References and Notes

1. M. H. Carr *et al.*, *Nature* **391**, 363 (1998).
2. K. K. Khurana *et al.*, *ibid.* **395**, 777 (1998).
3. R. T. Reynolds, S. W. Squyres, D. S. Colburn, C. P. McKay, *Icarus* **56**, 246 (1983).
4. S. Mood, *Astronomy* **11**, 16 (1983); R. T. Reynolds, C. P. McKay, J. F. Kasting, *Adv. Space Res.* **7** (5), 125 (1987); C. S. Powell, *Sci. Am.* **276**, 28 (January 1997); F. J. Dyson, *Atl. Mon.* **280**, 71 (November 1997).
5. R. T. Pappalardo *et al.*, *Nature* **391**, 365 (1998).
6. R. Greeley *et al.*, *Icarus* **135**, 4 (1998).
7. P. M. Cassen, R. T. Reynolds, S. J. Peale, *Geophys. Res. Lett.* **6**, 731 (1979); M. N. Ross and G. Schubert, *Nature* **325**, 133 (1987); G. W. Ojakangas and D. J. Stevenson, *Icarus* **81**, 220 (1989).
8. S. W. Squyres *et al.*, *Nature* **301**, 225 (1983).
9. S. Chang, in *The Chemistry of Life's Origins*, J. M. Greenberg *et al.*, Eds. (Kluwer Academic, Boston, 1993), pp. 259–299; R. Navarro-Gonzalez, M. J. Molina, L. T. Molina, *Geophys. Res. Lett.* **25**, 3123 (1998).
10. J. L. Kirschvink, in *The Proterozoic Biosphere: A Multidisciplinary Study*, J. W. Schopf and C. Klein, Eds. (Cambridge Univ. Press, Cambridge, 1992), pp. 51–52; P. F. Hoffmann, A. J. Kaufman, G. P. Halverson, D. P. Schrag, *Science* **281**, 1342 (1998).
11. H. Elderfield and A. Schultz, *Annu. Rev. Earth Planet. Sci.* **24**, 191 (1996).
12. V. Tunnicliffe, *Oceanogr. Mar. Biol.* **29**, 319 (1991); R. A. Lutz and M. J. Kennish, *Rev. Geophys.* **31**, 211 (1993).
13. T. M. McCollom and E. L. Shock, *Geochim. Cosmochim. Acta* **61**, 4375 (1997).
14. B. M. Jakosky and E. L. Schock, *J. Geophys. Res.* **103**, 19359 (1998).
15. G. A. Knauer, in *Interactions of C, N, P, and S. Biogeochemical Cycles and Global Change*, R. Wollast, F. T. MacKenzie, L. Chou, Eds. (Springer-Verlag, New York, 1993), pp. 211–231.
16. P. Jean-Baptiste, *Earth Planet. Sci. Lett.* **158**, 81 (1998).
17. K. Zahnle, L. Dones, H. F. Levison, *Icarus* **136**, 202 (1998).
18. R. W. Carlson *et al.*, *Science* **283**, 2062 (1999).
19. R. E. Johnson and T. I. Quickenden, *J. Geophys. Res.* **102**, 10985 (1997).
20. W.-H. Ip, *Icarus* **120**, 317 (1996).
21. D. T. Hall *et al.*, *Nature* **373**, 677 (1995).
22. Y. L. Yung and M. T. Leu, paper presented at the American Astronomical Society Division of Planetary Science Meeting 28, Tucson, AZ, 1996; M. L. Delitsky and A. L. Lane, *J. Geophys. Res.* **102**, 16385 (1997); R. E. Johnson and W. A. Jesser, *Astrophys. J.* **480**, L79 (1997).
23. N. A. Spaun *et al.*, *Geophys. Res. Lett.* **25**, 4277 (1998).
24. J. C. Priscu *et al.*, *Science* **280**, 2095 (1998).
25. J. Overmann, H. Cypiowka, N. Pfennig, *Limnol. Oceanogr.* **370**, 150 (1992).
26. G. S. Orton *et al.*, *Science* **274**, 389 (1996).
27. P. V. Hobbs, *Ice Physics* (Clarendon, Oxford, 1974).
28. G. D. Crawford and D. J. Stevenson, *Icarus* **73**, 66 (1988).
29. J. M. Moore *et al.*, *ibid.* **135**, 127 (1998).
30. R. Sullivan *et al.*, *Nature* **391**, 371 (1998).
31. J. A. Rathbun *et al.*, *Geophys. Res. Lett.* **25**, 4157 (1998).
32. E. L. Shock, in *Evolution of Hydrothermal Ecosystems on Earth (and Mars)* (Wiley, Chichester, England, 1996), vol. 202, pp. 40–60.
33. K. A. Maher and J. D. Stevenson, *Nature* **331**, 612 (1988).
34. N. H. Sleep, K. J. Zahnle, J. F. Kasting, H. J. Morowitz, *ibid.* **342**, 139 (1989).
35. E. L. Shock, *J. Geophys. Res.* **102**, 23687 (1997).
36. J. D. Anderson *et al.*, *Science* **281**, 2019 (1998).
37. F. Sohl and T. Spohn, *J. Geophys. Res.* **102**, 1613 (1997).
38. R. Greeley and B. D. Schneid, *Science* **254**, 996 (1991).
39. H. Y. McSween, *Meteoritics* **29**, 757 (1994).
40. C. Ballhaus *et al.*, *Nature* **348**, 437 (1990).
41. Extrusion or outgassing of sulfide and subsequent oxidation on the surface could be responsible for the observed sulfates [T. McCord *et al.*, *Science* **280**, 1242 (1998)].
42. W. Stumm and J. J. Morgan, *Aquatic Chemistry* (Wiley, New York, ed. 2, 1981).
43. J. C. Murrell, *FEMS Microbiol. Rev.* **88**, 233 (1992); C. G. Friedrich, *Adv. Microb. Physiol.* **39**, 235 (1998).
44. T. J. DiChristina, R. G. Arnold, M. E. Lidstrom, M. R. Hoffmann, *Water Sci. Technol.* **20**, 69 (1988); D. R. Lovley and E. F. Phillips, *Appl. Environ. Microbiol.* **51**, 683 (1988); C. R. Myers and K. H. Nealson, *J. Bacteriol.* **172**, 6232 (1990).

