

Use of Iodine for Water Disinfection: Iodine Toxicity and Maximum Recommended Dose

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Iodine is an effective, simple, and cost-efficient means of water disinfection for people who vacation, travel, or work in areas where municipal water treatment is not reliable. However, there is considerable controversy about the maximum safe iodine dose and duration of use when iodine is ingested in excess of the recommended daily dietary amount. The major health effect of concern with excess iodine ingestion is thyroid disorders, primarily hypothyroidism with or without iodine-induced goiter. A review of the human trials on the safety of iodine ingestion indicates that neither the maximum recommended dietary dose (2 mg/day) nor the maximum recommended duration of use (3 weeks) has a firm basis. Rather than a clear threshold response level or a linear and temporal dose–response relationship between iodine intake and thyroid function, there appears to be marked individual sensitivity, often resulting from unmasking of underlying thyroid disease. The use of iodine for water disinfection requires a risk–benefit decision based on iodine's benefit as a disinfectant and the changes it induces in thyroid physiology. By using appropriate disinfection techniques and monitoring thyroid function, most people can use iodine for water treatment over a prolonged period of time. **Key words:** goiter, iodine, thyroid disorders, thyroid hormones, water purification, water supply. *Environ Health Perspect* 108:679–684 (2000). [Online 21 June 2000]

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Iodine is an essential nutrient for optimal thyroid function in adults and for fetal, infant, and child development. Dietary supplementation, generally via iodized salt but occasionally via iodinated water, has decreased goiter and hypothyroidism due to iodine deficiency in most of the world. Data from supplementation programs and elsewhere indicate that adults need to ingest at least 150–200 µg iodine per day (1–3). Hollowell et al. (4) reported that the average American intake of iodine is near optimal. Nonetheless, ingestion of iodine in excess of the recommended daily intake level is common because of iodine in dietary sources such as dairy, eggs, meat, bread, and seaweed, or that in pharmacologic sources such as the cardiac antiarrhythmic drug amiodarone. Excess iodine may also disrupt normal thyroid function, but the maximum safe level for long-term ingestion remains undetermined. Experts suggest that 1–2 mg/day is safe for most people, yet empiric evidence suggests that much higher amounts are usually tolerated without problems (1,5).

The use of iodine to improve the microbiologic quality of drinking water in areas without safe public sources of potable water also contributes iodine levels in excess of the recommended maximum daily intake. Field water treatment is a necessity for millions of travelers, campers, military troops, and people living and working in underdeveloped areas, in addition to entire populations in disaster and medical relief situations (6). We reviewed published data on the effects of

consuming more than the daily recommended dose of iodine in an attempt to identify the maximum safe dose and duration of ingestion when iodine is used for water disinfection.

Iodine for Water Treatment

Iodine is a halogen, like chlorine, that exerts a biocidal effect through its chemical property as a strong oxidant. The active disinfectant species are elemental iodine and hypiodous acid (7,8). Iodide has no disinfectant activity; however, iodine is rapidly converted to iodide in the stomach and absorbed into the blood. Water disinfection with halogens is a first-order chemical reaction: the primary variables are aqueous concentration of halogen and the time it is in contact with the microorganism (9,10). In addition, different classes of microorganisms vary in their susceptibility to halogens. Bacteria are very sensitive, viruses are intermediate, and protozoan cysts are more resistant. Doses of iodine below 1 mg/L are effective for bacteria within minutes; however, at this concentration, it would take many hours to kill *Giardia* cysts. Although low doses can be used in controlled situations, recommended levels of iodine for point-of-use water disinfection in unmonitored field situations are higher to allow for unanticipated reactions with organic contaminants (halogen demand) and to allow a relatively short contact time (6).

Iodine has several advantages over chlorine for field use—including greater chemical stability of the product and less reactivity

with organic nitrogenous contaminants of residual concentrations in water—leaving higher free residual concentration in water and more acceptable taste in equipotent doses (11,12). Iodine is available in a variety of forms, including solutions (tincture of iodine, povidone, Lugol's, and saturated aqueous solution with iodine crystals), tablets, and iodine resins.

