PERSPECTIVES: ECOLOGY

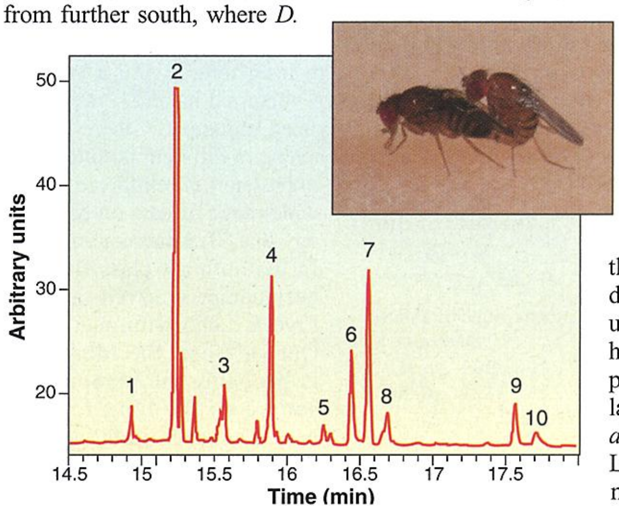
The Rapid Origin of Reproductive Isolation

Nick Barton

The part that natural selection plays in the origin of species has long been debated. It is easy to see that if two populations are kept separate—by mountains or ocean, for example—they will eventually become so different that they can no longer interbreed successfully. Their differences may have evolved by natural selection, but their reproductive isolation is merely a side effect of changes that emerged for other reasons. This view seems unsatisfactory to those who emphasize the positive aspect of selection in evolution. Both Alfred Russell Wallace (*1*) and Theodosius Dobzhansky (*2*) argued that natural selection would reinforce reproductive barriers between diverging populations. There has been little evidence, however, that selection has in fact contributed directly to the formation of new species (speciation) in this way. Reports by Higgie *et al.* (*3*) and Hendry *et al.* (*4*), on pages 519 and 516 of this issue, provide examples from fruit fly and sockeye salmon populations showing that selection can produce the kind of isolation that separates species in the wild (*3*), and moreover, that it can do so within a very short time (a dozen or so generations) (*3, 4*).

The best evidence that selection has reinforced mating barriers as an adaptation to reduce interbreeding has been indirect: Where two species encounter each other in nature, their preference for their own kind is typically stronger than for species whose ranges do not overlap (*5*). In their report, Higgie *et al.* (*3*) give the first direct evidence that such a pattern can be generated by selection, and that it can be generated very quickly. They worked with *Drosophila serrata* and *Drosophila birchii*, fruit flies that are almost indistinguishable in morphology and produce viable and fertile hybrid offspring in the laboratory. These sister species are found together in northeastern Australia, yet they rarely interbreed. Where their ranges do overlap, the two species differ in the mix of hydrocarbons on their cuticle (see the figure, this page). The strong correlation between mate choice and hydrocarbon profiles in hybrid offspring, and in flies perfumed with hydrocarbons from the other species, shows that mate choice is largely due to the scent of these chemicals (*6*). Most important, in southeastern Australia, beyond the range of *D. birchii* where only *D. serrata* is found, the hydrocarbons of *D. serrata* change abruptly, and there is a corresponding weakening of its mating preference (*3*).

Higgie *et al.* (*3*) set up experimental populations containing *D. birchii* together with *D. serrata* from either the north or the south of its range. After nine generations, Higgie *et al.* compared the cuticular hydrocarbons of *D. serrata* with those of control populations in which only one species was present. Little change was seen in *D. serrata* taken from the north, within the range of *D. birchii*; in contrast, *D. serrata* taken from further south, where *D. birchii* is absent in nature, tended to evolve hydrocarbons more similar to those of the northern *D. serrata*. (Females from three replicate populations evolved in this direction, as did males from two replicates. However, males from the remaining replicate evolved in the opposite direction.) The investigators did not test the consequences for mate preferences, but the strong correlation between hydrocarbons and mate choice in previous experiments suggests that selection has acted so as to reduce cross-mating between the species.



