Dietary Iodine Intake in Pregnancy
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Iodine deficiency is a worldwide health problem, which continues to affect even highly-developed countries and in its severe form is a leading cause of preventable mental retardation[1]. However, there is much debate on the consequences of less severe forms of iodine deficiency, in particular concerning its impact on neurological development in the fetus[2,3].

Because the fetal thyroid does not develop until 13 - 15 weeks gestation, neuropsychological development is entirely dependent on maternal thyroid hormone supply, which in turn relies among other variables on maternal dietary iodine intake[4].

Recent publications have shown that mild maternal hypothyroidism or even asymptomatic hypothyroxinaemia can result in an intellectual deficit[5] and/ or neuropsychomotor defects and may be associated with Attention Deficit and Hyperactivity Disorder (ADHD)[6]. In addition to iodine deficiency, children born to mothers having circulating antithyroid antibodies are at risk of impaired neurological development even if thyroid function is normal.

The introduction of Universal Salt Iodization (USI)7 represents a simple intervention which does not require any change in dietary habits, in particular table salt intake, but simply involves the addition of an iodinated substance, usually potassium iodide (KI) or potassium iodate (KIO3) to table salt at a concentration of 20 to 40 mg/Kg. Most countries have adopted a voluntary approach to salt iodization with the result that in Europe only 27% of households use iodised salt[7]. In the absence of USI it would probably be practical to target a programme of iodine prophylaxis at pregnant women in the form of mineral supplemented vitamin pills. Administration of iodide (~200 µg per day) would fulfil WHO recommended dietary iodine intake (200 - 300 µg /day). Iodine is easy to administer, inexpensive and has the potential of producing a dramatic effect particularly during the first trimester where brain development occurs1 but national experience in respect of opportunistic periconceptual folate supplementation has not been encouraging[8].

The case for iodine supplementation is strengthened by recent reports from North America and Australia, heretofore considered areas replete in iodine, of a decline in dietary iodine intake in both the general and pregnant populations[9], probably attributable to decreases in milk or bread iodine content. Studies on iodine intake in Ireland would be particularly relevant as only approximately 3.3% of total salt sales are in the iodised form (RHM Foods, Private Communication). Thus, dietary iodine intake in the Irish population is opportunistic and subject to changes based on dietary preferences. For example seafish and marine products are
iodine rich but levels of consumption vary widely. Milk and dairy products constitute a major iodine source but their content shows seasonal variation with a higher iodine content when cattle in winter housing are fed dietary supplements including iodine. Seasonal variation could contribute to lower dietary iodine intake in pregnant women

during summer months and particularly affect first trimester fetuses at that time. Maternal iodine status is not entirely dependent on dietary intake during gestation. If preconception iodine nutrition is adequate there will be sufficient stored thyroid hormone to support mother and fetus at least in the crucial first
trimester. However, if preconception dietary intake has been deficient the increasing demands of later pregnancy may produce a deficit which if unsupplemented may result in a hypothyroxinemic state.

Available data suggest that Ireland, in common with most European states is borderline iodine deficient[10,11] and that the pattern of iodine excretion would in the absence of iodine supplementation result in significant maternal negative maternal iodine balance[12]. As in the case of the United States, iodine intake in Ireland may not be in a steady state due to changes in dietary habits, food preparation and agricultural practice. Some Irish women and their babies may not be adequately protected against the potentially damaging effects of iodine deficiency suggesting that a fresh look at dietary iodine intake in Ireland is a timely priority.

There is much evidence that hypothyroidism or hypothyroxinaemia can result in impaired cognition in the neonate but the benefits of iodide supplementation in terms of neuropsychological or neuromotor development in the infants born to mothers living in areas of moderate or borderline dietary iodine intake remain to be confirmed. Screening for neonatal hypothyroidism as practiced in Ireland and most developed countries is very successful in detecting hypothyroidism arising from
thyroid agenesis or dysgenesis which can, if detected early, be treated successfully with replacement therapy. Unfortunately, neonatal hypothyroidism arising from significant early pregnancy maternal iodine deficiency can give rise to irreversible mental retardation in the neonate (1;2) . Although it cannot be said whether deleterious consequences in terms of neuropsychological development, could result if current dietary iodine intake in pregnant Irish women remain unchanged, the prospect of preventing subtle cognitive impairment in even a fraction of at - risk children merits more detailed investigation.

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References

1. Delange F. Iodine deficiency as a cause of brain damage. Postgraduate Medical Journal 2001; 77:217-220.
2. Glinoer D, Delange F. The potential repercussions of maternal, fetal, and neonatal hypothyroxinemia on the progeny. Thyroid 2000; 10:871-887.
3. Consensus Statement #2 American Thyroid Association Statement on Early Maternal Thyroidal Insufficiency: Recognition, Clinical Management and Research Directions . Thyroid 2005 ;15: 77-79
4. Glinoer D. The regulation of thyroid function in pregnancy: pathways of endocrine adaptation from physiology to pathology. Endocr Rev 1997; 18:404-433.
5. Haddow JE, Palomaki GE, Allan WC, Williams JR, Knight GJ, Gagnon J et al. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. New England Journal of Medicine 1999; 341:549-555.
6. Vermiglio F, Lo Presti VP, Moleti M, Sidoti M, Tortorella G, Scaffidi G et al. Attention deficit and hyperactivity disorders in the offspring of mothers exposed to mild-moderate iodine deficiency: A possible novel iodine deficiency disorder in developed countries. Journal of Clinical Endocrinology and Metabolism 2004; 89:6054-6060
7. WH0/, NHD01/1. 2001 Assessment of Iodine Deficiency Disorders and Monitoring their Elimination. Geneva.
8. Ward M,Hutton J,Mc Donnell R,Bachir N,Scallan E,O'Leary M,Hoey J,Doyle A,Delany V,Sayers G. Folic acid supplements to prevent neural tube defects: trends in East of Ireland 1996-2002. Ir Med J 2004; 97, 274-276..
9. Hollowell JG, Staehling NW, Hannon WH, Flanders DW, Gunter EW, Maberly GF et al. Iodine nutrition in the United States. Trends and public health implications: Iodine excretion data from National Health and Nutrition Examination Surveys I and III (1971-1974 and 1988-1994). J Clin Endocr Metab 1998; 83(10):3401-3408
10. Hetherton AM and Smyth PPA. Status of iodine deficiency in Ireland. in Iodine Deficiency in Europe - A Continuing Concern. NATO ASI Series , 317-322. 1993. New York, Plenum Press.
11. Smyth PPA, Hetherton AMT, Smith DF, Radcliff M, OHerlihy C. Maternal iodine status and thyroid volume during pregnancy: Correlation with neonatal iodine intake. J of Clin Endocr Metab 1997; 82:2840-2843.
12. Smyth PPA. Variation in iodine handling during normal pregnancy. Thyroid 1999;9 :637-642.