (Sina et al., 1983). Results from *in vitro* tests are not transferable to the *in vivo* situation, where copper ions are generally bound to protein or amino acid ligands.

Male Wistar rats were implanted with subcutaneous osmotic pumps that continuously administered saline, copper(II) chloride, the copper chelate cupric nitrilotriacetate (Cu-NTA), or NTA for 3 or 5 days. Copper was delivered at a rate of 4 mg/kg of body weight per day, which maintained serum copper levels 30-70% higher than in the untreated controls for copper(II) chloride or 100-120% higher than in controls for Cu-NTA. Hepatic and renal DNA levels of 8-hydroxy guanosine, a deoxyguanosine oxidation product associated with mutagenesis and carcinogenesis, were significantly elevated in the copper-exposed animals (Toyokuni & Sagripanti, 1994). Chromosomal aberrations and micronuclei were observed in the bone marrow of inbred Swiss mice exposed to copper (from copper sulfate) concentrations of 0, 1.3, 2.6, or 5.1 mg/kg of body weight by intraperitoneal or subcutaneous injection (Bhunya & Pati, 1987).

5.6 Carcinogenicity

Copper and its salts do not appear to be animal carcinogens based on limited long-term exposure data. In one study, two strains of mice (18 per sex per group per strain) were exposed to copper

hydroxyquinoline (181 mg of copper per kg of body weight per day) by gelatin capsule until they were 28 days old, whereupon the compound was administered for an additional 50 weeks in the feed at 2800 mg/kg of feed (506 mg of copper per kg of body weight per day). No significant increases in tumour incidence were observed in either sex or either strain (BRL, 1968).

6. EFFECTS ON HUMANS

6.1 Acute exposure

The acute lethal dose for adults lies between 4 and 400 mg of copper(II) ion per kg of body weight, based on data from accidental ingestion and suicide cases (Chuttani et al., 1965; Jantsch et al., 1984; Agarwal et al., 1993). Individuals ingesting large doses of copper present with gastrointestinal bleeding, haematuria, intravascular haemolysis, methaemoglobinaemia, hepatocellular toxicity, acute renal failure, and oliguria (Agarwal et al., 1993).

At lower doses, copper ions can cause symptoms typical of food poisoning (headache, nausea, vomiting, diarrhoea). Records of published studies of gastrointestinal illness induced by copper from contaminated beverages plus public health department reports for 68 incidents indicate an acute onset of symptoms (Low et al., 1996). Symptoms generally appear after 15-60 minutes of exposure; nausea and vomiting are more common than diarrhoea. Copper concentrations were not available for all incidents. Among 24 outbreaks with quantitative data, the lowest copper concentrations were 3.5 and 3.8 mg/litre; background information on the samples analysed and analytical methods was limited. In 6 of the 24 cases (25%), the copper concentration was less than 10 mg/litre (Low et al., 1996). Data on the amount of beverage consumed were not available for any of these incidents.

An analysis of data from the US Centers for Disease Control regarding 155 reported cases of copper intoxication from drinking-water sources during the years 1977-1982 and 1991-1994 indicated that reported levels of copper in drinking-water were in the range of 4.0-156 mg/litre (US EPA, 1987; CDC, 1993, 1996).

6.2 Short-term exposure

Recurrent morning episodes of nausea, vomiting, and abdominal pain were reported by three of four family members exposed for over a year to drinking-water containing elevated levels of copper. Symptoms were associated with early-morning consumption of water, juice, or coffee. Copper concentrations measured on three occasions during the period covered by the complaint ranged from 2.8 to 7.8 mg/litre; only the 7.8 mg/litre sample was collected in the early morning (Spitalny et al., 1984). All samples were collected before the complaint was registered with the Department of Health. The median copper concentration for a series of samples collected after the complaint was 3.1 mg/litre, compared with a median value of 1.6 mg/litre for a series of samples collected from another house on the same service line but located closer to the beginning of the line.

Recurrent episodes of vomiting, diarrhoea, and abdominal cramps were reported among individuals consuming water from homes with water that exceeded the US EPA maximum contaminant level goal of 1.3 mg/litre in four case-studies (Knobeloch et al., 1994). Data on water use practices and symptoms were collected using a questionnaire. The incidence of symptoms was positively correlated with the copper concentration in the water samples, ingestion of first-draw water, and water intake. There was a negative correlation with consumer age and use of bottled water. The presence of various confounding factors suggests discretion when drawing conclusions based on the data presented.

A 26-year-old male presented with symptoms of cirrhosis, liver failure, and Wilson disease (Kayser-Fleischer rings) after more than 2 years of self-prescribed use of copper supplements (O'Donohue et al., 1993). The patient ingested 30 mg of supplemental copper per day for 2 years

and 60 mg/day for a poorly defined period of up to a year. Liver damage was extensive, and a transplant was required. The diseased liver had an average copper concentration of 3230 µg/g dry weight (normal 20-50 µg/g); tissue histopathology was similar to that seen in Indian childhood cirrhosis and Wilson disease. The patient's family medical history and evaluation of his parents and sisters for copper excretion suggested that he did not carry the Wilson disease gene. Liver damage apparently resulted from the prolonged daily exposure to over 10 times the US estimated safe and adequate daily intake for copper.

6.3 Long-term exposure

Long-term intake of copper in the diet in the range of 1.5-3 mg/day has no apparent adverse effects. Daily intake of copper below this range can lead to anaemia, neutropenia, and bone demineralization in malnourished children (NAS, 1989). Adults are more resistant than children to the symptoms of a copper deficiency. No studies of adverse effects of long-term exposure of humans to copper at concentrations greater than those that occur in the diet were identified.

