Journal of the American College of Toxicology
14(3):231–239, Raven Press, Ltd., New York
© 1995 Cosmetic Ingredient Review

Final Report on the Safety Assessment of Sodium Iodate[1]

Abstract: Sodium Iodate is an inorganic salt that is intended for use as an oxidizing agent in cosmetics, but no current uses have been reported. It is approved by the European Union for use as a preservative in rinse-off cosmetic products at concentrations no greater than 0.1%. Pure Sodium Iodate is a sufficiently strong oxidizing agent that it presents a fire risk near organic material, and it can react violently with aluminum, arsenic, carbon, copper, hydrogen peroxide, phosphorous, potassium, sulfur, and metal sulfides. Sodium Iodate is toxic to the retina; injection of 10^{-4} *M* Sodium Iodate into the vitreous of rabbit eyes inactivated the electroretinogram in 1 day. Acute toxicity studies in mice show that concentrations of 505 mg/kg delivered orally is expected to cause convulsions and death in half the animals. No mutagenic activity was seen in Ames tests. Exposure of repair proficient strains of *Escherichia coli* to ionizing radiation and Sodium Iodate increased the number of DNA single-strand breaks over those seen with exposure to ionizing radiation alone. Sodium Iodate combined with aflatoxin B_1 showed fewer mutations in the Ames test than did aflatoxin alone. These available data were not sufficient to support the safety of Sodium Iodate for use in cosmetic formulations. Additional data were considered necessary to evaluate the safety of this ingredient, including the purpose of use and likely concentration of use in cosmetics; 28-day dermal toxicity data; and animal irritation data as a function of dose. It cannot be concluded that this ingredient is safe for use in cosmetic products until the listed safety data have been obtained and evaluated. **Key Words:** Sodium Iodate—Oxidizing agent—Inorganic salt.

Sodium Iodate is an inorganic salt that functions as an oxidizing agent (Nikitakis, 1988).

CHEMISTRY

Definition and Structure

Sodium Iodate (CAS No. 7681-55-2) is the inorganic salt that conforms to the following formula (Nikitakis et al., 1991):

$$NaIO_3$$

Sodium Iodate is also known as Iodic Acid, Sodium Salt (Nikitakis et al., 1991; Registry of Toxic Effects of Chemical Substances [RTECS], 1992).

[1] Reviewed by the Cosmetic Ingredient Review Expert Panel.

Address correspondence and reprint requests to Dr. F. A. Andersen at Cosmetic Ingredient Review, 1101 17th Street, NW, Suite 310, Washington, D.C. 20036, U.S.A.

Physical and Chemical Properties

Sodium Iodate occurs as a white crystalline powder (Friel, 1974; Windholz, 1983) and has a molecular weight of 197.89 (Weast, 1982; Material Safety Data Sheet [MSDS], 1991). It is soluble in water (Greenberg and Lester, 1954; Grant, 1972), acetone (Hawley, 1971), and acetic acid; it is insoluble in alcohol (Weast, 1982). An aqueous Sodium Iodate solution is neutral (Windholz, 1983).

Sodium Iodate is an oxidizing agent and may ignite near organic materials (Hawley, 1971). It may react violently with aluminum, arsenic, carbon, copper, hydrogen peroxide, metal sulfides, organic matter, phosphorus, potassium, and sulfur (Sax, 1979). Sodium Iodate is incompatible with strong reducing agents and combustible materials (MSDS, 1991). Contact with combustible materials, flammable materials, or powdered metals can cause fire or explosion. The physical and chemical properties of Sodium Iodate are summarized in Table 1.

Manufacture and Production

Sodium Iodate is derived from the interaction of sodium chlorate and iodine in the presence of nitric acid (Hawley, 1971).

