

Evolution

An asymmetrical view of fitness

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EVOLUTION is often described as the movement of populations through an adaptive landscape¹: the mathematical nature of much of population genetics theory means that this is only too often a landscape with figures. The allegorical terrain is usually hilly, with most evolving populations at or near a higher or lower summit of adaptation and able to reach a higher peak only by passing through a valley of relative unfitness. Some of these are deep and almost impassable, separating differently adapted species. On each of the major eminences are smaller summits, and as evolution is by its nature opportunistic and responsive only to an immediate challenge, populations may become marooned on a low hillock when higher peaks are available all around them. How populations pay the evolutionary price of traversing from one adaptive peak to the next is at the centre of much of the theory of population genetics. On page 345 of this issue, Clarke and McKenzie² report the use of a novel measure of the disruption of development to assess the response of a population to the incorporation of an evolutionary novelty.

Much of the controversy about the nature of evolutionary change arises from the difficulty of measuring the cost of passing from one state of adaptation to another. Quite frequently, populations must respond to an environmental challenge by taking advantage of a new mutation with a major influence on fitness. The complexity of the process of development means that such mutations are likely to influence many aspects of the phenotype. Their pleiotropic effects may therefore mean that the population must undergo a period of reduced fitness during the process of adaptation, and in some cases that developmental constraints will limit its ability to evolve³. R. A. Fisher⁴ pointed out nearly 70 years ago that there will then be strong selection for modifier genes that minimize the harmful effects of a new mutation on the developing phenotype while maximizing those that allow adaptation to the new environment. In laboratory mice, for example, it is possible to

reduce the developmental disruption caused by a new morphological mutant by breeding from individuals who show relatively little abnormality. However, the mutant can be restored with its full virulence by crossing it into another genetic background so that amelioration of its effects has arisen from a restructuring of the genomes rather than as a result of change in the allele itself⁵.

The ascent of a new adaptive peak can also be seen in mutants of cultivated plants such as sweet peas and cotton in which older varieties have less disadvantageous side-effects than do those that have only recently arisen⁶. Until now, however, this process has not been observed in natural populations.

Because both sides of a bilaterally symmetrical animal are a product of the same genome, random deviations between left and right indicate how accurately that genome is able to control the process of development. Fluctuating asymmetry — which is to be distinguished from directional asymmetry such as the universal difference in position of left and right human testes familiar to most readers of *Nature* — can hence give an insight into the cost of evolving towards a new adaptive peak. It is this asymmetry that is investigated by Clarke and McKenzie in this issue².

The Australian sheep blowfly *Lucilia cuprina* is a major pest, and several attempts have been made to control it with insecticides. Resistance to dieldrin evolved within 2 years of its introduction in 1955, and this insecticide was then abandoned and replaced with diazinon. Diazinon resistance appeared by 1967, but this chemical is still in use. Flies resistant to dieldrin show significantly more fluctuating asymmetry in bristle number than do wild-type flies; but individuals who carry the gene conferring diazinon resistance are no more asymmetrical than are flies who have never been exposed to insecticides. Twenty years of evolution have allowed *Lucilia* populations to take advantage of a major mutation by adjusting to its disruptive effects on development. That this does indeed reflect a general adaptive shift rather than a change in the major gene itself is confirmed by the experiments of Clarke and McKenzie, who backcrossed the diazinon resistance gene into a laboratory stock of susceptible flies: the level of fluctuating asymmetry associated with diazinon resistance then exceeds even that of flies resistant to dieldrin.

Asymmetry hence shows the degree to which the population has escaped from the valley of relative unfitness which separates it from a new adaptive peak. Its success in making the transition is manifest in the ability of the diazinon resistance gene to persist in insecticide-free population cages containing susceptible flies from modern wild populations, an ability which disappears when diazinon resistance is

crossed into a genetic background which has never before experienced the mutant. The fitness modifiers responsible for the adaptive shift are not linked to the gene encoding diazinon resistance.

Fluctuating asymmetry has the potential to assess the state of adaptation of a population even when the nature of the selection involved is unknown. In some animals, there is a negative association between fluctuating asymmetry and heterozygosity as measured by gel electrophoresis of proteins⁹. This might reflect selection acting at the protein loci themselves, but is more likely to indicate that individuals with a high level of heterozygosity in the genome as a whole (a small part of which is sampled by electrophoresis) are more developmentally stable and hence better adapted⁹. Inbred, highly homozygous and relatively unfit mice have more fluctuating asymmetry than do those arising from crosses between strains¹⁰. For any highly heritable continuous character controlled by several genes, individuals at the extremes of the distribution of a population are more likely to be homozygous than are those near the mean¹¹, and there is evidence that in some cases at least such individuals have higher levels of fluctuating asymmetry¹². As selection nearly always acts against extreme values of continuous characters, this developmental disruption again indicates the position of an individual on its local peak of adaptation.

Species represent very different sets of adaptations, and it is usually possible to cross the valleys of unfitness which separate them only in the laboratory. In interspecific hybrids of trout there is, as might be expected, considerably more fluctuating asymmetry than in the parental stocks¹³. Habitat disturbance has led to hybridization between two species of sunfish *Enneacanthus* in New Jersey and Con-

necticut and once again the hybrids are more asymmetrical than are the parental species¹⁴.

Fluctuating asymmetry hence at least has the potential to provide insight into the state of adaptation of a population and, in principle, could be used to test the extent to which an evolutionary response to a changing environment has been successfully completed. By comparing its value for different characters, fluctuating asymmetry might suggest the strength of selection and the relative importance of traits even when their function is unknown. Comparisons of this kind in fossils and their descendants might even indicate whether evolution has been accompanied by a general increase in developmental stability¹⁵. Work on fluctuating asymmetry has the additional merit of providing information on the genetics of populations quickly and cheaply; and in the present bleak landscape for research in evolutionary biology this may turn out to be its greatest advantage. □

1. Wright, S. *Evolution and the Genetics of Populations. IV. Variability Within and Among Populations*. (Univ. Chicago Press, 1984).
2. Clarke, G.M. & McKenzie, J.A. *Nature* 325, 345-346 (1987).
3. Maynard Smith, J. *et al. Q. Rev. Biol.* 60, 265-287 (1985).
4. Fisher, R.A. *Am. Nat.* 62, 115-126 (1928).
5. Fisher, R.A. & Holt, S.B. *Ann. Eugen.* 12, 102-120 (1944).
6. Fisher, R.A. *Am. Nat.* 62, 571-574 (1928).
7. McKenzie, J.A. & Purvis, A. *Heredity* 53, 625-634 (1984).
8. Mitton, J.B. & Grant, M.C. *A. Rev. Ecol. Syst.* 15, 479-499 (1984).
9. Chakraborty, R. & Ryman, N. *Genetics* 103, 149-152 (1983).
10. Leamy, L.C. *Am. Nat.* 123, 579-593 (1984).
11. Sould, M. *Am. Nat.* 120, 751-764 (1982).
12. Sould, M. & Couzin-Roudy, J. *Am. Nat.* 120, 765-785 (1982).
13. Leary, R.F., Allendorf, F.W. & Knudsen, K.L. *Evolution* 39, 1318-1326 (1985).
14. Graham, J.F. & Felley, J.D. *Evolution* 39, 104-114 (1985).
15. Palmer, A.R. & Strobeck, C. *A. Rev. Ecol. Syst.* 17, 391-421 (1986).

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