

Washington Post

"How Science Responds When Creationists Criticize Evolution"

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Maybe you've encountered them, the perfectly nice people who stop you with a statement like, "Well, you know, evolution is just a theory, and it's very controversial, even among scientists."

Or maybe they say, "There's no way a bunch of gears and springs in a junk pile could suddenly fall together by accident and become a working watch. The existence of a watch tells you there had to be an intelligent watchmaker." Sometimes, they'll stump you by asserting that, on his deathbed, Charles Darwin renounced his theory of evolution.

Usually the people who say these things mean well. But the statements are based on a faulty understanding of biology. Unfortunately, many of us challenged by those who call themselves creationists are not well prepared to respond.

But science has good answers to these challenges to the theory of evolution. First, there's absolutely no controversy within science about the reality of evolution. There is a well accepted, solidly established body of evidence showing that evolution is real and, although knowledge of some mechanisms is incomplete, much is known about how evolution works. From this body of information we've drawn answers to some of the most common challenges issued by creationists.

Take that last one, about [Darwin on his deathbed](#). Not only is it irrelevant to whether evolution is true, the statement is false. For one thing, Darwin would have had no motive to recant. Before the great naturalist died in 1882, he had the satisfaction of knowing that the Church of England and several other Christian denominations had declared there to be no conflict between his theory and the churches' teachings. Indeed, Darwin, an evolutionist to the end, was laid to rest in the hallowed ground of Westminster Abbey.

In the generations since, most major denominations within the Christian, Jewish and Islamic traditions have found Darwinian evolution compatible with their religious beliefs. The statement by Pope John Paul II in November that evolution was more than a hypothesis was the fourth acceptance of evolution by the Roman Catholic Church.

For some people, fundamentalist Protestants most prominently, the issue likewise has been settled but with the opposite verdict.

For them, Genesis, however poetic, never uses metaphor or simile to tell the story of how the world and its inhabitants came to be. To them, the Bible is a scientific document to be taken literally. If science makes a claim counter to the Bible, creationists say it is automatically understood that science is wrong and a literal reading of the scripture is right.

Creationists believe that God created each kind of living thing independently and instantaneously about 6,000 years ago and all during the first six days.



Evolutionists believe that all living things are descended, through a cumulative series of genetic changes, from one common ancestor, or perhaps a few ancestors. The first ancestors would have been primitive, self-replicating, cell-like structures that arose more than 3.5 billion years ago. Evolutionists are, however, quite far from explaining how the first living thing arose.

Here with are criticisms you may hear, drawn from creationists and their literature, and responses based on what scientists have learned. What follows is not an attack on creationism but a defense of evolutionism.



Evolution is just a theory; it hasn't been proved.

Well, yes, [evolution is a theory](#), but not in the way that critics think. When scientists refer to it as the “theory of evolution,” the wording does not mean that they doubt it’s true. Evolution has been nailed down about as solidly as anything can be in science.

The confusion arises because in science “theory” means more than “hypothesis.” A hypothesis is a speculation or a prediction. Experiments or observations are needed to verify it. A theory, on the other hand, is a broad explanation for a class of phenomena. It generally is bigger and grander than a single hypothesis, even one that has passed all tests.

Thus, atomic theory is the coherent set of explanations of the structure and behavior of atoms. Einstein’s theory of relativity has passed every experimental test but still is called a theory.

In science, an explanation becomes a theory if it is internally consistent, always agrees with observations and can be used to make testable predictions (hypotheses). Within a theory may be “laws,” which can be expressed more tersely, often with mathematical equations.

So, has evolution been proved true? Strictly speaking, no. It is an accepted fact of scientific logic that you can never prove something true. Experiments and observations can only falsify theories or hypotheses.

Scientists insist on many tests of a hypothesis, the results all tending in the same direction before they accept it as probably true. The more evidence, the more acceptable it is and the higher the probability of truth.

Still, in science there is no such thing as 100 percent certainty. The evidence for evolution is so overwhelming that scientists say the probability of it being true approaches 100 percent. The fact that creationists say they are fully 100 percent certain of their view is based not on scientific evidence but, as their own literature says, on their faith in the literal truth of Genesis, which gives rise to doubts about the scientific case.

In fact, evolution has massive amounts of supporting evidence from many fields of science—*anatomy, geology, animal behavior, paleontology and even molecular biology.*

The odds against random chance producing a complex organism from lifeless ingredients are astronomical.

If [chance](#) were the only factor, this would be true. But chance is only one of two key players, and the other, natural selection, is decidedly not random. It favors species better adapted to their environments and kills off those less suitable. The process applies to all living things.

Here's how it works. Every generation usually produces more offspring than can survive, given limited supplies of food, water, space and other resources in a given habitat. For no species are Earth's natural resources unlimited. Individuals must compete with other members of their own species for these resources.

The offspring, however, are slightly different from one another in genetic endowment. Because of mutations in genes—here's the only random part—siblings differ in various subtle ways.

As a result, individuals that happen to inherit traits that give them an advantage automatically will be more likely to survive than their relatives lacking the trait.

They probably will have more offspring, and the offspring will inherit the genetic trait.

Far from being random, natural selection ensures that the only players in what Darwin called the "struggle for existence" are those that have passed all previous tests.

There are no transitional fossils.

The fossil record is rife with gaps where evolution says there should be intermediate forms.

Far from it. Paleontologists have found [many transitional fossils](#) representing intermediate forms in the evolution of one major form of life into another.

There are, for example, excellent skeletons of extinct animals showing the transitions from primitive fish to bony fish, from fish to amphibian (the first four-legged creatures walked on the ocean bottom, not on land), from amphibian to reptile, from reptile to mammal (it happened about the time the first dinosaurs were arising), from reptile to bird (the bird-sized *Archaeopteryx* specimen from southern Germany, for example, has feathers and dinosaurlike teeth) and even from land animal to whale (there are fossil whales with four legs, and modern whales

still have remnants of hind legs buried in their flesh; their front legs have changed into flippers).

There is abundant fossil evidence showing transitional diversifications among mammals into rodents, bats, rabbits, carnivores, [horses](#), elephants, manatee, deer, cows and many others. One of the most finely divided sequences of transitions documents the evolution of apelike creatures through half a dozen intermediate forms into modern humans.

Perhaps the oldest known transitional sequence involves the horse. It starts about 55 million years ago with a terrier-sized creature that had four toes in front and three in back. This is the famous species once called Eohippus, but now, for technical reasons, renamed Hyracotherium.

The lineage evolved through at least 14 steps, each represented in the fossil record by a successful species, until the modern horse, a pony-sized Equus, the genus to which modern horses belong, appeared about 4 million years ago.

Still, gaps in the fossil record will keep paleontologists busy for decades. Most kinds of fossils are extremely rare. After all, to become a fossil, the species not only must exist, but individuals also must die in places where conditions are right for preservation. In other words, the skeleton must be buried in sediments with preservative properties before scavengers or weathering can destroy it. Then only a few of those places will undergo erosion or uplifting that exposes the long-buried remains.

The fossil record shows that species do not evolve but exist for millions of years without changing.

It is true that most species appear to persist unchanged through time. Although some evolutionists once thought that continuous gradual change might be the rule, it is now clear that species are more stable.

They come into existence after relatively brief periods of rapid change in a small sub-population of a preexisting species. After only a few centuries or a few millennia of change, the new species persists with little further change for long periods, sometimes millions of years. This varying tempo is called "[punctuated equilibrium](#)."

The periods of change generally coincide with episodes of environmental instability such as sudden climate change. A desert may become a wetland; a warm climate may turn cool. The change may wipe out species not suited to the new conditions and create opportunities for new "lifestyles" to emerge.

So, if individuals happen to have inherited a mutation that "pre-adapted" them to the new regime, they automatically prosper at the expense of their brothers.

Once the environment stabilizes, so do the species in it.

Evolution has never been observed.

Yes, it has, and not just the rise of minor changes but of [whole new species](#).

Strictly speaking, evolution is simply a change in the frequency with which specific genes occur in a population. By this token, there is the well known example of the peppered moth of Britain.

In 1848, 98 percent of these moths were gray, a color that hid them from birds when they perched on gray lichens that covered tree trunks. Darker-winged variants were rare and tended to be eaten by birds. Then as the Industrial Revolution's smokestacks killed the lichens and darkened tree trunks, the gray moths stood out and were eaten

while the darker mutants survived.

Gradually, the moth became a predominantly dark-winged species and, by 1898, gray individuals were less than 5 percent of the total. Now that air pollution controls have taken effect, lichens are growing back, and the peppered moth again is becoming a chiefly gray species.

In that case, evolution by natural selection occurred but did not create a new species. Gray moths still could interbreed with black moths, proving that they belonged to the same species.

The rise of bona fide new species has, however, been documented in such laboratory-reared species as the fruit fly, of which eight new species have been found. Also, six new species of other insects have been seen to form. In such free-living species as mice, a new species has emerged on the Faeroe Islands in the last 250 years.

In recent years, scientists also have documented evolution of a new species of marine worm, called a polychate. And among plants, at least a dozen new species have been seen to arise over the last 50 years, including a new species of corn.

Natural selection cannot change one species into another because it can work only on variation already present in the species.

Yes, but new variation is being generated continuously by mutations. In the case of the peppered moth, genes for light and dark wings were present in the population, and nature merely favored one over the other. Nonetheless, the variation had to have arisen at some point in the past.

Each gene is the code telling a cell how to make a particular protein. Once made, the protein carries out specific functions that make one kind of cell different from another and, sometimes, one organism different from another. Thus mutations can change the whole organism's form and function.

Although creationists sometimes assert that [all mutations are harmful](#), this is not so.

Mutations happen all the time, primarily as a result of simple errors in the gene-copying process that makes sperm and eggs. Not only can a tiny change be introduced into a gene, but an entire functional module from one gene can be copied and inserted into a different gene, creating in one step a protein with radically new properties. This is one way in which very large changes can occur suddenly.

Most mutations are neutral because most of the genome is noncoding DNA—the so-called “junk DNA.” Most of the rest are harmful, killing the cell or perhaps the whole embryo long before birth. But the rare mutation will confer an advantage. Quite often, however, the advantage will be irrelevant in the current environment of the species.