Iodine resins offer additional advantages for field use because the resins are an extremely stable form of iodine that can be incorporated into a wide range of filters and act as a demand disinfectant with limited dissolution in water (13). Little iodine is released into aqueous solution, however: as water passes through and microorganisms contact the resin, iodine is aided by electrostatic forces and binds to microorganisms. The residual iodine concentration with iodine resins is much less than concentrations from the recommended doses of tablet or liquid forms of iodine (Table 1). Iodine resin filters usually incorporate two additional stages: microfiltration to remove *Cryptosporidium* oocysts that are resistant to halogen disinfection, and granular activated charcoal to further reduce the concentration of iodine in effluent water.

Iodine has been used to ensure the safety of potable water since the 1940s, when the military developed a tablet formulation for use by troops in the field (10). Widespread use followed in the civilian population. There are no accurate figures for the number of civilian or military personnel who use iodine for water disinfection. A survey of manufacturers reveals that in 1998 approximately 60,000 iodine resin devices were sold for individual or small-group civilian use. In addition, the leading manufacturer sold more than 300,000 bottles of iodine tablets. This does not include iodine sold in other forms, such as tincture of iodine, povidone, or iodine crystals in aqueous solution.

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Table 1. Residual iodine in demand-free water using recommended doses of available product.

Iodine products	Recommended dose per liter of water ^a	Concentration of iodine
Iodine tablets (tetraglycine hydroperiodide)	1–2 tablets	8–16 mg/L
Emergency drinking water germicidal tablet		
Potable Aqua ^b		
Globaline ^c		
2% iodine solution (tincture)	0.25–0.5 mL (5–10 gtt)	4–8 mg/L
10% povidone-iodine solution	0.35–0.70 mL (8–16 gtt)	4–8 mg/L
Saturated iodine crystals in water		
Polar Pure ^d	13–26 mL	4–8 mg/L
Pentaiodide resin at room temperature	—	1–2 ppm
Triiodide resin		
At room temperature	—	0.2 ppm
At 42°C	—	1 ppm
At 71°C	—	6–10 ppm
After granular activated charcoal	—	0.01 ppm

Data from Powers (14) and Marchen and Fina (15). gtt, drops.

^aLower dose in clear, warm water (< 15°C), higher dose in very cold or cloudy water. Disinfection efficacy is a function of iodine concentration and contact time as well as a function of the water temperature. ^bWisconsin Pharmaceutical, Jackson, WI. ^cVan Ben Industries, Long Island, NY. ^dPolar Equipment, Saratoga, CA.

Because of the ill-defined risk of iodine affecting thyroid function and because other means of water treatment are often available, the World Health Organization (WHO) and the U.S. Environmental Protection Agency recommend that iodine be used for short-term or emergency use only for water treatment (16–18). However, many people use iodine for much longer periods because of the convenience and effectiveness of the products. Despite the extensive use of iodine for both pharmacologic preparations and water disinfection over the past 50 years, there are remarkably few reports of resulting clinical thyroid disorders (19). Recently, goiters discovered among a group of Peace Corps volunteers in Africa were linked epidemiologically to the use of iodine resin filters for water disinfection (20).

Thyroid Effects of Excess Iodine Ingestion

Physiologic effects. The physiologic regulation of the thyroid gland by iodine is complex, involving feedback mechanisms at several biochemical and physiologic steps that depend on the amount of iodine and the rate of administration. In most instances, these autoregulatory mechanisms effectively handle excess iodine intake.

In animal studies, acute iodine administration has induced different responses based on the amount of iodine. Single low doses of iodine (2.5–50 µg) given intraperitoneally to rats had little effect on the metabolism of iodine relative to organification. The accumulation of iodine by the thyroid gland and the incorporation of iodine into triiodothyronine (T₃) or tetraiodothyronine (T₄) increased proportionately with the dose received. At serum levels > 50 µg/dL, rats had decreased uptake of thyroidal iodide, decreased organification, and decreased production of T₃ and T₄ from newly iodinated

amino acids (21). With large doses of iodide, the iodide accumulation and the absolute rate of organification both decreased. The inhibition of iodine organification is called the Wolff-Chaikoff (W-C) effect. Wolff and Chaikoff (22) and Wolff et al. (23) further demonstrated that when rats maintained high levels of serum iodine for > 40 hr, the inhibition of iodine on organification in the thyroid ceased and the thyroid resumed its normal rate of hormone synthesis, preserving the euthyroid state.

