

Iodine Nutrition, Nodular Thyroid Disease, and Urinary Iodine Excretion in a German University Study Population

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We determined the influence of different nutritional factors on the urinary iodine excretion in an East German university population. First, we assessed iodine excretion in spot urine samples. Second, we measured iodine content in the university canteen meals, where approximately 20% of the probands had regular meals. Third, we used a special food questionnaire to assess for other sources of nutritional iodine intake, namely iodine tablets, fish consumption, etc. Fourth, we determined the actual prevalence of goiter and thyroid nodules in our probands by high-resolution ultrasonography. The mean urinary iodine excretion in our cohort was $109 \pm 81 \mu\text{g/g}$ level indicating a borderline adequate iodine intake (100–200). The frequency of thyroid nodules was 30% and the frequency of goiter 11%. Thyroid volumes greater than 18 mL and 25 mL were considered to be enlarged in adult women and men respectively. Urinary iodine excretion was not related to the presence of goiter or thyroid nodules. In addition urinary iodine excretion did not vary with regular consumption of canteen meals, which contained approximately 50% of the daily recommended iodine intake. In contrast probands with regular supplementary intake of iodine tablets had significantly higher values of urinary iodine excretion ($169 \pm 130 \mu\text{g/g}$) compared to participants without ($103 \pm 87 \mu\text{g/g}$). No other single nutritional factor (e.g., salt, milk, or bread) had a statistically significant impact on urinary iodine excretion or was able to raise the urinary iodine excretion above the level of marginal iodine deficiency. In summary, the nutritional iodine intake in a Saxonian study population was found to be close to the margin of iodine deficiency. This shows insufficient supplementation of iodine through iodized salt/industrialized food production.

Introduction

IODINE DEFICIENCY remains a major health problem in many European countries after the turn of the century, despite many efforts to improve nutritional iodine supply and an increasing awareness in the general population about the role of iodine in the prevention of thyroid disease.

The annual health care costs for thyroid disease in Germany have recently been estimated to amount to 1.3 billion U.S. dollars (1) with up to 30% of the adult German population displaying an enlarged thyroid gland and up to 50% presenting thyroid nodule(s) according to a large survey by Hampel et al. in 1995 (2).

Several studies have recently readdressed the issue of iodine supply in Germany in different age groups. Gärtner et al. (3) investigated urinary iodine excretion in selected risk groups for iodine deficiency. The mean daily iodine excretion was $134 \mu\text{g/d}$ in 278 men 50–70 years of age, $117 \mu\text{g/d}$ in 288 women 50–70 years of age, $125 \mu\text{g/d}$ in 772 conscripts, and $74 \mu\text{g/d}$ in 53 breast-feeding women (3). Rendl et al. (4) have investigated a group of schoolchildren in Würzburg and found that the proportion of samples with concentrations below $100 \mu\text{g/L}$ or below $50 \mu\text{g/L}$ was 15.4% and 4.3%,

respectively (4). Liesenkotter et al. (5) have screened a cohort of children 3–15 years of age from Berlin and found a mean iodine concentration of $115.8 \mu\text{g}$ iodine per gram creatinine (5).

While these studies point to a significant improvement in the general iodine supply in different areas of Germany, the underlying reasons for this improvement (e.g., altered nutrition, usage of iodinated salt) and its consequences for thyroid pathologies are less obvious. To clearly assess a population's iodine status, the World Health Organization (WHO) recommended monitoring the iodine content in salt at the production level, measurement of the urinary iodine concentration and carrying out surveys with approximately 900 probands in local circumstances as the most suitable method for iodine status assessment at the community level (6). Therefore, we aimed to investigate how far consumption of different iodine containing food might be reflected in a difference in urinary iodine excretion as one important indicator of adequate iodine supply. Thus, we screened a cohort of 805 employees and students of the University of Leipzig, an East German city in a former area with iodine deficiency (7) for prevalence of nodular thyroid disease. In this cohort we analyzed urinary iodine excretion in relation to nutri-

tional iodine supply by university canteen meals, for which the iodine content was determined, and/or consumption of low- or high-iodine containing food assessed by a special food questionnaire and/or additional iodine intake through iodine tablets.

