The MAK Collection for Occupational Health and Safety

Iodine and inorganic iodides

Assessment Values in Biological Material – Translation of the German version from 2016

M. Nasterlack[1], H. Drexler[2,*], A. Hartwig[3,*], MAK Commission[4,*]

1 *68526 Ladenburg, Germany*

2 *Head of the working group "Assessment Values in Biological Material" of the Permanent Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Deutsche Forschungsgemeinschaft, Friedrich-Alexander-Universität Erlangen-Nürnberg, Institute and Outpatient Clinic of Occupational, Social, and Environmental Medicine, Henkestraße 9–11, 91054 Erlangen, Germany*

3 *Chair of the Permanent Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Deutsche Forschungsgemeinschaft, Institute of Applied Biosciences, Department of Food Chemistry and Toxicology, Karlsruhe Institute of Technology (KIT), Adenauerring 20a, Building 50.41, 76131 Karlsruhe, Germany*

4 *Permanent Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Deutsche Forschungsgemeinschaft, Kennedyallee 40, 53175 Bonn, Germany*

***** *email: H. Drexler (hans.drexler@fau.de), A. Hartwig (andrea.hartwig@kit.edu), MAK Commission (arbeitsstoffkommission@dfg.de)*

Keywords: iodine; inorganic iodides; biological reference value; BAR; background exposure

Citation Note: Nasterlack M, Drexler H, Hartwig A, MAK Commission. Iodine and inorganic iodides. Assessment Values in Biological Material – Translation of the German version from 2016. MAK Collect Occup Health Saf [Original edition. Weinheim: Wiley-VCH; 2018 Jul;3(3):1535-1545]. Corrected republication without content-related editing. Düsseldorf: German Medical Science; 2026. https://doi.org/10.34865/bb755356e2218_w

Republished (online): 06 Mar 2026

Originally published by Wiley-VCH Verlag GmbH & Co. KGaA; https://doi.org/10.1002/3527600418.bb755356e2218

Manuscript completed: 16 Oct 2014

Published (online): 26 Jul 2018

The commission established rules and measures to avoid conflicts of interest.

Iodine and inorganic iodides

BAT Value Documentation

M. Nasterlack[1], H. Drexler[2, *], A. Hartwig[3, *], MAK Commission[4, *]

DOI: 10.1002/3527600418.bb755356e2218

Abstract

In 2015 the German Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area has evaluated iodine [CAS No. 7553-56-2] and inorganic iodides. Available publications are described in detail.
There is no reproducible quantitative relation between iodine exposure, absorption and resulting body burden. The association between iodine absorption and the incidence of thyroid dysfunction seems to be U-shaped. Therefore, no sharp boundary can be drawn between iodine oversupply and iodine deficiency relating to individual health. No harmless range could be indicated, so a BAT value (biological tolerance value) for iodine and inorganic iodides could not be derived.
There are major regional differences due to different geological deposits of iodine, different concentrations of iodine in drinking water, iodine-containing aerosols and nutritional habits. Furthermore, there is a high variability in the urinary analysis. Therefore, a BAR ("Biologischer Arbeitsstoff-Referenzwert") for iodine and inorganic iodides could not be derived.

Keywords

iodine; potassium iodide; sodium iodide; ammonium iodide; ammonium iodate; magnesium iodide; calcium iodide; occupational exposure; biological tolerance value; BAT value; EKA; biological reference value; BAR; toxicity

Author Information

[1] Konrad-Seel-Straße 1, 68526 Ladenburg, Germany
[2] Chair of the Working Group "Setting of Threshold Limit Values in Biological Materials", Deutsche Forschungsgemeinschaft; Institute and Outpatient Clinic of Occupational, Social and Environmental Medicine, Friedrich-Alexander University Erlangen-Nürnberg (FAU), Henkestr. 9–11, 91054 Erlangen, Germany
[3] Chair of the Permanent Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Deutsche Forschungsgemeinschaft, Department of Food Chemistry and Toxicology, Institute of Applied Biosciences, Karlsruhe Institute of Technology (KIT), Adenauerring 20a, Building 50.41, 76131 Karlsruhe, Germany
[4] Permanent Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Deutsche Forschungsgemeinschaft, Kennedyallee 40, 53175 Bonn, Germany

