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The instability of dietary iodine supply over time in an affluent society

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Abstract. In the Bernese region, where goiter was formerly endemic, alimentary salt has been supplemented by increasing amounts of potassium iodide (KI): 5, 10, 20 mg KI/kg in 1922, 1965 and 1980 respectively. Ioduria rose from <30 μg I/g creatinine in 1920 to >100 μg I/g creatinine in the 1980s. In 1992 ioduria was estimated in 55 healthy volunteers (group A and individual B) and 234 thyroid carcinoma patients after thyroidectomy: hypothyroid patients with (C) and without thyroid remnants (D) and euthyroid patients on T4** substitution (E). The arithmetic mean iodine excretion of the healthy volunteers in group A and individual B was found to be 87 ± 40 μg I/g creatinine. This is insufficient according to the recommendations of the WHO. In all groups, the iodine excretion reached the recommended level only in some members: 24% (A, B), 19% (C), 38% (D) and 81% (E).

It was thought in the 1980s that in a formerly iodine-deficient society, iodinated salt would continue to provide an adequate supply of iodine. However, iodine intake in this affluent society has proved to be unstable. This can be attributed to modifications of eating habits, which include a reduction of total salt consumption, combined with a growing consumption of manufactured food of cosmopolitan origin, prepared using salt containing little or no iodine.

Key words. Unstable iodine supply; iodine; alimentation; goiter; salt; thyroid; thyroid neoplasms.

The understanding of problems related to iodine deficiency involves the evaluation of the local geographical setting.

During the last glaciation, the Wurm period, most central parts of all continents were covered with glaciers[1]. At the end of the period the subglacial water streams resulting from the melting ice masses eroded the superficial parts of the earth's crust, and leached out its mineral iodide, thus inducing a permanent iodine scarcity in water and soil. Extensive agriculture in densely populated areas also contributed[2]. In regions where this happened, goiter was endemic[3–12]. One such area is the Bernese region, where the present study was conducted[13–17].

The intake of dietary iodine can be assessed by measuring iodine excretion. According to the guidelines of the World Health Organization (WHO), the iodine supply of a population is sufficient if the mean daily urinary excretion exceeds 100 μg iodine/g creatinine (or 100 μg iodine/l, assuming a 24-hour urinary creatinine value of about 1.5 mg and a mean 24-hour urine volume of 1500 ml), but should preferably lie between 150 and 300 μg I/g creatinine[18]. Provided 150 μg iodide is ingested daily in a given population, the incidence of goiter is expected to drop below 3%, which represents the proportion induced by causes other than iodine deficiency[19–21].

Experimental goiter prophylaxis began at the turn of the century. Local experiments with sodium iodide tablets in schoolchildren were successful in Switzerland[6,22,23] and in the United States[4,5]. Therefore, to lower the incidence of goiter and cretinism in Switzerland on a broader scale, a salt iodination program with 5 mg KI/kg alimentary salt (=3.75 mg iodide) was approved in some cantons in 1922. Although the results in schoolchildren and in army recruits were spectacular[7,24,25], this measure only spread to involve the whole country by 1952. In 1962, iodine in salt was doubled to 10 mg KI/kg (=7.5 mg iodide). After a reduced salt intake[26] and a still insufficient iodine excretion (93 μg iodine/g creatinine, ref. 34) had been registered in the general population, salt iodination was again increased to 20 mg KI/kg table salt in 1980 (=15 mg iodide). Thereafter, 4 successive Swiss studies in 1981, 1984, 1985 and 1988 beautifully documented a progressively increasing iodine intake: 119, 127, 143 and 160 μg iodine/g creatinine respectively (refs 27–30, table 1, fig. 1).

The aim of our study, 12 years after the last nation-wide increase of salt iodination up to 20 mg KI/kg, was to reevaluate the real iodine intake of selected groups within the Bernese population via their urinary excretion of iodine, to see whether the 1992 data would confirm the results of Swiss studies in the 1980s. Iodine

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** Abbreviations. TSH = thyroid stimulating hormone, T3 = triiodothyronine, T4 = thyroxine, WHO = World Health Organization.

Table 1. Review of studies with factors related to iodine uptake in Switzerland from 1922 to 1992.

Year of KI change in salt	Study year	Population studied	n	Urinary iodine µg/g creatinine (a)	Goiter prevalence (grades II–IV) % (b)	Thyroid volume g (c)	24 h thyroid uptake of radioiodine % (d)	Reference
1922	1921	Kaisten		18 ± 8 (µg/d)	62	-	-	v. Fellenberg 1925[17]
5 mg/kg	1925	Effingen		64 ± 27 (µg/d)	1	-	-	v. Fellenberg 1925[17]
	1949	Z, autopsies	122	-	89	55	-	Gerber 1980[84]
	1956	B, elderly hospital pts	12	-	-	-	42	Steck 1972[26]
1965	1968	B, elderly hospital pts	66; 54	-	37 (20–80)	-	32	Steck 1972[26]
10 mg/kg	1975	B, hospital pts	101	-	10	-	-	Bürgi 1992[35]
	1978	B, hospital pts	77	66 ± 35	34	-	-	Geiser 1978[83]
	1979	B, S, healthy adults	770	93 ± 44	-	-	27	Schmid 1980[34]
	1979	Z, autopsies	74	-	34	32	-	Gerber 1980[84]
1980	1981	S, healthy adults	27	119 ± 76	-	-	22	Eberhard 1983[27]
20 mg/kg	1984	Nat. adult patients	112	127 ± 67	-	-	-	Mordasini 1984[28]
	1985	B, females (delivery)	101	143 ± 88	2	-	-	Bürgi 1990[29]
	1985	L, autopsies	66	-	31	20	-	Bohnhoff 1988[85]
	1988	B, school children	245	160 ± 80	0	-	-	Supersaxo 1991[30]
	1992	B, healthy adults	54	87 ± 40	-	-	34	Als (this paper)
		B, Tx, hypo, no tr	67	105 ± 64	-	-	-	Als (this paper)
		B, Tx, hypo, with tr	30	80 ± 27	-	-	-	Als (this paper)