In humans, iodide intake of > 2 mg/day results in a proportionate decrease in the organification of thyroidal iodide, but there is no evidence that the overall amount of organic iodination is decreased (24). There are adequate data to demonstrate that thyroid ¹³¹I uptake or thyroid clearance of iodide decreases with increases in serum iodide levels. Studies to limit radioactive iodine uptake in the event of a nuclear accident or fallout have demonstrated rapid suppression of uptake after the administration of iodide (25,26). Single doses > 10 mg suppress the uptake of radioactive iodine to ≤ 1.5% within 24 hr, and daily doses of ≥ 15 mg will maintain uptake below 2% (25).

The limitations of *in vivo* measurement make it difficult to demonstrate the same biochemical mechanism of the W-C effect and escape from it in humans (27). Regardless of the mechanism, an escape from the inhibitory effects of prolonged excess levels of iodine is also seen in humans. For this reason, most people can tolerate high doses of iodine without developing thyroid abnormalities.

Clinical effects. Hyperthyroidism. Iodine-induced hyperthyroidism can be caused by underlying thyroid disease or by the consumption of iodine by people with prior iodine deficiency (5,28,29). During the worldwide campaign to eliminate endemic goiter and associated cretinism,

Stanbury et al. (30) reported that some people would develop hyperthyroidism after receiving only small amounts of iodine supplements. The average incidence of hyperthyroidism was 1.7%, but ranged as high as 7% in Sweden. Any increase in iodine intake will cause some increase in the incidence of hyperthyroidism in a previously iodine-deficient population (31,32). Most cases result from underlying multinodular thyroid disease when autonomous nodules that are not suppressed by increased iodine uptake produce excessive thyroid hormone (31,32). Most of these cases resolve spontaneously and the incidence of hyperthyroidism drops to presupplementation levels within a few years. The elderly are at more risk because they have a higher incidence of multinodular goiter. In areas without endemic goiter, hyperthyroidism as a result of iodine is much less common. Hyperthyroidism may also be induced in people with Graves disease, especially after antithyroid therapy, and in approximately 2% of patients taking amiodarone (33) or other pharmacologic sources of iodine. There is also concern that iodine may induce autoimmune thyroid disease and both papillary and follicular thyroid cancer (1,29).

Reports of hyperthyroidism from the ingestion of iodine used for water disinfection are rare. Liel and Alkan (34) reported on two travelers who became hyperthyroid, presumably because of using iodine tablets for water disinfection. Both were from iodine-sufficient areas and tested positive for antithyroid peroxidase antibodies and negative for antithyroglobulin antibodies. The mother and sister of one of the travelers had Hashimoto thyroiditis (34). The course of the thyroid dysfunction for both travelers was relatively mild and self-limited.

A female Swiss traveler developed mild and reversible hyperthyroidism after using iodine for water purification. The traveler had a previous hemithyroidectomy for a non-toxic nodule, had evidence of autoimmune disease, and tested positive for antithyroid peroxidase antibodies. The traveler's transient hyperthyroidism was followed briefly by subclinical hypothyroidism but reverted to euthyroid status a few months later, when her iodine intake was reduced (35).

Iodine-induced hypothyroidism and/or goiter. Hypothyroidism from excessive iodine intake is much more common than hyperthyroidism. Hypothyroidism is attributed to the prolonged suppression of thyroid hormone production as the result of excess iodine levels, but the mechanism through which iodide goiter is produced is not well understood (36,37). Apparently, patients with iodide goiter require less iodide to inhibit the organic binding of iodine and do

not escape the inhibitory effects of iodides as do those who do not develop goiter (38,39). The problem may result from the thyroid gland's inability to limit the uptake of iodine when large amounts are available, after which the accumulated iodine inhibits the synthesis of hormone (36). Histologic studies suggest that many people who develop iodide goiter have underlying thyroid disease (40). Iodine-induced hypothyroidism or goiter is more common in several groups but may occur with or without underlying thyroid disease. The following people are at increased risk for iodine-induced hypothyroidism (5,28,37,41):

