Fact Sheet Date: June 1998

NEW YORK STATE
- HUMAN HEALTH FACT SHEET -

Ambient Water Quality Value for Protection of Sources of Potable Water

SUBSTANCE: Zinc **CAS REGISTRY NUMBER:** Not Applicable

AMBIENT WATER QUALITY VALUE: 2,000 ug/L

BASIS: Non-oncogenic

INTRODUCTION

The ambient water quality value applies to the water column and is designed to protect humans from the effects of contaminants in sources of drinking water; it is referred to as a Health (Water Source) or H(WS) value.

Regulations (6 NYCRR 702.2) require that the water quality value be based on the procedures in sections 702.3 through 702.7. Potential water quality values are derived below, and the value of 2,000 ug/L selected for zinc as described under "Selection of Value."

Zinc is an essential nutrient in humans, necessary for both metabolism and in cell growth and division (ATSDR, 1994). Recommended dietary allowances (RDAs) are 15 mg/day for men and 12 mg/day for women, with higher values recommended for nursing and pregnant women.

PRINCIPAL ORGANIC CONTAMINANT CLASSES AND SPECIFIC MCL

A. Discussion

Zinc has a Specific MCL for New York State as defined in 700.1 of 5,000 ug/L. Zinc is not in a principal organic contaminant class as defined in 700.1.

The U.S. Environmental Protection Agency has established a secondary maximum contaminant level for zinc of 5,000 ug/L.

B. Derivation of Water Quality Value

Because the Specific MCL is based on aesthetic considerations, it is not a basis for a H(WS) value.

ONCOGENIC EFFECTS

U.S. EPA (1997) classifies zinc as D: Not Classifiable as to its carcinogenicity. Review of findings reported in ATSDR (1994) revealed no useable human or laboratory animal data that clearly indicate that zinc is oncogenic via any route of exposure. An ambient water quality value based on oncogenic effects could not be derived given the lack of data.

NON-ONCOGENIC EFFECTS

A. Data

Normal fetal growth and development requires zinc; zinc deficiency may cause fetal damage (ATSDR, 1994). Adverse developmental effects in humans due to exposure to zinc (10.6 mg zinc/kg/day) is suggested by a study by Kumar (1976) but information to assess its significance is lacking. McMichael et al. (1994) in a case-control study of neural tube defects concluded that women with very high serum zinc levels may have an increased risk of fetuses with this condition. The authors reported that "for both unmatched and adjusted matched analyses, mean maternal serum zinc concentration was higher in cases than controls (P = 0.02 and P = 0.03, respectively)."

Ingestion of 0.71 mg zinc/kg/day by humans for 12 weeks resulted in reduced levels of high-density lipoprotein (HDL) cholesterol, but not serum cholesterol, triglyceride or low-density lipoprotein (LDL) cholesterol (Black et al., 1988). ATSDR (1994) believes that 50 mg/day is the threshold LOAEL for zinc.

ATSDR (1994) derived a minimal risk level (MRL) of 0.3 mg/kg/day for intermediate-duration oral exposure to zinc; due to the lack of long-term studies, ATSDR also adopted this level as the chronic MRL. The basis for this MRL is hematological effects in women who consumed daily supplements of 50 mg zinc as zinc gluconate for 10 weeks (Yadrick et al., 1989). Specifically, the authors found a significant reduction in erythrocyte superoxide dismutase activity (47% decrease), hematocrit, and serum ferritin compared to pretreatment levels. Male volunteers who also received 50 mg zinc as zinc gluconate but for 6 weeks exhibited a 15% decrease in erythrocyte superoxide dismutase activity (Fisher et al., 1984).

U.S. EPA (1997) derived an oral reference dose (RfD) for zinc of 0.3 mg/kg/day based on the same study as ATSDR used for their MRL. U.S. EPA applied an uncertainty factor of 3 to the LOAEL of 1.0 mg/kg/day identified from the Yadrick et al. (1989) data. U.S. EPA's derivation is attached as Exhibit I. The derivation of the RfD is comparable to the derivation of an Acceptable Daily Intake (ADI) under New York's regulations (6 NYCRR 702). The Department agrees with EPA's determination and believes that an ADI of 0.3 mg zinc/kg/day is the appropriate basis for an ambient water quality value protective against non-oncogenic effects.