The table shows changes in salt iodination in Switzerland in 1922, 1965 and 1980 and presents data from various studies on
a) urinary iodine excretion;
b) goiter prevalence (mean average of the whole group, all ages confronted);
c) thyroid volumes (autopsies of persons aged 41–50 years, as the mean average age of our group A probands was 48 years), when known; and
d) 24-h radioiodine uptake in the thyroid (expressed in % of total administered amount of ^{123}I or ^{131}I; in Berne the examined patients (n = 130, aged 18–91 years, mean average 64 years, median: 66 years) had been referred with a clinical suspicion of hyperthyroidism, but were found euthyroid thereafter on the basis of blood serum values of TSH and/or TRH).

The different groups of probands and patients originate from various geographical locations, with a different iodine content in soil and drinking water. Thus, data comparison between different towns indicates only a trend, whereas longitudinal data comparison within one town, as for instance Berne, leads to safer conclusions.
(B = Berne, L = Lausanne, Z = Zürich, S = Solothurn, Nat. = nationwide, pts = patients, Tx = thyroidectomy, hypo = hypothyroid, tr = thyroid remnants). Iodine excretion in 1921 and 1925 is expressed in µg/day, later on in µg/g creatinine (arithmetic mean ± 1SD).

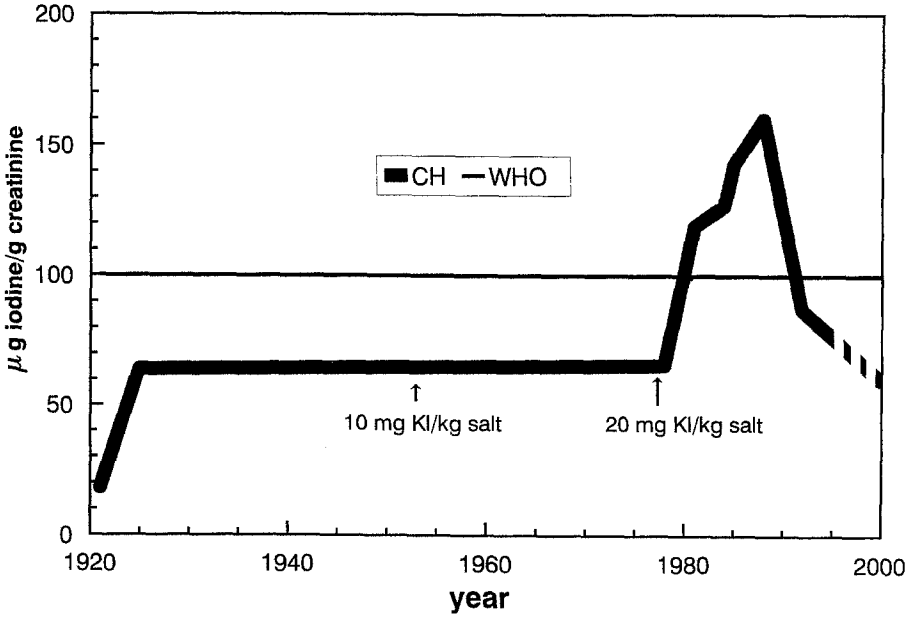

Figure 1. Historical evolution of urinary iodine excretion in Switzerland from 1920 to 1992 (refs 26–30, 34, 83). Subgroups of the Bernese population are still/again living in a situation of iodine deficiency. The horizontal black line represents the minimal daily excretion of iodine as recommended by the WHO: 100 µg I/g creatinine[18].

Table 2. Characteristics of the 4 groups of volunteers and patients and of individual B.

Group	Persons	Specification	n	Sex ratio	Age* years	Thyroidal status	TSH* mU/l
A	Healthy volunteers (hospital staff, friends)	Control population	54	36 F 18 M	48 (19–75)	normal EUTHYROID	1.50
B	Healthy volunteer	Intra-individual variation of results (over 12 months)	1	1 F	32	normal EUTHYROID	1.80
C	THYROID CARCINOMA differentiated	6 weeks after thyroidectomy; no T4 for 6 weeks, no T3 for 2 weeks	30	22 F 8 M	49 (18–82)	thyroid remnants after surgery HYPOTHYROID	62.80
D	THYROID CARCINOMA differentiated	6 weeks after thyroidectomy and after ^{131}I-ablation; no T4 for 6 weeks, no T3 for 2 weeks	67	51 F 16 M	66 (18–84)	no thyroid remnants HYPOTHYROID	76.30
E	THYROID CARCINOMA differentiated	After thyroidectomy and after ^{131}I-ablation; substitution with T4 (0.15 mg/d)*	137	93 F 44 M	54 (18–88)	no thyroid remnants EUTHYROID	0.80

The classification from A to E mirrors the evolution of hormonal status during the classical treatment course of differentiated thyroid carcinoma (*arithmetic mean and range). TSH = thyroid stimulating hormone.

excretion was measured in a group of 55 healthy subjects and in a larger group (n = 234) of thyroidectomized patients who were under surveillance after initial treatment of their differentiated (papillary and follicular) thyroid carcinoma.