But consider this scenario. Suppose that the world of the chicken suddenly became flooded and the hapless birds had to float on the water and paddle about. Because most chickens don't have much webbing between their toes, they wouldn't move very fast. But if one odd chicken happened to be born with a little webbing between its toes, a trait perhaps disadvantageous on land, it could paddle faster and maybe escape predators more easily. Obviously, this chicken would be more likely to survive and to raise a family of web-toed chicks.

Life contains structures and systems too complex to have evolved gradually, step by step.

This is one of the oldest criticisms made by creationists, and recently it has been revived. Darwin himself anticipated it, citing the eye with all its “inimitable contrivances” as a structure that may seem too complex to have arisen through a series of steps, each conferring sufficient advantage that it would be favored for survival.

Darwin said that, if it could be demonstrated that any structure exists in nature that could not have arisen by natural selection, “my theory would absolutely break down.”

Since then, biologists have vindicated Darwin by discovering many examples of primitive eyes among various species, ranging from the simplest eye spots of a few light-sensitive cells through progressively more complex forms to the complete, highly sophisticated mammalian eye.

Together, these discoveries show how a series of many cumulative steps could create a human eye. In fact, biologists now know that eyes arose and evolved independently at least 40 times.

Last year, [Michael Behe](#), a biochemist, published *Darwin's Black Box*, raising the argument again. Within science it was widely dismissed for its tactic of argument from ignorance.

Behe essentially contends that if you can't imagine how something could have happened naturally, then that is proof that the thing must have happened supernaturally. In science, ignorance is no more evidence than was Darwin's astonishment about the eye.

Behe cites several structures and processes, some inside cells and some involving whole organ systems, that he says are “irreducibly complex” and therefore must have arisen by special creation, by God creating the whole thing at one stroke.

One, for example, is a series of at least seven chemical reactions that must occur within blood for it to clot and stop a wound from bleeding. In this scientifically well-known “cascade” of reactions, substance A first acts on substance B, changing it into a form that can act on substance C, which then is changed so it can act on substance D and so on. Obviously, the system works well.

Behe argues, however, that it is inconceivable that the cascade could have evolved from some simpler form with fewer steps because all steps now are essential. Since each step requires the participation of several components, Behe writes, not only is the entire blood-clotting system irreducibly complex, but so is each step in the cascade.

As it happens, scientists have deduced the nature of an evolutionary path that a primitive blood-clotting mechanism could have followed to evolve the more complex cascade. The process is biologically plausible and uses well-known mechanisms that exist in all cells for duplication and modification of existing genes followed by inactivation of the old gene.

The mutations required at the beginning of the process are neither beneficial nor deleterious, but once they occur, they produce a blood-clotting system that can be controlled more precisely. This is beneficial since a runaway blood-clotting mechanism could turn the entire bloodstream into one massive clot.

The same events that turn a one-step process into a two-step process could be repeated indefinitely, scientists have found, adding still finer control at each step, conferring yet greater advantage.

The fact that this problem has been addressed, incidentally, refutes one of Behe's contentions. He says evolutionists never try to explain how complex systems might have arisen through incremental changes. In fact, scientific literature includes numerous such instances.

Evolution violates the Second Law of Thermodynamics.

A [complete answer](#) would require lots of mathematics and a deeper understanding of the Second Law than can be described here. The short answer, however, is: no, it doesn't.

The Second Law can be stated in many different ways, but the most relevant is that order cannot emerge from disorder in a closed system. In other words, a random jumble cannot spontaneously assemble itself into some orderly structure without tapping some outside energy source. Some creationists say this means that life cannot evolve from simple to complex. Complex life forms would have to have been created separately.

Earth, however, is not a closed system. It receives huge amounts of energy from the sun and from chemical bonds within compounds, and this energy allows life to evolve.

If the Second Law truly prohibited local emergence of increased order, there would be no ice cubes. The greater orderliness of water molecules in ice crystals than in the liquid state is purchased with the expenditure of energy at the generator that made the electricity to run the freezer. And that makes it legal under the Second Law.

Creation science is genuine science.

The philosophical underpinnings of creation science automatically place it in a very different realm from natural science. The natural sciences (biology, chemistry, physics and the like) begin with the assumption that nothing should be accepted as true for purposes of research unless it can be demonstrated reliably through observation or experiment.

Creation science starts with the assumption that Genesis is literally true. “God’s inerrant word,” as recorded in the Bible, “must always prevail” over anything that natural science says, according to Henry M. Morris, founder and recently retired director of the country’s largest and most influential creationist organization, the Institute for Creation Research in El Cajon, California.

The institute’s literature describes the institute as “a Christ-focused creation ministry.”

(Boyce Rensberger is the author of *Life Itself: Exploring the Realm of the Living Cell*, Oxford University Press, 1997, 290 pp.)

The Lady Hope Story:

◆ ◆ ◆ A Widespread Falsehood ◆ ◆ ◆

File pulled from the Talk.Origins Archive, with additional information from the [Autobiography of Charles Darwin](#) and [The Darwin Legend](#), by James Moore.

by **Simon Yates**

A few more details on the spread of the story and its subsequent rebuttal, taken from the book [The Survival of Charles Darwin: A Biography of a Man and an Idea](#), by Ronald W. Clark, (published by Weidenfeld & Nicholson, 1985), p. 199.

“Shortly after his death, Lady Hope addressed a gathering of young men and women at the educational establishment founded by the evangelist Dwight Lyman Moody at Northfield, Massachusetts. She had, she maintained, visited Darwin on his deathbed. He had been reading the Epistle to the Hebrews, had asked for the local Sunday school to sing in a summerhouse on the grounds, and had confessed: ‘How I wish I had not expressed my theory of evolution as I have done.’ He went on, she said, to say that he would like her to gather a congregation since he ‘would like to speak to them of Christ Jesus and His salvation, being in a state where he was eagerly savouring the heavenly anticipation of bliss.’

“With Moody's encouragement, Lady Hope's story was printed in the *Boston Watchman Examiner*. The story spread, and the claims were republished as late as October 1955 in the *Reformation Review* and in the Monthly Record of the Free Church of Scotland in February 1957. These attempts to fudge Darwin's story had already been exposed for what they were, first by his daughter Henrietta after they had been revived in 1922. ‘I was present at his deathbed,’ she wrote in the *Christian* for February 23, 1922. ‘Lady Hope was not present during his last illness, or any illness. I believe he never even saw her, but in any case she had no influence over him in any department of thought or belief. He never recanted any of his scientific views, either then or earlier. We think the story of his conversion was fabricated in the U.S.A. . . . The whole story has no foundation whatever.’” (Ellipsis is in the book)

Clark's source for Lady Hope's supposed quotations of Darwin is given as [Down, the Home of the Darwins: The Story of a House and the People Who Lived There](#), by Sir Hedley Atkins KBE, published by Phillimore for the Royal College of Surgeons of England, 1974. Henrietta's rebuttal is referenced more fully as: Mrs. R B Litchfield, "Charles Darwin's Death-Bed: Story of Conversion Denied," *The Christian*, February 23, 1922, p. 12.

Charles Darwin (1809-1882)

"By further reflecting that the clearest evidence would be requisite to make any sane man believe in the miracles by which Christianity is supported,—and that the more we know of the fixed laws of nature the more incredible do miracles become,—that the men at that time were ignorant and credulous to a degree almost incomprehensible by us,—that the Gospels cannot be proven to have been written simultaneously with the events,—that they differ in many important details, far too important, as it seemed to me to be admitted as the usual inaccuracies of eye witnesses;—by such reflections as these, which I give not as having the least novelty or value, but as they influenced me, I gradually came to disbelieve in Christianity as a divine revelation. The fact that many fake religions have spread over large portions of the earth like wildfire had some weight with me. But I was very unwilling to give up my belief; I feel sure of this, for I can remember often and often inventing day-dreams of old letters between distinguished Romans, and manuscripts being discovered at Pompeii or elsewhere, which confirmed in the most striking manner all that was written in the Gospels. But I found it more and more difficult, with free scope given to my imagination, to invent evidence which would suffice to convince me. Thus disbelief crept over me at a very slow rate, but was at last complete. The rate was so slow that I felt no distress, and have never since doubted even for a single second that my conclusion was correct."

(Charles Darwin in his [Autobiography of Charles Darwin](#), Dover Publications, 1992, p. 62.)

Sir Francis Darwin (1848-1925)

"Lady Hope's account of my father's views on religion is quite untrue. I have publicly accused her of falsehood, but have not seen any reply. My father's agnostic point of view is given in my *Life and Letters of Charles Darwin*, Vol. I., pp. 304-317. You are at liberty to publish the above statement. Indeed, I shall be glad if you will do so. Yours faithfully, Francis Darwin. Brookthorpe, Gloucester. May 28, 1918."

(Quoted from James Moore, [The Darwin Legend](#), Baker Book House, MI. 1994, p. 21.)

Charles Darwin (1809-1882)

"I think that generally (& more & more as I grow older), but not always, that an agnostic would be the most correct description of my state of mind."

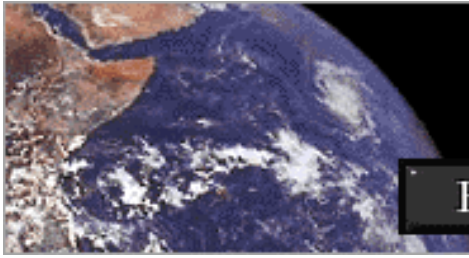
(Quoted from Adrian Desmond and James Moore, [Darwin: The Life of a Tormented Evolutionist](#), New York: W. W. Norton & Company, 1991, p. 636.)