[*] Email: H. Drexler (hans.drexler@fau.de), A. Hartwig (andrea.hartwig@kit.edu), MAK Commission (arbeitsstoffkommission@dfg.de)

Iodine and inorganic iodides

Chemical name	Molecular formula	CAS number	Molecular weight [g/mol]	Melting point [°C]	Vapour pressure [hPa]	Solubility in water [g/l]
Iodine	I_2	7553-56-2	253.81	114*	0.31 (at 25 °C)*	0.33*
Potassium iodide	KI	7681-11-0	166.00	686*	1.3 (at 745 °C)*	1430*
Sodium iodide	NaI	7681-82-5	149.89	662*	1.3 (at 767 °C)*	1793*
Ammonium iodide	NH_4I	12027-06-4	144.94	405*	1.3 (at 211 °C)*	1542*
Ammonium iodate						
Magnesium iodide	MgI_2	10377-58-9	278.11	637*	no data	1480*
Calcium iodide	CaI_2	10102-68-8	293.89	740*	no data	no data

* cited from Hartwig (2014), translated

BAR (2015)	**not established**
MAK value (2014)	**not established**
Peak limitation	–
Absorption through the skin (2013)	H
Sensitization	–
Carcinogenicity	–
Embryotoxicity	–
Germ cell mutagenicity	–

For iodine and inorganic iodides, a supplement to the MAK documentation is available, which in turn is based on an IPCS report by the WHO (Hartwig 2014, translated; WHO 2009).

The following general information on the metabolism and toxicokinetics of iodine are based on the data found in the above-mentioned documents.

1 Metabolism and Toxicokinetics

1.1 Absorption and distribution

Inhaled iodine is rapidly absorbed and almost 100% of it is retained. In contrast, dermal absorption is very low (Greim 2006, translated).

After ingestion of organic and inorganic iodide, iodine is almost completely absorbed through the small intestine. The normal serum level is between 1 and 5 μg/l. The thyroid gland is the main storage organ for iodide, although it also accumulates in the salivary and mammary glands and in the stomach mucosa. A specific sodium-dependent iodide transporter in the basolateral membrane of the thyroid follicular cells (sodium-iodide symporters) is responsible for active transport into the thyroid gland (BfR 2004).

Transport and storage result in a thyroid:serum gradient of 40:1 (Burman and Wartofsky 2000). The thyroid-stimulating hormone (TSH) generated by the pituitary gland stimulates the iodide transport (Spitzweg and Heufelder 1999). In the thyroid gland, iodide is oxidized by peroxidase and bound to tyrosine, forming 3-monoiodotyrosine and 3,5-diiodotyrosine (iodination). In the case of iodine deficiency, a relatively greater amount of monoiodotyrosine is formed. The thyroid gland hormones thyroxine (T_4) and triiodothyronine (T_3) are the products of a coupling reaction of two molecules 3,5-diiodotyrosine (T_4) or one molecule diiodotyrosine and one molecule 3-monoiodotyrosine (T_3) (BfR 2004).

1.2 Metabolism and elimination

The iodine content in the body of a healthy adult is estimated to be 10–20 mg. In the thyroid gland, which acts as a storage organ, 70% to 80% of the total iodine quantity is found. In thyroid glands enlarged as a result of iodine deficiency, iodine contents of less than 1 mg were found (BfR 2004).

A part of the iodide is released again by deiodinases from the thyroid gland and the other tissues. Some hormone derivatives are excreted via the biliary pathway. Some of the iodine becomes thus available again via the enterohepatic circulation. Per day, about 20 μg is eliminated with the faeces (11%) and the major portion (89%) with the urine (BfR 2004). Absorption and elimination of iodine are in equilibrium (Burman and Wartofsky 2000; DeGroot 1966). During nursing, the mammary gland stores iodine to a greater extent and passes it on with the milk to supply the newborn infant. When the supply of iodine is sufficient, the thyroid gland stores no more than 10% of the iodine intake. In an iodine deficiency situation, it can amount to as much as 80% (Zimmermann 2009).

