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Original article

Evolution of urinary iodine excretion over eleven years in an adult population

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SUMMARY

Background & aims: Few prospective cohort studies have evaluated dietary iodine intake and urinary iodine concentrations in the general adult population. We assess the evolution of urinary iodine excretion and factors that may influence it in an adult population followed for 11 years.
Methods: A population-based cohort study was undertaken in Pizarra (Spain). In the three study phases (baseline ($n = 886$), and 6 ($n = 788$) and 11 years later ($n = 501$)), participants underwent an interview and a standardized clinical examination that included a food questionnaire, and thyroid hormone and urinary iodine determinations. Subjects with thyroid dysfunction, palpable goiter or urinary iodine excretion >400 μg/L were excluded.
Results: Urinary iodine increased over the years (100.6 ± 70.0 μg/L at baseline vs. 125.4 ± 95.2 μg/L at 6 years and 141.6 ± 81.4 μg/L at 11 years; $p < 0.0001$). Urinary iodine was significantly higher in subjects who reported iodized salt consumption and in subjects with a higher intake of dairy products ($p < 0.05$). Consumption of iodized salt (Risk ratio (RR) = 1.23, 95% CI [1.01–2.05]) and dairy products (RR = 2.07, 95% CI [1.01–4.23]), and a baseline urinary iodine concentration ≥100 μg/L (RR = 1.26, 95% CI [1.04–1.53]) were significantly associated with urinary iodine concentrations ≥100 μg/L at 11 years. There is no correlation between thyroid function (TSH, free triiodothyronine or free thyroxine levels) and urinary iodine concentrations in conditions of iodine sufficiency.
Conclusions: The increase in urinary iodine concentrations over eleven years is associated with an increase in iodized salt intake and with the dairy products intake, and possibly with a higher iodine content of dairy products. However, individual variability in urinary iodine excretion was not fully explained by dietary iodine intake alone; previous urinary iodine concentrations were also important.

1. Introduction

Iodine is a micronutrient that is essential to synthesize thyroid hormones. The consequences of both iodine deficiency and iodine excess are well known [1]. Iodine deficiency is one of the most frequent public health problems all over the world, though it could easily be corrected. International health organizations are working to eradicate iodine deficiency, and there have been worldwide improvements in iodine nutritional status over the last years [1].

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Historically, Spain was considered an iodine-deficient area [2], but recent studies, especially those assessing iodine intake in children, have reported an increased dietary iodine intake in the Spanish population [3].

The urinary iodine excretion measured in a random urine sample is used to assess the iodine nutritional status in population studies. The advantages and disadvantages of this determination are well known and have been discussed recently [4]. The disadvantages include a high intra-individual variability, which has been associated with dietary iodine intake. However, the collection of a random urine sample is easier than a 24 h sample in population studies. Urinary iodine excretion reflects around 70% of dietary iodine intake, but it has a wide range of values, even in subjects within the same community [5]. However, the impact of endogenous factors on individual variability of urinary iodine excretion has not been fully investigated [6].

Most studies assessing the evolution of dietary iodine intake in a population usually compare data from previous cross-sectional studies performed in that population. In a longitudinal population-based study of the DanThyr cohort, the mandatory program for iodization of salt initiated in 2000 in Denmark led to significant changes in the thyroid function in a population with mild-moderate iodine deficiency [7]. Another longitudinal study initiated 3 years after the introduction of iodized salt in China with a 5-year follow-up has been published [8]. In Spain, a recent cross-sectional, population-based survey comprising a representative sample of the adult Spanish population [9] found the mean urinary iodine concentration to be 117.2 μg/L, and reported high inter-individual variability and regional disparities in urinary iodine excretion. However, only a few prospective cohort studies have assessed dietary iodine intake and urinary iodine concentrations in the general adult population [10].

The aims therefore of this study were to assess the evolution of urinary iodine excretion in an adult population and to evaluate the factors possibly affecting urinary iodine excretion in adults over an 11 year follow-up in the Pizarra Study.