45. We thank Y. Yung and E. Shock for useful discussions.

PERSPECTIVES: BEHAVIOR

Selfish Sentinels

Daniel T. Blumstein

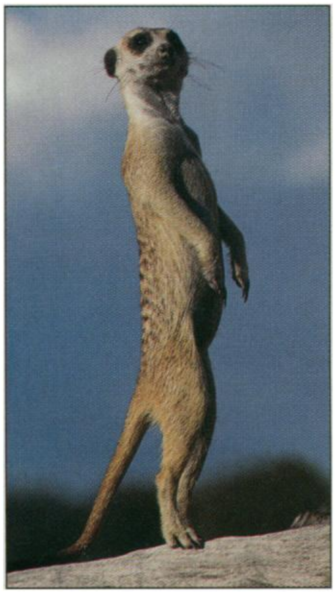
The selfless behavior of sentinels—the guards who take turns in keeping watch, putting themselves at risk for the benefit of others—has always been a human activity that people want to believe is found in other species too. In a variety of birds and mammals (*1*) that live in social groups, it is well documented that certain individuals act as guards while others forage for food and go about their daily routines. In some species, individuals trade-off sentinel duties in a coordinated fashion (*2*), perhaps to spread the danger evenly because guards are believed to be exposed to a greater risk of predation (*3*). What selection processes could explain the existence of such a potentially risky behavior in a large number of unrelated species? The popular view has been that sentinel behavior is influenced primarily by kin selection, that is, individuals tend to engage in behavior that benefits their relatives. But, on page 1640 of this issue, Clutton-Brock and colleagues present an elegant study in the African mongoose (*Suricata suricatta*) that dispels the myth of kinship and instead supports the opposing view that sentinel behavior is a selfish not selfless activity (*4*).