Criteria for assessing iodine nutrition based on urinary iodine excretion according to the WHO and the International Council for Control of Iodine Deficiency Disorders (6,8,9) are described in Table 1.

Methods and Materials

Eight hundred five students and employees of the University of Leipzig participated in the study. The probands were contacted by posters, university press, and the university intranet Website. All 805 participants lived in the area of Leipzig, Saxony, and all volunteers were German. Probands were asked to give a spot urine sample and to complete a questionnaire.

The study protocol was approved by the local ethic committee. All participants gave written informed consent. Our study was performed in April 2002.

The iodine/creatinine ratios in spot urine samples were determined in 792 probands to minimize bias through kidney function decline with age (10,11). We converted the WHO values (mg/L) into micrograms of iodine per gram of creatinine, that is the iodine deficiency stages I (II, III) are equivalent to UI and creatinine concentrations of 50–99 µg/g (25–49 µg/g, < 25 µg/g), because there was a normal distribution of the data (Table 1; 6) and determination of iodine/creatinine ratios in spot urine samples that has been suggested as the gold standard for the assessment of iodine status of a population (10,11).

Iodine concentration was measured manually according to the protocol described by Sandell and Kolthoff (12), which is based on the catalytic role of iodine in the reduction of ceric ammonium sulphate in the presence of arsenious acid. All urinary samples were digested before the Sandell-Kolthoff reaction to remove interfering substances as previously described (12). Creatinine was measured as described by Jaffe et al. (13).

The iodine content in 14 representative canteen meals was determined by inductively coupled plasma-mass spectrometry (ICP-MS) with an ELAN 6000 (Perkin Elmer, Boston, MA) (14). Thus, food was freeze-dried and subsequently disintegrated in a 25% solution of TMAH (tetramethylammoniumhydroxid; Tamapure-AA; Tama Chemicals, USA) over 3 hours at 80°C. Drinks were diluted at a ratio of 1:20 with bidistilled water and were directly subjected to ICP-MS analysis. For iodine analysis by ICP-MS a matrix-adapted cali-

bration was used in order to avoid potential matrix effects. After TMAH digestion samples were spiked with different concentrations of potassium-iodide (Ultrapure, Johnson Matthey ALFA Products, Karlsruhe) and used for calibration. To avoid signal drifting, tellurium (100 µg/L) was used as an internal standard. The neighborhood of the measured isotope and the internal standard in mass spectrometry allowed a correction of mass-dependent matrix effects. Iodine content in each sample was corrected for the ¹²⁷I/¹²⁸Te ratio. In addition, two reference materials (milk powder, bovine liver) were disintegrated in parallel with the samples and measured after every tenth sample.

The food questionnaire completed by all 805 participants asked whether the probands had regular canteen meals (> 3 per week) and assessed frequencies of intake of food with high iodine content, such as milk and milk products, marine fish, cereals, special meat products, usage of iodized table salt (15–17), and intake of iodine-containing tablets. Additionally, we obtained a history of thyroid disorders (probands and family members), previous exposure to contrast medium (computed tomography [CT] scan, cardiac catheterization), pregnancy and breast feeding, as well as smoking.

Ultrasound was performed using a high-resolution real-time instrument (7.5 MHz). Thyroid volumes and volumes of thyroid nodules were calculated according to the spherical ellipsoid formula: volume = π/6 × anteroposterior diameter (cm) × width (cm) × length (cm) (18). Thyroid volumes greater than 18 mL were considered to be enlarged in adult women and thyroid volumes greater than 25 mL were considered to be enlarged in adult men, which corresponds to the mean +3 standard deviation (SD) in iodine sufficient populations (19) and guaranteed, that gender specific values for goiter are above the 97th percentile of thyroid volumes found in iodine replete control population (6).

Statistical analysis was performed using SPSS procedures (version 10.0, SPSS Inc., Chicago, IL).