In addition to the measurements of iodine excretion, changes in the intake of iodized salt and other iodine-containing food in Switzerland were assessed using available statistical and other information.

Subjects and methods

Subjects

289 persons; 55 volunteers and 234 patients with thyroid carcinoma, were investigated from October 1991 through November 1992 (table 2). No volunteer or patient entering the study was on any special diet. Informed oral consent was given.

Group A. The 54 subjects were healthy volunteers, hospital staff and friends of the first author, with a fairly high educational and socioeconomic level, living in an urban environment. Their clinical histories did not reveal any signs or symptoms of thyroid disease or a history of radio-opaque dye ingestion. No acute or chronic disease was registered.

Individual B. One healthy female volunteer, who was investigated repeatedly (n = 9), to assess intra-individual variability.

Groups C, D, E. Thyroid carcinoma patients from the whole administrative area of Berne (about one million inhabitants). Most of them were of rural origin. The classification of these groups from C to E outlines the evolution of hormonal status during thyroid ablation in the classical treatment course of thyroid carcinoma.

C) 30 patients with differentiated thyroid carcinoma, at least 6 weeks after thyroidectomy, with thyroid remnants of variable extent (3–10 g) not yet eradicated with radioiodine. Either they had never had hormone substitution, or T4 and T3 substitution had been withdrawn 6 and 2 weeks, respectively, before the investigation.

D) These 67 patients had no thyroid remnants after thyroidectomy and eradication with radioiodine (evidence from ultrasonography and ^{131}I scan); T4 and T3 substitution had been withdrawn 6 and 2 weeks, respectively, before the study.

E) Most of the patients (n = 137) were seen after operation and thyroid eradication. They were devoid of thyroid remnants, but euthyroid (as assessed by TSH, TT3 and TT4 values) under T4 substitution.

Methods

All laboratory analyses were performed at the Department of Clinical Chemistry, Inselspital, University of Berne. The complete 24-hour urines were collected on an outpatient basis, and total iodine (nmol/l) and creatinine (μg/l) were measured in an aliquot which had been frozen immediately after sampling. The plastic and glass utensils used for collecting and manipulating urine samples were carefully washed with iodine-free detergent. During the period of urine sampling, venous blood was taken for the determination of thyroid stimulating hormone (TSH) in mU/l, which was performed in our laboratory on duplicate samples (RIA Kallestad). Samples were diluted at 1:10 if the value exceeded 50 mU/l. Creatinine (in μmol/l) was determined with the picric acid method after Jaffé[31]. Total iodine (nmol/l) was determined using a Technicon Autoanalyzer. The same wet-ash method (based on the Kolthoff-Sandell reaction[32]) was used as in the previous studies of iodine excretion performed in the Bernese region since 1975[27–30,34,83]. The analyses were carried out by an

Table 3. Urinary excretion of iodine.

Group	Ioduria/24 h		Creatininuria g/24 h	Iodine/Creatinine	
	nmol/24 h	µg/24 h		nmol/mmol	µg/g
A (Volunteers)	902 *(846)* (343–2566) 0.43	114	1.6	77 *(74)* (29–224) 0.42	87
					p(A, C) > 0.05
B (One normal longitudinally)	823 *(697)* (482–2030) 0.54	104	1.6	62 *(48)* (28–151) 0.58	70
					p(A, C) > 0.05
C (After surgery)	908 *(846)* (425–2117) 0.44	115	1.8	71 *(67)* (4–137) 0.35	80
					p(C, D) < 0.05
D (Surgery + ^{131}I ablation)	1041 *(914)* (343–2745) 0.48	132	1.6	94 *(83)* (43–446) 0.61	105
					p(D, E) < 0.05
E (Substitution)	1582 *(1365)* (484–6250) 0.51	200	1.4	132 *(117)* (37–435) 0.5	148

Urinary excretion of iodine (arithmetic mean, (*median*), (range), coefficient of variation) in nmol/24h, in µg/24h, in nmol/mmol creatinine and in µg/g creatinine, creatininuria in g/24h in the 4 groups of volunteers and patients and in individual B. The calculation of a statistically significant difference between individual B and the patient/volunteer groups has been omitted.
The indicated results of the arithmetic mean values of µg iodine/g creatinine have been calculated by summing up and then averaging all concerned n values. A division of the arithmetic means of the ioduria in µg/24 hours by the creatininuria in g/24 hours would not yield the same results.

experienced scientist who had also been involved in these earlier trials[33].

In order to be able to compare different data sources, we adapted our terminology to that used in previous Swiss studies[26–30,34,83] and by the WHO[18] and expressed the urinary iodine excretion as nmol/mmol creatinine or as µg/g creatinine (conversion factors from the molar to the metric system for urine: 0.89). For information, the absolute daily excretion of iodine as nmol/24 h and as µg/24 h is also given in table 3.

Statistical treatment

To test whether or not a statistically significant difference existed between arithmetic mean results of the groups evaluated we carried out statistical analyses using the Wilcoxon-Rank sum test. For descriptive purposes, the results are expressed as the arithmetic mean, plus/minus standard deviation (SD) or range, unless otherwise indicated.