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Evolution is a Fact and a Theory

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When non-biologists talk about biological evolution they often confuse two different aspects of the definition. On the one hand there is the question of whether or not modern organisms have evolved from older ancestral organisms or whether modern species are continuing to change over time. On the other hand there are questions about the mechanism of the observed changes... how did evolution occur? Biologists consider the existence of biological evolution to be a *fact*. It can be demonstrated today and the historical evidence for its occurrence in the past is overwhelming. However, biologists readily admit that they are less certain of the exact *mechanism* of evolution; there are several *theories* of the mechanism of evolution. Stephen J. Gould has put this as well as anyone else:

In the American vernacular, "theory" often means "imperfect fact" - part of a hierarchy of confidence running downhill from fact to theory to hypothesis to guess. Thus the power of the creationist argument: evolution is "only" a theory and intense debate now rages about many aspects of the theory. If evolution is worse than a fact, and scientists can't even make up their minds about the theory, then what confidence can we have in it? Indeed, President Reagan echoed this argument before an evangelical group in Dallas when he said (in what I devoutly hope was campaign rhetoric): "Well, it is a theory. It is a scientific theory only, and it has in recent years been challenged in the world of science - that is, not believed in the scientific community to be as infallible as it once was."

Well evolution is a theory. It is also a fact. And facts and theories are different things, not rungs in a hierarchy of increasing certainty. Facts are the world's data. Theories are structures of ideas that explain and interpret facts. Facts don't go away when scientists debate rival theories to explain them. Einstein's theory of gravitation replaced Newton's in this century, but apples didn't suspend themselves in midair, pending the outcome. And humans evolved from ape-like ancestors whether they did so by Darwin's proposed mechanism or by some other yet to be discovered.

Moreover, "fact" doesn't mean "absolute certainty"; there ain't no such animal in an exciting and complex world. The final proofs of logic and mathematics flow deductively from stated premises and achieve certainty only because they are NOT about the empirical world. Evolutionists make no claim for perpetual truth, though creationists often do (and then attack us falsely for a style of argument that they themselves favor). In science "fact" can only mean "confirmed to such a degree that it would be perverse to withhold provisional consent." I suppose that apples might start to rise tomorrow, but the possibility does not merit equal time in physics classrooms.

Evolutionists have been very clear about this distinction of fact and theory from the very beginning, if only because we have always acknowledged how far we are from completely understanding the mechanisms (theory) by which evolution (fact) occurred. Darwin continually emphasized the difference between his two great and separate accomplishments: establishing the fact of evolution, and proposing a theory - natural selection - to explain the mechanism of evolution.

- Stephen J. Gould, "Evolution as Fact and Theory"; Discover, May 1981

Gould is stating the prevailing view of the scientific community. In other words, the experts on evolution consider it to be a *fact*. This is not an idea that originated with Gould as the following quotations indicate:

Let me try to make crystal clear what is established beyond reasonable doubt, and what needs further study, about evolution. Evolution as a process that has always gone on in the history of the earth can be doubted only by those who are ignorant of the evidence or are resistant to evidence, owing to emotional blocks or to plain bigotry. By contrast, the mechanisms that bring evolution about certainly need study and clarification. There are no alternatives to evolution as history that can withstand critical examination. Yet we are constantly learning new and important facts about evolutionary mechanisms.

- Theodosius Dobzhansky "Nothing in Biology Makes Sense Except in the Light of Evolution", *American Biology Teacher* vol.35 (March 1973) reprinted in *Evolution versus Creationism*, J. Peter Zetterberg ed., ORYX Press, Phoenix AZ 1983

Also:

It is time for students of the evolutionary process, especially those who have been misquoted and used by the creationists, to state clearly that evolution is a FACT, not theory, and that what is at issue within biology are questions of details of the process and the relative importance of different mechanisms of evolution. It is a FACT that the earth with liquid water, is more than 3.6 billion years old. It is a FACT that cellular life has been around for at least half of that period and that organized multicellular life is at least 800 million years old. It is a FACT that major life forms now on earth were not at all represented in the past. There were no birds or mammals 250 million years ago. It is a FACT that major life forms of the past are no longer living. There used to be dinosaurs and Pithecanthropus, and there are none now. It is a FACT that all living forms come from previous living forms. Therefore, all present forms of life arose from ancestral forms that were different. Birds arose from nonbirds and humans from nonhumans. No person who pretends to any understanding of the natural world can deny these facts any more than she or he can deny that the earth is round, rotates on its axis, and revolves around the sun.

The controversies about evolution lie in the realm of the relative importance of various forces in molding evolution.

- R. C. Lewontin "Evolution/Creation Debate: A Time for Truth" *Bioscience* 31, 559 (1981) reprinted in *Evolution versus Creationism*, op cit.

This concept is also explained in introductory biology books that are used in colleges and universities (and in some of the better high schools). For example, in some of the best such textbooks we find:

Today, nearly all biologists acknowledge that evolution is a fact. The term THEORY is no longer appropriate except when referring to the various models that attempt to explain HOW life evolves... it is important to understand that the current questions about how life evolves in no way implies any disagreement over the fact of evolution.

- Neil A. Campbell, *Biology* 2nd ed., 1990, Benjamin/Cummings, p.434

Also:

Since Darwin's time, massive additional evidence has accumulated supporting the fact of evolution - that all living organisms present on earth today have arisen from earlier forms in the course of earth's long history. Indeed, all of modern biology is an affirmation of this relatedness of the many species of living things and of their gradual divergence from one another over the course of time. Since the publication of *The Origin of Species*, the important question, scientifically speaking, about evolution has not been whether it has taken place. That is no longer an issue among the vast majority of modern biologists. Today, the central and still fascinating questions for biologists concern the mechanisms by which evolution occurs.

- Helena Curtis and N. Sue Barnes, *Biology* 5th ed. 1989, Worth Publishers, p.972

One of the best introductory books on evolution (as opposed to introductory biology) is that by Douglas J. Futuyma, and he makes the following comment:

A few words need to be said about the "theory of evolution," which most people take to mean the proposition that organisms have evolved from common ancestors. In everyday speech, "theory" often means a hypothesis or even a mere speculation. But in science, "theory" means "a statement of what are held to be the general laws, principles, or causes of something known or observed", as the Oxford English Dictionary defines it. The theory of evolution is a body of interconnected statements about natural selection and the other processes that are thought to cause evolution, just as the atomic theory of chemistry and the Newtonian theory of mechanics are bodies of statements that describe causes of chemical and physical phenomena. In contrast, the statement that organisms have descended with modifications from common ancestors - the historical reality of evolution - is not a theory. It is a fact, as fully as the fact of the earth's revolution about the sun. Like the heliocentric solar system, evolution began as a hypothesis, and achieved "facthood" as the evidence in its favor became so strong that no knowledgeable and unbiased person could deny its reality. No biologist today would think of submitting a paper entitled "New evidence for evolution"; it simply has not been an issue for a century.

- Douglas J. Futuyma, op. cit., p.15

There are readers of these newsgroups who reject evolution for religious reasons. In general these readers oppose both the *fact* of evolution and *theories* of mechanisms, although some anti-evolutionists have come to realize that there is a difference between the two concepts. That is why we see some leading anti-evolutionists admitting to the fact of "microevolution" - they know that evolution can be demonstrated. These readers will not be convinced of the "facthood" of (macro)evolution by any logical argument and it is a waste of time to make the attempt. The best that we can hope for is that they understand the argument that they oppose. Even this simple hope is rarely fulfilled.

There are some readers who are not anti-evolutionist but still claim that evolution is "only" a theory which can't be proven. This group needs to distinguish between the fact that evolution occurs and the theory of the mechanism of evolution.

We also need to distinguish between facts that are easy to demonstrate and those that are more circumstantial. Examples of evolution that are readily apparent include the fact that modern populations are evolving and the fact that two closely related species share a common ancestor. The evidence that *Homo sapiens* and chimpanzees share a recent common ancestor falls into this category. There is so much evidence in support of this aspect of primate evolution that it qualifies as a fact by any common definition of the word "fact".

In other cases the available evidence is less strong. For example, the relationships of some of the major phyla are still being worked out. Also, the statement that all organisms have descended from a single common ancestor is strongly supported by the available evidence, and there is no opposing evidence. However, it is not yet appropriate to call this a "fact" since there are reasonable alternatives.

Finally, there is an epistemological argument against evolution as fact. Some readers of these newsgroups point out that nothing in science can ever be "proven" and this includes evolution. According to this argument, the probability that evolution is the correct explanation of life as we know it may approach 99.9999...9% but it will never be 100%. Thus evolution cannot be a fact. This kind of argument might be appropriate in a philosophy class (it is essentially correct) but it won't do in the real world. A "fact", as Stephen J. Gould pointed out (see above), means something that is so highly probable that it would be silly not to accept it. This point has also been made by others who contest the nit-picking epistemologists.

The honest scientist, like the philosopher, will tell you that nothing whatever can be or has been proved with fully 100% certainty, not even that you or I exist, nor anyone except himself, since he might be dreaming the whole thing. Thus there is no sharp line between speculation, hypothesis, theory, principle, and fact, but only a difference along a sliding scale, in the degree of probability of the idea. When we say a thing is a fact, then, we only mean that its probability is an extremely high one: so high that we are not bothered by doubt about it and are ready to act accordingly. Now in this use of the term fact, the only proper one, evolution is a fact. For the evidence in favor of it is as voluminous, diverse, and convincing as in the case of any other well established fact of science concerning the existence of things that cannot be directly seen, such as atoms, neutrons, or solar gravitation

So enormous, ramifying, and consistent has the evidence for evolution become that if anyone could now disprove it, I should have my conception of the orderliness of the universe so shaken as to lead me to doubt even my own existence. If you like, then, I will grant you that in an absolute sense evolution is not a fact, or rather, that it is no more a fact than that you are hearing or reading these words.

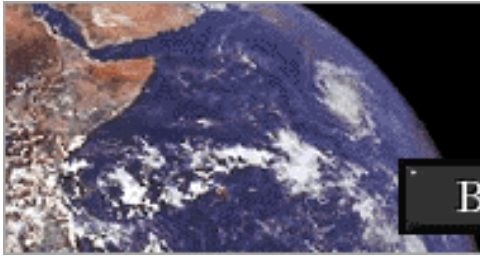
- H. J. Muller, "One Hundred Years Without Darwin Are Enough" *School Science and Mathematics* 59, 304-305. (1959) reprinted in *Evolution versus Creationism* op cit.