TABLE 1. *Chemical and physical properties of Sodium Iodate*

Characteristic	Description	Reference
Physical appearance	White crystalline powder	Friel (1974); Windholz (1983)
	Rhombic white crystals	Sax (1979); Weast (1982)
Molecular weight	197.89	Weast (1982); MSDS (1991)
	197.9	Grant (1972); Windholz (1983)
	197.92	Sax (1979)
Melting point	Decomposes	Sax (1979); Weast (1982)
Solubility	Soluble in water	Greenberg and Lester (1954); Hawley (1971); Grant (1972)
	Soluble in acetone	Hawley (1971)
	Soluble in acetic acid	Weast (1982)
	Insoluble in alcohol	Greenberg and Lester (1954); Hawley (1971); Weast (1982)
pH Value (aq. solution)	Neutral	Windholz (1983)
Reactivity	Oxidizing agent; fire risk near organic material	Hawley (1971)
	May react violently with Al, As, C, Cu, H_2O_2, metal sulfides, organic matter, P, K, S	Sax (1979)
	Incompatible with strong reducing agents and combustible materials; contact with combustible materials, flammable materials, or powdered metals can cause fire or explosion	MSDS (1991)
Density	4.277 (at 17.5°C)	Sax (1979); Weast (1982)

Analytical Methods

Sodium Iodate can be determined by first adding one drop of starch T.S. to 1 ml of a 1:10 solution of the sample and then adding several drops of 20% hypophosphorous acid, which results in a transient blue color (Informatics, Inc., 1973).

Ultraviolet Absorbance

Published data on the ultraviolet absorbance of Sodium Iodate were not found.

Impurities

Sodium Iodate contains approximately 99% $NaIO_3$ (Windholz, 1983).

USE

Cosmetic

The product formulation data submitted to the Food and Drug Administration (FDA) in 1993 indicated that Sodium Iodate was not contained in any cosmetic formulations (FDA, 1993).

International

Sodium Iodate is a preservative approved for use by the European Union (EU) at a concentration of 0.1% or lower in rinse-off products only (EEC Cosmetic Directive, 1990).

Noncosmetic

Sodium Iodate has medical uses (Hawley, 1971). It has been used as an antiseptic in treating diseases of the mucous membranes (Friel, 1974; Windholz, 1983). Saturated aqueous solutions of Sodium Iodate have been used as a dressing for ulcers and septic wounds and dilute solutions for suppurative and gangrenous affections of mucous membranes, eczema, and cystitis (Greenberg and Lester, 1954). A powdered form of Sodium Iodate has been used for otorrhea.

The World Health Organization (WHO) recommends that iodate salts be used as an additive in table salt to prevent goiter (Gosselin et al., 1976). Sodium Iodate, anhydrous or hydrated, is generally recognized as safe for use as a trace mineral added to animal feed (Rothschild, 1990).

GENERAL BIOLOGY

Absorption, Distribution, Metabolism, Excretion

Pigmented rabbits were dosed by i.v. injection with a retinotoxic dose, 30 mg/kg, of I^{131}-Sodium Iodate in physiological saline (Orzalesi and Calabria, 1967). The concentration of Sodium Iodate in the blood, where it was contained primarily in the plasma fraction, decreased gradually. Iodate quickly penetrated into the anterior chamber and vitreous body of the globe and was eliminated slowly. Large

amounts of radioactivity were found in the thyroid gland, liver, and kidneys; small amounts were found in the muscle, choroid, lens, and retina. Iodate uptake by the retinal pigment epithelium seemed high, but the amount may have been exaggerated by contamination from the choroid.

Albino guinea pigs were used to determine the permeability effects of 0.02 *M* Sodium Iodate in 0.85% saline given by intracutaneous injection with circulating Evan's blue dye (Steele and Wilhelm, 1967). Sodium Iodate produced a monophasic reaction and induced a strong early response lasting 30 to 40 h after dosing. After 16 to 20 h, a small (3-4 mm in diameter) area of central necrosis at the site of injection was observed.

Ocular Effects

Sodium Iodate is a retinotoxic compound (Armstrong et al., 1982). Ocular injection of 10^{-4} *M* or more of Sodium Iodate into the vitreous of the globes of New Zealand albino rabbits inactivated the electroretinogram (ERG) in 1 day.

In rabbits, Sodium Iodate produces a pigmentary degeneration of the retina in the globe (Orzalesi and Calabria, 1967), and iodate causes black pigmentation of the retina (Informatics, Inc., 1973). In a study in which rabbits were dosed with Sodium Iodate by i.v. injection, atrophy and marked pigmentation of the retina were observed (Wada, 1960).

Additional data on possible adverse ocular effects of Sodium Iodate, including (but not limited to) retinal, blood-retinal barrier, pigment epithelium, rhodopsin regeneration, and ERG effects, are available (Sorsby and Reading, 1964; Sorsby, 1966; Clifton and Makous, 1973; Davson, 1973; Flage, 1983; Amemiya, 1977; Nilsson et al., 1977a,b,c; Olsen et al., 1979; Ringvold, 1978; Textorius and Welinder, 1981; Campochiaro et al., 1986; Korte et al., 1986; Kitano et al., 1988; Taura and Reddy, 1988; Korte et al., 1989).