Review of dietary sources of iodine

The change in the intake of iodine from a number of important dietary sources, especially salt, was estimated using statistical information from the Swiss Federal Statistical Office and from the food industry.

Results and discussion

Values for urinary iodine excretion

The arithmetic mean urinary iodine excretion of group A volunteers was 87 ± 36 µg iodine/g creatinine (table 3). The urinary iodine excretion of subgroups of the Bernese population in 1992 was below the minimal recommended WHO standard of 100 µg iodine/g creatinine in 76%, 81% and 62% of patients in groups A, C, D, respectively. The longitudinal data from individual B showed an insufficient iodine excretion on 8 out of 9 occasions (89%, fig. 2). In group E, thyroidectomized patients with T4 substitution, the percentage whose excretion was below the recommended value decreased to 19%. Values indicative of a severe iodine deficiency (< 50 µg iodine/g creatinine) were found in 11%, 12% and 3% of patients in groups A, C and D, respectively, and in 4 out of 9 samples in individual B. The arithmetic mean urinary iodine excretion in group D patients (105 µg/g creatinine) was near the lower threshold of WHO recommendations. Iodine depletion after withdrawal of thyroid hormone in thyroidectomized patients (group D) was dramatically mirrored by a 29% difference in the arithmetic mean urinary iodine excretion ($p < 0.05$) between groups E and D. The arithmetic

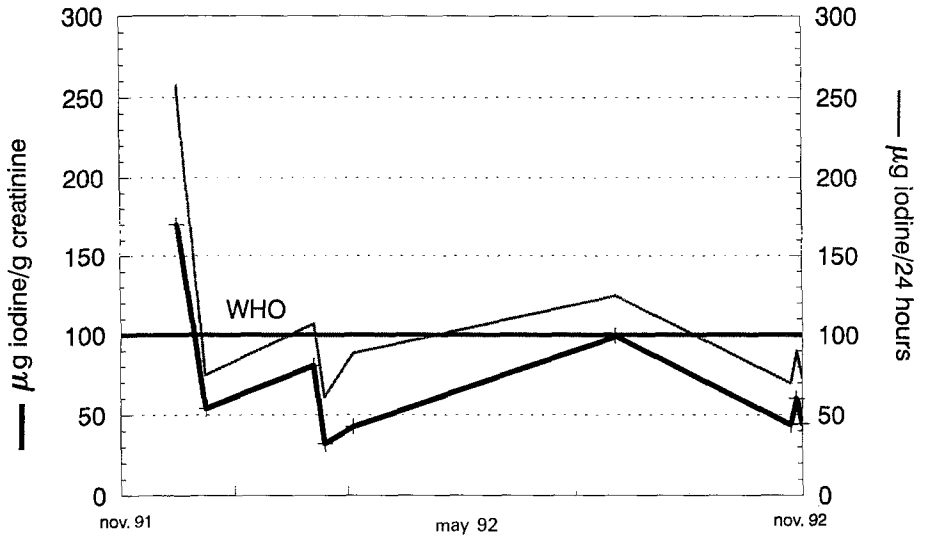

Figure 2. Intra-individual variability of urinary iodine excretion (individual B). Results (μg I/g creatinine and μg I/24 hours) on the basis of 9 successive measurements in the same individual (B) over 11 months. Only 1 out of 9 samples satisfied the minimal WHO recommendations: 100 μg I/g creatinine (ref. 18, see horizontal fat line). As both excretion curves are roughly parallel, the influence of creatininuria is not significant.

mean and median values for urinary iodine in nmol/24 h and in nmol/mmol creatinine, together with the ranges and coefficients of variation, are given in table 3. The median value systematically lies below the arithmetic mean value.

Women in groups A, D, and E excreted on average more iodine/g creatinine than men; 18%, 23% and 21%, respectively. In group C the increase was 82%, but the number of patients in this group was rather low. No significant 4-seasonal difference was registered ($p > 0.05$) for any of the groups. The 24-hour arithmetic mean urinary creatinine excretion was 1.65 ± 0.44 g in all groups combined. It was higher in men than in women: 54%, 57%, and 56% for groups A, D, E, respectively (table 3). In group E, the urinary creatinine excretion was insignificantly lower than in group A ($p = 0.59$, table 3), with an identical female/male sex ratio (table 2).

Frequency of iodine deficiency

The results described above show that, in general, the groups investigated in Berne in 1992 showed an iodine excretion considerably lower than that recommended by the WHO. Only 24% of the healthy persons (group A) excreted more than the recommended minimum of 100 μg I/g creatinine (table 3, fig. 1). Furthermore, measurements of urinary iodine excretion over time in one single person (intra-individual variability, case B, fig. 2) demonstrated that on only one occasion out of 9 evaluated was it within the WHO-sufficient range. In group D (no thyroid remnants), urinary iodine excretion was slightly higher than in group C (with thyroid remnants) and the mean excretion did reach the minimum recommended threshold. However, in all the groups A, C, and D a considerable number of individuals (76%, 81%, and 62%, respectively) excreted less than 100 μg iodine/g creatinine. Some individuals (11%, 12% and 3% in groups A, C, D, respectively) excreted even less than 50 μg iodine/g creatinine; they presented a severe iodine deficiency. The fact that the median value of urinary iodine excretion was systematically below the arithmetic mean value points even more strongly to the existence of iodine-deficient subgroups within this Bernese population.