2 Critical Toxicity

As a component of the thyroid hormones, iodine is an essential element. The daily requirement of an adult human is given as 150–200 μg (WHO 2009).

An increased ingestion of iodine can lead to disorders of the thyroid function. Possible pathological consequences are hyperthyreosis, autoimmune diseases

(Basedow's disease and Hashimoto's thyroiditis), but also hypothyreosis and goitre (Hartwig 2014, translated; WHO 2009) (see also Section 3.2).

Exposure to very high amounts of iodide aerosols can produce so-called "iodinism", which may be accompanied by symptoms such as swelling of the salivary glands, gingivitis, metallic taste, irritation of the conjunctiva, eyelid oedema, iodine rhinitis, acneiform skin rashes and irritation of the gastrointestinal tract (Otto et al. 2002).

Excessively high iodine doses (> 300 mg/day) can induce skin lesions (acneiform exanthema (iododerma)) in humans (WHO 2009).

3 Exposure and Effects

3.1 Relationship between external and internal exposure

There are no generalizable data available for inhalation exposure to iodine or inorganic iodides and the resulting blood/plasma levels or urine concentrations. Intake of iodine by inhalation, however, increases total elimination (Smyth et al. 2011).

In a study (n = 85) involving inhalation and dermal exposure of workers to potassium iodide aerosols, concentrations of 34–2710 µg iodine/l were measured in the blood and 52–48 696 µg iodine/g creatinine in the urine (Otto et al. 2002).

In a study with 117 surgeons and surgical nurses regularly using iodine-containing hand scrub solutions and 92 non-exposed control persons, higher concentrations were found in the first group (142 µg iodine/l urine [12–822 µg/l urine] versus 89 µg/l urine [10–429 µg/l urine]) (Erdogan et al. 2013).

No other studies on this subject are available. No reproducible quantitative relationship between iodine exposure or intake and resultant internal exposure can therefore be derived.

3.2 Relationship between internal exposure and effects

The association between iodine intake and the presence of functional thyroid disorders results in a U-shaped curve (Laurberg et al. 2010). Therefore, no clear-cut delineation can be made between an oversupply or a deficiency of iodine with regard to related effects on individual health. Rather, the starting point for iodine supply is just as decisive a factor for the occurrence of iodine-associated disorders as the frequency of corresponding genetic predispositions in the affected population.

In populations, in which iodine deficiency has been prevalent for a long time, high rates of functional thyroid autonomy are associated with a lower tolerance threshold when the iodine intake is increased. A sudden increase in iodine intake can thus lead to iodine-induced thyroid gland dysfunction, without the value recommended by the WHO being exceeded. In genetically predisposed persons, the increase in iodine intake can produce Hashimoto's thyroiditis (autoimmune thyroiditis) via an increase in thyroid antibodies. The extent of this increase depends on the severity of an existing iodine deficiency and the amount of iodine supplementation. A rapid increase in iodine intake enhances the observed effects (Andersson et al. 2010; Zimmermann 2009). According to Laurberg et al. (2010), the risk of some iodine-as-

sociated diseases can, however, be correlated with the iodine intake, assessed from the mean iodine urinary excretion (see Table 1).

Table 1 Association between the iodine intake of the general population and an increase of disease risk in the population (from Hartwig 2014, translated; according to Laurberg et al. 2010)

Iodine supply	Mean urinary iodine concentration [µg/l]	Disease/effect
Severe iodine deficiency	< 25	cretinism goitre hypothyroidism
Moderate iodine deficiency	25–50	low intelligence quotient goiter hypothyroidism hyperthyroidism
Mild iodine deficiency	50–100	goitre hyperthyroidism
Optimal	100–200	
Higher than adequate (high normal)	200–300	iodine-induced hyperthyroidism immune hyperthyroidism (Basedow's disease) hypothyroidism autoimmune thyroiditis (Hashimoto's thyroiditis)
Excessive	> 300	iodine-induced hyperthyroidism immune hyperthyroidism (Basedow's disease) hypothyroidism autoimmune thyroiditis (Hashimoto's thyroiditis) goitre thyrotoxicosis