Pharmacological Effects

Twelve male albino Wistar rats were given 0.2% Sodium Iodate in the drinking water for 2 days and then dosed with 10 mg/kg thiourea by i.p. injection (McCreesh and Mann, 1958). The control group of 27 male albino Wistar rats was dosed with 10 mg/kg thiourea without pretreatment. Sodium Iodate greatly diminished the hyperglycemia that normally follows thiourea administration.

ANIMAL TOXICOLOGY

Acute Toxicity

Oral

The oral mean lethal dose (LD_{50}) of 6% Sodium Iodate for groups of 10 fasted female white Swiss mice was 505 mg/kg (Webster et al., 1957). Signs of intoxication included diarrhea, alternate hyperactivity and lassitude, followed by weakness, prostration, and dyspnea. At higher doses, death was often preceded by excitability and convulsions. Hemoglobinuria occurred in many of the animals.

Animals that survived 2 weeks following dosing underwent necropsy. At microscopic examination, lesions, such as degeneration of many parietal cells of the gastric glands, were observed in animals dosed with 463 mg/kg Sodium Iodate.

Parenteral

Using groups of 10 fasted female white Swiss mice, i.v. and i.p. (LD_{50}) of 3% Sodium Iodate were determined to be 108 and 119 mg/kg, respectively (Webster et al., 1957). The highest doses tolerated by 10 of 10 mice without mortality were 70 and 90 mg/kg by i.v. and i.p. administration, respectively. For both methods of dosing, signs of toxicity were similar to those previously observed following oral administration, except diarrhea was not observed.

The i.v. lowest lethal dose (LD_{LO}) of iodates for rabbits was 75 mg/kg (Maxwell, 1930). Signs of toxicity included convulsions, spastic paralysis, irritation of the mucous membranes, and albuminuria. The toxicity of iodates was greatly increased in the presence of iodides. The i.v. LD_{LO} of Sodium Iodate for dogs was 200 mg/kg (RTECS, 1992).

Black rabbits were dosed with Sodium Iodate by i.v. injection and killed 4, 20, or 80 days after dosing (Wada, 1960). (The number of animals and dose were not given.) A gross and histochemical examination of the organs was performed, revealing pyknosis and nuclear vacuolation, granulation of cells, and atrophy of the liver; swelling, vacuolation, and atrophy of the kidneys; atrophy of cardiac muscle; pulmonary lesions; and splenic and adrenal gland abnormalities. Some of the toxic effects produced by Sodium Iodate were reduced by coadministration of helenien, adrenocorticotropic hormone ACTH, methonin, or pantothenic acid.

MUTAGENICITY

Sodium Iodate showed no mutagenic activity by the Ames test, the micronucleus test using mouse bone marrow, or the recessive lethal test using *Drosophila* (Eckhardt et al., 1982). The concentration(s) tested were not given.

Antimutagenic Effects

The antimutagenic potential of 0.1 and 0.5 μmol Sodium Iodate against 1.28 nmol aflatoxin B_1 (AFB_1) was examined by plate incorporation assay performed according to the method of Maron and Ames (1983) using *Salmonella typhimurium* strains TA100 and TA98 (Francis et al., 1988). A plate to which Sodium Iodate was not added was used as control. Triplicate plates were used, and the assay was repeated at least once. Sodium Iodate had antimutagenic activity toward AFB_1 when tested using strain TA100, but not when using strain TA98. The ID_{50} (dose required to inhibit the mutagenic activity of AFB_1 by 50%) of Sodium Iodate using strain TA100 was 150 nmol.

Effect on DNA Repair

The effect of Sodium Iodate, a radiosensitizing agent, on DNA single-strand breakage repair following irradiation was examined using *Escherichia coli* strains

B/r, B_{s-1}, and pol A, a mutant strain that does not repair damaged DNA (Myers and Chetty, 1973). Sodium Iodate (10 m*M*) was added to the cultures, which were then irradiated aerobically with γ-rays from a ^{60}Co source at a dose rate of 7 krad/min. Assays with bacteriophage T4 were also done to study the effects of radiation on DNA without a bacterial enzyme system.