7. PROVISIONAL GUIDELINE VALUE

The IPCS Environmental Health Criteria monograph for copper (WHO) concluded that:

> The upper limit of the AROI [acceptable range of oral intake] in adults is uncertain but it is most likely in the range of several but not many mg per day in adults (several meaning more than 2 or 3 mg/day). This evaluation is based solely on studies of gastrointestinal effects of copper-contaminated drinking-water. A more specific value for the upper AROI could not be confirmed for any segment of the general population....The available data on toxicity in animals were considered unhelpful in establishing the upper limit of the AROI, due to uncertainty about an appropriate model for humans.

A copper level of 2 mg/litre in drinking-water will be protective of adverse effects of copper and provides an adequate margin of safety. Owing to limitations of the epidemiological and clinical studies conducted to date, it is not possible to establish a clear effect level with any precision. Thus, it is recommended that this guideline value for copper of 2 mg/litre remain provisional as a result of uncertainties in the dose-response relationship between copper in drinking-water and acute gastrointestinal effects in humans. It is also noteworthy that copper is an essential element.

It is stressed that the outcome of ongoing epidemiological studies in Chile, Sweden, and the USA may, upon publication, shed some light on more accurate quantification of effect levels for copper-induced toxicity in humans, including sensitive subpopulations.

Staining of laundry and sanitary ware occurs at copper concentrations above 1 mg/litre. At levels above 5 mg/litre, copper also imparts a colour and an undesirable bitter taste to water.

8. REFERENCES

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Copper

Copper is both an essential nutrient and a drinking-water contaminant. It has many commercial uses. It is used to make pipes, valves and fittings and is present in alloys and coatings. Copper sulfate pentahydrate is sometimes added to surface water for the control of algae. Copper concentrations in drinking-water vary widely, with the primary source most often being the corrosion of interior copper plumbing. Levels in running or fully flushed water tend to be low, whereas those in standing or partially flushed water samples are more variable and can be substantially higher (frequently >1 mg/litre). Copper concentrations in treated water often increase during distribution, especially in systems with an acid pH or high-carbonate waters with an alkaline pH. Food and water are the primary sources of copper exposure in developed countries. Consumption of standing or partially flushed water from a distribution system that includes copper pipes or fittings can considerably increase total daily copper exposure, especially for infants fed formula reconstituted with tap water.

Provisional guideline value	2000 µg/litre
Occurrence	Concentrations in drinking-water range from 0.005 to >30 mg/litre
Basis of guideline derivation	To be protective against adverse effects of copper and provide an adequate margin of safety in populations with normal copper homeostasis
Limit of detection	0.02–0.1 µg/litre by inductively coupled plasma/mass spectrometry; 0.3 µg/litre by inductively coupled plasma/optical emission spectroscopy; 0.5 µg/litre by flame atomic absorption spectrometry
Treatment achievability	Copper is not removed by conventional treatment processes. However, copper is not normally a raw water contaminant.
Additional comments	For adults with normal copper homeostasis, the guideline value should permit consumption of 2 or 3 litres of water per day, use of a nutritional supplement and copper from foods without exceeding the recommended dietary upper limit of 10 mg/day or eliciting an adverse gastrointestinal response. Owing to limitations of the available data for sensitive populations, it is not possible to establish a clear effect level with any precision. Thus, it is recommended that the guideline value for copper remain provisional. Staining of laundry and sanitary ware occurs at copper concentrations above 1 mg/litre. At levels above 2.5 mg/litre, copper imparts an undesirable bitter taste to water; at higher levels, the colour of water is also impacted.

Toxicological Review

IPCS concluded that the upper limit of the acceptable range of oral intake in adults is uncertain but is most likely in the range of several (more than 2 or 3) but not many milligrams per day in adults. This evaluation was based solely on studies of gastrointestinal effects of copper-contaminated drinking-water. However, the data on the gastrointestinal effects of copper must be used with caution, since the effects observed are influenced by temporal aspects of exposure and the concentration of ingested copper to a greater extent than the total mass or dose ingested in a 24-h period. Recent studies have delineated the threshold for the effects of copper in drinking-water on the gastrointestinal tract, but there is still uncertainty regarding the long-term effects of copper on sensitive populations, such as carriers of the gene for Wilson's disease and other metabolic disorders of copper homeostasis.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of copper greater than 1.5 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards retained this value as a maximum allowable or permissible concentration. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 1.0 mg/litre was established for copper, based on its laundry and other staining properties. The 1993 Guidelines derived a provisional health-based guideline value of 2 mg/litre for copper from the provisional maximum tolerable daily intake proposed by JECFA, based on a rather old study in dogs that did not take into account differences in copper metabolism between infants and adults. The guideline value was considered provisional because of the uncertainties regarding copper toxicity in humans. This guideline value was retained in the addendum to the Guidelines published in 1998 and remained provisional as a result of uncertainties in the dose–response relationship between copper in drinking-water and acute gastrointestinal effects in humans. It was stressed that the outcome of epidemiological studies in progress in Chile, Sweden and the USA may permit more accurate quantification of effect levels for copper-induced toxicity in humans, including sensitive subpopulations. Copper can also give rise to taste problems at concentrations above 5 mg/litre and can stain laundry and sanitary ware at concentrations above 1 mg/litre.

Primary Reference

IPCS (1998) *Copper*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 200).