In any meaningful sense evolution is a fact, but there are various theories concerning the mechanism of evolution.

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Transitional Vertebrate Fossils FAQ

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[Last Update: March 17, 1997]

Iwrote this FAQ as a reference for answering the "there aren't any transitional fossils" statement that pops up on talk.origins several times each year. I've tried to make it an accurate, though highly condensed, summary of known vertebrate fossil history in those lineages that led to familiar modern forms, with the known transitions *and* with the known major gaps both clearly mentioned. Version 6.0 of the FAQ has been almost entirely rewritten, with:

1. A completely rewritten introduction & conclusion, discussing what "transitional" means, why gaps occur, and what the fossil record shows.
2. A greatly expanded list of "chains of genera" for most groups, especially mammals.
3. References for documented species-to-species fossil transitions, mostly for mammals.
4. Explicit mention of the notable remaining gaps in the fossil record.

If you have questions about this FAQ or want to send email to the author, [click here](#).

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[PART 2](#) has transitions among mammals (starting with primates), including numerous species-to-species transitions, discussion, and references. If you're particularly interested in humans, skip to the primate section of part 2, and also look up the fossil hominid FAQ.

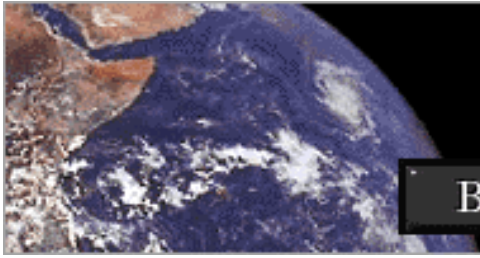
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Archaeopteryx FAQs

[All About Archaeopteryx](#)

Archaeopteryx is commonly cited as an example of a transitional fossil (i.e. a form showing characters common to two separate groups). This is disputed by anti-evolutionists, who claim that *Archaeopteryx* is a complete bird and thus cannot be transitional. This FAQ briefly describes the fossils and then discusses the large number of features shared between *Archaeopteryx* and dinosaurs. It has been claimed that *Archaeopteryx* could fly just like modern birds. The mechanics of flight are briefly described and it is shown that *Archaeopteryx* lacked features essential to be able to fly like modern birds. *Archaeopteryx*'s ancestry is discussed, along with the contentious position of *Protoavis*.

[On Archaeopteryx, Astronomers, and Forgery](#)

Most anti-evolution arguments about *Archaeopteryx* revolve around how it is a complete bird and thus not transitional. However, a group of people led by Prof. Fred Hoyle and Dr. N. Wickramasinghe have adopted a different tack by suggested that *Archaeopteryx* is a forgery. They claim that the feather impressions were forged onto a small reptile skeleton and implicate the then director of the Natural History Museum in London, Sir Richard Owen. Not unnaturally, these claims have been contested by the Natural History Museum. This FAQ details the claims made by both sides and shows that the suggestion of forgery is unsupported by the evidence.

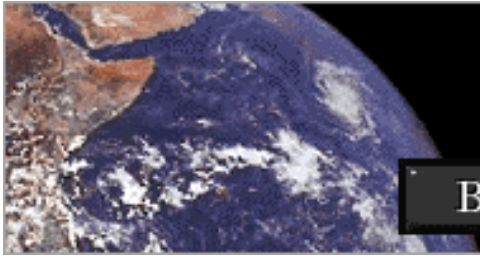
[Archaeopteryx: The Challenge of the Fossil Record](#)

The most detailed anti-evolution claims that have been made about *Archaeopteryx* occur in "Evolution: the Challenge of the Fossil Record" by Dr. D.T. Gish. This book, first published in 1985, is probably one of the best known sources of anti-evolution arguments. It has now been superseded by a new book, "Evolution: The Fossils Still Say NO", published in 1995. However, given the well known half-life of anti-evolutionary material years after it has been superseded, it is useful to look at the claims made in the 1985 book to see whether or not they hold up to scrutiny. They don't.

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Punctuated Equilibria

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[Last Update: February 4, 1996]

Outline

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0. Foreword

There are few components of modern evolutionary theory which seem so prone to misinterpretation as Niles Eldredge and Stephen Jay Gould's theory of punctuated equilibria (PE for short). In this matter, the person attempting to come to a better understanding of punctuated equilibria will find that he or she may be hampered by the popular writings of those same authors rather than helped. As in most cases, the primary literature remains the best source of information.

1. Summary of Punctuated Equilibria

The essential features that make up Punctuated Equilibria are as follows:

1. Paleontology should be informed by neontology.
2. Most speciation is cladogenesis rather than anagenesis.
3. Most speciation occurs via peripatric speciation.
4. Large, widespread species usually change slowly, if at all, during their time of residence.
5. Daughter species usually develop in a geographically limited region.
6. Daughter species usually develop in a stratigraphically limited extent, which is small in relation to total residence time of the species.
7. Sampling of the fossil record will reveal a pattern of most species in stasis, with abrupt appearance of newly derived species being a consequence of ecological succession and dispersion.
8. Adaptive change in lineages occurs mostly during periods of speciation.
9. Trends in adaptation occur mostly through the mechanism of species selection.

The theory of Punctuated Equilibria provides paleontologists with an explanation for the patterns which they find in the fossil record. This pattern includes the characteristically abrupt appearance of new species, the relative stability of morphology in widespread species, the distribution of transitional fossils when those are found, the apparent differences in morphology between ancestral and daughter species, and the pattern of extinction of species.

PE relies upon the insights of study of modern species for its principles. These studies indicate the importance of consideration of geography and interspecies interactions upon predictions of the distribution and abundance of transitional specimens. While Eldredge and Gould acknowledge that geological processes contribute to the "gappiness" of the fossil record, they also assert that PE is by far the more important consideration in that regard.

2. The Problem of Paleospecies

Paleontologists have to recognize species from their fossil remains. The problem of "What is a paleospecies?" led Niles Eldredge and Stephen Jay Gould to propose the theory of punctuated equilibria. The term "paleospecies" makes explicit the distinction between the classification of species from fossil remains and the process of recognizing species in modern populations. This problem involves geology, taphonomy, taxonomy, and -- though often ignored -- geography.

Mayr's Biological Species Concept uses the criterion of reproductive isolation to distinguish species in modern populations. Paleontologists who pursue taxonomic endeavors (which includes most of them) have to classify their finds generally based upon morphological features. The rareness of preservation of tissues containing DNA, or even of soft tissues, limits the range of diagnostic characters which may be utilized. The paleontologist has no access to such information. (Whether modern biologists really do have access to that information is a matter of some little debate in the literature.)

The fossil record is incomplete. This incompleteness has many contributing factors. Geological processes may cause to confusion or error, as sedimentary deposition rates may vary, erosion may erase some strata, compression may turn possible fossils into unrecognizable junk, and various other means by which the local fossil record can be turned into the equivalent of a partially burned book, which is then unbound, pages perhaps shuffled, and from which a few pages are retrieved. Beyond geology, there remains taphonomy -- the study of how organisms come to be preserved as fossils. Here, there are further issues to be addressed. Hard parts of organisms fossilize preferentially. The conditions under which even those parts may become fossilized are fairly specialized. All this results in a heavily skewed distribution of even what *parts* of organisms become fossilized, and that affects which features of morphology are available for use in classification. The issue of geography enters into all this, as a consequence of the fact that living lineages occupy ecological niches, and those niches are bound to certain features of geography.

Paleospecies, then, have to be recognized as species from morphology alone, where the available morphological characters are drawn from a skewed distribution, the pattern of fossilization is skewed, and the geographic correlates of fossilization are limited in extent.

3. Patterns of speciation from neontological study

Eldredge and Gould's insight into paleontological processes was to derive their understanding of paleospecies from living biological species. In this manner, it can be made clear what PE means for the concept of paleospecies.

First, modern species appear to have derived from cladogenesis, the splitting of a daughter species from an ancestral species rather than transformation of the ancestral species in toto. This is a multiplication of species, and without it, the diversity of the living systems that we see would be impossible.

Second, the mode of speciation most often seen is also identified from modern populations. That mode is allopatric speciation of peripheral isolates, or peripatric speciation in Mayr's terminology. Peripatric speciation states that a population of an ancestral species in a geographically peripheral part of the ancestral range is modified over time until even when the ancestral and daughter populations come into contact, there is reproductive isolation. While saltational speciation by change in ploidy is observed to occur in modern populations, this form of speciation is also known to be rare (except in plants). The incipient speciation of clinal forms has recently become very controversial, and is also likely to be rare in any case. Sympatric speciation, the production of a daughter species within the geographic range of the parent species, likewise is held to be a very rare event seen primarily in insect and parasite lineages.

Third, the frequency with which peripatric speciation occurs in modern lineages can be seen as "rare". This rarity is different from the rarity discussed in the last item. There, we compared how often certain modes of speciation were seen compared to other modes of speciation. Here, we want to know how common it is for a species to produce a daughter species by peripatric speciation. The answer is, "Not very common at all." This rarity means that a species may produce zero, one, or perhaps a few daughter species during its entire time span of existence.

Fourth, the period of transition between parent species and daughter species is short compared to the period of time a species exists as a distinct form. When a small sub-population is isolated from the rest of the population of a species, the particular set of variations in the sub-population is much smaller than that in the remainder of the population. These variations, when in conjunction with suitable features of geographic locale, climate, and resources, can lead to rapid development of reproductive isolation from the ancestral population. The reduction in variation due to small sub-population size is known as the "founder effect".

Fifth, significant adaptations developed or accentuated in the daughter species can lead to the rapid dispersal and establishment of a daughter species throughout the range of the ancestral species, or into new ranges. The ecological processes of dispersal and succession can occur very quickly compared to evolutionary processes of change.

Sixth, the principles of gene flow, genetic homeostasis, and large population size inhibit widespread ancestral populations from much directional (adaptive) change. Any adaptive change found in the ancestral population is likely to be small and unrelated to evolutionary trends.