The number of single-strand breaks for *E. coli* strains B/r and B_{s-1} after irradiation was greater in cultures to which Sodium Iodate had been added compared with those that were irradiated only. The main target of action was considered the cell membrane. For *E. coli* strain pol A, no difference was observed. The addition of Sodium Iodate decreased the survival of strain B/r but had no effect on the survival of strain pol A.

CLINICAL ASSESSMENT OF SAFETY

Toxicity

Iodate salts have a toxicity rating of 4, very toxic, with the implicit assumption that the lethal dose for humans is in the same class as the LD_{50} for test animals (Gosselin et al., 1976).

Sodium Iodate is more toxic than the chlorates (Greenberg and Lester, 1954). It can result in slow intoxication accompanied by inflammatory changes in the intestines and fatty degeneration of the liver.

SUMMARY

Sodium Iodate, an inorganic salt that occurs as a white crystalline powder or as rhombic white crystals, functions as an oxidizing agent. It is soluble in water, acetone, and acetic acid and insoluble in alcohol. Sodium Iodate, a quite reactive oxidizing agent, is derived from the interaction of sodium chlorate and iodine in the presence of nitric acid. It is approximately 99% pure.

In 1993, data submitted to the FDA did not report the use of Sodium Iodate in cosmetic formulations. It is approved by the EU for use as a preservative at concentrations no greater than 0.1% in rinse-off products only. Sodium Iodate has been used medically.

In pigmented rabbits dosed by i.v. injection with a retinotoxic dose of I^{131}-Sodium Iodate, Iodate quickly penetrated into the anterior chamber and vitreous body of the globe. Large amounts of radioactivity were found in the thyroid gland, liver, and kidneys. In one study using albino Wistar rats, Sodium Iodate greatly diminished the hyperglycemia that normally follows thiourea administration.

Sodium Iodate is a retinotoxic compound. It may adversely affect the retina, blood–retinal barrier, pigment epithelium, rhodopsin regeneration, and ERG.

The oral LD_{50} of 6% Sodium Iodate and i.v. and i.p. LD_{50}'s of 3% Sodium Iodate for female white Swiss mice were 505, 108, and 119 mg/kg, respectively. The i.v. LD_{LO}'s for rabbits and dogs were 75 and 200 mg/kg, respectively.

Sodium Iodate did not have mutagenic activity in the Ames, micronucleus, or recessive lethal test. Sodium Iodate had antimutagenic activity toward AFB_1 using *S. typhimurium* strain TA100 but not strain TA98. DNA single-strand breaks

were increased in cultures of *E. coli* strains B/r and B_{s-1} after irradiation when Sodium Iodate was added compared with those irradiated without Sodium Iodate addition. Sodium Iodate decreased the survival of strain B/r, but it had no effect on *E. coli* strain pol A.

In humans, iodate salts are rated as very toxic, with the lethal dose assumed to be of the same magnitude as the LD_{50} for test animals. Sodium Iodate is more toxic than the chlorates.

DISCUSSION

Section 1, paragraph (p), of the *Cosmetic Ingredient Review (CIR) Procedures* states that "A lack of information about an ingredient shall not be enough to justify a determination of safety." In accordance with Section 30(j)(2)(A) of the *Procedures,* the Expert Panel informed the public of its decision that the data on Sodium Iodate were insufficient to determine whether Sodium Iodate, for possible purposes of cosmetic use, is either safe or unsafe. The Expert Panel released a "Notice of Insufficient Data" announcement on August 23, 1993, outlining the data needed to assess the safety of Sodium Iodate. The types of data required included:

1. Purpose of use and likely concentration of use in cosmetics
2. 28-Day dermal toxicity data
3. Animal irritation dose-response data

No offer to supply the data was received. In accordance with Section 45 of the *CIR Procedures,* the Expert Panel will issue a "Final Report—Insufficient Data." When requested data are available, the Expert Panel will reconsider the final report in accordance with Section 46 of the *CIR Procedures,* "Amendment of a Final Report."

Additionally, the Expert Panel recognizes that ocular irritation data are lacking; however, because of the structure of Sodium Iodate, the ingredient would be an ocular irritant. The information summarized in this report concerning ocular effects produced by Sodium Iodate substantiated that Sodium Iodate is harmful to the eyes. Therefore, it is the opinion of the Expert Panel that Sodium Iodate should not be contained in formulations designed for use around or near the eye.