The only exception to the general tendency towards iodine deficiency was shown by group E. In these patients, hormonal substitution with synthetic T4 (0.15 ± 0.04 mg T4/day) provided an average iodine supply of 98 ± 3 μg I/day in addition to iodine from other sources.

The evidence of inadequate iodine intake in group A, and in repeated measurements in individual B, were of prime importance for assessing the iodine intake of the normal population. In the 97 thyroidectomized hypothyroid patients of groups C and D, urinary iodine excretion accurately reflected iodine intake, because the physiological detour of iodine via organification was not present. The arithmetic mean results of groups C and D, with a larger case number than group A and individual B, supported the conclusion that iodine intake was generally below the WHO recommended level. Another prospective study, done in Berne over the same time span, including about 200 pregnant women and 71 healthy volunteers, supports the present conclusions (Brander et al., unpubl. data).

Group A and individual B might be considered not to be typical for the Bernese population in general, since they came from an urban area and had a rather high educational and socioeconomic level. Their dietary habits tended to be somewhat different from those of a

more rural population; owing to health-consciousness their consumption of pickled and smoked meat was moderate. However, most of the members of groups C, D and E were from rural areas, and possibly had a higher intake of smoked and pickled meat. The fact that the results showed the same trend in these groups indicates that the inadequacy of iodine intake in group A and individual B was not likely to be due to a special feature of the eating habits of this group.

Comparison of the results with those of previous studies

Previous Swiss studies based on urine samples, between 1981 and 1988, reported a sufficient iodine excretion in the cities of Berne[29,30], Solothurn[27] and nation-wide[28] since the last modification of salt iodination in 1980. We therefore have to consider why our 1992 results (table 1, fig. 1) from the same geographic area[29,30] showed a different picture.

One question that has to be considered is the possible influence of differences in the socioeconomic status of the study subjects, which could affect dietary habits, and also differences in geographical origin, which could lead to variation in the iodine content of the local soil, and hence of the drinking water and locally-produced foodstuffs. Our group A and individual B were of about the same socioeconomic status (hospital staff and friends of the first author) as those in reference 27 (table 1). They also all originated from a pre-Alpine region. The patients and volunteers of references 29, 30 differed, however, in age, sex distribution and the state of pregnancy[29]. In reference 28, the patients had been recruited nation-wide. Even if the comparison with these previous studies may suffer from some bias, it can still usefully be made.

The urinary creatinine excretion in previous Swiss studies[28,34] did not significantly differ from our group A results, and though in group E the 24-hour urinary creatinine excretion was slightly lower, the difference was not significant ($p_{A,E}$: 0.59). The small difference between groups A and E could be related to a not fully understood interference by the hormonal T4 substitution. As the urinary excretion of iodine in group E (in μg I/g creatinine) was significantly higher ($p_{D,E}$: <0.05) than in the other groups, there is no significant influence of renal function which might explain the observed divergences in the urinary excretion of iodine.

Data apart from measurements of urinary iodine can also provide indications of the level of iodine intake in a population; for example the prevalence of goiter, thyroid volume and the percentage of radioiodine uptake in the thyroid at 24 hours (table 1). In Switzerland, such data are scarce, and they have mostly been collected retrospectively, at variable time intervals and within highly variable patient/proband populations (often on distinct population than that used to measure the urinary excretion of iodine), and in different places: in Berne, Zürich, Lausanne and Solothurn, and nation-wide. A longitudinal comparison of data originating from towns with a different iodine content in soil and drinking water (for instance Berne and Solothurn) has to be considered with caution. At the most, it could indicate a trend. For Berne, the data collected during the 1980s and '90s are not numerous enough to allow a conclusion. Thus, urinary iodine excretion remains the most reliable monitor of iodine supply.

The divergence between the present results and previous ones is extremely unlikely to be due to the methodology used. Firstly, the iodine determinations were performed under exactly the same technical conditions and by the same experienced scientist[33] as those in the previous Swiss studies[27–30,34,83]. Secondly, as the arithmetic means and the standard deviations of the 24-hour urine collections (our study) and of urine samples (former Swiss studies) in general do not differ significantly[36–38], the results for urinary excretion of iodine of all existent studies can be reliably compared. Moreover, each of our groups A, C, D and E contained a sufficient number of volunteers/patients to produce reproducible arithmetic means and standard deviations for urinary iodine excretion; i.e., each of our patient groups was reliable per se[36].

Nutrition yesterday and today

Iodine intake depends on the amount of iodine present in the diet. When Hunziker investigated iodine excretion and goiter prevalence in rural areas in 1922, he found that there were sharply delineated areas of alimentary autarchy[13,17,39–41]. A goiter-free village with a high iodine content in rock, soil and drinking water could be only 8 km removed from a village where iodine resources were severely deficient and goiter was endemic. In more recent times, the influence of the geographical inhomogeneity of iodine distribution has been reduced, because iodine supply via food has become subject to more and more external influences.

Today, the quality, composition, number and volume of meals are different from those of former generations (ref. 42, fig. 3). One-person households increased from 18.7% in 1970 to 32% in 1990 (**Ref. A*), and the percentage of women working outside the home has risen. In parallel, more and more meals are eaten away from home[43]. This 'eating-out' is illustrated by the fact that as much as 60% of the total Swiss bread production nowadays is consumed outside the home, whereas at the beginning of the century, most bread was eaten around

* Ref. A: in 'Statistische Quellenwerke der Schweiz-Volkszählung 1970', Eidgenössisches Statistisches Amt Bern 1972, Heft 476, Table 2.01, pp. 12–13; 'Volkszählung 1980', Bundesamt für Statistik Bern 1983, Heft 702, Table 5.01, p. 172; and 'Eidgenössische Volkszählung 1990, Haushalte und Familien, geographische Tabellen', Bundesamt für Statistik Bern 1993, Table 7.003-00, p. 108.