B. Derivation of Water Quality Value

As provided in Part 702, an ambient water quality value of 2,000 ug/L can be derived for zinc from the ADI of 0.3 mg/kg/day [300 ug/kg/day], using a 70 kg adult consuming 2 liters of water per day and allocating 20% of the ADI to drinking water:

$$\text{Water Quality Value} = \frac{(300\ \text{ug/kg/day})(70\ \text{kg})(0.2)}{(2\ \text{L/day})}$$

$$= 2{,}100\ \text{ug/L, rounded to } 2{,}000\ \text{ug/L}$$

CHEMICAL CORRELATION

A value based on chemical correlation is not applicable because data are sufficient to evaluate zinc based on section 702.5 and no information was found to indicate that correlation based on section 702.4 is warranted.

SELECTION OF VALUE

The H(WS) value is designed to protect humans from oncogenic and non-oncogenic effects from contaminants in sources of drinking water. To protect for these effects, regulations (6 NYCRR 702.2(b)) require that the value be the most stringent of the values derived using the procedures found in sections 702.3 through 702.7. The non-oncogenic value of 2,000 ug/L (702.5) is the most stringent value derived by these procedures and is the ambient water quality value for zinc.

REFERENCES

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6 NYCRR (New York State Codes, Rules and Regulations). Water Quality Regulations, Surface Water and Groundwater Classifications and Standards: Title 6 NYCRR, Chapter X, Parts 700-705. Albany, NY: New York State Department of Environmental Conservation.

10 NYCRR (New York State Codes, Rules and Regulations). Public Water Systems: Title 10 NYCRR, Chapter 1, State Sanitary Code, Subpart 5-1. Albany, NY: New York State Department of Health, Bureau of Public Water Supply Protection.

U.S. EPA (Environmental Protection Agency). 1997. On-Line as of June 12. Integrated Risk Information System (IRIS). Cincinnati, OH.

Yadrick, M. K., M.A. Kenney and E. A. Winterfelt. 1989. Iron, copper and zinc status: Response to supplementation with zinc or zinc and iron in adult females. Am. J. Clin. Nutr. 49:145-150. [As cited in ATSDR, 1994 and U.S. EPA, 1997].

New York State Department of Environmental Conservation
Division of Water
SJS
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EXHIBIT 1
ORAL REFERENCE DOSE FOR ZINC

Taken from the On-Line Integrated
Risk Information System (IRIS)
U.S. Environmental Protection Agency
(As of June 12, 1997)

1 - IRIS
NAME - Zinc and Compounds
RN - 7440-66-6

RDO -

- ORAL RFD SUMMARY :

NOTE: This RfD for the soluble salts of zinc supplies adequate zinc to meet the requirements in adolescents and adults over a lifetime without any concurrent physiological impairment. It does not supply the Recommended Daily Allowance (RDA) to those members of the population who have greater requirements for a short, less-than-lifetime duration, for example, infants, pre-adolescent children, or, possibly, lactating women. For short-term requirements in infants, pre-adolescent children, and lactating females, refer to the RDAs (NRC, 1989).

At a Workshop on the "Risk Assessment of Essential Elements" (Herndon, VA; March 10-12, 1992), several nutritionists commented on the derivation of the zinc RfD. The most relevant comment raised the issue of zinc bioavailability from various media. Dr. Harold Sandstead (1992) summarized this viewpoint and suggested the following values for zinc RfDs from various media: zinc supplements - 0.25 mg/kg/day; "omnivores" - 0.7 mg/kg/day; and vegetarians - 1.7 mg/kg/day. The proposed RfD for individuals consuming supplements, which is roughly comparable to soluble salts of zinc, is quite similar to the RfD verified by EPA's RfD/RfC Work Group. This agreement between the nutritionists and the toxicologists gives the EPA greater confidence in the verified RfD.

Critical Effect	Experimental Doses*	UF	MF	RfD
47% Decrease in erythrocyte superoxide dismutase (ESOD) concentration in adult females after 10 weeks of zinc exposure Human Diet Supplement Study Yardick et al., 1989	NOAEL: None LOAEL = 59.72 mg/day (1.0 mg/kg/day)	3	1	3E-1 mg/kg/day

*Conversion Factors: The dose conversion factors were based on a 60-kg reference female body weight. Total dose was derived from estimations from the FDA Total Diet Study for 1982-1986, plus reported supplemental dose. For example, for the Yadrick et al., 1989 study, the dose is 1.0 mg/kg-day based on 50 mg zinc supplement plus 9.72 mg/day zinc from the diet (total of 60), divided by the assumed average body weight of the participants (60 kg).

