

Iodine Deficiency and Endemic Goiter: A National Tragedy

Iodine deficiency causes severe hormone-induced physiological damage to fetus and newborn resulting in cretinism, stunting, deaf-mutism, malformed limbs, spastic motor disorders, poor vision and goiter (swelling of the thyroid gland on the neck), as well as milder forms of mental and physical impairment. There was developmental lag in children of goitrous mothers in goiter endemic areas(1).

Iodine is a micronutrient, a trace element that occurs naturally in the soil; highest levels are from seaweed. Although replenished somewhat by the cycle of evaporation and rain, the earth's soil is gradually being stripped of iodine (soil erosion). Because the human body cannot retain iodine (unlike most other micronutrients), the vegetables and pulses that are iodine-rich along with iodized salt must be consumed daily.

There is no region in India free of iodine deficiency—the Himalayan regions of India and Bhutan; Gangetic belt and districts with polluted water supply / not consuming iodized salt and higher consumption of thiocyanates in diet. In this issue of the Journal, Chandra, *et al.*(2) in a well designed study in district level school/preschool children show that poor iodine level in salt consumed remains is a major cause of persisting high prevalence of goiter (30%).

In 1981, Gopalan (Nutrition Foundation of India) was requested by the Ministry of Social Welfare to undertake a rapid evaluation of the current status of the National Goiter Control Program, and on the basis of the data, provide Government of India, a blue print for intensification of the program. This blueprint was based on a nation wide survey (Himachal Pradesh, Punjab, Jammu & Kashmir, Uttar Pradesh—undivided, Bihar, West Bengal, Assam, Nagaland, Manipur, Gujarat, Rajasthan), for prevalence and severity of goiter, possible etiology, administrative aspects for salt production-iodization, packaging, distribution problems and finally availability of iodine in salt at consumer level and directions for use of parenteral iodine(3,4).

In 1997, after more than a decade of activism by health experts and international development agencies, India enacted its own Universal Salt Iodization Code. Legislation and Professor V Ramalingaswami's efforts need mention(5). But in July 2000, only two years after passing the code, the Indian government repealed it—surrendering to pressure from the highly vocal small salt producer's lobby, as well as to widespread arguments that mandatory iodization was coercive, unfair and an act of multinational exploitation.

The decision to repeal the iodization laws appalled the nation's doctors and scientists. Some pointed out that the repeal would not affect the rich, who would continue to buy iodized salt, but the poor, who would be unaware of the risk of using the slightly cheaper salt. The Indian Medical Association and Indian National Science Academy said that the repeal "re-imposes a serious public health burden," and lamented that the laws should have stood for the next century. The present study indicates that biochemical iodine deficiency due to intake of less iodine is still the main cause for the persistence of IDD.

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KN Agarwal,

Hon Scientist, National Science Academy,

President,

Health Care & Research Association

for Adolescents,

D-115, Sector-36, NOIDA 201301,

Uttar Pradesh, India.

Email: adolcare@hotmail.com

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