- those with underlying thyroid problems, including current or prior thyroiditis, previous treatment for Graves disease, previous subtotal thyroidectomy for benign nodules, and previous treatment with interferon- α
- fetuses, preterm neonates, and newborn infants who are at risk because of the placental transfer of iodide from mothers treated with iodides
- those with endemic goiter due to very high dietary iodine intake—mainly described in coastal areas of Japan
- those with other conditions, including
 - elderly people without clinical thyroid disease who may have subclinical hypothyroidism (i.e., elevated TSH but normal free T_4 levels) (subclinical hypothyroidism is very common, affecting 5–10% of adults over the age of 50)
 - patients with certain nonthyroid diseases such as chronic dialysis and cystic fibrosis, especially those taking sulfasoxazole
 - patients taking medications containing iodine, formerly iodide expectorants but, more recently, amiodarone
 - patients taking lithium
 - people with a family history of goiter or thyroiditis (suggested by a few case reports).

Excess iodine ingestion may be causally related to autoimmune thyroid disease (1,5). However, a recent study of Japanese subjects who excreted an average of 1.5 mg/day iodine in their urine demonstrated no correlation between antithyroglobulin antibodies or hypothyroidism and levels of urinary iodine (42).

There are three types of reports and studies of iodine-induced hypothyroidism and goiter: case reports, cross-sectional studies of cohorts with high iodine intake, and experimental studies. Case reports help elucidate the natural history and pathophysiology of hypothyroidism, but do not have a population-time denominator to determine the incidence (19,36,38). Paris et al. (38) discussed 11 patients with iodide goiter that developed after a minimum of 13 months of

ingesting high but unspecified amounts of iodide for asthma. The patients' iodine uptake by the thyroid rapidly recovered when they stopped taking iodide. In one patient who resumed taking iodide after the goiter had resolved, the goiter reappeared within 5 weeks. Harrison (36) studied four patients with iodide goiters who had been taking iodine for asthma in doses of 24–1,270 mg/day for 4–13 years. Their goiters resolved spontaneously after they stopping taking iodine.

In the few cross-sectional population studies reporting iodine-induced goiter, the incidence of goiter is variable and does not correlate well with the quantity of iodine or with the level of hypothyroidism. In some coastal regions of Japan, residents who eat a traditional diet rich in seafood and seaweed have an average iodine intake of 1–5 mg/day, with some residents consuming as much as 20–40 mg/day. A few people developed goiters at this intake level (39,43). Those who ingested 3–16 mg/day iodine had a 10% incidence of goiter, but clinical hypothyroidism in this population was rare (43,44).

Mu et al. (45) found that > 60% of school children in central China developed goiter from ingesting water with natural iodine content of 462 μ g/L and that all were clinically euthyroid. Assuming they ingested an average of 2 L water/day, this is the lowest documented iodine dose to produce goiter, even accounting for the lower body weight of the children. In this study, estimated intake was not specified and other dietary sources of iodine were not excluded. In another study, 44 (46%) Peace Corps volunteers who ingested an average of at least 50 mg iodide per day over a period of up to 1 year (from the use of iodine resins for water disinfection) had enlarged thyroids, but 68% of these had normal thyroid function tests (20).

Amiodarone, used for serious cardiac dysrhythmias, contains 37% organic iodide by weight that releases 9–10 mg free iodine from a 300-mg dose. Various investigators have reported an incidence of hypothyroidism of 2–10% among American patients taking amiodarone (33). The pathophysiologic mechanism by which amiodarone leads to iodine-induced goiter may include the inducement of antithyroid antibodies and direct effects of the medication rather than simple suppression by iodine (32,39,46). Among patients taking amiodarone, goiter was present in 60% of hypothyroid patients and 39% of euthyroid patients in Italy and in 22% of hypothyroid patients and 17% of euthyroid patients in the United States (46). The difference in goiter rates may be related to mild underlying iodine deficiencies in the Italian patients.