As mentioned we used means instead medians for statistical analysis because there was a symmetrical distribution of the data. Moreover, the statistical bias in large cohorts (> 500 probands) is lower when using mean values than median values as mean values better describe metric variables than the median values (17,20).

Results

Urinary iodine excretion

The urinary iodine excretion, iodine intake, and prevalence of nodular thyroid disorders was determined in a co-

TABLE 1. ASSESSMENT OF IODINE INTAKE BASED ON URINARY EXCRETION (6,8,9)

<i>Urinary iodine excretion (µg iodine/g creatinine)^a</i>	<i>Iodine deficiency stage</i>	<i>Assessment of iodine intake/nutrition</i>
> 300	—	Excessive/risk of adverse health
200–299	—	More than adequate/risk of iodine induced hyperthyroidism
100–199	0	Adequate/optimal
50–99	I	Insufficient/mild iodine deficiency
25–49	II	Insufficient/moderate iodine deficiency
< 25	III	Insufficient/severe iodine deficiency

^aWHO values (mg/L) were converted into µg iodine/g creatinine.

hort of 805 students and employees of the University of Leipzig. Table 2 shows the features of the study population.

Urinary iodine excretion was analyzed in 792 participants. Because the number of female employees is higher than the number of males, we enrolled more females in our study. The mean urinary iodine excretion was found to be $109 \pm 81 \mu\text{g}$ iodine per gram creatinine ($\mu\text{g/g}$) for all probands and differed significantly for males ($79 \pm 44 \mu\text{g/g}$) and females ($116 \pm 86 \mu\text{g/g}$) (*t* test, $p < 0.001$). When we converted the urinary iodine excretion of our cohort to micrograms per liter the mean UI in our population was found to be $104 \mu\text{g/L}$. This suggests (1) a borderline adequate iodine intake ($100\text{--}200 \mu\text{g/L}$ or $\mu\text{g/g}$, Table 1) and (2) that conversion of UI stages (Table 1) from $\mu\text{g/L}$ to $\mu\text{g/g}$ does not result in a statistical bias in our population.

Sixty-four percent of males and 40% of females were younger than 30 years.

Proportions of probands with adequate and moderate to severe iodine deficiency according to the WHO stages are given in Table 2. Moreover, 283 (36%) probands had mild iodine deficiency with urinary iodine excretions between $50\text{--}99 \mu\text{g/g}$, 69 (9%) had inadequate high urinary iodine excretions between $200\text{--}299 \mu\text{g/g}$, and 27 (3%) had urinary iodine excretions greater than $300 \mu\text{g/g}$.

Urinary iodine excretion ($\mu\text{g/g}$) correlated positively with age ($p < 0.01$, Spearman $\rho = 0.151$) while creatinine excretion alone correlated negatively with age ($p < 0.01$, Spearman $\rho = -0.232$).

Excessive iodine excretion ($>300 \mu\text{g/g}$ according to WHO criteria) (6,17) was observed in 27 probands as a result of contrast medium exposure ($n = 2$) within the previous 4 months, intake of iodine tablets ($n = 13$), or of unknown cause ($n = 12$). Data of these probands were excluded from further statistical analysis.

Mean urinary iodine excretion in a subgroup of pregnant ($n = 10$) or breast feeding females ($n = 16$) was 138 ± 130

$\mu\text{g/g}$, whereby only five females in this group took iodine tablets, despite recommendations for iodine supplementation during pregnancy (21). The urinary iodine excretion at different ages is shown in Table 3. Urinary iodine excretion in females increases with age.

Iodine content in meals

Analysis of the iodine content in 14 representative meals prepared with iodinated salt at the university canteen showed a mean iodine content of $72 \mu\text{g}$ per meal corresponding to nearly half of the WHO recommended daily iodine intake level for adults ($150 \mu\text{g/d}$) (6). Of note, iodine content in the individual menus differed considerably, ranging from $18\text{--}136.5 \mu\text{g}$ per meal (Table 4). One hundred four (28 males and 76 females) of the 805 probands in our cohort had regular lunches (≥ 3 times per week) at the university canteen since 1997.

