

Table 52.8 Magnitude and global prevention approach to iodine deficiency disorders

Globally, about 740 million people are affected by goiter, and more than 2 billion living in 130 countries are estimated to be at risk for iodine deficiency disorders (IDD).

Iodine deficiency is the world's leading cause of preventable mental retardation and impaired psychomotor development in young children. In its extreme form, iodine deficiency causes cretinism.

Iodine deficiency may be associated to alterations in the progeny's psychoneuro-intellectual, developmental prognosis.

Salt iodization is the preferred strategy in eliminating IDD as a public health problem, and universal iodization is the target for the beginning of the twenty-first century.

The addition of encapsulated Fe to iodized salt improves the effectiveness of iodine in goitrous children where the prevalence of anemia is high.

School health programs that include deworming, feeding, giving an adequate supply of iron and iodine supplements, as well as health education, all have potentially beneficial effects on the health and education of schoolchildren.

Oppenheimer (2002) revealed the following conclusions and recommendations described in Table 52.7.

Iodine and Fe Fortification and Parasitosis

Freeman *et al.* (2001) evaluated the effectiveness of salt fortified with diethylcarbamazine (DEC) and iodine to eliminate Bancroftian filariasis and iodine deficiency. During this 1-year period all consenting residents of Mison, Haiti ($n = 1932$) were given salt fortified with 0.25% DEC and 25 ppm of iodine. *Wuchereria bancrofti* microfilaria prevalence and intensity, antigenemia and urinary iodine were measured before and after the first year salt distribution began. To measure the effect of DEC-fortified salt on adult worm motility, 15 microfilaria-positive men were examined with a scrotal area ultrasound. Entomologic surveys were conducted to determine the *W. bancrofti*-infected *Culex quinquefasciatus* proportion. After one year's treatment, the prevalence and intensity of microfilaremia were reduced by more than 95%, while antigenemia levels were reduced by 60%. The motility of adult worms, detected by ultrasound, was not significantly decreased by DEC-fortified salt. The proportion of vector mosquitoes carrying infectious stage larvae decreased significantly from 2.3% in the nine months before the intervention to 0.2% in the last 3-month follow-up period. Iodine deficiency, which had been moderate to severe, was eliminated after one year of iodized salt consumption. The DEC-fortified salt was well accepted by the community and reduced microfilaremia; its low-level transmission had no reported side-effects. Based on their results, they concluded that salt co-fortified with DEC and iodine should be considered as a concurrent intervention for lymphatic filariasis and iodine deficiency elimination programs.

Zimmermann *et al.* (2002) remind us that, in less industrialized countries, children are at high risk for both goiter and anemia, and that ID adversely affects thyroid metabolism, reducing the efficacy of iodine prophylaxis in areas

of endemic goiter. As a result, they carried out a study to verify that cofortification of iodized salt with Fe would improve iodine efficacy in goitrous children with a high prevalence of anemia. This 9-month, randomized, double-blind trial included 6–15-year-old children ($n = 377$). They were given iodized salt (iodine 25 $\mu\text{g/g}$ salt) or dual-fortified salt with iodine (iodine 25 $\mu\text{g/g}$ salt) and Fe (1 mg Fe/g salt, as ferrous sulfate microencapsulated with partially hydrogenated vegetable oil). The dual-fortified salt group's Hb and Fe status improved significantly ($P < 0.05$) when compared to the iodized salt group. At 40 weeks, an ultrasound measured the mean decrease in thyroid volume in the dual-fortified salt group (–38%); it was twice that of the iodized salt group (–18%) ($P < 0.01$). Compared with the iodized salt group, serum T_4 was significantly increased ($P < 0.05$) and in the dual-fortified salt group, the prevalence of hypothyroidism and goiter decreased ($P < 0.01$). They concluded that the addition of encapsulated Fe to iodized salt improves iodine efficacy in goitrous children where the prevalence of anemia is high.