4. Application of neontology to paleontology

Now we are ready to apply these concepts from the biology of extant organisms to that of fossil organisms. This proceeds on the simple inference that past life went about its business in much the same way as present life does.

Some of the predictions of Punctuated Equilibria are as follows:

(A) Punctuated Equilibria postulates that speciation events comprise most of the evolutionary change seen in adaptation. This is a consequence of the inhibitory effects of gene flow, genetic homeostasis, and large population sizes (6 above). The adaptations of newly speciated daughter populations are forever excluded from the ancestral population because of reproductive isolation (2 above).

(B) PE explains the abrupt appearance of new species in the fossil record. The splitting of lineages (1 above) in the mode of allopatric speciation (2 above) followed by ecological dispersal and succession (5 above) would result in geologically abrupt appearance of the daughter species everywhere except the limited geographic area where the speciation took place. Most speciation takes place as peripatric speciation, which is confined to a limited geographic region, and after which ecological principles argue for relatively rapid reintroduction and spread into new habitats for the daughter species. Since the critical change occurs in such a small region and in such a limited population, the probability of finding specimens which document the transition from ancestral to daughter species is very low. A population which can exploit resources untapped by current populations will grow and spread at somewhere near its theoretical intrinsic rate of increase. The cases of introduced species in modern times (the starling in North America, for example) demonstrate the extreme rapidity in which a species may spread across large geographic extents.

(C) PE explains the relative stasis of most species. A species may produce a few daughter species during its duration (3 above). Large interbreeding populations are unlikely to change much due to genetic homeostasis and gene flow from far-flung parts of the range (6 above) [Eldredge & Gould emphasize homeostatic mechanisms over gene flow.].

(D) PE asserts "species selection" as the way in which major adaptive trends proceed. Closely related species are often likely to overlap in niche space (5 above). Ecological processes may cause the displacement and possible extinction of certain species due to competition with other species. If adaptive change in large populations is largely inhibited (6 above), then each species represents a "hypothesis" that is "tested" in competition. This is one of the more controversial points in PE.

(E) PE also makes a statement concerning the pattern of fossils found. This pattern has both geographic and stratigraphic components. If peripatric speciation is the mode of speciation, then the place where transitional fossils between a parent and daughter species will be found will be limited in geographic region (2 above). Because the time needed for transition from parent to daughter species is short compared to the total residence time of either, the stratigraphic extent of transitional fossil sequences will be very brief (4 above).

5. PE vs. Phyletic Gradualism

Punctuated Equilibria could have been advanced simply upon the basis of features of geology, taphonomy, geography, and taxonomy. However, that is not how Eldredge and Gould chose to do it. Instead, they codified what they saw as an inaccurate and incorrect "picture" of the fossil record, labelled it as "phyletic gradualism", and demonstrated that their PE was to be preferred on several points.

The essential features of "phyletic gradualism" are described by Eldredge and Gould.

In this Darwinian perspective, paleontology formulated its picture for the origin of new taxa. This picture, though rarely articulated, is familiar to all of us. We refer to it here as "phyletic gradualism" and identify the following as its tenets:

1. New species arise by the transformation of an ancestral population into its modified descendants.
2. The transformation is even and slow.
3. The transformation involves large numbers, usually the entire ancestral population.
4. The transformation occurs over all or a large part of the ancestral species' geographic range.

These statements imply several consequences, two of which seem especially important to paleontologists:

1. Ideally, the fossil record for the origin of a new species should consist of a long sequence of continuous, insensibly graded intermediate forms linking ancestor and descendant.
2. Morphological breaks in a postulated phyletic sequence are due to imperfections in the geological record.

[E&G 1972]

While it is nice to have terminology by which obtuse opponents of a theory may be conveniently labelled, there is no actual point to even bringing up "phyletic gradualism" in establishing PE.

Eldredge and Gould quoted from Darwin in their 1972 paper to establish their concept of phyletic gradualism. They claim that Darwin set the task of later workers to search out confirmation of phyletic gradualism. That view is, unfortunately, just so much hokum.

Nothing can be effected, unless favourable variations occur, and variation itself is apparently always a very slow process. The process will often be greatly retarded by free intercrossing. Many will exclaim that these several causes are amply sufficient wholly to stop the action of natural selection. I do not believe so. On the other hand, I do believe that natural selection will always act very slowly, often only at long intervals of time, and generally on only a very few of the inhabitants of the same region at the same time. I further believe, that this very slow, intermittent action of natural selection accords perfectly well with what geology tells us of the rate and manner at which the inhabitants of this world have changed. [Charles Darwin, *Origin of Species* 1st Edition 1859, p.153]

The above quote from Darwin also shows that Darwin did not embrace three of the four antecedent conditions that Eldredge and Gould specified for phyletic gradualism, and the single one that Darwin did embrace is also a tenet of any theory of speciation. Some people may embrace phyletic gradualism, but it is incorrect to attribute the concept to Charles Darwin. The quote above is notable on several points. The "free intercrossing" bit is easily recognizable as a forerunner of the concept of gene flow, though Darwin was probably concerned there with blending inheritance. Darwin makes explicit that there is no constancy of rate implied with the comment on intermittent action. Darwin also recognized that change would be more likely to occur in a sub-population. Whether Darwin meant by "of the same region" much the same thing as the modern concept of allopatric speciation is disputable.

Darwin did think that a daughter species arose from a population of the parent species. So do punctuated equilibrist. Darwin did think that the transformation would be slow, but he did not think that it would be "even". Darwin did not think that the transformation would involve large numbers, and certainly not the entire parent population. Darwin did not think that the transformation would occur across the entire ancestral range.

But on the view of all the species of a genus having descended from a single parent, though now distributed to the most remote points of the world, we ought to find, and I believe as a general rule we do find, that some at least of the species range very widely; for it is necessary that the unmodified parent should range widely, undergoing modification during its diffusion, and should place itself under diverse conditions favourable for the conversion of its offspring, firstly into new varieties and ultimately into new species. [Charles Darwin, *Origin of Species* 1st Edition 1859, p.391]

It is difficult to extract meaning from the above without recognition that Darwin was well aware of the importance of geographical distribution in the production of new species.

Only a small portion of the world has been geologically explored. Only organic beings of certain classes can be preserved in a fossil condition, at least in any great number. Widely ranging species vary most, and varieties are often at first local, -- both causes rendering the discovery of intermediate links less likely. Local varieties will not spread into other and distant regions until they are considerably modified and improved; and when they do spread, if discovered in a geological formation, they will appear as if suddenly created there, and will be simply classed as new species. [Charles Darwin, *Origin of Species* 1st Edition 1859, p.439]

The above quote comes from the famous section on the imperfection of the geological record. However, Darwin makes it clear that geographic location makes a difference in the finding of intermediate forms. "Both causes" in the above could not make discovery of intermediate links less likely if Darwin expected the transformation of the entire parent population.

Gould has said that history cannot be done on the basis of selective quotes and qualifying footnotes, but rather must be a matter of general tenor and understanding. However, I think the onus is upon Eldredge and Gould to demonstrate that phyletic gradualism actually exists outside of their description.

There have been persons that have advanced positions that could more or less be termed "phyletic gradualism". A feature of Gould and Eldredge 1977 is the discussion of various purported examples of change in the phyletic gradualist mode. Of many examples, G&E only found one to meet their criteria for establishing phyletic gradualism. Most examples were disputed by G&E because the original work ignored the geographical dimension.

Richard Dawkins has a chapter in "The Blind Watchmaker" which goes into some detail on how "phyletic gradualism" is in many ways a strawman of Eldredge and Gould's creation. It is a recommended read.

6. Common errors in discussion of PE

Many errors can be found in discussion of the concept of PE. G&E 1977 point out several of these.

PE is not mutually exclusive of phyletic gradualism. Gould and Eldredge take pains to explicitly point out that PE is an expansive theory, not an exclusive one (1977).

PE sometimes is claimed to be a theory resting upon the lack of evidence rather than upon evidence. This is a curious, but false claim, since Eldredge and Gould spent a significant portion of their original work examining two separate lines of evidence (one involving pulmonate gastropods, the other one

involving Phacopsid trilobites) demonstrating the issues behind PE (1972). Similarly, discussion of actual paleontological evidence consumes a significant proportion of pages in Gould and Eldredge 1977. This also answers those who claimed that E&G said that PE was unverifiable.

PE is essentially and exclusively directed to questions at the level of speciation and processes affecting species. The basis of PE is the neontological theory of peripatric speciation. The criteria by which "punctuations" are recognized by Gould and Eldredge involve temporal issues and geographic issues. PE is not expected to be as useful at lower or higher levels of change.

PE is by no means either synonymous with "saltationism", nor did Gould's essay on Richard Goldschmidt "link" PE with Goldschmidt's "hopeful monster" conjecture. Gould wrote an article that has caused much confusion. "Return of the hopeful monsters" sought to point out that a hatchet job had been done on some of the concepts that Richard Goldschmidt had formulated. The discussion of systemic mutations as mutations which affect rate or timing of development has caused many people to assume that Gould was somehow linking PE to this concept. A close reading of the article shows this to not be the case.

Gould and Eldredge did not specify any particular genetic mechanism for PE. PE does not require large scale mutations.

PE is not a saltational theory of evolution. The emphasis upon applying consequences of peripatric speciation to paleontology shows this critique to be unfounded. PE is no more saltational than peripatric speciation is in study of modern organisms.

7. References

Dawkins, Richard. 1986. *The Blind Watchmaker*. New York, New York: W.W. Norton Co.

Eldredge, N., & Gould, S. J. 1972. Punctuated equilibria: an alternative to phyletic gradualism. In: *Models In Paleobiology* (Ed. by T. J. M. Schopf).

Gould, S. J., & Eldredge, N. 1977. Punctuated equilibria: the tempo and mode of evolution reconsidered. *Paleobiology*, 3, 115-151.

Gould, S. J. 1980. Return of the Hopeful Monster. In: *The Panda's Thumb*. New York, New York: W.W. Norton Co. pp. 186-193.

