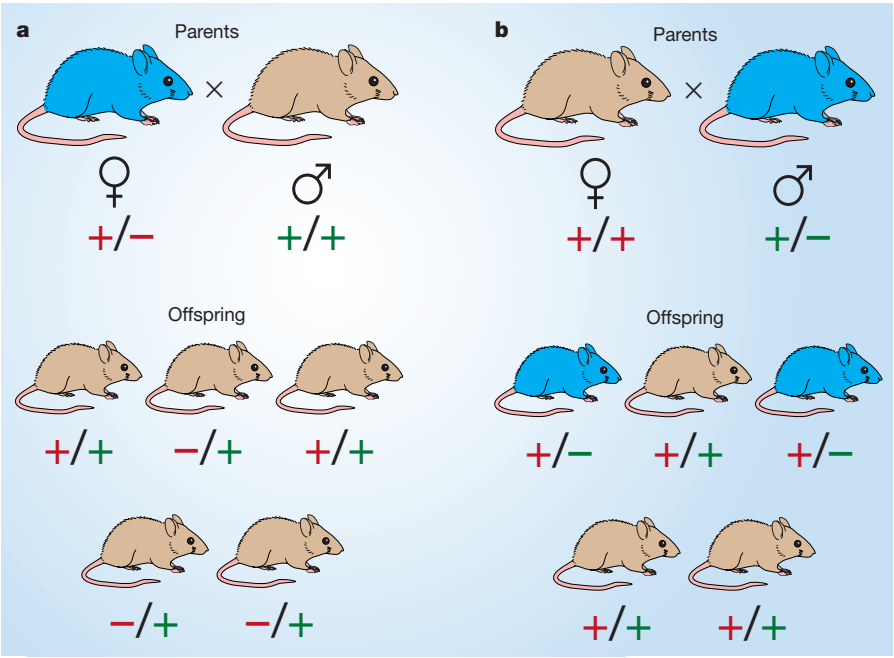


Figure 1 **Expressing *Peg3* or a reporter gene.** a, **In this cross, the mother expresses a reporter gene (β-galactosidase, which produces blue pigment) and not *Peg3* because she has inherited the mutated gene (−) from her father. She also has a silent copy of the wild-type *Peg3* gene (+) from her mother. When crossed to a fully wild-type male, her offspring will all express wild-type *Peg3* because they inherit the wild-type gene copy from their father, regardless of which copy they inherit from their mother. In this cross, a mutant mother is raising normal babies.** b, **A normal mother is mated to a *Peg3* mutant male that expresses the reporter gene he inherited from his own father. The babies from this cross will inherit a silent wild-type gene copy from their mother. But some will have their father's mutant copy and will thus express the reporter gene, while others will have their father's wild-type gene and will be normal. Here, a normal mother is raising a mixture of normal babies and babies that don't express *Peg3*. Other crosses were made that resulted in litters with all mutant offspring or offspring with two wild-type copies of the gene.**

versa (Fig. 1). To ensure that the gene is silenced, and to recognize which individuals are expressing *Peg3* and which are not, Curley *et al.* used mice with a 'targeted mutation', where the function of *Peg3* is disrupted by insertion of a 'reporter gene' that turns cells blue if the new gene is expressed instead of *Peg3*. The mutation was created in an otherwise genetically identical inbred strain of mice, so that the only difference was the presence or absence of *Peg3* expression. This is a powerful experimental approach, as mothers can have mixed litters of offspring that express or lack *Peg3* (Fig. 1).

Using these lines, the authors looked for the gene's effects on maternal behaviour by comparing mutant mothers (those that had the reporter gene instead of *Peg3*) and wild-type mothers (those with functional *Peg3*) rearing all wild-type offspring. They also compared wild-type and mutant offspring reared by wild-type mothers to examine how *Peg3* influenced offspring traits.

The results were striking on both counts. Although they had the same number of pups, mutant mothers ate less and gained less weight in their pregnancy, had less fat reserves after giving birth, and produced less milk. Mutant pups were less competent at suckling, and performed less well even after weaning. They also lost out in sibling competition, as pups born into families with a mixture of wild-type and mutant offspring were smaller than counterparts born into exclusively wild-type or mutant families. The point here is that asymmetry among siblings resulted in greater competition compared with families of all equals.

Finally, newborn mice depend on their mother to keep their body temperature constant and high. Mothers lacking *Peg3* were poor nest-builders, and failed to retrieve offspring that wandered from the nest; *Peg3*-deficient offspring were delayed in developing self-thermoregulation. In the end, absence of *Peg3* expression led to smaller pups that had delayed reproduction and were less likely to survive. *Peg3* therefore affects the behaviour of both mother and offspring.

Genes such as *Peg3* that influence the fitness of both parents and offspring require us to consider simultaneous evolution in two generations, rather than the more typical single generation at a time. However, such shared genetic influences on mothers and offspring are consistent with previous theoretical[4,5] and empirical work[6–9] showing that genetic coadaptation of parent and offspring behaviour is to be expected and can evolve. A positive coadaptation as shown by Curley *et al.*, where offspring that are able to get the most from their mothers grow up to be good providers, is also seen in parental care and offspring begging in a bird[6] and an insect[9]. So selection acts through both the mothers and the offspring[9]. However, in the case of *Peg3*, only the father's gene is selected, so mothers may be working harder than they might if both copies of the gene were expressed.

Demonstrating the effect of a gene using targeted mutagenesis (the focus of molecular behaviour genetics) is not the same as demonstrating genetic influences on natural variation in behaviour (the focus of evolutionary behaviour genetics). In addition, parenting and infant behaviour are likely to be influenced by many genes rather than by a single gene. Evidence for the effects of natural variation in *Peg3* on parent–offspring interactions in wild populations must await further study, but other genetic investigations of parental performance in different strains of mice also implicate *Peg3* as one of many influences on parenting and infant survival[10]. Thus, the effect is not solely due to a complete disruption of the gene: normal levels of variation at this genetic locus seem to be important, at least in differences among laboratory strains.

Is the behaviour of mother and offspring evolving because of conflict or as a coadaptation? The studies of *Peg3* in mice provide direct evidence that parent and offspring behaviour can have a common genetic basis, and support the idea that both conflict and coadaptation between parents and offspring can be accommodated. Or, as today's parents learned from the Rolling Stones, you may not always get what you want, but you might just find that you get what you need. ■

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**Correction**

In Julian Krolik's News and Views article, "Dust-filled doughnuts in space" (*Nature* **429,** 29–30; 2004), it was incorrectly stated that the observations described were the first to be made of an extragalactic source using infrared interferometry. In fact, the first such observations were published by M. Swain *et al.* (*Astrophys. J.* **596,** L163–L166; 2003), using the Keck interferometer to resolve the core of NGC 4151 at 2.2 micrometres.