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IODINE DEFICIENCY DISORDERS

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Abstract
This paper reviews present knowledge on disorders induced by iodine deficiency. The Recommended Dietary Allowance of iodine is 50 μg/day from 0 to 12 months, 90 μg/day from 1 to 6 years, 120 μg/day from 7 to 12 years, 150 μg/day during adolescence and adulthood and 200 μg/day during pregnancy and lactation. When the physiological requirements of iodine are not met in a given population, a series of functional and developmental abnormalities occur in all ages, which are grouped under the general heading of iodine deficiency disorders among that goiter is only the visible "top of the iceberg". Iodine deficiency is the leading cause of preventable mental retardation and neurointellectual deficits in schoolchildren. One of very important of health consequence of iodine deficiency is an elevated thyroid uptake of radioiodine that aggravates the risk of thyroid cancer in case of a nuclear accident. In addition to health problems, iodine deficiency has a negative impact on domestic animals, resulting in a reduction of productivity with negative economic consequences. As a solution for these disorders the most logical approach is the introduction of iodine prophylaxis that public health care aspects include the planning and monitoring of prophylactic campaigns, the technical aspects of production and distribution of iodised salt and the other methods of iodine prophylaxis. The last recommendation by WHO-UNICEF-ICCIDD is that, in order to provide 150 μg/day of iodine via iodised salt and considering the average salt intake and the loss of iodine from production site to the household and cooking, the iodine concentration in salt at the site of production should range between 20 to 40 mg of iodine per kilogram of salt
Key words: iodine deficiency disorders, iodine prophylaxis, iodised salt

Introduction

Iodine is a trace element present in the human body in minute amounts (15-20 mg, essentially in the thyroid gland). The primary source of iodine is seawater. Iodide ions at the surface of the seas oxidize by the sunlight to elemental iodine, which is volatile. From the surfaces of the seas iodine evaporates into the atmosphere. Iodine returns to the earth in the form of rain, and through the rivers, return to the sea. This is the natural cycle of iodine. The iodine content in food for human consumption depends on the natural availability of iodine in the place of production. Namely, the content of iodine in vegetables and animal products for human consumption depends on the iodine content in the local soil and water. The highest iodine content is found in seawater and sea animals like fishes; however, sea salt does not contain enough iodine to maintain human health because it evaporates together with the water during its production. Consequently sea salt requires iodine fortification. The iodine concentrations in drinking water vary widely; therefore drinking water is of

limited importance as a source of iodine for human health providing at most 10% of the body's needs.

Iodine is necessary for synthesis of the thyroid hormones thyroxine and triiodothyronine that play a decisive role in the metabolism of most cells of the organism and in the process of early growth and development of most organs, especially of the brain and nervous system, as well as many physiological processes including thermogenesis and production of body energy. (1)

The Recommended Dietary Allowance of iodine is 50 μg/day from 0 to 12 months, 90 μg/day from 1 to 6 years, 120 μg/day from 7 to 12 years, 150 μg/day during adolescence and adulthood and 200 μg/day during pregnancy and lactation (2). When the physiological requirements of iodine are not met in a given population, a series of functional and developmental abnormalities occur (Table 1), among which goiter is only the visible "top of the iceberg".

Table 1

Ages	Disorders
Fetus	Abortions Stillbirths Congenital anomalies Increased per natal mortality Increased infant mortality Neurological cretinism: deaf mutism, spastic diplegia squint Myxoedematous cretinism: mental deficiency, dwarfism, hypothyroidism Psychomotor defects
Neonate	Goiter Neonatal hypothyroidism
Infant, child and adolescent	Goiter, Retarded mental and physical development, Juvenile hypothyroidism
Adult	Goiter and its complications Iodine –induced hyperthyroidism Decrease fertility rate
All ages	Goiter Hypothyroidism Impaired mental function Increased susceptibility to nuclear radiation

The most critical period is from the second trimester of pregnancy to the third year after birth (3). Normal levels of thyroid hormones are for optimal development of the brain. In areas of iodine deficiency, where thyroid hormone levels are low, brain

development is impaired. These complications, which constitute an hindrance to the development of populations, are grouped under the general heading of Iodine Deficiency Disorders (IDD). In addition to health problems, iodine deficiency has a negative impact on domestic animals, resulting in a reduction of productivity with negative economic consequences.

