Iodine is abundant around us but mostly contained in compartments dissociated with human consumption. If natural diet included 1 or 2 teaspoonsful of sea-bottom mud per week, there would be no iodine deficiency. On the other hand, to obtain the same amount of iodine from rainwater or most other fresh-water sources, daily intake of water has to be in the order of 30 liters or more (1). Accordingly, iodine-deficiency disorders have been common in most countries, with severe consequences for public health and economic development (2).

The two last decades have witnessed a fundamental improvement in world iodine nutrition. Main contributors to this impressive development have been national committees and health care officials in many countries, as well as the United Nations Children’s Fund (UNICEF) and the World Health Organization (WHO). However, the main driving force has been a nongovernmental organization: the International Council for Control of Iodine Deficiency Disorders (ICCIDD). It is not the topic of this editorial to describe in detail the roles of key persons behind the achievements of ICCIDD. The history and main efforts are well understood from the website www.iccid.org, which also gives insight into the excellent work of the executive director John Dunn, and the great loss inflicted by his recent sudden death. (See obituary in this issue).

The development in world iodine nutrition is illustrated in the report of Pretell et al. in the current issue of the journal (3). The ThyroMobil visited 163 sites in 13 Latin American countries. Urinary iodine content was mildly below optimal in one country (Guatemala), within optimal range in many countries, and mildly above optimal in a few countries. The report shows that severe iodine deficiency has been eradicated in Latin America because of the iodization of salt. However, it also illustrates the importance of continuous monitoring of iodine nutrition. Unforeseen changes in iodine nutrition had occurred and severe iodine deficiency may well return if no monitoring exists. Furthermore, optimal prevention of thyroid disease is more than eradication of severe iodine deficiency. The basic and most important task of iodine fortification programs is to prevent developmental brain damage, but even mild iodine deficiency causes a high prevalence of disease, especially nonnontoxic multinodular goiter, and toxic multinodular goiter with subclinical and overt hyperthyroidism in the elderly. On the other hand, if iodine intake exceeds the level necessary to prevent iodine-deficiency disorders this seems to be associated with an unnecessary high occurrence of subclinical and overt hypothyroidism (4).

Thus, prevention of thyroid disease by iodine supplementation may occur at several levels. It is obligatory that severe iodine deficiency should be avoided, and international guidance and assistance should be offered to countries who cannot achieve this by their own forces. If possible, prevention should be extended to a level above this. Iodine intake of every individual cannot be regulated, but population iodine intake should be watched and regulated. There should be monitoring of iodine fortification of salt for human consumption and use of salt, on levels of iodine in mineral mixtures used for feeding dairy cows and other domestic animals, on use of absorbable iodine containing chemicals in cosmetics, etc. This should be combined with studies of relevant population groups, to see that iodine intake is within the optimal levels.

All our knowledge indicates that such relatively simple and inexpensive measure may reduce the incidence and prevalence of thyroid disease, and the consequences of undiagnosed thyroid function abnormalities in the population.

References