Human experimental studies provide the best dose–response data, but most are of limited duration (Table 2). In several recent studies, researchers administered approximately 30 mg iodine/day for 1–12 weeks to healthy volunteers. The results of all studies showed that patients had statistically significant increases in TSH levels, but only some of the patients showed significant decreases in levels of T_3 and T_4 . However, despite these changes, TSH, T_3 , and T_4 levels remained within the normal range and rapidly reverted to normal when patients stopped taking the iodine supplements (49,51–54). In two of the studies, researchers also found that thyroid volume increased (52,54). In both studies, thyroid size rapidly reverted to normal after cessation of excess iodine. Studies administering even higher doses of iodine to subjects obtained similar results (47,51,55). Paul et al. (56) looked at the minimum dose that would cause alterations in thyroid function and found that 1,500 μ g/day increased TSH levels whereas 500 and 750 μ g/day did not. None of these experimental studies followed patients long enough to detect clinical hypothyroidism or goiter.

The basis for existing recommendations of maximum safe intake level of iodine are studies by Freund et al. (48) and Thomas et al. (57), who iodinated the water to three Florida prisons. They found minimal changes and no clinical problems when water with 1 mg/L iodine was provided to prisoners for up to 3 years. However, prisoners given water containing 5 mg/L iodine for 2 months had a significant decrease in iodine uptake and serum protein-bound iodine. After the addition of 1 mg/L iodine in prison water for 15 years, the investigators did not find any subjects in whom the use of iodinated water caused a decrease in serum concentrations of thyroxine below normal values, and they did not find any instances of allergic reactions. In addition, there were no instances of neonatal goiter in 181 full-term infants born to women who were incarcerated while pregnant and who delivered their infants while in prison (48).

Morgan and Karpan added 12–19 mg/L iodine to the water supply of a military base for 26 weeks and reported no evidence of altered thyroid activity or other abnormalities. Unfortunately, the only published report of their study (50) contains no details of their results, and the original data are not available.

Other toxic effects of iodine. In a recent review of toxic effects of iodine, Pennington (58) found that sensitivity reactions including rashes and acne may occur with usual supplementation levels of iodine. Given the necessity of iodine, it is not clear why some people react to certain forms of the substance, such as the iodine in salt. Extrathyroidal side

Table 2. Summary table of experimental evidence for iodine use in drinking water.

Iodine dose/day (mg)	Subjects	Time (days)	Person-weeks	Ref
Low dose			16,666	
1.5 ^a	8	14	16	(47)
2–4 ^b	29–133	37 months	16,640	(48)
8 ^a	4	17	10	(49)
Intermediate dose			878	
10–15 ^b	70	60	630	(48)
		(2 months)		
12–20 ^c	NS	182	NS	(50)
		(26 weeks)		
10–20 ^d	4	90	52	(49)
20–30 ^{**}	12	14	24	(51)
25 ^a	11	28	44	(39)
27 ^{a,e}	10	30	17	(52)
32 ^a	7	7	7	(53)
32 ^{a,e}	8	90	104	(54)
High dose			113.5	
70 ^{a,f}	14	14	28	(51)
1,140 ^a	4	80	44	(47)
72 ^a	10	11	15	(55)
360 ^g	9	12–19	18	(55)
100 ^h	5	12	8.5	(25)

Low, intermediate, and high dose defined as 1–10, 10–35, and > 35 mg/day, respectively. NS, not specified, but included all personnel on a military base.