- ORAL RFD STUDIES :

Yadrick, M.K., M.A. Kenney and E.A. Winterfeldt. 1989. Iron, copper, and zinc status: Response to supplementation with zinc or zinc and iron in adult females. Am. J. Clin. Nutr. 49: 145-150.

The oral RfD is based on a clinical study which investigated the effects of oral zinc supplements on copper and iron balance. This study is supported by several other studies which indicate that zinc supplementation can alter copper balance. The effects on copper and iron biochemistry are considered of concern since long-term iron or copper deficiency could result in significant adverse effects. For example, zinc supplementation therapy with megadoses of up to 5 g/day, as well as smaller amounts of 150 mg/day, taken for 1 to 2 years have produced copper deficiency anemia (Fischer et al., 1984). In addition, several studies have investigated the effects of zinc supplementation on the high-density lipoprotein (HDL) levels of adult males. These have been added as supporting studies because the observed change in HDL values in males may be significant since a sustained decrease in HDL concentrations may be associated with increased risk of coronary artery disease when combined with a parallel increase in low-density lipoprotein (LDL) cholesterol.

A 10-week study of zinc supplementation in 18 healthy women given zinc gluconate supplements twice daily (50 mg zinc/day, or 1.0 mg/kg-day, see below) resulted in a decrease of erythrocyte superoxide dismutase (ESOD) activity (Yadrick et al., 1989). ESOD concentrations declined over the 10-week supplementation period and at 10 weeks

were significantly different ($p<0.05$) from values during the pretreatment period. By 10 weeks, ESOD activity had declined to 53% of pretreatment levels. Change in enzyme activity is considered a better indicator of altered copper status than a measure of metal concentration in tissue or plasma. This has been documented by studies in rats fed copper-deficient or high-zinc diets, in which copper metalloenzyme activity is greater and precedes changes in plasma or tissue levels of copper (L'Abbe and Fischer, 1984a,b). Ceruloplasmin concentrations were not altered. Serum zinc was significantly increased. There was also a significant decline in serum ferritin and hematocrit values at 10 weeks. Such a decrease could pose a significant risk to the iron status of women.

No measurements were made of dietary zinc or copper in this study. However, a level of dietary zinc can be estimated at 9.72 mg/day for females (20- to 30-years old) from the results of the FDA Total Diet Study for 1982-1986 (Pennington et al., 1989). The LOAEL of 1.0 mg/kg-day was calculated from the sum of these dietary estimates and the supplemental zinc dose using an assumed body weight of 60 kg for adult females, as shown in the conversion factor section.

Support for considering the intake of 50 mg/kg-day supplemental zinc as a threshold LOAEL is provided by Fischer et al. (1984) which also suggests that zinc affects copper balance at doses of 0.95 mg/kg-day in males. Healthy men given 25 mg of zinc as gluconate twice daily for a 6-week period displayed a significant decrease ($p <0.05$) in erythrocyte superoxide dismutase (ESOD) activity at the end of 6 weeks exposure. There were no differences between serum copper levels or ceruloplasmin activity in the 13 members of the supplement group compared with controls. Serum zinc levels were significantly increased in the supplement group after 2 weeks.

Prasad et al. (1978) fed a patient with sickle cell anemia supplements of 150 to 200 mg zinc/day for 2 years. The supplement resulted in copper deficiency; serum copper and plasma ceruloplasmin levels were decreased. When copper was administered, the plasma ceruloplasmin levels became normal. In a follow-up study, of 13 patients on zinc therapy (similar treatment levels assumed), 7 patients had ceruloplasmin levels at the lower limit of normal after 24 weeks of dosing.

In a 9-week study, Festa et al. (1985) fed nine male students diets containing 2.6 mg copper/day and 1.8-20.7 mg zinc/day for 1- to 2-week periods. This study indicated that fecal copper excretion was influenced by the amount of zinc in the diet and the length of time it was administered. Typically, after 1-2 weeks at 18.5 mg/day (just 3.5 mg/day higher than the adult RDA), subjects lost significantly more copper in the feces. Plasma copper concentrations were unchanged.

