**Peg3 and the Conflict Hypothesis**

Li et al. (1) reported that knockouts of the imprinted gene Peg3, like knockouts of the related imprinted gene Peg1/Mest (2), disrupted maternal care behavior in mice. Although this second report strengthens the link between genomic imprinting and offspring care, Li et al. are incorrect to conclude that their finding provides support for the conflict hypothesis (3) for the evolution of genomic imprinting.

Li et al. argued that paternal and maternal genomes have different interests regarding the level of maternal care, as the “paternal interest is best served by prolonged care,” while it is in the female’s interest to achieve the next pregnancy “in the shortest possible time.” At first sight the argument looks similar to the conflict hypothesis, which proposes that paternally inherited alleles demand greater nutrient resources from the mother when females have more than one mate over their reproductive life, because they have lower relatedness among siblings than do maternally inherited alleles (3).

However, the paternally expressed Peg3 affects the behavior of daughters, not of the current mate. So it is not until the following generation, that of the grandchildren, that any benefits of increased maternal care occur. This is inconsistent with a conflict-based explanation, because relatedness asymmetries do not carry over between generations; a gene that is transmitted from a father to his daughter is as likely to be transmitted to the daughter’s offspring as it is a gene that the same daughter received from her mother. Put another way, grand-offspring are equally related to their maternal grandmother and to their maternal grandfather. So there is no evolutionary reason to expect differential expression of paternally and maternally inherited genes that affect the fitness of grand-offspring through maternal care behavior. This is true for autosomal and X-linked genes. The reduced fetal size of knockouts of both Peg1 and Peg3 is predicted by the conflict hypothesis, but other reasons may need to be invoked to explain imprinting of genes controlling maternal care.

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References


Response: Before we examined the function of the Peg3 gene (1), we would not have predicted that it would affect maternal behavior; that it does is of interest regardless of its imprinted status. It is also true that this observation may not fit easily into the conflict hypothesis for the evolution of genomic imprinting. Indeed, we made no explicit claims in support of the conflict hypothesis or otherwise, but simply offered possibilities that might explain the observation.

Still, Peg3 mutation also retarded fetal growth in mutant embryos in utero, an observation entirely consistent with the conflict hypothesis for an imprinted gene that shows expression of the paternal allele (2). Peg3 is widely expressed in mesodermal, endodermal, and neural tissues. The expression is first seen in E 6.5 embryos (which, incidentally, is remarkable for being detected in the anterior visceral endoderm and the primitive streak), and persists into adulthood primarily in the brain (1). The gene may well have a variety of diverse functions in development, of which, so far, we have glimpsed only some. If imprinting confers an advantage on any of these functions, this condition may become fixed, regardless of whether it is beneficial for all of the functions. Further positive effects of imprinting on other functions of Peg3 would only improve its chances of spreading within the population.

Mice in the wild, unlike their laboratory counterparts, exhibit complex social interactions within their communities. It is unclear if imprinting of genes to regulate maternal behavior would be advantageous under these circumstances. We agree with the view of Haig (3) that this aspect requires additional tests before the evolutionary significance of imprinting for behavior can be judged properly. This example illustrates the need for further experiments to determine the variety of functions that imprinted genes may have. So far, the functions of relatively few genes are known. The debate on imprinting and functions of the Peg3 gene and its effect on maternal behavior is itself a positive outcome, whatever the final verdict.

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Réferences


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References

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