

**Genetic Drift**

Genetic drift is a microevolutionary force, a consequence of the finite size and structured nature of natural populations. Genetic drift was emphasized by the American school of population genetics founded by Sewall Wright (1889–1988); their counterparts in England drew inspiration from Ronald A. Fisher (1890–1962), emphasizing large unstructured populations whose microevolution would be governed by natural selection.

Genetic drift has three roles in contemporary evolutionary theory: First, its original formulation, as the imperfect ability of finite-sized populations to replicate themselves genetically, either through time or when subdivided. Deviations from the expected (parental or grand average) allele frequencies will vary in magnitude in inverse proportion to the size of the population: The smaller the population, the larger the deviation from expected values. Wright (1932:358–359) invoked this mathematical property as a “trial and error

mechanism" enabling a population to cross a "valley" and "continually find its way from lower to higher peaks" in his metaphor of the adaptive landscape. In 1942, Julian S. Huxley (1887–1975) promoted this as a major evolutionary force, calling it the "the Sewall Wright Effect" (Huxley 1942).

The second role for genetic drift is in founder-effect speciation, proposed by Ernst Mayr (1904–) (Mayr 1954). Here, a founding population, because of its small size, does not provide an adequate genetic representation of the ancestral population from which it was derived. Some alleles present in the parental population may be absent in the daughter population, and the frequencies of alleles in the daughter population may be quite different from those in the parental population. Consequently, the process of speciation may involve genetic drift to a very large extent, producing a temporal pattern of discontinuity in the history of the gene pool, which Stephen J. Gould (1980) has described as "the Wright Break." However, speciation may not be the ultimate result of a founder effect. Its effects in the gene pools of local populations may include elevated frequencies of ordinarily rare genetic pathologies, such as Ellis-van Creveld Syndrome among the Pennsylvania Amish (McKusick et al. 1964) or porphyria among White South Africans (Dean 1971).

The third role of genetic drift is in governing the spread of alleles not affected by natural selection, alleles that afford no net difference in fitness to the organism possessing them when compared with their alternatives. In the absence of selective forces, their spread will be governed solely by random forces. This role was initially explored by the Japanese geneticist Motoo Kimura (1968), and, due to the widespread recognition of "junk" in the genome, it is now paradigmatic within the study of molecular evolution.

While most molecular evolution is governed by genetic drift because the genetic variation is not phenotypically expressed, the most interesting aspects of human evolution involve features of the phenotype. Here, too, however, genetic drift was probably operating in small structured populations of foraging hominids. It has been documented in contemporary rural populations by the British human biologist Derek Roberts (1968) on the island of Tristan da Cunha and by the Italian human geneticist Luca Cavalli-Sforza (1969) in the Parma Valley, Italy.

It may be noted that although Wright

did not introduce the term "drift" to the study of biological microevolution until the 1930s, the word had been used in a somewhat analogous sense by historical linguists for at least a decade (cf. Sapir 1921).

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*See also* Evolutionary Theory; Molecular Anthropology

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#### **Genetics (Mendelian)**

Genetics is the study of the transmission of hereditary units across generations. Though named for the Bohemian cleric Gregor Mendel (1822–1884), whose work was published in 1866, Mendelian genetics actually dates only from the turn of the century, when Mendel's work was "rediscovered" by Carl E. Correns (1864–1933), and secondarily by Hugo de Vries (1843–1935) and Erich Tschermak von Seyfeneck (1871–1962), neither of whom appreciated its significance immediately (Bowler 1989). Mendel's discoveries were "premature" because the study of heredity was not yet conceptually divorced from the study of development. These fields were made distinct toward the end of the nineteenth century by biologists such as August Weismann (1834–1914), which then permitted Mendel's work strictly on heredity to take on significance. Two outspoken proponents of Mendelism soon emerged: William Bateson

(1861–1926) in England, and Thomas Hunt Morgan (1866–1943) in the United States.

Mendel's own work on peas involved the demonstration that hereditary units retained their integrity every generation (i.e., particulate inheritance) rather than being transformed every generation, as implied by blending inheritance. The two "laws" that now bear his name were codified as such by the early Mendelians as rallying points for the new science of genetics (itself named by Bateson in 1906). Indeed, Mendel's work was generally formulated as a *single law*, until divided into the familiar pair of laws by Morgan in his *A Critique of the Theory of Evolution* (1916).

Mendel's Law of Segregation governs the transmission of a single gene, formalizing the inference that the life cycle involves the formation of haploid gametes and their reconstitution into diploid zygotes every generation. Mendel's Law of Independent Assortment governs the transmission of two genes, formalizing the inference that they segregate into gametes and combine into zygotes in a statistically random fashion with respect to each other. This is recognized to be strictly applicable only to genes on different chromosomes.

Mendelian genetics developed a now widely familiar jargon: The genetic information encoding a particular characteristic is called a *gene* (shortened by Wilhelm Johannsen [1857–1927] [1909], from Hugo de Vries' [1848–1935] [1889] "*pangen[e]*," itself taken from Charles Darwin's [1809–1882] [1868] "*pangenesi*s"). The variant forms of a gene are called *alleles* (shortened from "*allelomorph*"); an individual bearing two different alleles is *heterozygous*; an individual bearing identical alleles is *homozygous*; an allele expressed in a heterozygote is *dominant*; an allele whose effect is suppressed in a heterozygote is *recessive*; an individual's genetic constitution is its *genotype*; the expression of the genetic information, forming the outward characteristics or features of the organism, is its *phenotype*.