8. Acknowledgements

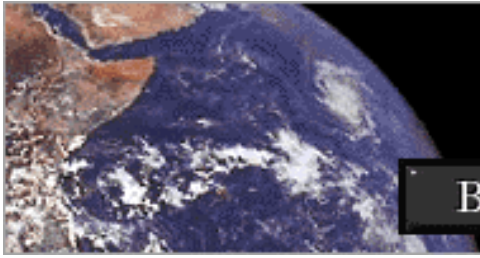
Chris Colby provided a cogent and detailed critique of the first draft, pointing out the "gene flow vs. homeostasis" argument in E&G 1972 among many other points. Many of his points remain to be incorporated, which says more about the extent of my free time rather than the importance of those points.

Chris Nedin suggested several changes incorporated here, including expansion of the discussion of Darwin in relation to phyletic gradualism. I still need to check out *The Beak of the Finch* for examples of (relatively) rapid evolutionary change.

I thank the Oxford Text Archive for providing a free e-text of *Origin of Species*, first edition. This makes looking up interesting passages much easier than in the paper versions.

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Some More Observed Speciation Events

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By Chris Stassen

Here is a short list of referenced speciation events. I picked four relatively well-known examples, from about a dozen that I had documented in materials that I have around my home. These are all common knowledge, and by no means do they encompass all or most of the available examples.

Example one:

Two strains of *Drosophila paulistorum* developed hybrid sterility of male offspring between 1958 and 1963. Artificial selection induced strong intra-strain mating preferences.

(Test for speciation: sterile offspring and lack of interbreeding affinity.)

Dobzhansky, Th., and O. Pavlovsky, 1971. "An experimentally created incipient species of *Drosophila*", *Nature* 23:289-292.

Example two:

Evidence that a species of fireweed formed by doubling of the chromosome count, from the original stock. (Note that polyploids are generally considered to be a separate "race" of the same species as the original stock, but they do meet the criteria which you suggested.)

(Test for speciation: cannot produce offspring with the original stock.)

Mosquin, T., 1967. "Evidence for autopolyploidy in *Epilobium angustifolium* (Onagraceae)", *Evolution* 21:713-719

Example three:

Rapid speciation of the Faeroe Island house mouse, which occurred in less than 250 years after man brought the creature to the island.

(Test for speciation in this case is based on morphology. It is unlikely that forced breeding experiments have been performed with the parent stock.)

Stanley, S., 1979. *Macroevolution: Pattern and Process*, San Francisco, W.H. Freeman and Company. p. 41

Example four:

Formation of five new species of cichlid fishes which formed since they were isolated less than 4000 years ago from the parent stock, Lake Nagubago.

(Test for speciation in this case is by morphology and lack of natural interbreeding. These fish have complex mating rituals and different coloration. While it might be possible that different species are inter-fertile, they cannot be convinced to mate.)

Mayr, E., 1970. *Populations, Species, and Evolution*, Massachusetts, Harvard University Press. p. 348

By James Meritt

Someone writes:

I have a friend who says since we have never seen a species actually split into two different species during recorded history that he has trouble believing in the theory of evolution. Is this bogus and have humans seen animals bred into different species? (The various highly bred english dogs come to mind but I suppose this would be easier to find in vegetation. Corn, wheat strains? Donkeys and mules?)

This is bogus. We've seen it happen naturally *without* our tampering with the process. From the FAQ:

"Three species of wildflowers called goatsbeards were introduced to the United States from Europe shortly after the turn of the century. Within a few decades their populations expanded and began to encounter one another in the American West. Whenever mixed populations occurred, the species interbred (hybridizing) producing sterile hybrid offspring. Suddenly, in the late forties two new species of goatsbeard appeared near Pullman, Washington. Although the new species were similar in appearance to the hybrids, they produced fertile offspring. The evolutionary process had created a separate species that could reproduce but not mate with the goatsbeard plants from which it had evolved."

The article is on page 22 of the February, 1989 issue of *Scientific American*. It's called "A Breed Apart." It tells about studies conducted on a fruit fly, *Rhagoletis pomonella*, that is a parasite of the hawthorn tree and its fruit, which is commonly called the thorn apple. About 150 years ago, some of these flies began infesting apple trees, as well. The flies feed and breed on either apples or thorn apples, but not both. There's enough evidence to convince the scientific investigators that they're witnessing speciation in action. Note that some of the investigators set out to prove that speciation was not happening; the evidence convinced them otherwise.

By Anneliese Lilje

Just a smattering of a *huge* database of articles (1991 only):

1. Bullini, L and Nascetti, G, 1991, Speciation by Hybridization in phasmids and other insects, *Canadian Journal of Zoology*, Volume 68(8), pages 1747-1760.
2. Ramadevon, S and Deaken, M.A.B., 1991, The Gibbons speciation mechanism, *Journal of Theoretical Biology*, Volume 145(4) pages 447-456.
3. Sharman, G.B., Close, R.L, Maynes, G.M., 1991, Chromosome evolution, phylogeny, and speciation of rock wallabies, *Australian Journal of Zoology*, Volume 37(2-4), pages 351-363.
4. Werth, C. R., and Windham, M.D., 1991, A model for divergent, allopatric, speciation of polyploid pteridophytes resulting from silencing of duplicate- gene expression, *AM-Natural*, Volume 137(4):515-526.
5. Spooner, D.M., Sytsma, K.J., Smith, J., A Molecular reexamination of diploid hybrid speciation of *Solanum raphanifolium*, *Evolution*, Volume 45, Number 3, pages 757-764.
6. Arnold, M.L., Buckner, C.M., Robinson, J.J., 1991, Pollen-mediated introgression and hybrid speciation in Louisiana Irises, P-NAS-US,

7. Nevo, E., 1991, Evolutionary Theory and process of active speciation and adaptive radiation in subterranean mole rats, *spalax-ehrenbergi* superspecies, in Israel, *Evolutionary Biology*, Volume 25, pages 1-125.

... on and on to about #50 if you like...

There are about 100 each for every year before 1991 to 1987 in my database.

By L. Drew Davis

A List of Speciation References

- Weiberg, James R.. Starczak, Victoria R.. Jorg, Daniele. Evidence for rapid speciation following a founder event in the laboratory. *Evolution*. V46. P1214(7) August, 1992.
- Kluger, Jeffrey. Go fish. (rapid fish speciation in African lakes). *Discover*. V13. P18(1) March, 1992.
- Hauffe, Heidi C.. Searle, Jeremy B.. A disappearing speciation event? (response to J.A. Coyne, *Nature*, vol. 355, p. 511, 1992). *Nature*. V357. P26(1) May 7, 1992.
Abstract:

Analysis of contact between two chromosomal races of house mice in northern Italy show that natural selection will produce alleles that bar interracial matings if the resulting offspring are unfit hybrids. This is an important exception to the general rule that intermixing races will not tend to become separate species because the constant sharing of genes minimizes the genetic diversity requisite for speciation.

- Barrowclough, George F.. Speciation and Geographic Variation in Black-tailed Gnatcatchers. (book reviews) *The Condor*. V94. P555(2) May, 1992.
- Rabe, Eric W.. Haufler, Christopher H.. Incipient polyploid speciation in the maidenhair fern (*Adiantum pedatum*; *Adiantaceae*)? *The American Journal of Botany*. V79. P701(7) June, 1992.
- Nores, Manuel. Bird speciation in subtropical South America in relation to forest expansion and retraction. *The Auk*. V109. P346(12) April, 1992.
Abstract:

The climatic and geographic history of the Pleistocene and Holocene periods modified the distribution of the bird population in the South American forests. Forest birds are found dispersed in the Yungas and Parane areas with only minimal infiltration of the Chaco woodland, indicating an atmospheric change during the interglacial periods. In the Chaco lowlands, the interactions between non-forest birds reveal the existence of presence of a forest belt along the Bermejo and Pilcomayo rivers.

- Kondrashov, Alexey S.. Jablonka, Eva. Lamb, Marion J.. Species and speciation. (response to J.A. Coyne, *Nature*, vol. 355, p. 511, 1992). *Nature*. V356. P752(1) April 30, 1992.
Abstract:

J.A. Coyne wrongly asserted that neodarwinism includes allopatric evolution but not sympatric evolution. Allopatric evolution occurs among geographically isolated populations, whereas sympatric evolution occurs within one species' entire population. Both are neodarwinian since each results from natural selection of genetic variation. Also, Coyne failed to recognize that the molecular models used to illustrate how genetic changes bring on speciation are most useful when researchers acknowledge that both inherited epigenetic and genetic changes affect speciation.

- Spooner, David M.. Sytsma, Kenneth J.. Smith, James F.. A molecular reexamination of diploid hybrid speciation of *Solanum raphanifolium*. *Evolution*. V45. P757(8) May, 1991.

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- Miller, Julie Ann. Pathogens and speciation. (Research Update). *BioScience*. V40. P714(1) Nov, 1990.
- Barton, N.H. Hewitt, G.M. Adaptation, speciation and hybrid zones; many species are divided into a mosaic of genetically distinct populations, separated by narrow zones of hybridization. Studies of hybrid zones allow us to quantify the genetic differences responsible for speciation, to measure the diffusion of genes between diverging taxa, and to understand the spread of alternative adaptations. (includes related information) *Nature*. V341. P497(7) Oct 12, 1989.
- Wright, Karen. A breed apart; finicky flies lend credence to a theory of speciation. *Scientific American*. V260. P22(2) Feb, 1989.
- Coyne, Jerry A. Orr, H. Allen. Patterns of speciation in *Drosophila*. *Evolution*. V43. P362(20) March, 1989.
- Feder, Jeffrey L. Bush, Guy L. A field test of differential host-plant usage between two sibling species of *Rhagoletis pomonella* fruit flies (Diptera: Tephritidae) and its consequences for sympatric models of speciation. *Evolution*. V43. P1813(7) Dec, 1989.
- Soltis, Douglas E. Soltis, Pamela S. Allopolyploid speciation in *Tragopogon*: insights from chloroplast DNA. *The American Journal of Botany*. V76. P1119(6) August, 1989.
- Coyne, J.A. Barton, N.H. What do we know about speciation?. *Nature*. V331. P485(2) Feb 11, 1988.
- Barton, N.H. Jones, J.S. Mallet, J. No barriers to speciation. (morphological evolution). *Nature*. V336. P13(2) Nov 3, 1988.
- Kaneshiro, Kenneth Y. Speciation in the Hawaiian *drosophila*: sexual selection appears to play an important role. *BioScience*. V38. P258(6) April, 1988.

