Format: Abstract

Full text links

ELSEVIER FULL-TEXT ARTICLE

J Theor Biol. 2014 Aug 21;355:61-7. doi: 10.1016/j.jtbi.2014.03.041. Epub 2014 Apr 8.

Pregnancy sickness and parent-offspring conflict over thyroid function.

Forbes S[1].

Author information

1 Department of Biology, University of Winnipeg, 515 Portage Avenue, Winnipeg, MB, Canada R3B 2E9. Electronic address: s.forbes@uwinnipeg.ca.

Abstract

Pregnancy sickness is widespread in human mothers but its etiology, somewhat surprisingly, remains unclear. Human chorionic gonadotropin (**hCG**) has long been considered a prime hormonal suspect, but the correlation between pregnancy sickness and **hCG** levels is imperfect resulting in uncertainty about its causal role. As others have noted part of this uncertainty likely stems from the structural and functional diversity of **hCG**. One enigmatic role of **hCG** is its action as a thyroid stimulator during early gestation. Native **hCG** is weakly thyrotropic but is produced in prodigious quantities and suppresses the production of thyroid stimulating hormone (TSH) but not curiously when TSH levels are in the higher deciles. Higher levels of **hCG** induce higher maternal production of thyroxine (T4). **hCG** thus appears to augment and sometimes even supplant TSH in the regulation of thyroid hormone in early gestation. This has lead to the suggestion that **hCG** serves as a backup system, albeit incomplete, for the production of essential thyroid hormone during pregnancy. Another interpretation, however, is that **hCG**, produced by the embryo, serves as a second control circuit for the thyroid during pregnancy. If so, it serves embryonic interests that are at odds with maternal interests (maternal-embryo conflict) under conditions of iodine deficiency. Iodine is an essential micronutrient for neurodevelopment and thyroid function, and has been in short supply for most humans over most of our evolutionary history. Iodine deficiency during gestation has severe impacts on embryo neuromotor development, but also induces thyroid disease in mothers, impairing her future reproductive prospects. Under this view, embryos use **hCG** to push mothers to release more thyroid hormone. **hCG**, however, is produced outside the normal maternal thyroid control circuit and thus is not subject to a normal negative feedback. **hCG** also serves multiple functions simultaneously therefore its production is likely not fine-tuned for thyroid function per se. **hCG** levels may remain high even when thyroid hormone production is more than sufficient to meet the needs of mother and embryo. Instead, the system appears to be regulated at the back end by clearing surplus hormone using placental Type II (D2) and Type III (D3) deiodinases. As maternal thyroid hormone levels rise, placental D3 is upregulated, shunting more T4 and T3 into a deactivating pathway. The metabolites that result, particularly the inert metabolite of T4, reverse T3, are correlates of surplus thyroid hormone production and thus are strong candidates for the proximate triggers of pregnancy

sickness. Nausea and vomiting of early pregnancy thus arises as a by-product of an antagonistic pleiotropy between mother and embryo over the allocation of iodine: when dietary iodine is scarce, a benefit accrues to the embryo at a cost to mother; when iodine is plentiful, pregnancy sickness ranging from frequently mild to occasionally severe, is a sequelae of undiminished embryonic demands. If pregnancy sickness serves as a marker of thyroid function, an absence of first trimester nausea and vomiting sickness may indicate a higher priority for testing of thyroid function to avert the inimical effects of hypothyroidism during gestation.

KEYWORDS: Human chorionic gonadotropin; Iodine; Maternal-embryo conflict; Morning sickness

PMID: 24721478 DOI: 10.1016/j.jtbi.2014.03.041

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

LinkOut - more resources