The major difficulties faced by Mendelism lay not so much in the area of heredity as in the area of evolution. Where Mendelism implied transition across discontinuous character states (wrinkled versus round, green versus yellow), evolution seemed to be far more continuous in its patterns. The biometric school, led by Karl Pearson (1857–1936) and Walter F.R. Weldon (1860–1906), studied evolution without reference to underlying genetic mechanism, and the Mendelians

had a genetic mechanism that did not seem to produce the continuous variation encountered in nature. Some Mendelians, such as de Vries, proposed saltational models of evolution, wherein species jumped across character states in a single generation.

Alternatively, as first proposed by G. Udny Yule (1871–1951) and subsequently developed by Ronald A. Fisher (1890–1962), the continuous pattern of natural variation could be explained under Mendelian inheritance if several genes, each with small additive effects, were assumed to determine the characteristics in question. Fisher's work was more influential in England than in the United States, where the "multiple-factor hypothesis" had been accepted for some time.

In the "fly room" at Columbia University, New York, Thomas Hunt Morgan assembled a group that included Calvin B. Bridges (1889–1938), A.H. Sturtevant (1891–1970), H.J. Muller (1890–1967), and Theodosius Dobzhansky (1900–1975). They utilized the fruit fly (*Drosophila melanogaster*) to reconcile Mendelian heredity to the fact that observable cellular structures called chromosomes were known to be intimately involved in transporting genetic information across generations. It was through this research that each gene was recognized as occupying a specific locus on a chromosome, that many loci were linked on the same chromosomes, and that crossing-over occurred between homologous chromosomes, breaking up combinations of alleles on individual chromosomes, and bringing new combinations of alleles into existence. So persuasive and all-encompassing was this paradigm that the idea of "jumping genes" demonstrated by Barbara McClintock (1902–1992) in corn in 1950—genetic elements that move from locus to locus—was not accepted for several decades.

In pre-Mendelian times, it was well known that some traits "run in families," though the specific rules governing them had not been elucidated. Pierre Louis Moreau de Maupertuis (1698–1759), for example, tracked the inheritance of polydactyly through a kindred in his *Physical Venus* (1746). With the discovery of the Mendelian laws, the transmission of several diseases was soon found to be consistent with them. After Archibald E. Garrod (1857–1936) tracked the inheritance of a metabolic disorder known as alkaptonuria, Bateson, in 1902, suggested it to be a simple Mendelian recessive. The following year (1903), at Harvard University,

William Curtis Farabee (1865–1925), properly attributed brachydactyly to a dominant allele, and albinism to a recessive allele.

With the recognition that there was a fundamental law governing heredity of unit traits, human geneticists began to see unit traits everywhere. Paramount among them was Charles Benedict Davenport (1866–1944), a leader of the eugenics movement, who argued that immorality, promiscuity, criminality, and other nonnormative behaviors were caused by a simple Mendelian recessive allele for "feeble-mindedness." Likewise, the Phoenicians' "love of the sea" became attributable to an allele for "thalassophilia."

In some cases, of course, geneticists were correct in attributing diagnosably medical pathologies to genetic causes. Another major insight came shortly after the turn of the century, with the recognition that blood groups were inherited in a simple Mendelian fashion. The presumption of genes "for" specific characters, however, has often been limited only by the imagination of the geneticist, resulting in rampant reification of genes. In 1989, for example, the president of the American Society of Human Genetics proposed a gene governing divorce, through reasoning very similar to Davenport's nearly a century earlier (Comings 1989).

The exploitation of Mendelian genetics throughout the twentieth century, principally from the study of fruit flies and diseases, has resulted in something of a paradox in human genetics. There are three broad classes of Mendelian genes known in humans: those resulting in diseases when mutated; those governing biochemical minutiae, such as blood-group antigens and various forms of enzymes; and those governing phenotypic minutiae (such as earlobe shape, eye color, and tongue rolling). Of the latter class, most are oversimplifications, and some are simply false (e.g., tongue rolling; Fogle 1987).

Virtually nothing, however, is known about the genetic basis for the normal range of human variation: height, body build, facial conformation, forms of intelligence, and the like. Most of the human genome, on the contrary, appears to be occupied by diseases, to judge from the contemporary genetic maps. The explanation for this involves what can be called the "pathology paradigm." The rise of Mendelian genetics, and its spectacular successes in fruit flies and medical genetics, has built a research program in which a pathology is isolated and traced back to a locus with a dysfunctional allele (white eyes in *Drosophila*

or cystic fibrosis in humans). The function of the "normal" allele is inferred from the gross-est phenotype of the pathology. Thus, one can construct a map of the genetic diseases in humans with relatively little information on either what the actual activity of the gene is or nonpathological variation in the species. Consequently, although there is widespread agreement that most of the normal range of phenotypes in humans is caused by genes with complex interactions (epistasis), each with compound physiological effects (pleiotropy), genes are still viewed conceptually as independent entities with a single discrete function. Ernst Mayr (1959) criticized the application of this model in microevolutionary studies as "beanbag genetics"—which has left unexplored many of the interesting questions to evolutionary biologists and anthropologists.

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See also Dobzhansky, Theodosius; Davenport, Charles Benedict; Farabee, William Curtis; Mayr, Ernst; Mendel, Gregor Johann

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