From talk.origins

A talk.origins participant writes:

1) Speciation occurred in a strain of *Drosophila paulistorum* sometime between 1958 and 1963 in Theodosius Dobzhansky's lab. He wrote this up in:

Dobzhansky, T. 1973. Species of *Drosophila*: New Excitement in an Old Field. *Science* 177:664-669

2) A naturally occurring speciation of a plant species, *Stephanomeria malheurenensis*, was observed in Burns County, Oregon. The citing is:

Gottlieb, L. D. 1973. Genetic differentiation, sympatric speciation, and the origin of a diploid species of *Stephanomeria*. *American Journal of Botany* 60(6):545-553

3) In the 1940's a fertile species was produced through chromosome doubling (allopolyploidy) in a hybrid of two primrose species. The new species was *Primula kewensis*. The story is recounted in:

Stebbins, G. L. 1950. *Variation and Evolution in Plants*. Columbia University Press. New York

4) Finally, two workers produced reproductive isolation between two strains of fruit flies in a lab setting within 25 generations. I don't have the paper handy, so I can't give the species. The partial citing of the paper is:

Rice and Salt 1988. *American Naturalist* 131:911-

Dobzhansky got a subpopulation of *D. paulistorum* to speciate in his lab. The reference is:

Dobzhansky and Pavlovsky, 1957 An experimentally created incipient species of *Drosophila*, *Nature* 23: 289-292

See also:

Weinberg, et. al, 1992 Evidence for rapid speciation following a founder event in the laboratory, *Evolution* 46: 1214. (This isn't a full paper, just a note -- it describes what is probably speciation of a type of polychaete worm.)

Another talk.origins participant writes:

There are two distinct strains of *Rhagoletis pomonella*, the apple maggot fly. One infests the apple, the other the hawthorn. They have different breeding times--as the fruits flower at different times--and so they do not interbreed in the real world. I do not know if they could interbreed in the laboratory. Since the fly is not found in Europe, and the apple is an import from Europe, the only presumption is that the apple strain is a speciation off the original hawthorn strain.

Yet another talk.origins participant writes:

I do not currently have references to cite for the speciation of fish, however I have a couple for the case of rats. Genus *Rattus* currently consists of 137 species [1,2] and is known to have originally developed in Indonesia and Malaysia during and prior to the Middle Ages [3]. ([1] is the only source I have consulted.)

[1] T. Yosida. *Cytogenetics of the Black Rat*. University Park Press, Baltimore, 1980.

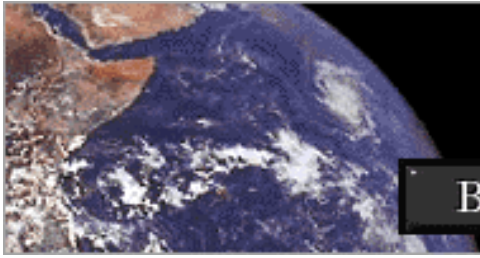
[2] D. Morris. *The Mammals*. Hodder and Stoughton, London, 1965.

[3] G. H. H. Tate. "Some Muridae of the Indo-Australian region," *Bull. Amer. Museum Nat. Hist.* 72: 501-728, 1963.

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Are Mutations Harmful?

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[Last update: May 23, 1999]

Outline

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People often ask questions such as "Doesn't evolution depend on mutations, and aren't most mutations harmful?" and "Are there favorable mutations?". In this FAQ we try to answer these questions. Briefly:

- Mutations happen.
- They happen with great regularity.
- Almost all mutations are neutral.
- Of the remainder, benefit/harm depends on circumstances

Biology is complicated; the jargon of the biological sciences is formidable. In this FAQ I have tried to answer common questions in simple language for the lay reader. At the same time I have tried also to provide material in greater depth for the reader who wants scientific substance.

Q: Doesn't evolution depend on mutations and aren't most mutations harmful?

A: No. Most mutations are neither harmful nor helpful.

That's the short answer. The long answer is that mutations can be neutral (neither helpful nor harmful), strictly harmful, strictly helpful, or (and this is important) whether they are harmful or helpful depends on the environment. Most mutations are either neutral or their effect depends on the environment. Let's look at an example of a mutation which may be harmful or helpful, depending upon circumstances.

English peppered moths come in two varieties, light and dark. Before the industrial revolution dark moths were very rare. During the worst years of the industrial revolution when the air was very sooty dark moths became quite common. In recent years, since the major efforts to improve air quality, the light moths are replacing the dark moths.

This is a classic example of natural selection; the variations in a species which are better suited to the environment survive and reproduce more effectively than those which do not.

It can be very tricky to determine why some variations better fit their environments than others. The peppered moth is a good example. In a famous paper Kettlewell proposed the following explanation:

Birds eat the kind of moth they can see the best.

In England before the Industrial Revolution trees are often covered with light colored lichens. As a result light moths were favored because they were hard to see on the bark of trees whereas the dark moths were easy to see; birds ate the dark moths. During the worst years of the Industrial Revolution the air was very sooty so tree bark was dark because of soot. Dark moths were hard to see whereas the light moths were easy to see; birds ate the light moths. As a result the dark moths became common and the light moths became rare.

Kettlewell's explanation (which makes for an appealing story) has not stood the test of time. Peppered moths seldom rest on exposed areas of the trunks of trees. Moreover the distribution of dark moths might not be well correlated with tree color except in the areas which Kettlewell studied. Some more recent studies indicate that peppered moth melanism is very well correlated with the amount of SO₂ (sulfur dioxide) in the air. [5]

None-the-less, before the Industrial Revolution a mutation which changed light moths into dark moths was an unfavorable (harmful) mutation whereas during the dark years it was a favorable (helpful) mutation.

To see why most mutations are neither harmful nor helpful it helps to know a bit about what mutations actually are. A mutation is a change in the genetic material that controls heredity. The genetic material is contained in chromosomes. Plants and animals have two copies of each chromosome whereas bacteria only have one copy. Organisms which have two copies of each chromosome are called diploids. Those which only have one copy of each chromosome are called haploids.

Chromosomes are divided into genes, each gene being a stretch of DNA, i.e., a sequence of nucleotides (A,G,C,T for short). The location of a gene is called a locus. (*The position of a nucleotide within a gene is called a site. Don't mix up locus and site.*) At a given locus you may find that the DNA sequence is different from one critter to another in some small way. These are usually known as different alleles although sometimes they are confusingly called different genes. Let's call them different alleles so that we don't get confused; besides that's the standard term.

If we look at populations of animals and plants we find that there are multiple alleles at 10-20% of the genes. In other words if we look at a given locus in all the members of a population about 10-20% of the time we will find more than one sequence at that locus. There can be more than two alleles within a population for a given gene locus.

Our peppered moths have a gene which controls whether the moth is light or dark.[1] Since moths are diploids each moth has two copies of the gene. If both copies of a given gene are the same allele then the moth is said to be homozygous for that gene. If the two copies are different alleles then the moth is said to be heterozygous for that gene. If both alleles are the same then the moth will be light or dark, depending on which allele it has. People sometimes say "which gene it has" but that is confusing because it mixes up genes and alleles. If a moth has two different alleles (i.e. if it is heterozygous) then the hue depends on which allele is dominant. In the case of peppered moths dark is dominant, i.e., a heterozygote will be dark rather than light.

Now let's talk about how a gene might change, i.e., how one allele might change into another. There are a number of ways this might happen. We might get a point mutation, one nucleotide being replaced by another. A section might get swapped end for end. A section might be snipped out. A section might be inserted. Or the entire gene might be duplicated. See the next [section](#) for a fuller description of the different kinds of mutations and their effects.

What is the consequence when one of these things happens? Most of the time the change either has no perceptible effect at all, or it is fatal. Coding genes map into proteins using the genetic code. The genetic code is redundant (the technical term is degenerate), i.e., different triplets of nucleotides will produce the same amino acid. Because of the redundancy a point mutation may have no effect at all on the protein being coded for; these are known as silent mutations. If the sequence is altered by snipping or swapping the result is likely to be fatal because the coding sequence [the readout in terms of triplets] will be messed up. However this isn't always true because there are processes that snip and insert sections of DNA into genes in a way that doesn't mess up the coding sequence.

Suppose we have one of these mutations that isn't fatal but isn't silent. What happens as a result is that we get a slightly different protein. Most of the time the new protein works very much the same as the old protein - it catalyzes the same reactions. Sometimes it's functional capability changes; it now catalyzes a different reaction. When this happens there may be another protein which also handled the original task; in this case we've added a capability. If there wasn't we lost the original capability and replaced it by a new one. Changes in enzymes (proteins that catalyze reactions) are seldom an all or nothing proposition.[3]

Gene duplication is important because it is a way to get new genes. Once a gene has been duplicated one copy can change while the other remains the same.

Genes vary a great deal with respect to how much they can be changed without the changes harming the organism. Some genes, such as those that encode the basic metabolism and the components of the replication, transcription, and translation machinery, are hard to change without harm. We see very little

variation in them from one organism to another. Such genes are said to be conserved.

"What is the net result," you may ask. Some mutations are fatal or very bad. These mutations get eliminated immediately. Some are silent and don't count. Sometimes a mutation is definitely advantageous; this is rare but it does happen. Almost all mutations which aren't silent and which aren't eliminated immediately are neither completely advantageous nor deleterious. The mutation produces a slightly different protein, and the cell and the living organism work slightly differently. Whether the mutation is helpful or harmful depends on the environment; it could be either.

