Sir,

We appreciate the article written by Shrestha et al. titled, “Iodine status among subclinical and overt hypothyroid parents by urinary iodine assay” in the last issue of our journal.[3] They have recommended reappraisal of salt iodization campaign to monitor for iodine excess which could be related to hypofunctioning of thyroid gland. However, there are certain clarifications requested to the authors regarding their observations that fortified iodine levels in consumed salt may be a major causative factor for thyroid dysfunction. The role of excess iodine in inhibiting release of thyroid hormone needs to be addressed carefully. Especially, if excess iodine should cause thyroid dysfunction, the actual source of excess iodine among the study population needs to be well defined and it could be in food, water, and salt,[3] and dose–response relationships inclusive of borderline excess intake and susceptibility factors for subclinical hypothyroidism and related thyroid antibody status need to be discussed.

This study would have been more informative if goiter characteristics of the study population were also included. Implementation of universal salt iodization (USI) program has been successful in improving iodine deficiency status in many developing countries, and decreasing goiter rates after an increase in urinary iodine concentrations following USI programs have been reported recently in China and Uganda.[3] Recent publication documenting increasing goiter rates at 13.5% and only 18.2% of households using iodized salt in the state of Tamil Nadu as per the survey done by Pandav et al.[4] strongly support and justify increase in iodine ppm content of consumed salt by our vulnerable populations.

There are several publications demonstrating drinking water and food with high iodine content accounting for excess of iodine while iodized salt may be the least contributor as per reports. The role of goitrogens is crucial in that there is improper utilization of iodine by thyroid gland, and to overcome this, appropriate increases in salt iodization are necessary.

As per literature search by Leung and Braverman[5] at one end of spectrum, people without any evidence of thyroid disease almost always remain euthyroid even if they consumed excess iodine and there is an escape from acute inhibitory effects of excess intrathyroidal iodide on organification process and, subsequently, thyroid hormone synthesis and adaptation to Wolff–Chaikoff effect occurs. At the other end, although underlying mechanism of iodine-induced hypothyroidism is controversial, it could be attributed to failure to adapt to Wolff–Chaikoff effect most probably due to a damaged thyroid as a result of previous pathological insults.

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Letters to the Editor

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