But if these two processes are required for structural and functional plasticity, how are they balanced? That is, how are the distinct molecular cascades underlying exocytosis and actin cytoskeletal reorganization coordinated? Perhaps evolution has perfectly balanced their rates, or maybe there is a physical link between the two systems. For instance, receptors delivered to the synapse from the recycling endosomes could stabilize the actin cytoskeleton and thereby provide a simple accounting process to balance changes in synaptic strength and spine size. Maybe when we fully understand how spine size and synapse strength are coordinated will we be poised to comprehend why spine size matters.

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EVOLUTION

The Puzzle of Human Sociality

Human cooperation may have evolved as a consequence of genetic relatedness, culture, or language within groups.

Robert Boyd

The scale and complexity of human societies present an important evolutionary puzzle. In every human society, people cooperate with many unrelated individuals. Division of labor, trade, and large-scale conflict are common. The sick, hungry, and disabled are cared for, and social life is regulated by commonly held moral systems that are enforced, albeit imperfectly, by third-party sanctions. In contrast, in other primate species, cooperation is limited to relatives and small groups of reciprocators. There is little division of labor or trade, and no large-scale conflict. No one cares for the sick, or feeds the hungry or disabled. The strong take from the weak without fear of sanctions by third parties. On page 1569 of this issue, Bowles (*1*) provides one explanation for the commonness of costly, prosocial behavior in human societies.

The behavior of other primates is easy to understand. Natural selection only favors individually costly, prosocial behavior when the beneficiaries of the behavior are disproportionately likely to share the genes that are associated with the behavior. Selection can favor altruism toward close relatives because recent common descent provides a cue of genetic similarity. The small size of primate families limits the size and complexity of the groups that can be formed through this process. Thus, standard evolutionary theory provides a perfectly good explanation for the behavior of other primates, but not humans.

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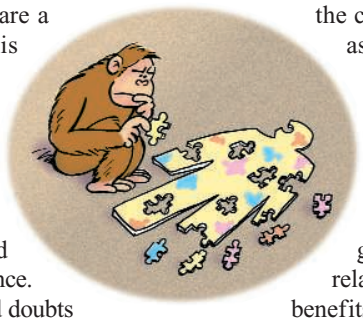
Bowles proposes that competition between genetically differentiated groups led to the evolution of our prosocial psychology. Limited migration between groups can lead to the buildup of genetic relatedness (which measures how much the possession of a particular gene in one individual predicts the presence of the same gene in a second individual) among group members. This means that group membership can also be a cue that allows assortative interaction—genes that cause you to help members of your group can be favored because other group members are disproportionately likely to carry the same genes, even though you do not share a recent common ancestor. This is an old idea. A version appears in *The Descent of Man* (*2*) and has reappeared many times since then. It has never gained much traction, however, because there have been good reasons to doubt its importance. First, theoretical work raised doubts about levels of genetic relatedness being high enough to favor prosocial behavior toward group members (*3*). Second, limited migration generates more competition within groups than between groups. This means that helping others in your own group reduces your own relative fitness and the fitness of your descendants. In some plausible models of the evolution of altruism when migration is limited, this effect exactly balances increases in relatedness, eliminating selection for altruism toward group members (*4*). Finally, the benefits of success in intergroup competition seems too small and the costs too large to allow cooperation to evolve. After all, other primates live in similar groups, but show little evidence of group-level cooperation.



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Bowles meets these objections with a combination of data and theory. First, he has assembled data on the amount of genetic differentiation among human hunter-gatherer groups (or put another way, the level of relatedness within such groups). These data show that the level of relatedness within such groups is substantially higher than previously supposed, a bit below that of cousins. This means that the cooperation will be favored as long as the benefits to individuals are about 10 times the cost. Second, because competition occurs between groups and successful groups are able to colonize the territories of extinct groups, competition among relatives does not attenuate the benefits derived from cooperation. Third, intergroup competition is common in small-scale societies, so the benefits derived from collective efforts to compete with other groups are plausibly substantial. Finally, Bowles notes that human foraging groups typically have culturally transmitted norms and practices, including food sharing and socially imposed monogamy, which reduce fitness differences within groups. He makes the original and interesting argument that such "leveling mechanisms" act like redistributive taxes to reduce the disadvantage of engaging in costly

prosocial behavior. The absence of these kinds of leveling mechanisms in primate groups may explain why human societies differ from those of other primates.