In an intervention study with schoolchildren 8–10 years old in Malawi, Furnee *et al.* (1997) examined the relationship of intestinal parasite treatment and oral iodized oil efficacy. Severely iodine-deficient schoolchildren with a single parasitic infestation, either *A. lumbricoides* ($n = 44$), hookworm ($n = 42$), or *Entamoeba histolytica* ($n = 24$), were randomly allocated to receive or not receive treatment before taking a 1 ml oral supplement (490 mg Iodine) of iodized ethyl esters from poppyseed oil. After supplementation, urinary iodine concentrations were measured regularly, to define time intervals indicating moderate iodine deficiency before urinary iodine concentrations returned to 0.40 mmol/l. Treatment with metronidazole for *E. histolytica* increased the protection period from 2.0 to 21.0 weeks ($P < 0.05$). For all untreated children, the duration effect was 9.2 weeks shorter ($P < 0.001$) than for their treated peers (16.8 weeks). They concluded that, by interfering with absorption, intestinal parasitic infestations reduce the efficacy of oral supplementation with iodized ethyl esters (Table 52.8).

Summary Points

Magnitude

- Iodine deficiency is the world's leading cause of preventable mental retardation and impaired psychomotor development in young children. In its extreme form, iodine deficiency causes cretinism. Globally, about 740 million people are affected by goiter and more than 2 billion living in 130 countries are estimated to be at risk for IDD.

Fetal growth

- Nutritional deficiencies are important determining factors for fetal growth, body composition and childhood development. Some stages are more vulnerable than others, and the most vulnerable stages may differ according to particular nutritional deficiencies. The intrauterine period is the most vulnerable for long-term neurological and cognitive deficit caused by iodine deficiency.

Immediate postnatal period

- Along with Fe deficiency, the immediate postnatal period (infants) is probably the most vulnerable to iodine deficiency. Iodine deficiency may be associated with alterations in the progeny's psychoneuro-intellectual, developmental prognosis. The risk of abnormal development is further enhanced because mother and offspring are exposed to iodine deficiency during gestation and the postnatal period.

ID = Iron Deficiency,
IDA = Iron Def. Anemia

Fe deficiency

- ID and IDA are estimated to affect about half of the world's population, and young children are among the most severely affected. ID adversely affects thyroid metabolism by reducing iodine prophylaxis efficacy in regions with endemic goiter. The degree of ID may affect thyroid hormone status. The therapeutic response in goitrous children to oral iodized oil is weakened when compared to Fe-sufficient children. In anemic pregnant women endemic goiter may aggravate anemia. Chronic iodine deficiency favors subclinical hypothyroidism where, in the presence of anemia, severity increases, more so if anemia is paralleled by goiter.

Parasitosis

- The most common chronic subclinical childhood infections with global dispersion are parasites such as *A. lumbricoides*, *T. trichiuria*, and hookworms. ID and helminth infestation are two of the most common conditions afflicting children in less industrialized countries. There is a significant elevation in toxoplasmosis prevalence in children with grade II goiter and a mild elevation in those with grade I goiter. A possible relationship exists between iodine deficiency and toxoplasma infection. Hookworm species being transmitted in a community influence the burden of IDA and should be considered when prioritizing and

planning programs for hookworm and anemia control. By interfering with absorption, intestinal parasitic infestation reduces the efficacy of oral supplementation with iodized ethyl esters.

Prevention

- Salt iodization is the preferred strategy in eliminating IDD as a public health problem, and universal iodization is the target for the beginning of the twenty-first century. The addition of encapsulated Fe to iodized salt improves the effectiveness of iodine in goitrous children where the prevalence of anemia is high. School health programs that include deworming, feeding, giving an adequate supply of Fe and iodine supplements, as well as health education, all have potentially beneficial effects on the health and education of schoolchildren.

find deworming protocols!