Groups of 9, 13 or 9 healthy white men were administered 0, 50, or 75 mg/kg-day zinc as zinc gluconate, respectively, for 12 weeks (Black et al., 1988). The subjects were given instructions to avoid foods high in calcium, fiber and phytic acid, dietary constituents that have a negative impact on zinc absorption. Subjects were also told to restrict their intake

of zinc-rich foods in order to minimize the variation in daily dietary zinc. Three-day dietary records were collected on a biweekly basis. These records indicated that the dietary zinc intakes of the three treatment groups were 12.5, 14.0, and 9.5 mg/day for the groups receiving 0, 50, and 75 mg/kg-day supplement, respectively. Based on the average body weights for each treatment group, these doses correspond to a total zinc intake of 0.16, 0.85, and 1.10 mg/kg-day.

Biweekly blood samples were collected from all subjects and analyzed for total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, zinc, and copper. Urinary zinc and copper values were also determined. There was a general decline in the mean serum HDL-cholesterol for the 75-mg supplement group between weeks 6 and 12. HDL values for this group were significantly lower than those for the placebo group at weeks 6 and 12 ($p < 0.05$). When the mean HDL-cholesterol level of these subjects was compared to population percentile norms, there was a decline from the 92nd to the 77th percentile (Simko et al., 1984) in 6 weeks, followed by a relative stabilization of HDL values for the remaining 6-week test period. There was also a decline in the HDL values for the 50-mg group between weeks 8 through 12; however, this decline was not significantly different (p 0.05) from that for the controls until the 12th week of treatment. Over the 12-week period the HDL values for the 50-mg group declined from the 90th to the 77th population percentile norms. Serum zinc, copper, total cholesterol, LDL-cholesterol and triglycerides did not appear to be affected by treatment. While it is not absolutely certain that the 50-mg zinc/day supplement represents a clearly biologically significant endpoint, this level, when viewed collectively with other studies investigating effects on HDL-cholesterol, may signify the beginning of the dose-response trend. The significance of this change is unknown in light of an absence of increase in LDLs.

Zinc supplementation (160 mg as zinc sulfate) was found to lower HDL-cholesterol values in 11 healthy men when administered over 5 weeks (Hooper et al., 1980). A control group of eight subjects received a placebo. Fasting cholesterol, HDL-cholesterol, and triglycerides were determined on a weekly basis for 7 weeks and again 11 weeks after the end of supplementation. Dietary zinc levels were not measured; however, in the FDA Total Diet Study, adult males consumed an average of 16.41 mg/day during 1982-1987 (Pennington et al., 1989). Based on a 70-kg average body weight and 16.41 mg/day dietary zinc, the average dietary zinc intake for those receiving a supplement was 2.52 mg/kg-day.

After an initial HDL increase during the first 2 weeks of supplementation, HDL levels were significantly lower than those for the controls during weeks 4 through 7 ($p = 0.002$ to 0.0001). HDL levels returned to normal 11 weeks after supplementation had ended. The 11 subjects of this study had initial mean HDL values below average for their age category (23-35 years old). During the first 7 weeks of monitoring, their HDL percentile values fell from the 36th to the 8th population percentile norm. Percentile standings lower than 10 are associated with cardiovascular risk. Serum cholesterol, LDL-cholesterol, and triglycerides did not change significantly during the study; serum zinc levels increased during the supplementation period. Serum cholesterol values were normal.

A third study of the effects of zinc supplementation was conducted by Chandra (1984) in 11 adult men (ages not given). Zinc sulfate tablets were administered twice daily for a total zinc supplement intake of 300 mg/day. Average dietary zinc during the supplementation period was 10.1 mg/day, based on 24-hour recall data and 11.2 mg/day in the pre-test period. Thus, the daily zinc intake was 4.43 mg/kg-day for a 70-kg male during supplementation. Fasting serum cholesterol, HDL-cholesterol, LDL-cholesterol, and triglycerides were measured biweekly for 6 weeks; a final measurement of these parameters was conducted at 16 weeks. Total lymphocytes, T-lymphocytes, and B-lymphocytes were also measured. Lymphocyte activity was monitored through polymorphonuclear migration response to chemotactic phytohemagglutinin (PHA) stimulation and phagocytosis of opsonized bacteria.