In flagrante delicto. Gas chromatographic profile of hydrocarbons in the cuticle of the fruit fly *Drosophila serrata*. Individual hydrocarbons that are important for mate recognition are labeled 1 to 10. **(Inset)** Photograph of a male and female fruit fly (*D. serrata*) mating.

The interpretation is simple: *D. serrata* in the north had long been exposed to the presence of its sister species, and so did not evolve in response to the presence of *D. birchii* in the laboratory. In contrast, *D. serrata* from the south evolved in the laboratory in the same way as northern populations presumably had in the past. Selection for a shift in mate choice is strong: When *D. birchii* is present, the proportion of *D. serrata* males from the south that successfully inseminate females of their own species is reduced by nearly 50%, whereas there is no significant interference with insemination by males from the north. Thus, the speed of the response to selection is not surprising. This work is important mainly because it opens up the possibility of a detailed ecological and genetic analysis of how mating systems evolve in nature.

Species can evolve differences in mate choice by direct selection on that choice (as in the Higgie experiment) or as a side effect of other changes. As with so much else in the study of speciation, these possibilities are difficult to distinguish because we usually see only an accumulation of changes, most of which may have evolved long after speciation itself.

In their study, Hendry *et al.* (*4*) describe a remarkable example of reproductive isolation that has evolved in 13 generations or less. Like other freshwater fish, salmon often evolve distinct "ecotypes" that are adapted to spawn in different habitats (see the figure, next page). Such ecotypes have repeatedly evolved in postglacial lakes within the last ~10,000 years. Hendry *et al.* describe an example from Lake Washington in the northwestern United States, where, in 1937, a large population of sockeye salmon was established in a river feeding into the lake. Salmon were found spawning at a lake beach in 1957, and they now have a morphology distinct from the fish that breed in the river: River females are larger, allowing them to dig deeper nests, and river males have shallower bodies, allowing them to swim more efficiently. Hendry *et al.* show that nearly 40% of fish breeding at the beach were born in the river, and yet they differ genetically, at six microsatellite loci, from the native beach fish. These differences are small (of the same order as those typically found between neigh-

CREDIT: HYDROCARBON PROFILE COURTESY OF M. HIGGIE; PHOTOGRAPH COURTESY OF A. O'TOOLE/UNIVERSITY OF QUEENSLAND

The author is at the Institute of Cell, Animal and Population Biology, University of Edinburgh, Edinburgh, UK. E-mail: n.barton@ed.ac.uk

boring populations within a species) but could hardly be maintained if the beach and river fish interbreed at random, and their offspring are fully viable and fertile. Thus, within half a century, both adaptive differences in morphology and some degree of reproductive isolation have evolved.

It is well established that natural populations can respond rapidly to selection (*7*). It is also well known that in the laboratory, selection for reproductive isolation can produce a rapid response, provided that it is not opposed by genetic exchange between the diverging populations (*8*). However, attempts to create species in the laboratory, by selection on a single interbreeding population, have usually failed (*8*). Neither of the examples presented by the Higgie and Hendry groups provide evidence that reproductive isolation evolved by the splitting of a single population; indeed, such evidence is almost impossible to obtain. But, because reproductive isolation can evolve so quickly, to levels that allow further divergence even after contact, this difficulty is perhaps not important. Populations must often become temporarily isolated for a few tens of generations, and this may suffice to allow divergence within an essentially continuous geographic range.

Socking it to the competition. Male red sockeye salmon (*Oncorhynchus nerka*) competing to spawn with a female.

The two reports provide strong evidence for the rapid evolution of reproductive isolation. This raises the question: Why do we not see more species? It may well be that new species (that is, reproductively isolated populations) do form often, but that only rarely do they evolve sufficiently to be recognized as separate species by biologists or such that they find a distinct ecological niche. For ecologists, the question is then whether the number of established species that we see is determined by a balance between the rate of speciation and the rate of extinction (*9*), or instead is set by the range of distinct niches that are available. The realization that evolution occurs on time scales accessible to experiment and observation may help bring together evolutionary and ecological approaches to address such questions.

