surprising, nor evidence against genetic conflict, as Flaxman and Sherman claim; nor is evidence of genetic conflict over miscarriage expected in all eutherian mammals – only those with CGs.

The link between hCG and NVP grows ever stronger and more interesting. hCG is very likely to be subject to genomic imprinting, suggesting not only parent–offspring conflict, but also conflict between mother and her embryo's paternal genes [5,6] It is the type, as much as the quantity, of hCG that triggers NVP [7]. Recent epidemiological data confirm the link between twinning, fetal sex and hyperemesis [8,9]. Female fetuses and twins produce more hCG and trigger hyperemesis more often. Are daughters and twins in greater need of protection from dietary toxins than are sons and singletons? Or are trisomy 21 embryos or hydatidiform moles especially sensitive to pathogens? Or, is the common thread a hormonal linkage that revolves around a maternal–fetal conflict? I do not doubt that maternal dietary changes during pregnancy are adaptive, and might benefit both mother and embryo. The evidence is circumstantial, but, as Thoreau said, some circumstantial evidence is very strong. Could such adaptations be superimposed upon an initial genetic conflict? Quite possibly. But is embryo–maternal protection the direct antecedent of NVP? We have yet to find the trout in the milk.

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References

1 Flaxman, S.M. and Sherman, P.W. (2002) Is morning sickness maladaptive? *Trends Ecol. Evol.* 17 10.1016/S0169-5347(02)02557-0
2 Pigliucci, I.I. and Kaplan, I.I. (2000) The fall and rise of Dr Pangloss: adaptationism and the Spandrels paper 20 years later. *Trends Ecol. Evol.* 15, 66–70
3 Schmidt-Sarosi, C. *et al.* (1998) Chromosomal analysis of early fetal losses in relation to transvaginal ultrasonographic detection of fetal heart motion after infertility. *Fertil. Steril.* 69, 274–277
4 Homan, G. *et al.* (2000) Human chorionic gonadotropin as a predictor of outcome in assisted reproductive technology pregnancies. *Fertil. Steril.* 73, 270–274
5 Lustbader, J.W. *et al.* (1998) Structural and molecular studies of human chorionic gonadotropin and its receptor. *Rec. Progr. Horm. Res.* 53, 395–424
6 Haig, D. (1993) Genomic imprinting, human chorionic gonadotropin and triploidy. *Prenat. Diagn.* 13, 151
7 de-Groot, N. *et al.* (1993) Genomic imprinting and β-chorionic gonadotropin. *Prenat. Diag.* 13, 1159–1160
8 Basso, O. and Olsen, J. (2001) Sex ratio and twinning in women with hyperemesis or pre-eclampsia. *Epidemiology* 12, 747–749
9 del Mar Melero-Montes, M. and Jick, H. (2001) Hyperemesis gravidarum and the sex of the offspring. *Epidemiology* 12, 123–124

Are mothers battling embryos or pathogens?

In a recent Opinion article in *TREE* [1], Forbes champions Haig's [2,3] proposal that the maternal–fetal conflict causes nausea and vomiting during pregnancy (NVP), and criticizes the hypothesis that NVP serves a prophylactic function [4,5]. However, there are problems that plague Forbes' position.

Addressing the proximate cause of NVP, Forbes cites the link between female fetuses, associated with high levels of human chorionic gonadotropin (hCG), and NVP. However, the value of male and female offspring differs as a function of maternal rank [6]; therefore, if NVP is due to genetic conflict, the association between fetal sex and NVP should be conditional rather than invariant.

For the mother, the value of a given fetus is a function of her remaining reproductive potential [7,8]. If NVP is the result of maternal–fetal conflict, severity should therefore be inversely related to maternal age, yet little or no correlation occurs [9]. A mother's willingness to invest is influenced by her caloric reserves, whereas the fetus prioritizes investment over maternal maintenance [10]. Maternal–fetal conflict should therefore be greater in lightweight than in heavyweight mothers, yet, as Forbes notes, the latter are more prone to NVP.

Forbes concedes that the patterning of gestational dietary alterations suggests an adaptation, yet he argues that this is independent of NVP. However, as in nongestational aversions [11], the development of gestational aversions is tied to nausea and vomiting [4,12]. To assert that aversions are produced by a mechanism that does not involve nausea and vomiting is to posit an evolutionarily novel and hitherto undocumented adaptation.

The prophylaxis hypothesis accounts for the coordination of NVP with immunosuppression and the association between aversions and the risk of infection [4,5]. Challenges to this hypothesis include the lack of a clear association between NVP and progesterone, the keystone of immunosuppression [5]. Moreover, criticisms of the genetic conflict hypothesis also weaken the prophylaxis hypothesis, as they are compatible: Because the mother pays a cost (immunovulnerability) to provide a benefit (freedom from attack by the maternal immune system) to the fetus, the level of immunosuppression, and hence of NVP, can be contested [5].

Forbes' proposal to introduce hCG into rodents fails to test these hypotheses, because manipulation of a postulated proximate mechanism sheds no light on ultimate cause. However, if homologs of this mechanism exist in nonhuman primates, then, because maternal–fetal conflict varies with the degree of promiscuity [2,3], these mechanisms should vary across mating systems. Conversely, if NVP serves a prophylactic function: (a) behavioral analogs of pregnancy sickness should occur in meat-eating omnivores but not in carnivores or non-meat-eating species; and (b) across women, the severity of NVP should correspond with the degree of immunosuppression [5].

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References

1 Forbes, S. (2002) Pregnancy sickness and embryo quality. *Trends Ecol. Evol.* 17, 115–120
2 Haig, D. (1993) Genetic conflicts in human pregnancy. *Q. Rev. Biol.* 68, 495–532
3 Haig, D. (1996) Altercation of generations: genetic conflicts of pregnancy. *Am. J. Reprod. Immunol.* 35, 226–232
4 Flaxman, S.M. and Sherman, P.W. (2000) Morning sickness: a mechanism for protecting mother and embryo. *Q. Rev. Biol.* 75, 113–148
5 Fessler, D.M.T. (2002) Reproductive immunosuppression and diet: an evolutionary perspective on pregnancy sickness and meat consumption. *Curr. Anthropol.* 43, 19–39; 48–61
6 Trivers, R.L. and Willard, D.E. (1973) Natural selection of parental ability to vary the sex ratio of offspring. *Science* 179, 90–92
7 Trivers, R.L. (1974) Parent–offspring conflict. *Am. Zool.* 14, 247–262
8 Forbes, L.S. (1997) The evolutionary biology of spontaneous abortion in humans. *Trends Ecol. Evol.* 12, 446–450
9 Weigel, M.M. *et al.* (2000) Early pregnancy nausea and vomiting in a high-altitude Andean population. *Int. J. Gynaecol. Obstet.* 69, 9–21
10 Blurton Jones, N. (1978) Natural selection and birthweight. *Ann. Human Biol.* 5, 487–489
11 Bernstein, I.L. (1999) Taste aversion learning: a contemporary perspective. *Nutrition* 15, 229–234
12 Dye, L. *et al.* (1998) Food aversions and cravings during pregnancy: a temporal association with nausea and vomiting. *Int. J. Obesity* 22, S130

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