There was a significant decrease in serum HDL values during weeks 4 and 6 ($p<0.1$ and $p<0.01$, respectively) with a return to baseline levels at week 16 (Chandra, 1984). LDL-cholesterol levels were significantly increased ($p<0.05$) at week 6, but there were no significant changes in serum cholesterol and triglycerides. During the 6-week supplement administration period, the HDL percentile values fell from the 43rd to the 6th percentile, as estimated from the population percentile norms for 30- to 35-year-old males (Simko et al., 1984).

There were no significant changes in lymphocyte counts during the period of zinc supplementation, but polymorphonuclear response to PHA stimulation (chemotactic migration) and phagocytosis were impaired (Chandra, 1984). Plasma zinc values increased during the supplement administration.

- ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 3 was used, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient.

- ORAL RFD MODIFYING FACTOR :

MF -- 1.

- ORAL RFD COMMENTS :

Zinc is an essential nutrient with RDA values ranging from 5 to 15 mg/day for different age and sex categories (NRC, 1989). The RDA is an estimate of the zinc needed for growth, development, metabolism and tissue maintenance for over 98% of the healthy American population. For 79% of a 70-year lifetime (55 years), the proposed RfD of 0.3 mg/kg-day supplies adequate zinc to meet these requirements in adolescents and adults without any concurrent physiological impairment. It does not supply the RDA for infants, preadolescent children or, possibly, for lactating women.

The RfD of 0.3 mg/kg-day is expected to be without adverse effects when consumed on a daily basis over an extended period of time. It neither induces a nutritional deficiency in healthy, non-pregnant, adult humans consuming the average American diet nor causes undesirable inhibition of normal lipid transport.

When the three studies monitoring HDL-cholesterol are considered as a group, they show a consistent lowering of HDL-cholesterol levels in response to the addition of zinc to the diet, an effect which is reversed with cessation of the zinc supplementation. The data of Black et al. (1988) indicate that the depressed HDL values can persist for up to 12 weeks. Data are available from all 3 studies at 6 weeks. However, in the Hooper et al. (1980) study, the 6-week data represent HDL status 1 week after supplement administration ended. Additional data will be needed to clarify whether or not this change is significant with longer exposure.

Supplemental zinc does not appear to have the same effect on females that it has on males. Healthy adult females were given supplemental zinc doses of 0, 15, 50 or 100 mg/day zinc as zinc acetate for 60 days (Freeland-Graves et al., 1982). Plasma cholesterol, HDL-cholesterol, and zinc were monitored at biweekly intervals. A transitory decrease in HDL values was noted at 4 weeks, but only in the group receiving the 100-mg/day supplement (1.8 mg/kg-day based on a 60-kg body weight and 8.1 mg/day zinc in the diet [from diet records]). This decrease in HDL values was not apparent at 6 and 8 weeks. Serum zinc levels were also highest in these subjects at 4 weeks.

A very slight but statistically significant ($p = 0.04$) 2-mg/dL increase in HDL cholesterol was seen in a group of 22 elderly male and female subjects (sex ratio unknown) 8 weeks after they ceased using zinc supplements (Goodwin et al., 1985). Serum zinc values fell from 92 to 86 g/dL during the same period. The average supplement intake was 29.1 mg/day with a range of 17.5 to 52.2 mg/day. The increase in HDL value seemed to be greatest for the subjects with the highest ratings for physical activity. Although the data in this study are far from conclusive with regard to the relationship between zinc and HDL values, they do add to the weight of evidence which suggests that the impact of supplemental zinc on HDL levels is real.

- ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The level of confidence in the studies is medium since they are well-conducted clinical studies with many biochemical parameters investigated but only few numbers of humans were tested. The confidence in the overall database is medium since these studies are all of short duration. Medium confidence in the RfD follows.

- ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1990

The Drinking Water Health Advisory for Zinc has received internal Office of Water review.

Other EPA Documentation -- None

- REVIEW DATES: 09/21/89, 08/15/91, 09/11/91, 11/06/91

- VERIFICATION DATE: 11/06/91

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