4 Selection of Indicators

In all available studies on iodine intake and elimination, the determination of iodine was mostly from spot urine, sometimes from the morning urine, rarely from the 24-hour urine. In a study on spontaneous urine samples, the analytical values in the spot urine correlated poorly with the determined iodine excretion in the 24-hour urine (Ohira et al. 2008) even after creatinine adjustment. In one study, a good correlation between the iodine concentration in the morning urine (mg/l) and the iodine concentration in the 24-hour urine (mg/day and mg/l) was observed (Nagata et al. 1998*)*.

No relevant publications are available for the concentrations of iodine in serum or plasma.

5 Methods

For the analysis of urinary iodine concentrations various methods are available (Moreda-Piñeiro et al. 2011). Some studies use the Sandell-Kolthoff method, which is based on a colorimetric measurement of the reduction of Cer(+IV) by As(+III) (cerium-arsenite method) catalysed by iodine after acidic or basic digestion (Fallouch et al. 2003; Matthes et al. 1973). In other studies methods applying ICP-MS are used (for example Michalke and Witte 2015), more rarely also direct potentiometric measurement with an iodine-selective electrode.

6 Background Exposure

An overview of more recent studies, in which the iodine background exposure in Germany has been investigated, is given in Table 2. Generally valid statements on iodine excretion in the general population cannot be made. The individual iodine intake is influenced by geogenic iodine occurrence and the associated iodine concentration in the drinking water, dietary habits and even the presence of iodine-containing aerosols (Smyth et al. 2011).

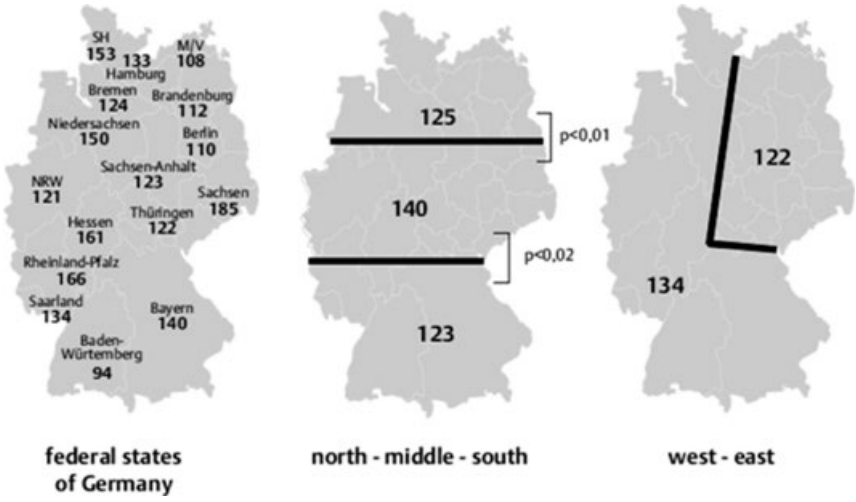

Figure 1 Medians for iodine excretion [µg/l] in various regions of Germany (Hampel et al. 2010)

Abbreviations: MV: Mecklenburg/Vorpommern (Western Pomerania); NRW: North Rhine-Westphalia; SH: Schleswig-Holstein
Niedersachsen: Lower Saxony; Sachsen-Anhalt: Saxony-Anhalt; Hessen: Hesse; Thüringen: Thuringia; Bayern: Bavaria; Rheinland-Pfalz: Rhinland-Palatinate

Table 2 Results of iodine monitoring in urine in Germany 1998–2013 (Gärtner et al. 2001 is not included, as redundant due to Manz et al. 2002; Brauer et al. 2006 is redundant due to Brauer et al. 2005; Remer et al. 2006 report only children aged 6–12 years)