Calculation of the nutritional iodine deficit

Using the food questionnaire (Table 5), the additional nutritional iodine intake (other than lunch) in these probands was estimated to be approximately $50 \mu\text{g/d}$. Based on these data we calculated an iodine intake of approximately $216\text{--}360 \mu\text{g}$ of iodine per week through central catering (Table 4) and approximately $350 \mu\text{g}$ iodine per week through food produced with iodized salt and other iodine containing products in this group (Table 5), resulting in a total iodine intake of $566\text{--}710 \mu\text{g}$ iodine per week. The recommended total iodine intake is $1050 \mu\text{g}$ iodine a week (6). Therefore, the remaining nutritional iodine deficit of the 104 probands would be 32%–46%.

Comparison of urinary iodine excretion between probands having regular lunches at the university canteen ($95 \pm 64 \mu\text{g/g}$) and a matched (age, gender, history of iodine exposure, intake of iodine tablets) control group of 400 probands

TABLE 2. IODINE EXCRETION, INTAKE, AND THYROID DISORDERS OF THE STUDY POPULATION

	Males	Females	All subjects
Population ^a	156 (19.4%)	649 (80.6%)	805 (100%)
Age ^b (years)	31.33 ± 12.07	35.42 ± 12.44	34.63 ± 12.46
Thyroid volume (mL) ^b	15.42 ± 10.28	12.12 ± 7.13	12.76 ± 7.94
Goiter ^a	11 (7%)	81 (12.5%)	92 (11.4%)
Thyroid nodules ^a	30 (19.9%)	201 (31.9%)	231 (29.6%)
Spot urine samples available ^a	153 (99.3%)	639 (98.5%)	792 (98.4%)
Overall iodine excretion (μg iodine/g creatinine) ^b	79 ± 44	116 ± 86^c	109 ± 81
Adequate iodine intake ^a (urinary excretion $100\text{--}200 \mu\text{g}$ iodine/g creatinine)	43 (27%)	201 (31%)	244 (31%)
Moderate to severe iodine deficiency ^a (urinary excretion $< 50 \mu\text{g}$ iodine/g creatinine)	44 (29%)	125 (20%)	169 (21%)
Daily iodine tablet ^a	4 (2.6%)	66 (10%)	70 (9%)
History of iodine containing contrast media ^a	18 (11.5%)	86 (13.3%)	104 (13%)
Radioiodine therapy ^a	1 (0.6%)	3 (0.5%)	4 (0.5%)
Other iodine containing medication ^a	1 (0.6%)	12 (1.8%)	13 (1.6%)
Thyroidectomy ^a	2 (1.3%)	14 (2.2%)	16 (2%)
Pregnancy/lactation ^a		27 (4%)	

^aNumber of patients.

^bMean \pm standard deviation.

^cOne female with history of iodine containing x-ray after January 1, 2002 and urinary iodine excretion $> 2000 \mu\text{g}$ iodine/g creatinine excluded.

TABLE 3. SUBGROUP ANALYSIS OF IODINE EXCRETION AT DIFFERENT AGES

	Males	Females	All subjects
Age < 25 years			
Spot urine samples available ^a	63	176	239
Iodine excretion (μg iodine/g creatinine) ^b	78.3 \pm 42.15	101.96 \pm 70.66	95.73 \pm 65.14
Age 25–35 years			
Spot urine samples available ^a	35	128	163
Iodine excretion (μg iodine/g creatinine) ^b	63.10 \pm 43.15	113.10 \pm 91.33	102.36 \pm 85.76
Age 35–45 years			
Spot urine samples available ^a	13	117	130
Iodine excretion (μg iodine/g creatinine) ^b	107.19 \pm 65.27	103.1 \pm 67.96	103.5 \pm 67.46
Age 45–55 years			
Spot urine samples available ^a	13	91	104
Iodine excretion (μg iodine/g creatinine) ^b	73.39 \pm 41.70	125.75 \pm 94.08	119.21 \pm 90.77
Age 55–65 years			
Spot urine samples available ^a	11	59	70
Iodine excretion (μg iodine/g creatinine) ^b	83.34 \pm 24.63	156.83 \pm 93.21	145.29 \pm 90.08

^aNumber of patients.
^bmean \pm standard deviation.

