may have evolved because it primarily maximizes personal fitness, not because of its effects on descendent and nondescendent kin. Behavioral ecology is replete with examples of elegant untested models. Over time, untested but theoretically compelling results are adopted into textbook truths. One subset of behavioral ecology that stands out in the proper integration of theory and empirical tests is the study of antipredator vigilance behavior (*8*).

Clutton-Brock and his collaborators, aided by a small army of research assistants, have now tested a number of the key assumptions of Bednekoff's sentinel model in the suricate mongoose populations of the Kalahari Gemsbok Park (South Africa). Also known as meerkats, suricates are a highly social, cooperatively breeding mongoose that, luckily for Clutton-Brock and company, were easily habituated to human observers. They live in groups of 3 to 30 animals that include both related and unrelated individuals. When foraging in the ground for invertebrates and small vertebrates, suricates are less able to detect predators. So, while other suricates forage, individuals take turns guarding the group from an elevated position (see the figure). When the sentinels detect predators, they emit alarm calls alerting the rest of the group to the presence of danger .

The investigators found that animals living in smaller groups (with fewer sentinels) had higher rates of predation than those living in larger groups. However, guard duty did not appear to be costly because during 2000 hours of observation, no sentinel suricate was ever observed being killed. In fact, the reverse was true: The sentinels were usually the first to detect a predator, and were conveniently located close to burrows down which they could readily escape. What then accounts for this apparently selfish guarding behavior?

If sentinel behavior in suricates is akin to alarm calling in ground squirrels and marmots, individuals should allocate their time to sentinel behavior as a function of the number of descendent kin (*9*) and nondescendent kin (*10*) that they live with. Alternatively, if sentinel behavior is similar to reciprocally altruistic mutual grooming in impala (*11*), then one would predict that individuals should take turns going on guard duty. However, if Bednekoff is correct, and sentinel behavior is a purely selfish activity, then animals should engage in sentinel behavior only when they have had enough to eat.

The Clutton-Brock study shows that neither kin selection nor reciprocity explains sentinel behavior in the suricates. Immigrants unrelated to all other group members were no more or less likely to guard than were individuals with many relatives around. Suricates did not seem to guard in successive bouts and the order of guarding was not constant, suggesting that there was no organized rota. But the nutritional state of suricates did have a large influence on sentinel behavior. Individuals who were given 25 g of supplementary food in the morning (boiled egg), spent 30% more time engaged in raised guarding than they did on the five previous days. And those fed 25 g/day for 30 days spent three times longer engaged in raised guarding than unfed individuals.

Ultimately, there is no reason to believe that any one mechanism should account for superficially complex social behavior in all species. It would be interesting to study the effects of food supplementation in dwarf mongooses (*Helogale undulata*)—species for which guarding has a documented predation risk and in which not all individuals guard (*3*)—to see whether sentinel behavior with an immediate direct cost and a possible kin-selected benefit is maintained by a mechanism other than personal benefit. However, as Bednekoff's model predicts, and as Clutton-Brock's suricate data illustrate, for at least one highly social species, animals selfishly guard others only once their bellies are full.

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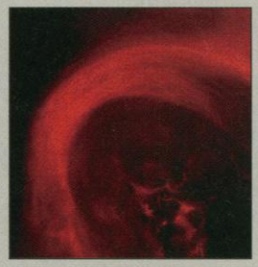
NOTA BENE: BIOMEDICINE

Pain-Killer Genes

Traumatic injuries are often followed by chronic nerve pain that remains long after the original injury has healed. Drugs to treat such neuropathic pain are often ineffective and can be associated with severe side effects. Wilson (*1*), Iadarola (*2*), and their colleagues now report two similar strategies for treating chronic pain, using viruses to deliver genes encoding pain-killer proteins to the central nervous system. Unlike analgesic drugs that are administered systemically, targeted delivery of a therapeutic pain-killer gene ensures that its protein product will be secreted in the vicinity of the nerves that conduct pain impulses.



To deliver the therapeutic gene to the spinal cord, the two groups developed different methods. Wilson and co-workers selected herpes simplex virus, which readily infects nerve cells, to transport the gene for human preproenkephalin (a precursor of Met-enkephalin, an opioid peptide with pain-killer activity) into mouse afferent nerves. They inoculated herpesvirus carrying the pain-killer gene into an abrasion in the mouse hindpaw. The virus traveled up the afferent nerves from the skin, taking up residence in the spinal cord. Here, proenkephalin was synthesized (red fluorescence in photo) and then processed into Met-enkephalin. Compared with mice inoculated with a marker gene, mice inoculated with the therapeutic gene took much longer to withdraw their hindpaw from a noxious stimulus, a measure of sensitivity to pain (hyperalgesia). Decreased pain sensitivity was observed for up to 6 weeks after inoculation of the therapeutic gene. That the pain-killing effect was at least partly due to Met-enkephalin was demonstrated by the restoration of hindpaw pain sensitivity after administration of naloxone, an opioid antagonist.

Iadarola's group took a different approach to deliver their therapeutic gene. They injected adenovirus—engineered to express the β-endorphin gene—directly into the cerebrospinal fluid (CSF) that bathes the spinal cord. β-endorphin (another pain-killer opioid peptide) was synthesized in the connective tissue cells of the pia mater (one of the membranes that protects the spinal cord). These cells secreted β-endorphin into the CSF. They found that injection of the therapeutic gene several days before testing resulted in a naloxone-reversible decrease in pain sensitivity.

Although these effects were transient, when applied repeatedly this gene delivery strategy may be applicable to chronic pain in humans.

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