Author	Period of investigation	Age	Sex	n	Median [µg/l]	MV [µg/l]	SD [µg/l]	Median [µg/g creatinine]	MV [µg/g creatinine]	SD [µg/g creatinine]
Meng et al. 1998	1989–1992	11–17	m/f	744	47			39	43	± 23.1
	1994	13–17	m/f	123	60			60	71	± 35.6
	1995–1996	11–17	m/f	982	98			101	125	± 47.5
Hampel et al. 2000	1993	10–18	m/f	212/188				73	85	± 39
	1995	10–18	m/f	865/1244				93	102	± 44
	1997	10–18	m/f	195/202				133	143	± 59
Manz et al. 2002	1996	50–70	m	278	99	107	± 73	73	83	± 45
		50–70	f	288	88	102	± 73	96	112	± 69
		recruits 17–21	m	772	83	95	± 65	57	65	± 40
Brauer et al. 2005	2002	31 ± 12	m	153					79	± 44
		35 ± 12	f	639					116	± 86
			m/f	792					109	± 81
Hampel et al. 2010	2005	18– >70	m/f	638/900	132					
Meisinger et al. 2012	2002–2006	25–88	m/f	1351/1154	110		IQ: 64–169			
	2006–2008	32–81	m/f	1292/1024	151		IQ: 97–214			
Völzke et al. 2013	1997–2001	37–65	m	2023	135		IQ: 91–187	120		IQ: 92–160
		35–61	f	2033	110		IQ: 63–170	155		IQ: 114–208

Abbreviations:
IQ = interquartile difference; m = male; f = female; m/f = no differentiation by gender made; MV = mean value; SD = standard deviation

The mean values of the iodine concentrations determined mostly in morning spot urine samples were, on a worldwide basis, given in the range of 59 µg/l in New Zealand (Thomson et al. 2001) up to 1500 µg/l in Japan (Nagata et al. 1998). In Germany, there are also considerable regional differences (see Table 2 and Figure 1). A general reference value for background exposure to iodine for Germany not be established. Regional variation up to 100% must here be taken into account. The table does not provide ranges of 95th percentiles, because all studies referenced here only report standard deviations of the mean, medians or interquartile ranges.

7 Evaluation

As, from a toxicological view point, no safe range can be given, the derivation of a BAT value for iodine is not possible. On account of the great regional differences, the derivation of a BAR ("Biologischer Arbeitsstoff-Referenzwert") is also not possible. In addition, there is also a high variability in urinalysis. The iodine concentration in urine permits no reliable statement to be made on the concrete status of the iodine supply in an individual. In a study undertaken once a month over one year, the iodine concentrations in spot urine varied by a factor of up to 26 times (Andersen et al. 2001, 2008), with variation coefficients of up to 86%, although variance could be reduced by creatinine correction and extrapolation to 24-hour urinary excretion. In a similar study with women from Switzerland, it was estimated that 10 consecutive spot urine or 24-hour urine samples would be necessary to establish the individual iodine status with an only 20% precision (König et al. 2011). Milhoransa et al. (2010) reported partly considerable variations in the 24-hour urinary iodine excretion on three different days within a week.

In the light of these uncertainties, a BAR for iodine and inorganic iodides cannot be established.

8 Interpretation of Data

A reference range for the iodine concentration in urine would have to be established on a regional basis; even within Germany, no uniform reference value can be derived.

Iodine intake and excretion correlate closely with each other with food being the main source of intake. In the light of the large variability of iodine excretion both during a single day and from day to day, no individual assessment of analytical values can be undertaken. Even if a reference range could be given, no reliable statement as to whether it has been met could be made from a single measurement (spot urine or 24-hour urine).

