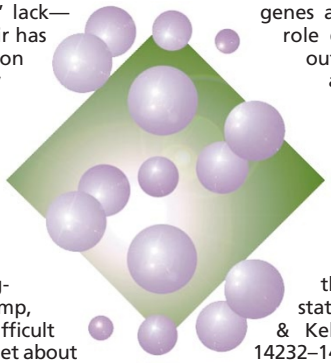
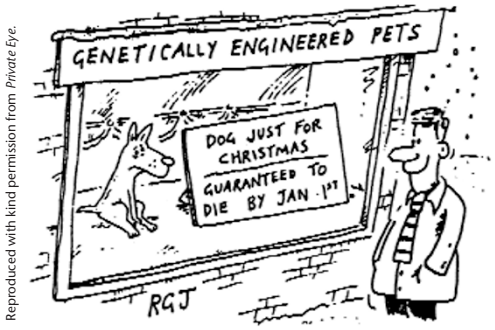
TOUCHING**base**

Twisty kats, twisted logic

An English proverb has it that "in a cat's eye, all things belong to cats." Vickie Ives Speir, of Marshall, Texas, appears to see things differently, for her breed of "twisty kats" lack—for the most part—functional front legs. Ms. Speir has interbred cats with radial hypoplasia, a condition that sometimes accompanies feline polydactyly and is characterized by the lack of the thicker long bone that normally runs between the wrist and elbow. One of her inspirations was to breed a cat "that would be less likely to run away and become wild", according to an article in *The Wall Street Journal*. She would appear to have achieved her aim, for "twisty kats" are unable to walk, run, sprint, jump and stretch like wild-type moggies. Speir asserts that they can walk, run and jump, although after a fashion, it would seem, for it is difficult to envisage how a cat with vestigial fore-arms can get about easily. "If they can clone a sheep", said Ms. Speir, "I can breed a cat that stands on its back feet." The French philosopher, Hippolyte Taine, had a point when he noted: "I have studied many philosophers and many cats. The wisdom of cats is infinitely superior."





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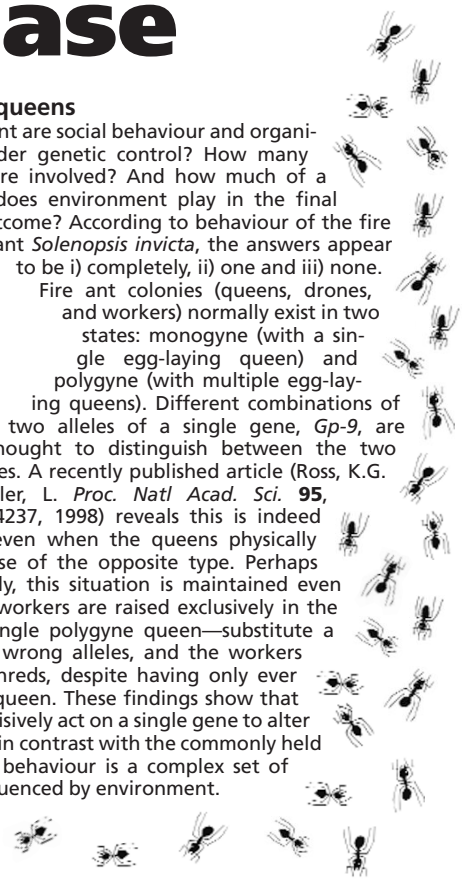
Genes for queens

To what extent are social behaviour and organization under genetic control? How many genes are involved? And how much of a role does environment play in the final outcome? According to behaviour of the fire ant *Solenopsis invicta*, the answers appear to be i) completely, ii) one and iii) none. Fire ant colonies (queens, drones, and workers) normally exist in two states: monogyne (with a single egg-laying queen) and polygyne (with multiple egg-laying queens). Different combinations of two alleles of a single gene, *Gp-9*, are thought to distinguish between the two states. A recently published article (Ross, K.G. & Keller, L. *Proc. Natl Acad. Sci.* **95**, 14232–14237, 1998) reveals this is indeed the case, even when the queens physically resemble those of the opposite type. Perhaps more surprisingly, this situation is maintained even when polygyne workers are raised exclusively in the presence of a single polygyne queen—substitute a queen with the wrong alleles, and the workers still rip her to shreds, despite having only ever known a single queen. These findings show that selection can decisively act on a single gene to alter social evolution, in contrast with the commonly held view that social behaviour is a complex set of traits heavily influenced by environment.



Mutation nomenclature standards

Standardization is never easy, but as genetic data accumulate in electronic databases around the internet, it is essential. After several meetings and extensive debate over the past two years, the "Nomenclature Working Group" has now published an article with recommendations for a nomenclature system for human gene mutations. It was published recently in *Human Mutation*, and is available on-line (http://journals.wiley.com/1059-7794/nomenclature.html.) *Nature Genetics* encourages all contributors to follow these recommendations.

"There's a study out that shows that not only are men genetically polygamous, unable to be faithful…they have no choice in the matter, but women are genetically wired so that they will fall for rich or prominent men. I used to think this was conniving on their part, but it's not…this is a purely genetic process…if they are caught in the wrong place at the wrong time, they can just say: "Well, look, it wasn't me." I'm just a lifeguard in the gene pool."

—Tom Wolfe, *Sixty Minutes*, 22 November 1998, Central Broadcasting Services

On the move with filamin 1

The cells that form the cortex originate in the deep proliferative ventricular zone of the brain and then migrate through a tangled jungle of cortical neurons before reaching the canopy of the developing brain, where they organize into the distinct layers of the cortex. A critical component of the migration machinery was recently revealed by Christopher Walsh and Jeremy Fox, of Harvard Institutes of Medicine, and colleagues who report that periventricular heterotopia (PH), a condition in which many neurons do not migrate, is caused by mutations in *FLN1*, encoding filamin 1 (*Neuron* **21**, 1315–1325, 1998). PH is an X-linked dominant disorder and heterozygous females suffer epilepsy as well as a spectrum of other symptoms, including vascular abnormalities. Hemizygous males die as embryos. The structure of filamin 1, which is reminiscent of dystrophin, suggests that it acts as a link between the cytoskeleton and membrane, binding with actin filaments and membrane receptors at its amino and carboxy termini, respectively. Human disorders such as double cortex syndrome and lissencephaly, characterized by disruption of the normal migratory trek of neurons, arise from deficiencies in genes that control the 'steering' of neurons. The neurons of PH patients, however, fail to move from 'first base', suggesting that filamin 1 acts as an 'engine' that initiates cell migration. The next step will be the identification of the 'keys' that start the engine; that is, the membrane receptor and associated ligand(s) that signal to filamin 1 to reorganize the actin filaments, a necessary prerequisite for cell locomotion. While this is the first time filamin 1 has been implicated in neuronal migration, it is known to have a role in the movement of several other cell types, including blood cells, melanocytes, leukocytes and tumour cells. Furthermore, the *FLN1* homologue from the slime mold *Dictyostelium discoideum* is required for cell motility and chemotaxis, and the *Drosophila* homologue is involved in the dorsal signalling pathway, suggesting that the molecular mechanics of cell movement are conserved throughout evolution.