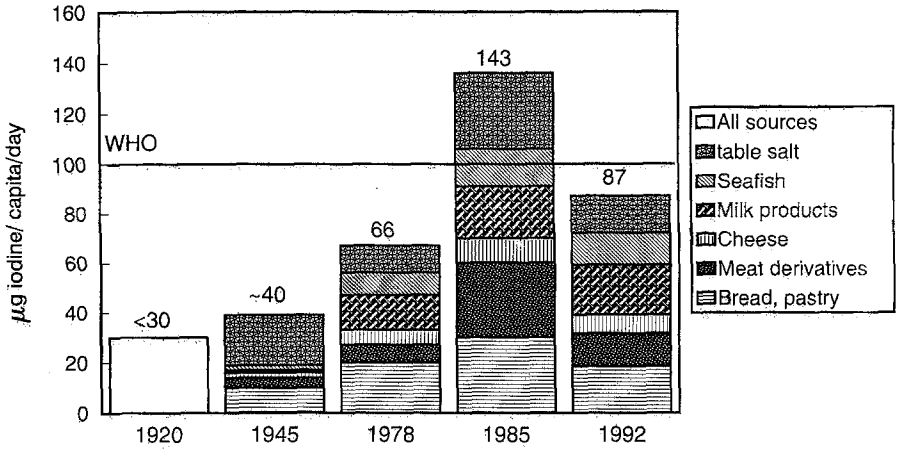

Figure 3. Estimation of iodine supply from basic foodstuffs from 1920 to 1992 in Switzerland (refs 29, 30, 42, 48, 52, 56, 61, 73, 77–79, 82, 83). Note a higher contribution of home-added kitchen salt in 1945 than later on, due to the smaller intake of industrially prepared foods. The changing alimentary habits over time (maximal meat intake in 1978, reduction of bread intake since 1920, increase of fish intake since 1920, increased intake of milk products and cheese since the 1960s, reduced salt intake) have been taken into account. In contrast to table 5, the real iodine content of salt has been considered here.

the family table ('Schweizerische Bäckereifachschule', pers. commun.).

Another factor which affects eating behaviour is a generalized 'back-to-nature' trend, pushed along by more and more insistent publicity, which makes people anxious to avoid conservation agents, toxins, microbial products or contaminants resulting from pollution. A recent awareness of diseases induced by a high intake of animal fat and salt, promoted mainly by cardiovascular research groups[44–52], has favored new alimentary trends based on fiber-rich, salt-poor diets.

Another development is that, as a result of improved conservation techniques, more and more industrially prepared foodstuffs are now being imported from other European countries and overseas (**Ref. B*). As a consequence, the variety of victuals offered for sale has increased enormously; for instance from 3000 to 6500 articles over 15 years in a popular Swiss megadistribution store[53]. In contrast to Swiss products, industrial food preparations in most European countries until the beginning of the 1990s were produced using salt which was not, or was only minimally, iodinated.

Iodized salt as a source of iodine

It has been shown that in Switzerland iodized salt is the main provider of iodine[34].

Salt distribution in Switzerland is regulated by a monopoly of production and trade. One federally-owned salt factory ('Vereinigte Schweizerische Rheinsalinen') in Schweizerhalle in north-western Switzerland provides the whole country with iodinated salt (table 4), with the exception of Canton Vaud. Table or kitchen salt is sold iodized (20 mg KI/kg) and non-iodized in retail shops. The two types are sold at the same price, and the packets are stacked near to one another on the same shelf. Salt for semi-industrial purposes (bread, cheese, smoked meat, tinned foods, prepared meals) is available in 25 or 50 kg bags, iodized at 0 or 20 mg KI/kg. Salt for industrial purposes (soups, broths, aromatizers), which is available in bulk in solid or fluid form in wagon containers, is iodized only at 0 or 10 mg KI/kg (table 4).

In 1992, 75% of the total alimentary salt was purchased by the food industry ('Schweizerische Rheinsalinen', pers. commun.). Swiss law allows manufacturers to use salt which is either uniodinated, or iodinated at 10 or 20 mg KI/kg. Legislation in many countries has prohibited the import of food with any iodine supplements (Scandinavia, Denmark), or with iodine supplements as high as those found in Switzerland (FRG until 1991). There-

Table 4. Salt distribution in Switzerland.

		1991 %	1992 %
1 kg packets (household)	non-iodized	9.2	14.6
	iodized (20 mg KI/kg)	90.8	85.4
25/50 kg bags (bread, cheese, meat products)	non-iodized	5.6	11.3
	iodized (20 mg KI/kg)	94.4	88.7
Loose (industrial)	non-iodized	17.7	17.5
	iodized (10 mg KI/kg)	82.3	82.5
Total	non-iodized	11.7	14.7
	iodized (10 mg KI/kg)	30.1	30.4
	iodized (20 mg KI/kg)	58.2	54.9

Percentages of (non-)iodized alimentary salt (10/20 mg KI/kg) sold in Switzerland by the 'Vereinigte Schweizerische Rheinsalinen' in 1991 and 1992. A partial explanation for a still too-low iodine supply to subgroups of the Bernese population in 1992 is given by the fact that only half of the consumed alimentary salt is iodized at 20 mg KI/kg.