(103 \pm 62 $\mu\text{g}/\text{g}$), who did not eat at the university canteen did not show statistically significant differences. The iodine intake between both groups as assessed by the food questionnaire was similar as well (data not shown).

Moreover, the analysis of the food questionnaire did not reveal a statistically significant influence of a single nutritional determinant (milk, meat, fish, cereal products) on the urinary iodine excretion.

For instance, 89% of all probands ($n = 805$) reported the use of iodinated salt at home (≥ 3 times per week) and consumption of fish (≥ 1 time per week) was reported by 98% of the participants (data not shown).

Daily intake of iodine tablets was reported by 70 probands ($n = 66$ females, $n = 4$ males) of the study population, and this was due to a history of goiter ($n = 39$) and/or thyroid nodules ($n = 31$) in 68 participants. Thyroid surgery had been performed in 8 of 70 probands. Urinary iodine excretion was significantly higher in all 69 available urine samples of these probands (169 \pm 130 $\mu\text{g}/\text{g}$) compared to controls without iodine tablet intake (103 \pm 87 $\mu\text{g}/\text{g}$, t test, $p < 0.05$).

Thyroid disorders

Goiter was present in 92 (11.4%) probands (7% males, 12.5% females). Thyroid nodules were found in 231 (29.6%)

TABLE 4. IODINE CONTENT IN FOURTEEN REPRESENTATIVE MENUS OF THE UNIVERSITY CANTEEN

No.	Menu	Weight (g)	I/100g Menu (μg)	I/Menu (μg)
I	Two eggs, potatoes, mustard sauce and broth	553	12	66.4
II	Meatball, potatoes and french beans	457	13.7	62.6
III	pork chop, mashed potatoes, sauerkraut and yoghurt	670	14.1	94.5
IV	Herbal curd, butter, potatoes, leek soup and yoghurt	520	16.9	88.1
V	deep fried pork, mashed potatoes and peas/carrots	379	18.5	70.2
VI	Fricassee with rice	327	15.4	50.4
VII	Russian soup with sausage and chocolate-pudding	635	18.4	117
VIII	Fish-fillet, mashed potatoes and sauce	410	22.5	92.1
IX	Roast, potatoes, sauerkraut, gravy and kiwi	533	8.3	44
X	Vegetarian cutlet, cauliflower and pasta	485	3.7	18
XI	Pasta, soup and chocolate-pudding	639	9.6	61.2
XII	Rib (pork), potatoes and french beans	442	11.8	52.2
XIII	Stew and rice with peas/carrots and kiwi	427	13.9	59.2
XIV	Souffl� (potatoes, tomatoes, cheese, sausage and haricot beans)	665	20.5	136.5
a	Juice	200	0.3	0.6

Weight \cong overall weight of the menu and weights of the meal constituents.
 I/100 g Menu \cong iodine content in 100 g of the menu (average).
 I/ Menu \cong overall iodine content of the selected menu.
^asupplementary to every menu.

TABLE 5. CONSUMPTION OF FOOD PRODUCTS WITH HIGH IODINE CONTENT (17)

Estimated iodine intake through daily use of following products	Frequencies of intake by the volunteers	Number of valid answers	Absolute/relative frequencies of answer yes	Absolute/relative frequencies of answer no
Iodized salt in household (20 μg)	≥ 3 times per week	98	96 (98%)	2 (2%)
Sea fish (140 μg)	≥ 1 times per week	98	96 (98%)	2 (2%)
Spinach (20 μg)	≥ 1 times per week	99	97 (98%)	2 (2%)
Coffee/tea (8 μg)	≥ 3 times per week	103	101 (98%)	2 (2%)
Milk (14 μg)	≥ 3 times per week	101	100 (99%)	1 (1%)
Bread (12 μg)	≥ 3 times per week	104	104 (100%)	—
Egg (10 μg)	≥ 1 times per week	102	79 (78%)	23 (22%)

Iodized salt based on 1 g.