^aAll values remained within clinical measures of normal limits. ^bSmall increase in protein-bound iodine, decrease in iodine uptake, T_4 unchanged. ^cNo values given: "No evidence of altered thyroid activity." ^dOne subject developed subclinical hypothyroidism and one developed subclinical hyperthyroidism. ^eMeasured thyroid volume increased but no goiter was apparent. ^f1.0 mg/kg/day. ^gOne subject developed subclinical hypothyroidism (TSH above normal range). ^hThree of five subjects developed subclinical hypothyroidism (TSH above normal range). ^{*}Statistically significant change in TSH, with or without change in T_3 or T_4 . ^{**}Changes in TSH, T_3 , and T_4 were not statistically significant.

effects of iodine include gastrointestinal (nausea, epigastric pain, and diarrhea), sialadenitis, and iodide fever. These toxic reactions require large doses of iodide, but dose–response relationships have not been established (59). It is unclear whether skin rashes and conjunctivitis are toxic or allergic reactions. Iodism refers to a chronic toxicity that occurs in all individuals when given iodine in sufficiently high doses over a long enough period of time—but the required dose and duration are highly individual. Symptoms resemble a sinus cold with gastrointestinal upset and acneform skin lesions, all of which resolve spontaneously when excess intake stops (60).

Dose response, threshold level, and significance of changes. Given the physiologic activity of iodine and marked individual sensitivity, especially in iodine-deficient populations or those with underlying thyroid disease, it is difficult to delineate a no-effect level, even for limited duration exposures. On the other hand, most people can tolerate pharmacologic doses of iodine for years with no ill effect (5). The reported incidences of goiter, hypothyroidism, and hyperthyroidism

vary so widely that they cannot be used to derive clear dose limits. Wolff (37) reviewed reported cases of iodide goiter and concluded that large amounts of iodine (from 18 mg to > 1 g/day) were required for prolonged periods (< 6 months in a few cases but ≥ 5 years of iodide therapy in many cases). Pennington (58) reviewed case reports to evaluate the levels at which oral doses of iodine cause adverse effects, and found that 49 people from 15 reports had adverse effects from iodine intakes ≤ 10 mg/day, including 19 with hyperthyroidism, one with goiter, and one with sensitivity reaction. Some of these individuals had underlying thyroid disease. Single 100-mg doses of potassium iodide for thyroid protection after a nuclear accident caused infrequent and mild extrathyroidal side effects and no long-term thyroid changes (61).

Experimental data (Table 2) suggest that ≥ 1.5 mg/day iodine will cause statistically significant changes in thyroid hormone assay. However, these changes are not clinically significant, so they questionably constitute a valid threshold effect level. Even at much higher doses the changes in thyroid function remain subclinical; that is, TSH levels were elevated but T_4 levels were within the normal range. Subclinical abnormalities in thyroid function are commonly identified by serologic diagnostic batteries, especially in elderly people, yet the significance and need for treatment are debated despite the persistence of these abnormalities in this population (62–64). In contrast, human experiments of iodine ingestion in healthy volunteers demonstrated reversible changes in thyroid function and goiter (47,51,52,54). Clinical hypothyroidism is rare among Japanese with endemic goiter due to long-term ingestion of excess iodine. In these cases, both thyroid function and thyroid size return to normal when excess iodine consumption is stopped, unless underlying thyroid disease is present (36,38,39,43).

The 10th edition of the *Recommended Dietary Allowances*, published in 1989 (65), suggested that the maximum allowable dietary dose of iodine be 1.0 mg/day for children and 2.0 mg/day for adults. These recommended limits were increased from 1.0 mg/day for adults in the previous (1980) edition (58,66) based primarily on the data from Freund et al. (48) and Thomas et al. (57). The National Academy of Sciences reached a similar conclusion by calculating a suggested no-adverse-response level from toxicity data (11), and noted that this was consistent with the data from both Freund et al. (48) and Morgan and Karpen (50). Most uses of iodine for field water treatment would exceed these recommended limits. Recommendations for short-term

emergency use of iodine are generally limited to approximately 3 weeks [Water and Sanitation for Health Project (67), Zoeteman (16), and the National Academy of Sciences (11)]. The WHO has sponsored long-term iodination of water for large populations at doses up to 0.3–0.4 mg/L to prevent iodine deficiency disorder. Although these doses may be bacteriocidal, they are unlikely to be adequate for the destruction of cysts and viruses. The details of adverse reactions were not provided in a summary report (3).