* Ref. B: General Direction of Swiss Customs, internal statistics on foreign trade: 'Aussenhandel nach Kapiteln des Zolltarifs, Commerce extérieur par chiffres du tarif douanier. 1. Gesamtverkehr-Trafic général' pages 520, 528, 886, 876.

fore the export-oriented Swiss food industry has had to use less iodinated salt. In 1992, 82% of industrial salt produced in wagon containers was iodized at only 10 mg, which represented a 20% increase compared to 1987. The percentage of salt iodized at the higher level of 20 mg KI/kg was only 58.2 in 1991 and 54.9% in 1992.

Thus, a substantial proportion of the salt consumed is little iodized (10 mg KI/kg) or not iodized at all.

Not only has the level of iodide in salt been dropping, but the consumption of salt has been decreasing steadily. This has been occurring since the beginning of the century[85]. In 1920 the arithmetic mean salt consumption was 30 g/capita/day, and it decreased to 17.5 g in 1964 and to 12 g in 1969 ('Schweizerische Rheinsalinen'). Estimations on the effective salt intake in Switzerland from 1980 to 1990 vary between 8 and 12 g[28,48,54–59]. In a study in 1987, the arithmetic mean daily salt intake over 7 days in 7 Swiss hospitals, for instance, was 7–9 g, depending on the quantity of food consumed, among patients in the general department taking no special diet[58]. The same result (8.2 g NaCl) was obtained in 1992 with a single day analysis in the Inselspital in Berne (Dept. of Dietetic Counseling, pers. commun.). Of course intra-individual variations are large; they have been estimated at 3–35 g/day[57]. Salt intake is probably strongly related to cultural tradition, and can vary widely between countries; thus, in the United States in 1968, Oddie described a salt intake of 6.5 g/capita/day, which was much lower than that in Switzerland in 1968 as well as 1992[63].

The general consumption of salt could have stabilized around 9 g/day since 1990, as mirrored by the constant quantities sold since then by the 'Vereinigte Schweizerische Rheinsalinen'. As, in affluent societies, about 60–90% of the daily salt intake seems to occur via processed foodstuffs[48,60–62], the reduction of salt intake is apparently the result of a reduction of the amount of salt in these foods. Another change leading to a lower consumption of sodium chloride is the new habit of using 'false salts' such as sodium glutamate (50% NaCl, iodized at 10 mg KI/kg) in cooking.

If an individual's total daily salt intake ($\approx$8–10 g) were provided exclusively via kitchen salt iodized at 20 mg/kg, a sufficient iodine supply (at least $20 \times 8 = 160$ µg/day) would be secured. However, the real situation is different. A realistic estimate (lithium marker technique, ref. 64) seems to be that 1–3 g of kitchen salt is used per day, providing about 15% of the total salt intake[61,65–68]. In Germany, where, as in Switzerland, the consumer can choose whether to buy iodized salt or not, the iodine excretion of subjects using iodized table salt (20 mg/kg) was compared with that of those using non-iodinized salt, and very little difference was found ($p > 0.05$, refs 68–70) either in iodine excretion or in goiter prevalence.

On the basis of the above figures, it can be calculated that if household salt were supposed to be the main vehicle for iodine supply, if as little as 1.5 g of salt is likely to be consumed per day, in order to yield the WHO-recommended iodine excretion[71] its iodine content would have to be relatively high ($\approx$66 mg iodine/kg). Figure 3 illustrates the relative contribution of table salt to the total daily iodine supply: it was much lower in 1992 than previously.

Other dietary sources of iodine

The main part of our daily salt/iodine supply comes from (semi-) industrially prepared foods[48,60–62,72], the most regular sources being milk and cheese, bread, pastry, meat preparations, fish derivatives, soups, condiments and the colorant E 127, containing erythrosine (refs 48, 61, 62, 72–74, fig. 3). Amounts in fresh fruit and vegetables are negligible. The iodine content of manufactured or semi-manufactured food depends largely on the amount of iodine in the salt used in its preparation. Most bakers, butchers and cheesemakers in Switzerland, questioned by telephone in 1993 on the use of iodized versus non-iodized salt, were unaware of the health implications of iodine (Nadray and Als, unpubl.). Indeed, they were rather afraid of any 'unnecessary' additives to traditional 'pure' products such as cheese and meat[75].

An estimate of the daily iodine intake via basic aliments in Switzerland was calculated on the basis that 100% of the salt used in preparation had been iodized at 20 mg KI/kg (table 5). The extrapolated value of 78–109 µg (arithmetic mean: 94 µg) daily iodine supply in table 5 still lies below the minimal recommended WHO threshold of 100 µg iodine. Furthermore, it is almost certainly over-optimistic; the real figure could be 30–50% lower, as only about half of all salt consumed is actually iodized at 20 mg/kg.

