# Too much or too little? The effect of iodine on thyroid diseases

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The thyroid hormone synthesis requires iodide uptake by the sodium-iodide symporter (NIS) and its oxidation by thyroid peroxidase (TPO). By weight, iodine constitutes 65% of thyroxine (T4) and 59% of triiodothyronine (T3). Normal subjects adapt to iodide intake by thyroid follicular cells autoregulation through the Wolff-Chaikoff effect and escape from the Wolff-Chaikoff effect. The recommended dietary allowance of iodide for adults in Taiwan is 140 μg.

Iodine deficiency may cause goiter, lead to hypothyroidism, and increase neonatal and infant mortality. The iodine nutrition at the community level may be assessed by measurements of urinary iodine, thyroid size, serum thyrotropin (TSH) and thyroglobulin. Prophylaxis and treatment of the iodine deficiency community can be achieved by adding iodine during the packaging or processing of salt. Regular monitoring of iodine nutrition is essential for sustaining iodine sufficiency.

The effects of iodide administration vary in subjects with different thyroid status or underlying disease process. Iodide administration may cause hyperthyroidism in patients with endemic goiter and iodide deficiency, or in patients with autonomously functioning goiter and iodide sufficiency. On the contrary, iodide administration may result in hypothyroidism in patients with chronic autoimmune thyroiditis. Iodide is recommended for the treatment in patients with Graves' hyperthyroidism for they are sensitive to the inhibitory effect of pharmacologic doses of iodide.