References

1. A. R. Wallace, *Darwinism* (Macmillan, London, 1889).
2. T. Dobzhansky, *Am. Nat.* **74**, 312 (1940).
3. M. Higgie, S. F. Chenoweth, M. W. Blows, *Science* **290**, 519 (2000).
4. A. P. Hendry, J. K. Wenburg, P. Bentzen, E. C. Volk, T. P. Quinn, *Science* **290**, 516 (2000).
5. J. A. Coyne, H. A. Orr, *Evolution* **51**, 295 (1997); M. A. Noor, *Am. Nat.* **149**, 1156 (1997).
6. M. W. Blows, R. A. Allan, *Am. Nat.* **152**, 826 (1998).
7. A. P. Hendry, M. T. Kinnison, *Evolution* **53**, 1637 (1999).
8. W. R. Rice, E. E. Hostert, *Evolution* **47**, 1637 (1993).
9. S. Hubbell, *The Unified Neutral Theory of Biodiversity and Biogeography* (Princeton Univ. Press, Princeton, NJ, in press).

CREDIT: VICTORIA MCCORMICK/ANIMALS, ANIMALS

PERSPECTIVES: AIDS

Preventing AIDS But Not HIV-1 Infection with a DNA Vaccine

Xuefei Shen and Robert F. Siliciano

Individuals infected with human immunodeficiency virus–1 (HIV-1), the cause of AIDS, develop strong immune responses against the virus but never completely eradicate the infection. The extraordinary mutation rate of HIV-1, its ability to evade immune responses by establishing a latent (silent) infection, and the progressive destruction of the CD4$^+$ T helper cells that it infects all contribute to the inability of the immune system to completely destroy this virus. These characteristics also complicate the development of vaccines to prevent HIV-1 infection. Indeed, it is currently unclear whether it will be possible to develop a vaccine that can actually prevent infection. Nevertheless, an important study by Barouch *et al.* (*1*) on page 486 of this issue suggests that vaccine-induced immune responses may control the virus effectively enough to prevent clinical disease even if they fail to prevent or eradicate infection.

Enhanced online at www.sciencemag.org/cgi/content/full/290/5491/463

Most vaccines against viruses prevent infection by inducing antibodies that stop the virus from infecting host cells. It is difficult to induce antibodies to HIV-1 that serve this protective role. This is a reflection of the enormous variation in the HIV-1 envelope (env) protein and the failure of most antibodies that recognize this protein to neutralize the virus. For these reasons, investigators developing vaccines to protect against AIDS have concentrated on boosting the response of CD8$^+$ cytolytic T lymphocytes (CTLs). These lymphocytes provide resistance to infection with HIV-1 by inducing lysis of virally infected cells. Numerous studies implicate virus-specific CD8$^+$ CTLs as crucial players in the control of HIV-1 replication (*2–4*). Confirmation of the importance of CTLs has come from a well-established animal model of AIDS: rhesus monkeys infected with simian immunodeficiency virus (SIV). Last year, Schmitz *et al.* (*5*) and Jin *et al.* (*6*) demonstrated that virus replication is not controlled in monkeys depleted of CD8$^+$ lymphocytes during SIV infection (see the figure).

Barouch and colleagues use a similar animal model to determine whether vaccine-induced immune responses, especially CTL responses, could prevent infection or ameliorate the course of disease. In this case, the virus used to infect rhesus monkeys was a chimeric simian/human immunodeficiency virus (SHIV) consisting of the SIV genome containing the HIV-1 *env* gene instead of the SIV *env* gene. Rhesus monkeys were immunized with a vaccine that contained DNA encoding the SIV gag and HIV-1 env proteins as well as human interleukin-2 (IL-2), a cytokine that enhances the immune response. Although immunized monkeys developed CTL responses that could be readily measured, they were not protected against intravenous challenge with a large dose of a highly pathogenic form of SHIV. However, the pattern of infection and the course of disease were altered markedly compared with control monkeys receiving a sham DNA vaccine. Sham-immunized animals generated only weak CTL responses after infection and showed high levels of viral replication and a rapid loss of CD4$^+$ T cells. There was clear disease progression in the control monkeys, and half of them died within 140 days of being exposed to SHIV. In contrast, monkeys immunized with optimal doses of the vaccine had a slightly blunted initial increase in virus levels in the blood (viremia) and then

---

The authors are in the Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA. E-mail: rsilicia@welch.jhu.edu