Although no carcinogenicity data were available, the Expert Panel did not consider it necessary to request such data based on the results of the mutagenicity assays contained in this report.

CONCLUSION

The safety of this ingredient has not been documented and substantiated. The CIR Expert Panel cannot conclude whether Sodium Iodate is safe for use in cosmetic products until the appropriate safety data have been obtained and evaluated.

Acknowledgment: Monice Zondlo Fiume, Scientific Analyst/Report Management Coordinator, prepared this report.

REFERENCES

Amemiya T. (1977) Postnatal maldevelopment of the retina. *Biol Neonate* 32:319–26.
Armstrong D, Hiramitsu T, Gutteridge J, Milsson SE. (1982) Studies on experimentally induced retinal degeneration. 1. Effect of lipid peroxides on electroretinographic activity in the albino rabbit. *Exp Eye Res* 35:157–71.
Campochiaro PA, Bryan JA III, Conway BP, Jaccoma EH. (1986) Intravitreal chemotactic and mitogenic activity. Implication of blood-retinal barrier breakdown. *Arch Ophthalmol* 104:1685–7.
Clifton L, Makous W. (1973) Iodate poisoning: early effect on regeneration of rhodopsin and the ERG. *Vision Res* 13:919–24.
Davson H. (1973) Effects of iodate on the blood-vitreous barrier. *Exp Eye Res* 16:373–5.
Eckhardt K, Gocke E, King M-T, Wild D. (1982) Mutagenic activity of chlorate, bromate, and iodate. *Mutat Res* 97:185.
EEC Cosmetic Directive. (1990) *Official Journal of the European Communities*. No. C 322/68.
Flage T. (1983) Changes in the juxtapapillary retinal pigment epithelium following intravenous injection of Sodium Iodate. A light and electron microscopic study using horseradish peroxidase as a tracer. *Acta Ophthalmol* 61:20–8.
Food and Drug Administration (FDA). (1993) Cosmetic product formulation data: Ingredient use by product category. Computer printout. Washington, DC: FDA.
Francis AR, Shetty TK, Bhattacharya RK. (1988) Modifying role of dietary factors on the mutagenicity of aflatoxin B_1: In vitro effect of trace elements. *Mutat Res* 199:85–93.
Friel JP, ed. (1974) *Dorland's Illustrated Medical Dictionary*. 25th ed. Philadelphia, PA: WB Saunders, 1431.
Gosselin RE, Hodge HC, Smith RP, Gleason MN, eds. (1976) Clinical Toxicology of Commercial Products. Acute Poisoning. 4th ed. Baltimore, MD: Williams and Wilkins, Section II: D87.
Grant J, ed. (1972) *Hackh's Chemical Dictionary*. 4th ed. New York: McGraw-Hill, 619.
Greenberg LA, Lester D. (1954) *Handbook of Cosmetic Materials*. New York: Interscience Publishers, 293.
Hawley GG, ed. (1971) *The Condensed Chemical Dictionary*. 8th ed. New York: Van Nostrand Reinhold, 803.
Informatics Inc. (1973) Scientific literature review on generally recognized as safe (GRAS) food ingredients—iodine and iodine salts. NTIS #: PB-223 849.
Kitano S, Hori S, Nagataki S. (1988) Transport of fluorescein in the rabbit eye after treatment with Sodium Iodate. *Exp Eye Res* 46:863–70.
Korte GE, Cushin S, Delman N. (1989) Permeability of regenerating and atrophic choriocapillaris in the rabbit. *Acta Anat* 134:144–50.
Korte GE, Gerszberg T, Pua F, Henkind P. (1986) Choriocapillaris atrophy after experimental destruction of the retinal pigment epithelium in the rat. *Acta Anat* 127:171–5.
Maron DM, Ames BN. (1983) Revised method for the *Salmonella* mutagenicity test. *Mutat Res* 113:173–215.
Material Safety Data Sheet (MSDS). (1991) MSDS on Sodium Iodate issued by J.T. Baker, Inc.*
Maxwell LC. (1930) The reaction of iodates in vivo. *J Pharmacol Exp Ther* 40:451–5.
McCreesh AH, Mann DE, Jr. (1958) Effect of orally administered sodium iodide and Sodium Iodate on blood sugar response to thiourea in rats. *J Am Pharm Assoc* 47:56–7.
Myers DK, Chetty KG. (1973) Effect of radiosensitizing agents on DNA strand breaks and their rapid repair during irradiation. *Radiat Res* 53:307–14.
Nikitakis JM, ed. (1988) *CTFA Cosmetic Ingredient Handbook*. 1st ed. Washington, D.C. Cosmetic, Toiletry, and Fragrance Association, 379.
Nikitakis JM, McEwen GN, Wenninger JA. (1991) *CTFA International Cosmetic Ingredient Dictionary*. 4th ed. Washington, D.C. Cosmetic, Toiletry, and Fragrance Association, 543–4.
Nilsson SEG, Knave B, Persson HE. (1977a) Changes in ultrastructure and function of the sheep pigment epithelium and retina induced by Sodium Iodate. I. The ultrastructure of the normal pigment epithelium of sheep. *Acta Ophthalmol* 55:994–1006.
Nilsson SEG, Knave B, Persson HE. (1977b) Changes in ultrastructure and function of the sheep pigment epithelium and retina induced by Sodium Iodate. II. Early effects. *Acta Ophthalmol* 55:1007–26.
Nilsson SEG, Knave B, Persson HE. (1977c) Changes in ultrastructure and function of the sheep

