**DOES PLACENTAL IODINE STORAGE COMPENSATE FOR LOW MATERNAL IODINE INTAKE?**


**SUMMARY**

**BACKGROUND**

The authors’ objective is to compare the iodine content of placentas from women giving birth at term in Ireland and Iran, areas with a median urinary iodine (UI) of 79 µg/L and 206 µg/L, respectively.

Controversy exists about the role of the placenta in transferring to the fetus iodine ingested by the mother. Although there is evidence of subclinical hypothyroidism or hypothyroxinemia in children born to women with borderline to low dietary iodine intake as assessed by urinary iodine excretion, the majority of mothers and infants show no ill effects from moderate iodine deficiency. Iodine transporters are present in the placenta; however, the proportion of iodine that crosses the placenta by active, as distinct from passive, diffusion or whether iodide stored in the placenta is available to the fetus or is directly related to the iodine content of the diet are unclear. The difference in UI between the two countries is almost certainly related to the policy of mandatory salt iodization undertaken in Iran; in Ireland, iodized salt represents <5% of total salt sales.

**METHODS**

Placental samples were obtained from 58 women from Ireland and 45 from Iran, delivered at term by elective pre-labor cesarean section, following uncomplicated singleton pregnancies, with normal neonatal outcomes. Tissue iodine content was determined from segments of cotyledons, approximately six per placenta, from different locations within each placenta, periphery and center. Results were expressed as nanograms of iodine per gram of tissue, wet weight.

**RESULTS**

The mean placental iodine values were 34 ng/g (range, 20 to 269) in women from Ireland and 187 ng/g (range, 20 to 400) in women from Iran (P<0.01). There was a wide variation in iodine values within individual Iranian women, with 42% of them having values <100 ng/g, while only 1% of Irish women had values >100 ng/g. The relationship of placental iodine (in nanograms per gram) to urinary iodine (in micrograms per liter) for the population of pregnant women in Ireland was 1:2, while in the pregnant women in Iran it was 1:1.

**CONCLUSIONS**

These findings, by demonstrating an apparent ability of the placenta to store iodine in a concentration-dependent manner, suggest a hitherto undetected role for the placenta. Whether placental iodine has a role in protecting the fetus from inadequacies in maternal dietary iodine intake is as yet unknown.

**COMMENTARY**

Several investigators in the past decade have studied the role of the placenta in iodine metabolism. The subject was nicely reviewed in a recent article by Smyth’s group (1). Both iodine transporters, sodium iodine symporter (NIS) and pendrin, have been identified in the placenta (2), as well as deiodinases D3 and to a lesser extent D2. Iodine transporters accumulate iodine in placental cell lines; NIS is up-regulated by human chorionic gonadotropin (3) and in rats on a low-iodine diet (4). Burns et al. (1) suggest that the placenta has a role not only in uptake but also in storing iodine as a possible means of protecting the fetus from inadequacies in maternal dietary iodine intake. Future research may be directed to a study of disturbances of iodine metabolism as the result of placentas being affected by obstetrical pathologies, such as gestational hypertension, prematurity, and placental abruption, among many others (5).

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References


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