References

- ACC/SNC. (2000). Fourth Report on the World Nutrition Situation. Geneva: ACC/SNC in collaboration with IFPRI. pp. 27–29.
- Albonico, M., Stoltzfus, R.J., Savioli, L., Tielsch, J.M., Chwaya, H.M., Ercole, E. and Cancrini, G. (1998). *Int. J. Epidemiol.* 27, 530–537.
- Beard, J.L., Borel, M.J. and Derr, J. (1990). *Am. J. Clin. Nutr.* 52, 813–819.
- Beard, J.L., Brigham, D.E., Kelley, S.K. and Green, M.H. (1998). *J. Nutr.* 128, 1401–1408.
- Black, M.M. (2003). *J. Nutr.* 133(11 Suppl 2), 3927S–S3931S.
- Brito, L.L., Barreto, M.L., Silva, R.deC., Assis, A.M., Reis, M.G., Parraga, I. and Blanton, R.E. (2003). *Rev. Panam. Salud Publica* 14(6), 422–431.
- Brooker, S., Peshu, N., Warn, P.A., Mosobo, M., Guyatt, H.L., Marsh, K. and Snow, R.V. (1999). *Trans. R. Soc. Trop. Med. Hyg.* 93, 240–246, [Abstract].
- De Andraca, I., Castillo, M. and Walter, T. (1997). *Nutr. Rev.* 55, 125–132.
- Dikson, R., Awasthi, S., Williamson, P., Demellweek, C. and Garner, P. (2000). *Br. J. Med.* 320, 1697–1701.
- Dunn, J.T. (2005). In: (eds M.E. Shils, M. Shike, A.C. Ross, B. Caballero and J. Cousins), *Modern Nutrition in Health and Disease*. Lippincott Williams & Wilkins, Philadelphia, PA, pp. 300–311.
- Eftekhari, M.H., Keshavarz, S.A., Jalili, M., Elguero, E., Eshraghian, M.R. and Simondon, K.B. (2006). *Asia Pac. J. Clin. Nutr.* 15, 50–55, [Abstract].
- Fernald, L.C. and Grantham-McGregor, S.M. (1998). *Am. J. Clin. Nutr.* 68, 691–698.
- Freeman, A.R., Lammie, P.J., Houston, R., Lapointe, M.D., Sztreit, T.G., Jooste, P.L., Brissau, J.M., Lafontant, J.G. and Addiss, D.G. (2001). *Am. J. Trop. Med. Hyg.* 65, 865–871.
- Furnee, C.A., West, C.E., van der Haar, F. and Hautvast, J.G. (1997). *Am. J. Clin. Nutr.* 66, 1422–1427.

Treated children w parasites and iodine deficiency. Iron and metronidazole improved efficacy.