2. Material and methods

2.1. Population

The study was carried out in a cohort of the Pizarra Study, a population-based prospective study undertaken in a town in Andalusia, southern Spain ($n = 1250$). The study population and the design of this survey have been described previously [11]. A total of 124 subjects did not attend or had missing data (Fig. 1). The first phase of the study (1995–1997) included 1126 individuals, aged 18–65 years, selected randomly from the municipal census of Pizarra. Of the original cohort, 976 (86%) were reassessed in 2001–2003 (second phase, 6 years after the first visit), and 634 (64.9%) were evaluated again in 2006–2008 (third phase, 11 years after the first visit).

For the purposes of the present study, patients were excluded if they were admitted to a hospital during the 2 weeks prior to the evaluation or into a geriatric institution, if they were pregnant, if they had a cancer, if they were receiving anti-thyroid or thyroid hormone therapy or if they had positive anti-thyroid peroxidase antibodies (TPO) (>50 IU/mL), or thyrotropin (TSH) (TSH<0.20 μIU/mL or TSH>5.0 μIU/mL), or urinary iodine excretion>400 μg/L, or palpable goiter at any study phase. The final sample size was 886 subjects in the first phase, 788 subjects in the second phase and 501 subjects in the third phase. No significant differences were found in the variables studied in each phase between the subjects included and those excluded in the study, except in thyroid function (data not shown).

The research has been carried out in accordance with the Declaration of Helsinki (2008) and all the participants gave their informed consent, and the study was reviewed and approved by the Ethics and Research Committee of Regional University Hospital, Malaga.

3. Procedures

At baseline and at the follow-up evaluations all the participants underwent an interview and a standardized clinical examination. The same methodology was used for all three phases. A standardized survey of health habits [12] and a food frequency questionnaire were administered at each phase [13]. Specifically, questions were asked systematically about the intake of iodized salt and other sources of iodine, such as the consumption of milk and other dairy products, fish or eggs [14]. Also, the consumption of different types of meats, fishes, vegetables, fruits, drinks, etc., was included [14]. Measurements were made in all the participants of weight and height, and the body mass index (BMI) was calculated (weight/height^2).

At all three study phases blood samples were collected after a 12 h fast. The serum was separated and frozen at −80 °C. Serum thyroid hormones were analyzed at the same time in an automated MODULAR ANALYTICS E170 analyzer (Roche Diagnostics GmbH, Mannheim, Germany). The TSH, free triiodothyronine (FT3) and free thyroxine (FT4) were measured by chemoluminescence (Roche Diagnostics GmbH). For TSH, the reference values in adults were 0.2–5.0 μIU/mL. For FT4, the reference values in adults were 10–22 pmol/L. For FT3, the reference values in adults were 3.10–6.8 pmol/L. The anti-TPO antibodies were measured by a radioimmunometric assay (Biocode S.A., Liege, Belgium) only at the first and third phases of the study. The iodine concentration in the first morning urine was measured after each study wave by the modified Benotti and Benotti technique [15]. Briefly, this method consists in a previous acid digestion of the urine sample with chloric acid at 110 °C for 90 min, and after, we made the Sandell–Kolthoff reaction, in which iodine acts as a catalyst for the reduction of cerium (IV) to cerium (III) by arsenic (III). The absorbance of each sample was measured at 420 nm with a spectrophotometer. The urinary iodine assay is subjected to a program of external quality assessment for the determination of iodine in urine of the Spanish Association of Neonatal Screening (AECNE) and of Ensuring the Quality of Iodine Procedures (EQUIP) Program. We perform these controls 3 times/year. The intra- and inter-assay coefficient of variation of the urinary iodine assay was 2.01% and 4.53%, respectively.

4. Statistical analysis

The statistical analysis was done with R free software. Continuous variables are presented as the mean, median and standard deviation, and categorical variables as percentages. A log transformation of urinary iodine concentration was used for analysis. The Spearman correlation coefficient was used to assess relationships between urinary iodine excretions at each study phase. Comparison between three or more groups was performed with the ANOVA test. To assess differences in urinary iodine excretion over time, a repeated measures ANOVA, adjusted by age and sex, was calculated only in subjects with data available for all three study phases. To assess whether urinary iodine excretion at the third phase was conditioned by urinary iodine excretion at the first and second phases of the study a Poisson regression analysis was done. The dependent variable was the urinary iodine excretion at the third phase. Two variables were created to be included as covariates for analysis: