The actual level of resource transfer will presumably lie somewhere between the two parental optima. Following a switch to monogamy, the new paternal optimum will coincide with the existing maternal optimum and selection on both sets of parental alleles will act to move the actual level of resource transfer to the new, uncontested equilibrium. In effect, selection will act to reduce the overall level of resource transfer from mother to offspring. How is this likely to affect imprinting?

For growth-promoting, maternally silenced genes (such as *Igf2*), loss of imprinting would increase gene dosage and resource demands, and therefore loss of imprinting of such genes will be counter-selected. Loss of imprinting of paternally silenced, growth-inhibitory genes (such as *Igf2r*) would be predicted to lead to reduced resource demands. Why was such a loss of imprinting not observed by Vrana *et al.*? We suggest that loss of imprinting of paternally silenced genes represents a minute proportion of the potential mutations that might achieve the desired reduction in resource transfer. Many hundreds, or perhaps thousands, of genes probably influence resource transfer, of which only approximately 50 (our current estimate) are imprinted. Moreover, even within the imprinted set, desirable mutations are as likely, or more likely, to alter aspects of gene function other than imprinting. For example, mutations in promoters, enhancers, sequences controlling mRNA splicing and translation, and sequences affecting RNA and protein stability may be selected. Therefore, we would not expect loss of imprinting to occur as a direct result of a switch to strict monogamy, unless the costs of maintaining it significantly outweigh those of removing it.

Hurst[6] suggests that the cost of hemizygosity (that is, the exposure of deleterious recessives) will promote diploid expression following a switch to monogamy. Deleterious recessives, however, will not provide significant selection in the short term because such deleterious alleles will not have accumulated at imprinted loci due to prior continual exposure in the hemizygous state. (We note a possible cost to maintaining imprinting, that of pathological loss of imprinting[7].) The costs of removing imprinting may be significant. If, through the action of mutations that do not affect imprinting, the actual level of resource transfer approaches the newly selected optimum favoured under monogamy, subsequent mutations that result in loss of imprinting will be counter-selected if they upset the newly acquired equilibrium. We suggest that this is likely because imprinted genes tend to occur in tight physical linkage, with complex sharing of regulatory elements[8]. Mutations that induce loss of imprinting without significantly disrupting the growth equilibrium may be rare. On balance, therefore, we suggest that selection for loss of imprinting may be very weak or effectively non-existent.

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Genetic conflicts and the private life of *Peromyscus polionotus*

One might think that the question whether a female *Peromyscus polionotus* has sexual relations with more than one partner is nobody's affair but her own. Hurst[1], however, argues that the coexistence of partner fidelity with genomic imprinting in *P. polionotus*[2] adds to evidence contradicting the 'conflict hypothesis', which states genomic imprinting has evolved because of the conflicting interests of maternal and paternal genomes[3,4].

P. polionotus is not strictly monogamous, but Hurst questions whether the low rate of partner exchange is sufficient to maintain imprinting given the cost of increased exposure to deleterious recessives when one allele is silent. Neither the selective cost of deleterious recessives nor the rate of partner change is known with precision, but a rough comparison suggests that paternal turnover is sufficient for the selective maintenance of genomic imprinting. At mutation–selection equilibrium, there is one selective death for each deleterious mutation[5]. Therefore, the selective force favouring the loss of imprinting is of the same order of magnitude as the mutation rate. By contrast, field data suggest that female *P. polionotus* frequently change partners (although not as often as *Peromyscus maniculatus*). Foltz[6] collected 61 pairs of consecutive litters of *P. polionotus*, and estimated that 12 subsequent litters (20%) had a different father from the mother's previous litter.

Inactivating mutations of several imprinted loci have phenotypes that Hurst believes contradict the conflict hypothesis[1]. I would prefer to wait until more is known about the normal functions of some of these loci before judging one way or the other, but will briefly comment on the case of paternally expressed *Mest*. Newborn *Mest*-deficient mice are growth retarded—consistent with predictions of the hypothesis—but *Mest*-deficient mothers neglect their young, which Hurst remarks as being "a behaviour that cannot obviously be explained by the conflict hypothesis." One may envisage, however, that if *Mest* has pleiotropic effects on growth and behaviour[7], imprinting might be maintained because of the gene's effects on fetal growth, with the imprinting of maternal behaviour a side effect. Nevertheless, one should not reject without test the possibility that natural selection has favoured imprinting of genes that affect maternal behaviour. Mice often form communal nests, with a preference for female relatives[8–10]. A mother's paternal genome would be selected to provide greater care for the mother's own offspring than would the mother's maternal genome, if by so doing the reproductive values of her own mother, her maternal half-sibs, or other maternal-side relatives were reduced[3].

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