for fission yeast tropomyosin [8]. When specific tropomyosin–actin filament populations emerge, they appear to favor recruitment of myosin-II [10–12]. *In vitro* studies have shown that tropomyosin promotes the activity of myosins (myosin-II and -V) that operate on formin-mediated filaments in fission yeast [12,13]. Interestingly, the acetylation of the single fission yeast tropomyosin isoform is incomplete and can provide further refinement of its function in the cell [14,15]. In contrast to its positive role with unbranched filaments, tropomyosin appears to be incompatible with many of the actin-binding proteins traditionally associated with the branched Arp2/3-mediated actin networks. Tropomyosin inhibits actin polymerization and branching by the Arp2/3 complex [16] and blocks severing by cofilin or gelsolin [17]. Furthermore, studies in fission yeast have shown that fimbrin displaces tropomyosin from branched filament networks at endocytic patches [18], preventing tropomyosin-mediated inhibition of cofilin and myosin-I at these actin structures [13,18]. There do appear to be exceptions to tropomyosin's apparent functional dichotomy, on the basis of recent studies in budding yeast [19,20], which may contribute to the functional diversification of tropomyosin isoforms in more complex cells.

In addition to advancing our understanding of stress fiber assembly and the role of tropomyosin, the work of Tojkander *et al.* [4] should motivate further investigations into the molecular mechanisms governing actin specification in non-muscle cells. Can mDia2 and other formins specify the recruitment of distinct tropomyosin isoforms? How do Tm4 and other isoforms differentially regulate myosin motors? Does α-actinin influence the accumulation of tropomyosin along filaments? Future investigations combining cell and *in vitro* studies should help us gain a better handle on these and other related questions.

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Genomic Imprinting: An Obsession with Depilatory Mice

Excessive grooming in mice has been promoted as a model of human obsessive-compulsive disorders. A recent paper adds *Grb10* to the list of genes with effects on behavioral hair loss, with the added twist that this time the gene is imprinted.

David Haig[1,*] and Francisco Úbeda[2]

The development of new psychopharmaceutical therapies is constrained by the lack of good animal models of human mental illness [1]. Excessive grooming and barbering behaviors in mice have been suggested as models of human compulsions, especially compulsive hair pulling or trichotillomania [2,3], but many questions remain about the interpretation of these behaviors in mice.

A recent paper in *Nature* [4] reports that paternal-specific expression of *Grb10* inhibits whisker removal in mice. *Grb10* is expressed exclusively from its maternal allele in most tissues of fetal mice, with the notable exception of the central nervous system, where it is the paternal (rather than the maternal) allele which is expressed. *Grb10* is also expressed from the paternal allele in adult brain [4]. This broad pattern of

expression is opposite to that of the well-known imprinted gene *Igf2*, which is expressed from its paternal allele in most fetal tissues but from its maternal allele in adult brain [5].

Mice with an inactivated maternal copy of *Grb10* had increased birth weight, while mice with an inactivated paternal copy had normal birth weight. An extensive behavioral analysis identified two peculiarities of mice with an inactivated paternal copy. First, these mice rarely backed-down in a test in which they and a control mouse entered a narrow tube from opposite ends. Second, mice with an inactivated paternal copy of *Grb10* 'barbered' the whiskers of cage mates at a higher frequency than did either wildtype or mice with the maternal copy inactivated [4]. This is not the first gene knockout to link backing-down and barbering behaviors. Mice with homozygous inactivation of *Dvl1* and *Plcb1* exhibit the opposite phenotypes to mice with paternal inactivation of *Grb10*, namely reduced barbering of cage mates and increased likelihood of backing-down in the tube test [6,7].

Grooming, Allogrooming and Barbering

In mammals, self-grooming, grooming of others (allogrooming), and barbering probably serve distinct adaptive functions. Self-grooming helps control ectoparasites such as lice. Laboratory mice are usually louse-free but wild mice commonly carry lice on their head and neck because this is where they cannot groom themselves [8]. Louse infestations develop over the body of solitary mice if self-grooming is prevented by placing a ruff around the mouse's neck or by preventing close apposition of the lower incisors that normally form an effective louse-comb [9].

Allogrooming helps control louse populations on the head, neck, and body of the groomed mouse. Mice with amputations of the hind feet are unable to groom themselves effectively. Amputees succumb to lethal louse infestations when kept alone but are able to keep each other largely louse-free when kept in stable groups, although not when group membership is regularly disrupted [10,11].

Barbering occurs during bouts of mutual grooming and involves plucking the whiskers of another mouse [12]. It appears to be an intrusion into the normal sequence of allogrooming and is commonly interpreted as an assertion of social dominance [2], although this interpretation is disputed [3]. In barbering strains, one mouse in each cage (the barber) has intact whiskers [13]. Barbered mice usually back-down when confronted by their barbers in the tube test [14].

Inactivation of *Hoxb8* causes hair loss due to excessive grooming and allogrooming but the mutant mice do not exhibit the loss of whiskers and facial hair associated with barbering [15]. Thus, grooming and barbering behaviors are genetically separable. Barbering occurs during allogrooming, however. Therefore, a mutation that affected the frequency of allogrooming might indirectly also affect the frequency of barbering. It would be useful to know whether the effects of inactivation of paternal *Grb10* are specific to barbering or also involve an increase in allogrooming.