Coffee/tea based on 200 mL.

Two hundred milliliters German cow milk.

Bread based on 1 slice (50 g).

probands ($n = 156$ single nodule, $n = 75 > 1$ nodule), including the 68 probands (6%) with known thyroid disease.

The age of the probands correlated positively with thyroid volume ($p < 0.01$, Spearman $\rho = 0.323$) and prevalence of thyroid nodules ($p < 0.01$, Spearman $\rho = 0.362$).

Females with nodular goiter ($n = 51$) had an insignificant lower urinary iodine excretion ($97 \pm 51 \mu\text{g/g}$) than females with nodular thyroid disease and normal volume ($n = 127$, $112 \pm 69 \mu\text{g/g}$, t test, $p > 0.05$). Similarly, comparison of urinary iodine excretion between males having nodular goiter ($n = 9$, $74 \pm 19 \mu\text{g/g}$) and males with nodular thyroid disease and normal volume ($n = 21$, $81 \pm 43 \mu\text{g/g}$) failed to show statistically significant differences (t test, $p > 0.05$).

Smoking and thyroid volume

Five hundred fifty-one (68%) probands answered the question for a history of smoking. Two hundred fifty-four probands (32%) failed to give an answer. Of 551 probands, 418 (76%) were nonsmokers and 133 (24%) were smokers. Mean thyroid volume was higher in smokers (13.9 mL) than in nonsmoking probands (12.5 mL) without reaching statistical significance (t test, $p = 0.085$, 2-tailed). Thyroid volume correlated positively with the reported packyears of the smokers (Pearson correlation coefficient 0.124 significant at the 0.01 level [2-tailed]).

Prediction of thyroid volume

Moreover, we investigated if the parameters age, smoking, gender, pregnancy or nursing period, and urinary iodine can predict the outcome variable thyroid volume. Linear regression analysis (analysis of variance [ANOVA]) reveals a prediction of thyroid volume through age ($p < 0.001$), gender ($p < 0.001$), and smoking ($p < 0.05$).

Discussion

Urinary iodine excretion is widely used as an indicator of the iodine nutritional intake. In our study we analyzed different components influencing urinary iodine excretion in the same adult population, that is, we assessed nutritional iodine intake by a food questionnaire, measured iodine content in canteen meals consumed by approximately 20% of our study population and assessed for other sources

of iodine intake (e.g., tablets or exposure to contrast medium).

The mean urinary iodine excretion in our cohort was within the lower range of adequate iodine intake (6,8,9), whereby the mean urinary iodine excretion was significantly lower in men than in women which is in agreement with previous reports from Saxony and other iodine-deficient areas (17,22,23). However this may also be because of regular intake of iodine tablets in 10% of females but only 3% of men in our study group.

The WHO definitions for adequate iodine status of a population are:

- Urinary iodine excretion between 100–200 $\mu\text{g/L}$ ($\mu\text{g/g}$), and
- Less than 20% of the study population with UI $< 50 \mu\text{g/L}$ ($\mu\text{g/g}$) (6).

Based on these criteria our study population showed borderline iodine deficiency because mean urinary iodine excretion was only 109 $\mu\text{g/g}$. Moreover, only a small proportion (31%) of the study group showed an optimal iodine intake (100–200 $\mu\text{g/g}$), whereas an unexpected high proportion (21%, $n = 169$) of all participants had urinary iodine excretions within the ranges of moderate to severe iodine deficiency (< 25 –49 $\mu\text{g/g}$, Table 1). We conclude that the alimentary iodine supply in the adult German population in Saxony is still close to the margin of iodine deficiency stage I (6). The high proportion of urinary iodine excretions below 50 $\mu\text{g/g}$ was astonishing because previous investigators in Germany, Völzke et al. (24) reported a frequency of 11% of probands with urinary iodine excretion below 50 $\mu\text{g/L}$ and Grüning et al. (22) reported a frequency of 12% of males and 8% of females of moderate to severe iodine deficiency in an adult population in Saxony. Rendl et al. (4) found an overall frequency of 4% of moderate to severe iodine deficiency in a younger population in Bavaria. In Austria greater than 40% of iodine status investigations did report a greater than 20% proportion of iodine deficiency ($< 100 \mu\text{g/g}$) 8 years after the introduction of 20 mg of potassium iodide per kilogram of salt (25). It has been established that the household usage of iodized salt with an average iodine content of 20 μg iodine per gram of salt is not sufficient to ensure adequate intake at the individual level because the mean calcu-