Iodine deficiency in the fetus

The iodine deficiency may make the mother hypothyroid with associated complications of inovulation, infertility, gestation hypertension, increased first trimester abortions, abnormal fetal positions, congenital anomalies, the varying manifestations of cretinism, and stillbirths. These damages may have adverse effects on her quality of life and her role in the family and community (4). Recent evidence indicates that the effects of iodine deficiency on the fetus, including abortion, stillbirth, congenital anomalies, and the varying manifestations of cretinism, probably arise from the lowered level of the blood T4 in the iodine deficient mother. The more severe reduction in the level of maternal T4, the greater threat to the integrity of the fetus.

The developing brain is the most vulnerable target for iodine deficiency. The need for adequate thyroid hormone and therefore, for iodine, in maturation of central nervous system is well established. Unfortunately, these adverse effects of iodine deficiency during development are generally permanent.

In iodine deficient environment there is an increased rate of these d complications which can be reduced by correction of the deficiency.

Iodine deficiency in the neonate

Neonatal hypothyroidism is a well-recognized cause of mental defects. This is due to the fact that the development of the brain is dependent on an adequate supply of thyroxine. Only about one third of normal brain development occurs before birth and the other two-thirds are completed in the first two years of life. Consequently, a normal level of thyroxine is very important both during pregnancy and in the first two years of life. The frequency of transient primary hypothyroidism is almost higher in areas of iodine deficiency. This syndrome is characterised by postnatally acquired severe hypothyroidism lasting for a few weeks and requiring substitutive therapy (5).

Iodine deficiency in the infancy and childhood

Iodine deficiency in children is characteristically associated with goiter (6). Euthyroid pubertal goiter is especially frequent in adolescents and occasionally requires substitutive therapy by thyroxine or iodine, because iodine metabolism is accelerated during this period of life. Children born and living in an iodine deficient area exhibit subtle or even overt neuropsychointellectual deficits as compared to controls living in the same ethnic, demographic nutritional, and socioeconomic system, except from that they are not submitted to iodine deficiency (7).

Iodine deficiency in the adult

The most common effect of iodine deficiency in adults is simple goiter with absence of classic clinical hypothyroidism. However, laboratory evidence of hypothyroidism with reduced T4 levels is common. This is often accompanied by normal T3 levels and raised TSH levels. In conditions of borderline iodine intake the thyroid function is frequently altered during pregnancy with progressive decrease of serum free T4 and consequently by an increase in serum TSH (6).

Iodine - induced hyperthyroidism

This condition is a complication of the correction of iodine deficiency occurring mainly in those over the age of 45 with long standing deficiency and cannot be entirely avoided even when supplementation is of only physiologic amounts of iodine (10 Delange). The possible reason for the development of iodine induced hyperthyroidism after iodine supplementation has now been identified: iodine deficiency increase thyrocyte proliferation and mutation rates. Possible consequences are the development of hyperfunctioning autonomous nodules in the thyroid, possible due to mutations TSH receptor, and hyperthyroidism after iodine supplementation (8)..

Increased susceptibility to nuclear radiation

Elevated thyroid uptake due to iodine deficiency aggravates the risk of thyroid irradiation and development of thyroid cancer in case of a nuclear accident (9) The best prophylaxis of radioiodine fallout from nuclear accidents is to increase the basal intake of iodine of the population (7).

Effects of Iodine Deficiency in Animals

Iodine deficiency has a negative influence on development of cattle breeding. In cattle, horses, sheep and pigs beside goiter iodine deficiency contributes to an increased rate of abortion. These animals, in area of iodine deficiency, have retarded growth, reduced fertility, poor meat yield and wool. Furthermore, human food products from such animals (milk and meat) contain low quantities of iodine. For those reasons salt for domestic animals also should be iodised.

The Socioeconomic Consequences of Iodine Deficiency

The health problems of iodine deficiency reflect on socioeconomic life. The population living in an area of iodine deficiency have less mental and working capacities. Moreover there will be an increased number of handicapped individuals who depend on others, for their care. High incidence of goiter caused by iodine deficiency requires complicated and expensive clinical and laboratory resources. In this way a poor public health care system becomes further impoverished.