The available data suggest that neither the maximum safe dietary dose of iodine nor the maximum safe period of consumption for water treatment are firmly established. Available data also are not adequate to define a linear and temporal dose response between iodine intake and thyroid function (Table 2). The strongest data suggest that low levels of iodine (1–5 mg/day) are safe for most people for years. It should be noted that Freund et al. (48) supplied 1 mg/L iodine in water, not 1 mg/day, and estimated the average daily intake as 2–4 mg/day. Well-controlled experimental trials have evaluated intermediate levels of iodine from 10 to 32 mg/day for up to 3 months. These levels consistently cause subclinical changes in thyroid function, and may cause thyroid enlargement, but clinical hypothyroidism was not observed. The data from Georgitis and McDermott (53) and LeMar et al. (54) suggest that 3 months of moderately high levels of iodine ingestion (32 mg/day) carries no more risk than 1 week of ingestion. Higher levels may be well tolerated, but Peace Corps data indicated that people who consumed > 30 mg/day for 6 months had a high incidence of goiter (20). In contrast, this incidence of goiter was not noted in populations that commonly used even higher doses of iodine as an expectorant (38,68), suggesting that some cofactor may be required for the development of goiter.

Recommendations for Iodine Use in Water Treatment

We are not advocating raising the daily minimum requirements of iodine, nor are we promoting the use of iodine for routine community water treatment in small remote communities or in developing countries, where there would be a higher incidence of adverse effects. Despite alternative methods of disinfection alternatives, iodine remains popular for individual and small-group field water treatment in situations where surface water is ingested or water quality from a distribution system is questionable. This discussion should be useful for the development of policy and procedures for agencies that place personnel in remote locations and

for stimulation of policy debate at agencies that regulate products containing iodine.

When iodine is considered the best means of water treatment, we recommend the following:

- Do not use an iodine-based water treatment method for people with increased susceptibility to iodine-induced thyroid disorders
 - pregnant women (fetus susceptible to goiter)
 - people with known hypersensitivity to iodine
 - people with a history of thyroid disease, even if controlled on medication
 - people with a strong family history of thyroid disease
 - people from countries or localities with chronic iodine deficiency.
- Examine the thyroid and do thyroid function tests on anyone planning to use iodine for prolonged periods (longer than 3 months) to ensure that they are initially euthyroid. It is not clear whether the presence of antithyroid antibodies is a contraindication to iodine use.
- Limit the use of water disinfection methods that produce moderately high levels of iodine (5–32 mg/L), such as iodine tablets, to 3 months.
- Euthyroid people can safely use iodine treatments that produce a low residual (≤ 1 mg/L) level, even for long periods of time. Current products containing iodine resin devices with a charcoal scavenger to remove residual iodine can meet this standard when used properly. Likewise, a two-step process of water treatment with iodine tablets or iodine solutions, then filtration by a device containing charcoal, can ensure safe levels of iodine and effective water treatment.
- Standards are needed to ensure the efficacy and safety of iodine-containing water purification products intended for prolonged use
 - the products must contain an effective iodine scavenger (usually granular activated charcoal) sufficient to decrease the residual to a level safe for long-term use (years)
 - the iodine scavenger must be effective throughout the life span of the device or there must be a reliable method to indicate when the iodine scavenger no longer has adsorptive capacity so that it can be replaced
 - the product must perform adequately without the need for high levels of residual iodine.

In summary, the use of iodine for water disinfection requires a risk–benefit decision similar to other medical interventions that are used prevent illness in high-risk populations. The paucity of accurate water quality data

for most parts of the world, and known issues of water quality where it is measured, mandate additional measures to ensure potable water in many situations (69). The risk for enteric infection (70) and the availability of other means of producing potable water should be weighed against the risk for and severity of thyroid disorder from iodine. Enteric disease rates from water with microbiologic contamination may be high and repeat infections can be anticipated because only limited immunity develops (71). Iodine is an effective, simple, and cost-efficient means of water disinfection. For temporary residents, relief workers, and travelers in areas where municipal water treatment is not reliable, the benefit of use exceeds the risk.

Although there is fair evidence that the current recommended upper limits of daily iodine ingestion are safe, there are limited data to suggest that this upper limit is the maximum safe level. Coupled with periodic monitoring of thyroid function, techniques that generate low levels (1–2 mg/L) of iodine in water can be safely used for years.

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