What's Imprinting Got to Do with It?

Grb10 is the first imprinted gene to be shown to affect grooming behaviors, although an earlier mapping study reported differences in grooming behavior between reciprocal crosses that might be explained by effects of imprinted genes [16]. Grooming behaviors in this earlier study showed linkage to the *p* locus, adjacent to the imprinted gene cluster associated with Prader-Willi and Angelman syndromes in humans. Prader-Willi syndrome is associated with compulsive skin-picking [17].

The kinship theory of genomic imprinting posits that imprinting is maintained by effects on the fitness of other individuals who are more closely related to an actor via the actor's mother or father [18]. For example, members of a litter have the same mother but may have different fathers. Conversely, members of neighboring litters may have different mothers but the same father when dominant males maintain harems. Another source of asymmetries of matrilineal and patrilineal relatedness within social groups is sex-biased dispersal. Predictions of the direction of imprinted effects on social behavior depend on the interplay between mating system and dispersal [19].

The evolutionary interpretation of the effects of imprinted genes on grooming behaviors hinges on whether the other mice affected are closer relatives to the depilatory mouse's mother or father and whether the underlying behaviors enhance or reduce the individual fitness of the barber and barbee.

Self-grooming is a public good because it not only reduces an individual's own parasites but also reduces the transfer of parasites to other individuals with whom the groomer comes into contact. Allogrooming is similarly a cooperative behavior that allows parasites to be removed from hard-to-reach areas, especially around the head and neck. Therefore, greater investment in hygiene-related behaviors, such as self-grooming and allogrooming, should be favored by whichever parental genome has higher relatedness within groups. If the primary effect of paternal-specific expression of *Grb10* is to reduce the frequency of allogrooming, then allogrooming would be predicted to have enhanced the fitness of individuals who were closer relatives of the allogroomer on the maternal side than on the paternal side.

By contrast, the assertion of social dominance is probably associated with less equal sharing of resources. Therefore, a greater propensity to assert dominance should be favored by the parental genome with lower relatedness within groups whereas a greater propensity to accept subordinate status should be favored by the genome with higher relatedness within groups. If the primary effect of paternal-specific expression of *Grb10* is to reduce the assertion of dominance, as expressed by whisker removal and backing-down in the tube test, then dominance relations are predicted to have involved individuals who were closer relatives on the paternal side than on the maternal side.

Detailed studies of *Grb10* knockouts should clarify whether the effects of paternal inactivation are specific to barbering or also affect the frequency of allogrooming. But ignorance of patterns of relatedness in wild mice will prevent a robust test of the kinship theory's predictions. Knowledge of the effects of imprinted genes on mouse behavior is rapidly outstripping our understanding of the social context of these behaviors in wild mice. If laboratory mice are to serve as useful models for disorders of human social interaction, then we need to know more about the social life of mice in their

'environment of evolutionary adaptedness'.

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Visual Perception: Bizarre Contours Go Against the Odds

A new study shows that the brain sometimes invents visual contours even when they would be highly unlikely to occur in the real world. This presents a challenge to theories assuming that the brain prefers the most probable interpretation of the retinal image.

Roland W. Fleming

How does the brain work out what is in our surroundings from the information on the retina? It's a question that has baffled scientists and philosophers for over a thousand years [1]. The key problem is that the retinal image is fundamentally ambiguous. For any given pattern of light that reaches our eyes, there are many possible scenes that could have created the image. Somehow the brain has to overcome this ambiguity and identify the one true state of the world. But how? Most researchers agree it would generally be a good idea for the brain to select the most *probable* interpretation of the image. However, as reported recently in *Current Biology*, Anderson *et al.* [2] have found that this is not what the brain always does.

Anderson *et al.* [2] created a motion display that causes the brain to 'invent' surface boundaries where none exist in the image. That in itself is not new: so-called 'illusory contours' have been discussed extensively since the Gestalt psychologists [3–5]. But here's the catch. Usually, illusory contours are the brain's way of rationally explaining the sudden disappearance of some feature or object. In our natural environment, when something in the retinal image ends abruptly, shrinks or disappears, one of the most likely explanations is that it is being hidden from view by some other surface, a so-called 'occluder'. When this occluder happens to match the background (in other words, when it is camouflaged) then the occluder itself produces no visible contrasts in the image. And this is why the brain creates illusory contours: it knows that the most probable explanation of the disappearing features is that something (which cannot itself be seen) is hiding them.

In the displays created by Anderson *et al.* [2], however, we experience vivid illusory contours even though the occluding surface is already clearly visible. In the centre of the display is a clearly visible square occluder. Surrounding the square, four circles oscillate in and out, each one nudging behind the square for a period during the motion. When a circle moves behind the square, a portion of it disappears from the image because it is hidden by the square. However, despite the presence of a clearly visible occluder that can fully account for the disappearance of circles' edges, observers experience an additional illusory contour that bulges and flexes over the top of the square. This is surprising because there is no rational reason for the brain to invent an additional occluder. The explanation for the missing parts of the circles is already visible, so nothing ought to be invented.

The authors argue that this finding has important theoretical consequences. There is a long tradition — dating back at least to Helmholtz [6] — of theories that pose perception as a process of