lated iodine intake of the population subgroups were below the WHO recommendations with a remaining iodine intake deficit of 30% (17).

Our data emphasize again, that borderline normal iodine excretions at the level of a population are consistent with inadequate iodine excretion in a significant proportion of this population. Therefore, the lower threshold for iodine sufficiency of a population with 100 $\mu\text{g/g}$ (Table 1) needs to be questioned, especially if there are further environmental or genetic goiter risks in a population.

The consumption of canteen meals, prepared with iodized salt, is thought to introduce a stable source of iodine intake. However, regular consumption of canteen meals, for which an average iodine content of 50% of the daily recommended intake was determined, did not result in higher urinary iodine excretion in 104 probands compared to 400 matched participants who did not eat at the canteen. This may in part be due to the highly variable iodine content in the canteen meals (Table 4). Our results suggest that nutritional intake of iodine-containing food was fairly similar throughout the study population. This was also reflected by the results of the special food questionnaire. No single nutritional determinant contributing to adequate urinary iodine excretion could be identified. This indicates a lack of selectivity of the questionnaire and a variable use of iodized salt in food production as illustrated by the variable (mean, $72 \pm 31 \mu\text{g}$) iodine content in canteen meals. Lack of standardized use of iodized salt in food production and lack of standardized assessment strategies in Europe as well as other continents (26) results in the necessity to monitor iodine deficiency by regular reevaluation in the same region.

Intake of iodine tablets was exclusively restricted to probands with a history of thyroid disease (goiter and/or thyroid nodules). In this subgroup urinary iodine excretion was significantly higher in comparison to probands who took no iodine tablets. Similarly, Gärtner et al. (3) have previously reported significantly higher levels of iodine excretion in 17 lactating women (114 $\mu\text{g/g}$) using iodine tablets compared to a control group of 53 women (74 $\mu\text{g/g}$), who did not (3). Because urinary iodine excretion was significantly influenced by intake of iodine tablets, this may lead to a bias of statistical analysis in a study population, if the percentage of probands with regular iodine tablet intake is not known (27).

The overall frequency of thyroid nodules (30%) was within but the frequency of goiters (11%) was below the ranges of previous investigations in Germany dating from 1995–2001 using the same cutoff values for defining goiter (2,22,24,28). It is tempting to speculate that our findings resulted from an improvement in the general iodine supply over the past years (17,29,30). The urinary iodine excretion as determined by the iodine/creatinine ratio is influenced by additional factors (e.g., muscle mass and physical activity; 17,31) and may be underestimated in younger and overestimated in older study populations because creatinine excretion is negatively correlated with age. However, we found it a suitable means to assess a large cohort. As expected, our data confirm that the individual risk for developing goiter rises with tobacco smoking (32).

Conclusion

Our data demonstrate a prevailing borderline iodine deficiency in the Saxonian population despite widely available

information on the effective prevention of iodine deficiency related thyroid diseases. Iodine supply through salt/usage of salt in food industry is still not enough in Germany.

Because only probands with regular iodine supplementation through iodine tablets showed sufficient urinary iodine excretions according to the WHO criteria, iodine tablets could presently remain the most efficient means to ensure adequate iodine intake in Germany in agreement with available recommendations for adolescents, pregnancy and nursing period (3,33,34).

Food manufacturers, outlets, restaurants and stores should be required to make available only iodized salt to customers. Moreover, these establishments should be monitored through health officers and nutrition scientists.

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