Monitoring the Iodine Prophylaxis

The indicators for monitoring the success of iodine prophylaxis in public health programmes to eliminate IDD are: salt iodisation, urinary iodine, thyroid size and neonatal TSH (10). The criteria for these indicators are the following:

- the prevalence of goiter among school children, established by palpation or ultrasound method is under 5%,
- the average value of iodine concentration in urine samples is higher or equal to 100 μg/L i.e., that less than 50% of samples have a concentration under 100 μg/L and that less than 20% of samples have a concentration under 50 μg/L,
- less than 3% of full blood samples have a concentration of TSH above 5 mU/L, and
- more than 90% of salt samples at household level contain efficiently iodised salt.

In order to proclaim "Elimination of IDD" in a country, the criteria of salt iodisation must be satisfied if this is the main method of prevention. Additionally, at least another two of the remaining criteria must be fulfilled.

A satisfactory and cheap pointer to the elimination of IDD is provided by results of neonatal systematic screening for congenital hypothyroidism based on the detection of elevated blood levels of TSH. This gives an insight into the iodine supply of pregnant women and neonates as the most vulnerable population groups.

Prevention and correction of IDD

The prevention and correction of IDD requires changes in eating patterns oriented to a higher consumption of food rich in iodine. This may however not be feasible in normal daily practice. Hence the addition of iodine to food or water has been recommended for preventing and correcting iodine deficiency. While several foods have been considered as possible vehicles for iodine ingestion salt, for human and animal consumption has become the most commonly accepted. Iodised salt should be made available not only for household salt but also for industrial food production, a strategy called Universal Salt Iodisation. The public health aspects of iodine prophylaxis including the planning and monitoring the prophylactic campaigns, the technical aspects of production and distribution of iodised salt and the other methods of iodine prophylaxis. The last recommendation by WHO-UNICEF-ICCIDD (11) is that, in order to provide 150 μg/day of iodine via iodised salt and considering the average salt intake and the loss of iodine from production site to household and cooking, the iodine concentration in salt at the site of production should range between 20 to 40 mg iodine per kilogram of salt.

Apstrakt

JOD DEFICITARNI POREMEĆAJI

U radu se iznose sadašnje znanje o poremećajima koji nastaju u organizmu usljed nedostatka joda. Dnevne fiziološke potrebe ljudskog organizma za jodom su u dobu od 0 do 12 mjeseci 50 μg/dan, od 1 do 6 godina 90 μg/dana, od 7 do 12 godina 120 μg/dan, u toku adolescencije i odraslom dobu 150 μg/dan i u toku trudnoće i laktacije 200 μg/dan. Kada navedeni fiziološke potrebe nisu zadovoljeni u određenoj populaciji dočiće do ozbiljnih funkcionalnih i razvojnih poremećaja u svim životnim dobima danas poznati kao jod deficitarni poremećaji, među kojima je vidljiva guša samo «vrh sante leda». Vodeći uzrok preventabilne mentalne retardacije i neurointelektualnog deficita u školske djece je jodni deficit, a jedna od veoma važnih posljedica njegovog deficita nastaje u slučaju nuklearne havarije kada je povećana tireoidna akumalacija radioaktivnog joda što ima za posljedicu povećanje rizika od razvoja tireoidnog karcinoma. Osim zdravstvenih problema jodni deficit ima negativne reperkusije na domaće životinje što rezultira u smanjenu produkciju sa negativnim ekonomskim posljedicama. Za rješenje ovih poremećaja najlogičniji pristup je uvođenje jodne profilakse čiji javno zdravstveni aspekti uključuju planiranje i praćenje profilaktičkih mjera, tehničke aspekte proizvodnje i distribucije jodirane soli ili drugih sredstava jodne profilakse. Da bi se obezbjedilo 150 μg joda na dan putem jodirane soli uzimajući u obzir prosječni dnevni unos soli u organizam i gubitke joda iz soli od mjesta proizvodnje do mjesta potrošnje kao i u procesu termičke obrade hrane prema posljednjim preporukama Svjetske zdravstvene organizacije, UNICEF-a i Internacionalnog vijeća za jod deficitarne poremećaje koncentracije joda u soli na mjestu proizvodnje treba da budu između 20 i 40 mg joda po kg soli.

Key words: jod deficitarni poremećaji, jodna profilaksa, jodirana so.

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