To the Editor:

One cannot deny the importance of goiter assessment in estimating the magnitude of iodine deficiency worldwide in previous decades. While a number of relatively rare pathologic disorders can result in an enlarged thyroid, when the prevalence of goiter is common in a population (>5%), this has most often been the result of inadequate iodine in the diet.

A recent article and editorial in Thyroid described the ThyroidMobile and its use in the assessment of thyroid volumes in populations (1,2). In this letter we would like to comment on the possible misclassification of the status of iodine deficiency disorders (IDD) in areas where salt iodization has recently been implemented when using measures of thyroid size.

Over the years there has been an increased understanding of the consequences of iodine deficiency. What transformed iodine deficiency from a condition focused on primarily by endocrinologists to an important public health issue was an awareness of the effect of iodine deficiency on the brain of the developing fetus and subsequent cognitive development. While cretinism has been known as a severe outcome of inadequate maternal iodine nutrition during pregnancy, the effect of iodine deficiency, even at mild and moderate degrees, in a population has been found to reduce the average IQ by 10%–15% (3). This message has been important for the public health community and policy makers as it became recognized that national development could be impeded by iodine deficiency.

The most common intervention to eliminate iodine deficiency in a population is to add a small amount of iodine (usually in the form of potassium iodate or potassium iodide) to salt. The marginal cost of adding iodine to salt and improvements in salt packaging is often passed on to the consumer and is usually around 5 to 10 cents per person per year, one of the most cost-effective public health interventions (4). One question that arises as more and more countries implement universal salt iodization (USI) programs is how to measure the impact of iodized salt on the prevalence of iodine deficiency. Historically, the most common indicator employed to measure progress towards USI has been thyroid size (usually by palpation and more recently through ultrasonography to estimate thyroid volume).

Palpation and visual examination of the thyroid to estimate the prevalence of goiter is the simplest method. However, there are a number of issues that need to be considered when interpreting goiter prevalence estimates. First, there can be large interobserver variability, and this variability increases as goiters become smaller and less common. Therefore, while goiter surveys can play an important role in identifying areas with a high prevalence of iodine deficiency, they are less reliable when the prevalence of goiter is low. Second, and perhaps more importantly, the development of goiter in an individual usually takes years; and once an individual’s iodine nutrition is corrected, it may take years for their thyroid to return to “normal” size. For some individuals the thyroid may never return to “normal” (5). This second factor is particularly relevant in areas where salt iodization programs are being implemented because goiter prevalence is a reflection of iodine status over 2 or more years, not current iodine nutrition status.

In some countries, local researchers have been attempting to find other causes of enlarged thyroid without realizing that this is a normal process in salt iodization programs. Measuring thyroid volume by ultrasonography also has interobserver variability (although perhaps less so than palpation). In addition, there have been limitations in the widespread application of ultrasonography because of the lack of an appropriate thyroid volume reference. Again, in spite of these limitations, the more important shortcoming of ultrasound in IDD surveillance is that thyroid volume estimates do not reflect current iodine status. Another unfortunate consequence of measuring thyroid size is that it focuses attention on the thyroid and not the most important organ that is affected by iodine deficiency—the brain.

More recently, the iodine concentration in urine has been adopted as the primary indicator for tracking progress in IDD control programs by the World Health Organization (WHO), United Nations Children’s Fund (UNICEF) and the International Council for Control of Iodine Deficiency Disorders (ICCIDD) (6). Because most of the iodine ingested is excreted in the urine, the measurement of iodine in the urine is a highly sensitive indicator of the iodine content in the diet. In an individual, the amount of iodine in the urine can be quite variable depending on a number of factors; therefore a single urinary iodine level is not very informative at the individual level. However, at the population level, the

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median level of urinary iodine from a representative sample of the population provides an estimate of the average amount of iodine in the diet of the population. If the iodine concentration of the population is adequate, then one can feel assured that the brains of newborns are being protected from IQ loss because of iodine deficiency.

In our experience, we have often found population surveys where the median urinary iodine concentration indicates adequate iodine nutrition and a high proportion of the population consuming adequately iodized salt, but the prevalence of goiter indicates either mild or moderate levels of iodine deficiency (7). This has lead to some confusion among policy makers and planners over the status and progress of national IDD elimination efforts.

There are some research issues in iodine nutrition that might include measures of thyroid volume and other indicators of thyroid function. However, in countries that have recently or are currently implementing universal salt iodization interventions, we do not recommend measures of thyroid size be collected in cross-sectional surveys to measure progress towards IDD elimination. We recommend reliance on median urinary iodine levels in populations to provide the basis for monitoring USI programs. Goiter rates should be interpreted with caution since they may not reflect current iodine status.

References

2. Dunn JT 2001 Correcting iodine deficiency is more than just spreading around a lot of iodine. Thyroid 11:363–364.

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