- Glinoe, D. (1993). *Ann. Endocrinol.* 64, 37–44, [Abstract].
- Grantham-McGregor, S. and Ani, C.C. (2001). In: (eds J.D. Fernstrom, R. Uauy, O. Arroyo), *Nutrition and Brain. Nestlé Nutrition Workshop Series and Performance Program*, Vol. 5. Nestec Ltd. Karger AG, Vevey, pp. 1–18.
- Grantham-McGregor, S. and Ani, C.C. (2002). In (eds R.E. Black and K. Fleisher), *Public Health Issues in Infant and Child Nutrition. Nestlé Nutrition Workshop Series, Pediatric Program*. Vol. 48. Nestec Ltd., Lippincott Williams & Wilkins, Philadelphia, PA, pp. 53–70.
- Hess, S.Y., Zimmermann, M.B., Arnold, M., Langhans, W. and Hurrell, R.E. (2002). *J. Nutr.* 132, 1951–1955.
- Hetzl, B.S. (1991). In (ed. B. Momcilovic), *Trace Elements in Man and Animals*. Zagreb: Institute for Medical Research and Occupational Health, University of Zagreb, Croatia, ISBN 86-81477-02-1, pp. 7-1–7-3.
- Huda, S.N., Grantham-McGregor, S.M., Rahman, K.M. and Tomkins, A. (1999). *J. Nutr.* 129, 980–987.
- Hurtado, E.K., Claussen, A.H. and Scott, K.G. (1999). *Am. J. Clin. Nutr.* 69, 115–119.
- Idjradinata, P. and Pollit, E. (1993). *Lancet* 341, 1–4.
- Khieu, V., Odermatt, P., Mel, Y., Keluangkhot, V. and Strobel, M. (2006). *Bull. Soc. Pathol. Exot.* 99, 115–118.
- Lozoff, B., Jiménez, E. and Wolf, A.W. (1991). *N. Engl. J. Med.* 325, 687–694.
- Lozoff, B., Klein, N.K., Nelson, E.C., McClish, D.K., Manuel, M. and Chacon, M.E. (1988). *Child Dev.* 69, 24–36.
- Martínez, H., Castañeda-Limones, R., González-Unzaga, M.A., Ramos-Hernández, R.I. and Velásquez-López, L. (2005). In: (eds R.H. Bourges, E. Casanueva and J.L. Rosado), *Recomendaciones de ingestión de nutrimentos para la población mexicana*. Editorial Medica Panamericana, México DF, pp. 301–316.
- Martínez-Salgado, H., Castañeda-Limones, R., Lechuga-Martin del campo, D., Ramos-Hernandez, R.J., Orozco-Lopez, M., Rivera-Dommarco, J., Mendoza, I. and Magos, C. (2002). *Gaceta Medica México* 138, 149–156.
- Nelson, C., Erikson, K., Pinero, D. and Beard, J.L. (1997). *J. Nutr.* 127, 2282–2288.
- Oppenheimer, S.J. (2002). In (eds R.E. Black and K. Fleischer), *Public Health Issues in Infant and Child Nutrition*. Nestlé Nutrition Workshop Series, Pediatric Program. Vol. 48. Nestec Ltd. Lippincott Williams & Wilkins, Philadelphia, PA, pp. 111–146.
- Palti, H., Meijer, A. and Adler, B. (1985). *Early Hum. Dev.* 10, 217–223.
- Pinero, D.J., Li, N.Q., Connor, J.R. and Beard, J.L. (2000). *J. Nutr.* 130, 254–263.
- Rivera, J., Shamah, T., Villalpando, S., González, T., Hernández, B. and Sepúlveda, J. (2001). Encuesta Nacional de Nutrición 1999. Estado nutricional de niños y mujeres en México. Instituto Nacional de Salud Pública. Cuernavaca, Morelos, México.
- Singh, S., Singh, N., Pandav, R., Pandav, C.S. and Karmarkar, M.G. (1994). *Indian J. Med. Res.* 99, 27–31.
- Smith, S.M., Finley, J., Johnson, L.K. and Lukaski, H.C. (1994). *Nutr. Res.* 14, 729–739.
- Stoltzfus, R.J., Chway, H.M., Montresor, A., Tielsch, J.M., Jape, J.K., Albonico, M. and Savioli, L. (2004). *J. Nutr.* 134, 348–356.
- Ulukanligil, M. and Seyrek, A. (2004). *Eur. J. Clin. Nutr.* 58, 1056–1061.
- UNICEF. The State of the World's Children. (2006). Available at: http://www.unicef.org/sowc06/pdfs/sowc06_fullreport.pdf [Accessed 17 March 2007].
- Vásquez-Garibay, E.M., Romero-Velarde, E., Nápoles-Rodríguez, F., Nuño-Cosío, M.E., Trujillo-Contreras, F. and Sánchez-Mercado, O. (2002). *Salud Publica México* 44, 195–200.
- Villalpando, S., García-Guerra, A., Ramírez-Silva, C.I., Mejía-Rodríguez, F., Matute, G., Shamah-Levy, T. and Rivera, J.A. (2003). *Salud Publica México* 45, S520–S529.
- WHO/UNICEF/ICCIDD (International Council for the Control of Iodine Deficiency Disorders) (1993). *Global Prevalence of Iodine Deficiency*. WHO, Geneva, Switzerland.
- Wolde-Gebriel, Z., West, C.E., Gebru, H., Tadesse, A.S., Fisseha, T., Gabre, P., Aboye, C., Ayana, G. and Hautvast, J.G. (1993). *Br. J. Nutr.* 70, 593–607.
- World Health Organization, United Nations Children's Fund & International Council for the Iodine Deficiency Disorders. Assessment of Iodine deficiency Disorders and Monitoring their Elimination. (2001). WHO/NHD/01.1. WHO, Geneva, Switzerland.
- Yajnik, C. (2002). *Nutr. Rev.* 64(5 pt. 2), S50–S51, Discussion S72–91.
- Yavuz, O., Yavuz, T., Kahraman, C., Yesildal, N. and Bundak, R. (2004). *J. Pediatr. Endocrinol. Metabol.* 17, 1443–1449.
- Zel'tser, M.E., Mezinova, N.N., Kobzar, N.N., Bazarbekova, R.B., Nazyrov, A.T., Kim, G.G., Nurbekova, A.A. and Nugmanova, M.I. (1994). *Probl. Endokrinol. (Mosk)* 40, 20–22, [Abstract].
- Zimmermann, M.B., Adou, P., Zeder, C., Torresani, T. and Hurrell, R.F. (2000). *Am. J. Clin. Nutr.* 71, 88–93.
- Zimmermann, M.B., Zeder, C., Chaouki, N., Torresani, T., Saad, A. and Hurrell, R. (2002). *Eur. J. Endocrinol.* 147, 747–753.