* Available for review from: Director, Cosmetic Ingredient Review, 1101 17th Street, N.W., Suite 310, Washington, D.C. 20036, U.S.A.

pigment epithelium and retina induced by Sodium Iodate. III. Early effects. *Acta Ophthalmol* 55:1027–43.

Olsen KJ, Ehlers N, Schonheyder F. (1979) Studies on the handling of retinotoxic doses of iodate in rabbits. *Acta Pharmacol Toxicol* 44:241–50.

Orzalesi N, Calabria GA. (1967) The penetration of I^{131} labeled Sodium Iodate into the ocular tissues and fluids. *Ophthalmologica* 153:229–38.

Registry of Toxic Effects of Chemical Substances (RTECS). (1992) Cincinnati, OH: National Institute of Occupational Safety and Health, RTECS Database; Sodium Iodate entry. Accessed online from the National Library of Medicine's Toxicology Data Network (TOXNET).

Ringvold A. (1978). The effect of Sodium Iodate on the ciliary body/iris in rabbits. *Exp Eye Res* 27:87–100.

Rothschild DL Jr. (1990) *The Food Chemical News Guide to the Current Status of Food Additives and Color Additives*. Washington, D.C.: Food Chemical News, 416.6.

Sax NI. (1979) *Dangerous Properties of Industrial Materials*. 5th ed. New York: Van Nostrand Reinhold, 984.

Sorsby A. (1966) Oxidizing agents as potentiators of the retinotoxic action of sodium fluoride, Sodium Iodate, and sodium iodoacetate. *Nature* 210:997–8.

Sorsby A, Reading HW. (1964) Experimental degeneration of the retina-XI. The effect of Sodium Iodate on retinal-SH levels. *Vision Res* 4:511–4.

Steele RH, Wilhelm DL. (1967) The inflammatory reaction in chemical injury. II. Vascular permeability changes and necrosis induced by intracutaneous injection of various chemicals. *Br J Exp Pathol* 48:592–607.

Taura T, Reddy VN. (1988) Effect of Sodium Iodate injection on the development of galactose cataract in the rat. *Ophthalmic Res* 20:286–92.

Textorius O, Welinder E. (1981) Early effects of Sodium Iodate on the directly recorded standing potential of the eye and on the c-wave of the DC registered electroretinogram in albino rabbits. *Acta Ophthalmol* 59:359–68.

Wada H. (1960) Experimental and pathological studies of the influence of Sodium Iodate and therapeutic effects of some drugs. *Kobe J Med Sci* 6(Suppl 1):15–7.

Weast RC. (1982) *CRC Handbook of Chemistry and Physics*. 63rd ed. Boca Raton, FL: CRC Press, B-148.

Webster SH, Rice ME, Highman B, Von Oettingen WF. (1957) The toxicology of potassium and sodium iodates: acute toxicity in mice. *J Pharmacol Exp Ther* 120:171–8.

Windholz M, ed. (1983) *The Merck Index. An Encyclopedia of Chemicals, Drugs, and Biologicals*. 10th ed. Rahway, NJ: Merck and Co., 1237.