9 References

Andersen S, Pedersen KM, Pedersen IB, Laurberg P (2001) Variations in urinary iodine excretion and thyroid function. A 1-year study in healthy men. Eur J Endocrinol 144: 461–465

Andersen S, Karmisholt J, Pedersen KM, Laurberg P (2008) Reliability of studies of iodine intake and recommendations for number of samples in groups and in individuals. Br J Nutr 99: 813–818

Andersson M, de Benoist B, Rogers L (2010) Epidemiology of iodine deficiency: salt iodisation and iodine status. Best Pract Res Clin Endocrinol Metab 24: 1–11

BfR (Bundesinstitut für Risikobewertung) (2004) Risikobewertung von Jod. In: Domke A, Großklaus R, Niemann B, Przyrembel H, Richter K, Schmidt E, Weißenborn A, Wörner B, Ziegenhagen R (eds.) BfR-Wissenschaft: Verwendung von Mineralstoffen in Lebensmitteln. Toxikologische und ernährungsphysiologische Aspekte, Part II, BfR-Hausdruckerei, Berlin Dahlem, 201–240

Brauer VFH, Brauer WH, Führer D, Paschke R (2005) Iodine nutrition, nodular thyroid disease, and urinary iodine excretion in a German university study population. Thyroid 15: 364–370

Brauer VFH, Schweizer U, Köhrle J, Paschke R (2006) Selenium and goiter prevalence in borderline iodine sufficiency. Eur J Endocrinol 155: 807–812

Burman KD, Wartofsky L (2000) Iodine effects on the thyroid gland: biochemical and clinical aspects. Rev Endocr Metab Disord 1: 19–25

DeGroot LJ (1966) Kinetic analysis of iodine metabolism. J Clin Endocrinol Metab 26: 149–173

Erdogan MF, Tatar FA, Unlütürk U, Cin N, Uysal AR (2013) The effect of scrubbing hands with iodine-containing solutions on urinary iodine concentrations of the operating room staff. Thyroid 23: 342–345

Fallouch S, Lejeune P-J, Barbaria J, Carayon P, Mallet B (2003) Urinary iodine analysis: an alternative method for digestion of urine samples. Clin Chem 50: 780–782

Gärtner R, Manz F, Grossklaus R (2001) Representative data of iodine intake and urinary excretion in Germany. Exp Clin Endocrinol Diabetes 109: 2–7

Greim H (ed.) (2006) Iod und anorganische Iodide. Gesundheitsschädliche Arbeitsstoffe, Toxikologisch-arbeitsmedizinische Begründungen von MAK-Werten, 40th issue, Wiley VCH, Weinheim

Hampel R, Gordalla A, Zöllner H, Klinke D, Demuth M (2000) Continuous rise of urinary iodine excretion and drop in thyroid gland size among adolescents in Mecklenburg-West-Pomerania from 1993 to 1997. Exp Clin Endocrinol Diabetes 108: 197–201

Hampel R, Bennöhr G, Gordalla A, Below H (2010) Urinary iodide excretion in adults in Germany 2005 meets WHO target. Exp Clin Endocrinol Diabetes 118: 254–257

Hartwig A (ed.) (2014) Iod und anorganische Iodide. Gesundheitsschädliche Arbeitsstoffe, Toxikologisch-arbeitsmedizinische Begründungen von MAK-Werten, 57th issue, Wiley VCH, Weinheim

König F, Andersson M, Hotz K, Aeberli I, Zimmermann MB (2011) Ten repeat collections for urinary iodine from spot samples or 24-hour samples are needed to reliably estimate individual iodine status in women. J Nutr 141: 2049–2054

Laurberg P, Cerqueira C, Ovesen L, Rasmussen LB, Perrild H, Andersen S, Pedersen IB, Carlé A (2010) Iodine intake as a determinant of thyroid disorders in populations. Best Pract Res Clin Endocrinol Metab 24: 13–27

Manz F, Böhmer T, Gärtner R, Grossklaus R, Klett M, Schneider R (2002) Quantification of iodine supply: representative data on intake and urinary excretion of iodine from the German population in 1996. Ann Nutr Metab 46: 128–138

Matthes W, Kiss T, Stoeppler M (1973) Zur Jodbestimmung im Nanogrammbereich nach der Sandell-Kolthoff-Reaktion. Fresenius J Anal Chem 267: 89–95

