RE: Urine Iodine Excretion in Patients with Euthyroid Nodular Disease

To the Editor: I read with interest the article by Cakir and coleagues in the March-April 2011 issue.[1] Having worked in the field of goiter,[2] I would like to comment on this article. The high prevalence of thyroid nodular disease throws light on the importance of the subject. The authors used urinary iodine concentration of spot urine samples to classify their individual patients into different grades of iodine deficiency or adequacy. Two aspects warrant clarification.

First, it is the median urinary iodine concentration rather than the individual values that are used in categorization of endemic goiter and this value is used for population groups rather than individuals. According to the median urinary iodine concentration, a population is described as iodine sufficient (median ≥100μg/L), mildly iodine deficient (50-199 μg/L), moderately iodine deficient (20-49 μg/L) or severely iodine deficient (<20 μg/L).[3] Individual values of urinary iodine concentration are important only as they contribute to the location of the median value of the population and have little if any significance as regard to the individuals iodine status. This stems from the fact that within the same subject the daily variability of urinary iodine concentration is large.[4] Day-to-day variation is of course even larger. Indeed, the urinary iodine concentration of a spot urine sample on any day may not tell about the iodine status of that individual yesterday and neither does it predict his iodine status tomorrow. Anderson and colleague[4] collected monthly urine samples for twelve months and measured urinary iodine excretion. In that study, wide intraindividual (between months) variation (range from 18 to 142 μg/day) was shown. This is further supported by other studies.[5] In the article, the subject of this letter, the authors used urinary iodine concentration in a single spot urine sample to classify their individual subjects into three iodine status groups to judge the effect of iodine status one year later on the volume of thyroid nodules. For this to be possible, it should be assumed that the subjects maintain the same iodine status throughout the follow-up year. Interestingly, two findings in Cakir and colleagues' study itself argue against this assumption. These include the lack of significant differences in the volumes of the thyroid lobes between the groups or within the groups between the start and the end of the study and the lack of differences in serum free T4 and TSH between the groups. Thus, it is rather difficult to conclude that iodine deficiency is the cause of enlargement of thyroid nodules in Cakir and colleagues' study since the patients' iodine status in the period before as well as during follow-up year is not known.

Second, the authors used urinary iodine values of <50 μg/L as indicating severe iodine deficiency while the World Health Organization and other international health institutions define severe iodine deficiency by values less than <20 μg/L.[5] However, this issue may be rendered immaterial since the authors used the cut-off values to define iodine status of individuals rather than populations. In conclusion, iodine status of Cakir's patients is indeed unknown. This being the case I believe that smoking and additional diseases may offer more likely explanations of Cakir's conclusion.

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DOI: 10.4103/0256-4947.84644

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