Cheese is mostly (81%) manufactured with iodized salt (20 mg KI/kg, table 5). The consumption of cheese and milk products has increased by a factor 8 over the last 30 years, and by 30% over the last 15 years, but due to changes in consumer demand and also to a modernized cheese-making process, the salt content has diminished in many cheeses and is maximal just below the rind[76]: for instance in the traditional Sbrinz and Emmental by 30 and 50% respectively, compared to the first half of the century[73]. In Gruyere, the most frequently eaten cheese in Switzerland (21% of the market, ref. 77), salt content has decreased by 20% in over 40 years[73]. Moreover, the cheese is manufactured with coarse, non-iodized salt, for technical reasons. In 1992, the arithmetic mean daily cheese consumption of 40 g/capita provided an arithmetic mean supply of 10 µg of iodine (ref. 77, and information from the 'Schweizerische Käseunion' and 'Genossenschaft für Weich- und Halbhartkäse').

Table 5. Estimation of arithmetic mean iodine supply (µg/capita/day) from basic foodstuffs in Switzerland.

	Average intake of aliment g/day	Average iodine content in µg/100 g aliment	Calculated iodine supply µg/day	References
Bread	130	18	24	'Richemont Bäckereifachschule' (IS) Blumenthal 1983[78]
Meat derivatives	50	34	17	'Ausbildungszentrum für die Schweiz. Fleischwirtschaft' (IS) Erard 1991[77] Arab 1982[48]
Cheese	40	25	10	'Schweiz. Käseunion' (IS) Sieber 1987[73] Erard 1991[77]
Milk + derivatives	350	3–12 7.5	(10–41) 26	Sieber 1991[42] Bürgi 1982[79] Erard 1991[77]
Sea-fish	15	120	17	Swiss Customs (food input IS) Spinnler 1984[56] Merke 1968[82]
Total			94 (78–109)	

Calculations have been done making the assumption that exclusively 20 mg KI/kg salt was used (IS = import statistics). In reality, only about 50% of salt is iodized at 20 mg KI/kg, and the effective daily iodine supply via bread, meat derivatives and cheese is probably much lower.

Bread is mostly (97%) prepared with salt iodized at 20 mg KI/kg ('Richemont Bäckereifachschule', pers. commun.) (table 5). However, bread consumption has decreased steadily over 3 generations from 400 g/capita/day at the beginning of the century, to 200 g in 1960, and to 130 g today (ref. 78, Swiss Federal Statistical Office). As the salt content of bread has also been reduced, by 15% over 6 years (from 2 to 1.7 g/100 g bread), this regular source of iodine supply has been further reduced. The calculated arithmetic mean daily intake of iodine through 130 g bread in 1992 was 24 µg/capita (ref. 42, 'Bäckereifachschule Luzern').

The iodine content of meat derivatives is variable. It may be up to 30 µg% in 'white' sausages (table 5), but in contrast, for the preparation of pickled meat, for instance, coarse non-iodized salt is preferred because of an unproven suspicion that iodine might induce an undesirable colour change ('Ausbildungszentrum für die schweizerische Fleischwirtschaft', Spiez, pers. commun.). As far as fresh animal products of Swiss origin are concerned, the supply of iodine through meat, milk and eggs is generally low: 3–12 µg iodine/100 g milk, depending on the artificial food adjuncts used in summer and winter, respectively[79]. It can, however, reach astonishing peaks (up to 4000 µg/kg milk) if highly iodized enteric or external antiseptics are applied, or mineral food supplements are fed to the cattle[80,81]; but this is rather exceptional in Switzerland[42]. Seasonal differences in iodine excretion, mainly in children drinking lots of milk, have been explained in this way; our results did not show significant seasonal differences in iodine excretion (fig. 2).

Over the last 15–20 years, although proportionally more pork and poultry has been consumed, the general consumption of meat has been decreasing in Switzerland. In 1992, the consumption averaged 150 g meat/capita/day, of which 1/3 was processed[42]. The calculated daily iodine intake through 50 g meat preparations/capita in 1992 gave an arithmetic mean of 17 µg (refs 42, 48, 77, 'Ausbildungszentrum für die schweizerische Fleischwirtschaft').

Sea-fish (but not the fish from Swiss lakes) is a good source of iodine. Consumption, calculated from the import statistics of the ministry of foreign trade, is 5.2 kg sea-fish/capita/year or 15 g/capita/day. Assuming a mean iodine content of 120 µg% (ref. 81), the calculated daily iodine intake via 15 g sea-fish/capita in 1992 gives an arithmetic mean of 17 µg (ref. 42, Swiss Federal statistics on foreign trade) (table 5).

Conclusions

As our pattern of food intake changes and diversifies, it is not surprising if the supply of one of its constituents, either naturally occurring or an artificially-added supplement, does not remain stable over time. This is clearly the case with iodine in the Berne region, and as a result the problem of iodine deficiency has arisen again. A solution to the problem seems to lie in the utilization of iodized salt and naturally iodine-rich foods, such as sea-fish.

An open market for foodstuffs could have various effects on iodine supply. On the one hand, it could mean that foods which have a naturally high iodine-content because they have been produced in areas which have

iodine-rich soils (grains, vegetables, animal food) would enter the country. On the other hand, if the national regulations of exporting and importing countries for the use of iodized salt do not change, the result could be a further reduction in the dietary iodine supply.

The former alimentary autarchy of the village or region no longer exists. Altered dietary habits in the context of extensive international food exchanges have led to the supply of iodine becoming unstable and uncontrollable. This has serious implications in a society where iodine-deficiency existed formerly, and was only rectified by the widespread use of iodinated salt. Therefore, the sources of iodine supply should be diversified and improved; the present level of salt iodination should be increased; there should be a systematic iodination of salt used in manufactured food; naturally iodine-rich aliments should be promoted and, last but not least, the addition of iodine to fertilizers should be considered.

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