Make no mistake. This is not a "group selection" hypothesis that competes with "kin selection" hypotheses [see the Review by Nowak (*5*) on page 1560 of this issue for a discussion of conditions that favor the evolution of cooperative behavior]. Both concepts are equivalent frameworks for describing the same evolutionary process. The group (also known as multilevel) selection approach describes all natural selection as going on in a series of nested levels: among genes within an individual, among individuals within a group, and among groups. The kin selection approach accounts all fitness effects back to the individual gene. Bowles adopts the multilevel selection framework, but you can pose exactly the same argument in a kin selection framework and if you do your sums properly, you will get exactly the same answer. The real questions are: Are amounts of genetic variation observed among contemporary human foraging groups representative of the Pleistocene hominin populations in which distinctively human behavior probably evolved? Were the benefits of success (survival) from intergroup competition in ancestral human populations large enough to compensate for the individual costs of participating in such contests? And, did the kinds of leveling mechanisms observed among contemporary foragers exist and work in the same way in ancestral populations?

The role of leveling mechanisms is especially tricky. In other primate species, access to resources is usually regulated by social dominance. Dominant males monopolize mating and dominant females get better access to food, sleeping sites, and so on. There is little dominance among human foragers, and access to resources is more egalitarian. Thus, it seems likely that the variance in reproductive success in human foraging groups is lower than in other primates. However, at least some of the leveling mechanisms that we see in human groups seem to require a degree of prosociality not seen in other primates. Food sharing and dispute resolution, for example, could rest on exactly the same prosocial impulses that Bowles seeks to explain. It is certainly fair to invoke reproductive leveling to explain the stability of extended altruism among humans, but whether it is sufficient to explain its origin is not yet clear.

The main competing explanations for the distinctive level of human cooperation do not suffer from this potential liability. Some authors have argued that theory of mind, spoken language, and other cognitive innovations have allowed humans to build larger coalitions among nonkin than other primates (*6*). Others have proposed that rapid cultural adaptation generated cultural variation among groups, and intergroup competition subsequently favored the spread of culturally transmitted group-beneficial beliefs and practices (*7*). In both cases, the triggering factor (such as language or social learning) is supposed to have evolved for some other reason; cooperation and prosocial preferences arose as a side effect. Of course, there is no reason why these hypotheses need be mutually exclusive. Language or culture may have led to the evolution of leveling mechanisms, which then potentiated the spread of prosocial genes because these mechanisms reduced the costs of cooperation.

Research into evolutionary processes that spawned our uniquely cooperative societies may help us understand the nature of our social preferences. Bowles's hypothesis is consistent with suggestions that people have innate, prosocial motivations, and that these feelings are elicited by cues of common group membership. Other hypotheses seem to fit more easily with alternative views of human nature. These are old questions, but still important ones. The kind of quantitative empirical work that Bowles has done will help answer them.

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ATMOSPHERE

An Ancient Carbon Mystery

Mark Pagani, Ken Caldeira, David Archer, James C. Zachos

Sudden global warming 55 million years ago provides evidence for high climate sensitivity to atmospheric CO_2, but the source of the carbon remains enigmatic.

About 55 million years ago, Earth experienced a period of global warming that lasted ~170,000 years (*1*). This climate event—the Paleocene-Eocene Thermal Maximum (PETM)—may be the best ancient analog for future increases in atmospheric CO_2. But how well do we understand this event?

Temperature records from the tropics to the poles indicate that at the start of the PETM, global temperatures increased by at least 5°C in less than 10,000 years (*2*). The rise in surface temperature was associated with changes in the global hydrological cycle (*3*) and a large decrease in the $^{13}C/^{12}C$ ratio of marine (*4*) and terrestrial carbonates (*5*) and of organic carbon (*3*). This carbon isotopic excursion indicates that changes in the global carbon cycle were linked to global warming.

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Furthermore, the ocean's carbonate compensation depth—the depth above which carbonate accumulates on the sea floor— rose substantially at the start of the carbon isotope excursion (*5*). This change is consistent with ocean acidification associated with a rapid influx of CO_2. Although the change in ocean chemistry was not uniform throughout the ocean (*6*, *7*), the confluence of isotopic and sedimentological data supports the conclusion that atmospheric CO_2 was the primary greenhouse gas driving the PETM. Yet, the source of the CO_2 remains a mystery.

Biological responses to global warming during the PETM include changes in the ecology of marine organisms, a mass extinction of benthic foraminifera (*4*, *8*), and a global expansion of subtropical dinoflagellates at the earliest onset of the event (*9*). Global warming also coincides with the appearance of modern orders of mammals (including primates), a transient dwarfing of mammalian species, and a migration of large mammals from Asia to North America (*8*).

According to one hypothesis, the PETM was caused by the release of ~2000 PgC from the destabilization of methane hydrates (which would subsequently oxidize to form CO_2) (*10*). However, it is unlikely that meth-