Meisinger C, Ittermann T, Wallaschofski H, Heier M, Below H, Kramer A, Döring A, Nauck M, Völzke H (2012) Geographic variations in the frequency of thyroid disorders and thyroid peroxidase antibodies in persons without former thyroid disease within Germany. Eur J Endocrinol 167: 363–371

Meng W, Schindler A, Horack S, Lux E, Muche A (1998) Renale Jodausscheidung bei Schülern in Ostdeutschland. Eine prospektive Studie von 1989 bis 1996. Med Klin (Munich) 93: 347–351

Michalke B, Witte H (2015) Characterization of a rapid and reliable method for iodide biomonitoring in serum and urine based on ion chromatography-ICP-mass spectrometry. J Trace Elem Med Biol 29: 63–68

Milhoransa P, Vanacor R, Weber Furlanetto T (2010) Intra- and interindividual iodine excretion in 24 hours in individuals in southern Brazil: a cross-sectional study. Ann Nutr Metab 57: 260–264

Moreda-Piñeiro A, Romarís-Hortas V, Bermejo-Barrera P (2011) A review on iodine speciation for environmental, biological and nutrition fields. J Anal At Spectrom 26: 2107–2152

Nagata K, Takasu N, Akamine H, Ohshiro C, Komiya I, Murakami K, Suzawa A, Nomura T (1998) Urinary iodine and thyroid antibodies in Okinawa, Yamagata, Hyogo, and Nagano, Japan: the differences in iodine intake do not affect thyroid antibody positivity. Endocr J 45: 797–803

Ohira S, Kirk AB, Dyke JV, Dasgupta PK (2008) Creatinine adjustment of spot urine samples and 24 h excretion of iodine, selenium, perchlorate, and thiocyanate. Environ Sci Technol 42: 9419–9423

Otto A, Letzel S, Jaekel-Reinhard A, Drexler H (2002) Belastung und Beanspruchung durch Kaliumjodid am Arbeitsplatz. In: Rettenmeier AW, Feldhaus C (eds.) Dokumentationsband über die 42. Jahrestagung der Deutschen Gesellschaft für Arbeitsmedizin und Umweltmedizin e. V., Druckerei Rindt, Fulda, 94–97

Remer T, Fonteyn N, Alexy U, Berkemeyer S (2006) Longitudinal examination of 24-h urinary iodine excretion in schoolchildren as a sensitive, hydration status-independent research tool for studying iodine status. Am J Clin Nutr 83: 639–646

Smyth PPA, Burns R, Huang RJ, Hoffman T, Mullan K, Graham U, Seitz K, Platt U, O'Dowd C (2011) Does iodine gas released from seaweed contribute to dietary iodine intake? Environ Geochem Health 33: 389–397

Spitzweg C, Heufelder AE (1999) Der Natrium-Jodid-Symporter der Schilddrüse. Entdeckung, Charakterisierung, klinische Relevanz und Perspektiven. Dtsch Med Wochenschr 124: 1077–1084

Thomson CD, Woodruffe S, Colls AJ, Joseph J, Doyle TC (2001) Urinary iodine and thyroid status of New Zealand residents. Eur J Clin Nutr 55: 387–392

Völzke H, Craesmeyer C, Nauck M, Below H, Kramer A, John U, Baumeister S, Ittermann T (2013) Association of socioeconomic status with iodine supply and thyroid disorders in Northeast Germany. Thyroid 23: 346–353

WHO (World Health Organization) (2009) Iodine and inorganic iodides: human health aspects. IPCS – Concise International Chemical Assessment Document 72, WHO, Genf

Zimmermann MB (2009) Iodine deficiency. Endocr Rev 30: 376–408

Authors: M. Nasterlack, H. Drexler (Chair of the Working Group "Setting of Threshold Limit Values in Biological Materials", Deutsche Forschungsgemeinschaft), A. Hartwig (Chair of the Permanent Senate Commission for the Investigation of Chemical Compounds in the Work Area, Deutsche Forschungsgemeinschaft), MAK Commission (Permanent Senate Commission for the Investigation of Chemical Compounds in the Work Area, Deutsche Forschungsgemeinschaft)
Accepted by the Working Group: 16 October 2014