Enhanced online at www.sciencemag.org/cgi/content/full/284/5420/1633

The author is in the Departments of Biology and Psychology, Macquarie University, Sydney, NSW 2109, Australia. E-mail: dan@galliform.bhs.mq.edu.au

The insight that individuals may enhance fitness by engaging in activities, such as coordinated sentinel behavior, that benefit their relatives has revolutionized the study of animal behavior in the past few decades (*5*). Reciprocal altruism (*6*), whereby individuals take turns in allocating time to sentinel duties, is also likely in highly social species in which individuals can easily recognize each other and therefore keep track of those shirking guard duty. Unfortunately, assumptions and presumptions about selfless sentinel behavior have led some to believe that it is more common and potentially more complex than it really is. More importantly, the assumed mechanisms, although theoretically convenient, may in fact not be true.

Recently, Bednekoff has questioned a variety of assumptions often made about the selfless behavior of sentinels (*1*). He developed a convincing model to explain how apparently coordinated guarding could emerge from individually selfish antipredator behavior. He first noted that there was no concrete evidence that sentinels were actually more likely to be killed while on guard than their less vigilant, foraging group mates. Turning this assumption around, Bednekoff pointed out that sentinels may detect and avoid approaching predators more readily than foraging animals. Thus, rather than being exposed to an increased risk of predation, sentinels might actually be safer than the rest of the group. Such an explanation could account for cases where individuals compete for sentinel positions (*7*). He also noted that sentinel behavior could be influenced by a sentinel's nutritional state. Hungry animals would be less likely to engage in sentinel behavior than their better-fed comrades. Based on these two assumptions, he concluded that complex sentinel behavior could take place in the absence of any kin-selected benefits.



Attention! Sentinel behavior among suricates (a type of mongoose), rather than being a selfless act that helps to save a sentinel's relatives, confers a benefit on the guards themselves. They are usually the first to detect predators and are closer than their foraging colleagues to burrows, down which they can readily escape.

Bednekoff's theoretical findings are at the forefront of contemporary behavioral ecological studies that seek to properly identify the role and scope of kin selection and to provide alternative mechanisms to explain the evolution of behaviors. In this case, sentinel behavior 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.

References and Notes

1. P. A. Bednekoff, *Am. Nat.* **150**, 373 (1997).
2. K. J. McGowan and G. E. Woolfenden, *Anim. Behav.* **37**, 1000 (1989).
3. O. A. E. Rasa, *S. Afr. J. Sci.* **83**, 587 (1987).
4. T. H. Clutton-Brock *et al.*, *Science* **284**, 1640 (1999)
5. W. D. Hamilton, *J. Theor. Biol.* **7**, 1 (1964).
6. R. L. Trivers, *Q. Rev. Biol.* **46**, 35 (1971).
7. A. Zahavi, in *Cooperative Breeding in Birds*, P. B. Stacey and W. D. Koenig, Eds. (Cambridge Univ. Press, Cambridge, UK, 1990).
8. P. A. Bednekoff and S. L. Lima, *Trends Ecol. Evol.* **13**, 284 (1998).
9. D. T. Blumstein *et al.*, *Anim. Behav.* **53**, 173 (1997).
10. P. W. Sherman, *Science* **197**, 1246 (1977).
11. B. L. Hart and L. A. Hart, *Anim. Behav.* **44**, 1073 (1992).

12. Supported by the Australian Research Council.

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.

References

1. S. P. Wilson *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **96**, 3211 (1999).
2. A. A. Finegold, A. J. Mannes, M. J. Iadarola, *Hum. Gene Ther.*, in press.

—ORLA SMITH