If you think about it, life has to work this way - mutations (changes in the genetic material) are happening all the time. The average human being has about 50-100 mutations, of which about 3 matter, i.e., they actually change a protein. If the typical mutation were deleterious life would go extinct in short order. [\[4\]](#)

Although most mutations are neither uniformly helpful nor harmful they may be either helpful or harmful in a particular environment. Environments are always changing, and each member of a population lives in a slightly different environment from the other members. Some organisms live; some do not. Some reproduce; some do not. The alleles of those that live and reproduce get passed on. Any difference in the organism which is favorable with respect to the environment will prosper.

It is important to realize that mutations do not occur in response to the environment. They simply happen. Quite often a mutation occurs within a population and then disappears because the organism had no offspring or didn't happen to pass the mutation on to its offspring; this can happen even if the mutation is beneficial. Sometimes a mutation will get established within a population by chance even though it doesn't offer an advantage; this is known as genetic drift. [\[8\]](#)

It is also important to realize mutations do not happen just once. They happen rarely but they keep happening over and over again within a species. In effect a mutation gets more than one bite at the apple; if it doesn't catch on the first time it appears it gets another chance. [\[9\]](#)

Q: Are there favorable mutations?

A: There are, but it can be hard to tell.

For a number of reasons it is not simple to give examples of favorable mutations. First of all, as we have seen, traits [\[6\]](#) may be favorable or unfavorable, depending upon the environment. Secondly it is not usually known to what extent a trait is genetically fixed and to what extent it reflects a reaction to the environment. Thirdly we don't usually know what genes effect which traits. Moreover a mutation may be favorable in the sense that it permits survival in an unfavorable environment and yet be unfavorable in a better environment.

However there are a number of good examples:

1. *Antibiotic resistance in bacteria*

In modern times antibiotics, drugs that target specific features of bacteria, have become very popular. Bacteria evolve very quickly so it is not surprising that they have evolved resistance to antibiotics. As a general thing this involves changing the features that antibiotics target.

Commonly, but not always, these mutations decrease the fitness of the bacteria, i.e., in environments where there are not antibiotics present, they don't reproduce as quickly as bacteria without the mutation. This is not always true; some of these mutations do not involve any loss of fitness. What is more, there are often secondary mutations that restore fitness.

Bacteria are easy to study. This is an advantage in evolutionary studies because we can see evolution happening in the laboratory. There is a standard experiment in which the experimenter begins with a single bacterium and lets it reproduce in a controlled environment. Since bacteria reproduce asexually all of its descendents are clones. Since reproduction is not perfect mutations happen. The experimenter can set the environment so that mutations for a particular attribute are selected. The experimenter knows both that the mutation was not present originally and, hence, when it occurred.

In the wild it is usually impossible to determine when a mutation occurred. Usually all we know (and often we do not even know that) is the current distribution of particular traits.

The situation with insects and pesticides is similar to that of bacteria and antibiotics. Pesticides are widely used to kill insects. In turn the insects quickly evolve in ways to become immune to the pesticides.

2. *Bacteria that eat nylon*

Well, no, they don't actually eat nylon; they eat short molecules (nylon oligomers) found in the waste waters of plants that produce nylon. They metabolize short nylon oligomers, breaking the nylon linkages with a couple of related enzymes. Since the bonds involved aren't found in natural products, the enzymes must have arisen since the time nylon was invented (around the 1940s). It would appear this happened by new mutations in that time period.

These enzymes which break down the nylon oligomers appear to have arisen by frameshift mutation from some other gene which codes for a functionally unrelated enzyme. This adaptation has been experimentally duplicated. In the experiments, non-nylon-metabolizing strains of *Pseudomonas* were grown in media with nylon oligomers available as the primary food source. Within a relatively small number of generations, they developed these enzyme activities. This would appear to be an example of documented occurrence of beneficial mutations in the lab.

3. *Sickle cell resistance to malaria*

The sickle cell allele causes the normally round blood cell to have a sickle shape. The effect of this allele depends on whether a person has one or two copies of the allele. It is generally fatal if a person has two copies. If they have one they have sickle shaped blood cells.

In general this is an undesirable mutation because the sickle cells are less efficient than normal cells. In areas where malaria is prevalent it turns out to be favorable because people with sickle shaped blood cells are less likely to get malaria from mosquitoes.

This is an example where a mutation decreases the normal efficiency of the body (its fitness in one sense) but none-the-less provides a relative advantage.

4. *Lactose tolerance*

Lactose intolerance in adult mammals has a clear evolutionary explanation; the onset of lactose intolerance makes it easy to wean the young. Human beings, however, have taken up the habit of eating milk products. This is not universal; it is something that originated in cultures that kept cattle and goats. In these cultures lactose tolerance had a strong selective value. In the modern world there is a strong correlation between lactose tolerance and having ancestors who lived in cultures that exploited milk as a food.

It should be understood that it was a matter of chance that the lactose tolerance mutation appeared in a group where it was advantageous. It might have been established first by genetic drift within a group which then discovered that they could use milk. [9]

5. *Resistance to atherosclerosis*

Atherosclerosis is principally a disease of the modern age, one produced by modern diets and modern life-styles. There is a community in Italy near Milan (see Appendices II and III for biological details) whose residents don't get atherosclerosis because of a fortunate mutation in one of their forebearers. This mutation is particularly interesting because the person who had the original mutation has been identified.

Note that this is a mutation that is favorable in modern times because (a) people live longer and (b) people have diets and life-styles that are not like those of our ancestors. In prehistoric times this would not have been a favorable mutation. Even today we cannot be certain that this mutation is reproductively favorable, i.e., that people with this mutation will have more than the average number of descendents. It is clear, however, that the mutation is personally advantageous to the individuals having it.

6. *Immunity to HIV*

HIV infects a number of cell types including T-lymphocytes, macrophages, dendritic cells and neurons. AIDS occurs when lymphocytes, particularly CD4+ T cells are killed off, leaving the patient unable to fight off opportunistic infections. The HIV virus has to attach to molecules that are expressed on the surface of the T-cells. One of these molecules is called CD4 (or CD4 receptor); another is C-C chemokine receptor 5, known variously as CCR5, CCCKR5 and CKR5. Some people carry a mutant allele of the CCR5 gene that results in lack of expression of this protein on the surface of T-cells. Homozygous individuals are resistant to HIV infection and AIDS. The frequency of the mutant allele is quite high in some populations that have never been exposed to AIDS so it seems likely that there was prior selection for this allele. (See [Appendix IV](#))

For a description of the recent literature consult the [OMIM site](#) for CCR5.

Types of mutations and their effects

Mutations are changes in the genome (genetic constitution). There are quite a number of ways in which mutations can happen. They also differ in the way that they impact evolution.

Mutations which occur when the genome is copied during reproduction are known as vertical transfer mutations. They are called vertical transfer mutations because they are transferred from ancestor to descendent along vertical lines of descent. In the original work on population genetics it was assumed that all mutations were vertical transfer mutations.

Horizontal transfer mutations occur when DNA is moved from one organism to another. Horizontal transfer can be a major source of evolutionary novelty. It is important because new genes can be propagated much more rapidly by horizontal transfer than by vertical transfer. If evolution is depicted by the tree, vertical genetic movement is the transmission of genes down branches; horizontal genetic movement is the transmission of genes between the branches.

Intra-organism transfer mutations occur when genes or parts of genes move around within an organism.

Strictly speaking, hybrids (mating across species) are not mutants. In many groups of species, particularly among plants, genes are transferred from one species to another via hybrids.

Types of mutations:

1. Point mutations

The most common type of copying error is the point mutation. In this form of mutation the nucleotide at a site is replaced by a different nucleotide. When people talk about mutation rates they are usually talking about rates of point mutations.

Effects of point mutations: Point mutations in junk DNA are common but have no effect. Sometimes point mutations in regulatory regions have no effect and sometimes they alter the expression of some genes.

2. Additions and deletions

During copying a segment of DNA may be deleted or a new segment may be inserted. Typically this happens as a result of chromosome breakage or realignment. (See below.) Additions and deletions can also be produced by certain types of horizontal transfer.

Effects of additions and deletions: If the length of the new or deleted segment is not a multiple of three the translation will be garbled after the point at which the insertion/deletion occurred because the frame reading is now misaligned. This is known as a frameshift mutation. In some genes there are segments that may be duplicated as a block. This is known as tandem duplication.

3. Chromosomal duplication

Sometimes one or more chromosomes are duplicated during reproduction; the offspring get extra copies of those chromosomes.

Effects of chromosomal duplication: Duplicating only one chromosome is generally disadvantageous; an example in human beings is Down's syndrome. Having multiple copies of all of the chromosomes is known as polyploidy. Polyploidy is rare in fungi and animals (although it does occur) and is common in plants. It has been estimated that 20-50% of all plant species arise as the result of polyploidy.

Gene duplication is very common; it is important because it provides a way to evolve new capabilities while retaining the old capabilities. All intermediate stages can be found in nature, from a single gene with alternate alleles to nearly identical duplicated genes with slightly different functional alleles to gene families of evolutionarily related genes with different functionalities.

4. Chromosomal breakage and realignment

During reproduction a chromosome may break into two pieces or two chromosomes may be joined together. A section may be moved from one part of the chromosome to another or may be flipped in orientation (inverted). This is the mechanism by which deletions, duplications and transpositions may occur.

Effects of chromosomal breakage and realignment: Quite often these types of changes do not affect the viability of the organism (the genes are still there; they're just in different places) but, in sexually reproducing species, they may make it less likely for the organism to produce viable, fertile offspring.

5. Retroviruses

Certain viruses have the ability to insert a copy of themselves into the genome of a host. The chemical that make this possible (reverse transcriptase) is widely used in genetic engineering.

Effects of retroviruses: Usually this is a way for the virus to get the host to do the work of reproducing the virus. Sometimes, however, the inserted gene mutates and becomes a permanent part of the host organism's genome. Depending on the position of the viral DNA in the host genome, genes may be disrupted or their expression altered. When insertions occur in the germline of multicellular organisms, they can be passed on vertically.

6. Plasmids

Plasmids are little pieces of circular DNA that are passed from bacterium to bacterium. Plasmids can be transferred across species lines.

Effects of plasmid transfer: Plasmid transfer is an important way of spreading useful genes such as those which confer resistance to antibiotics. Plasmid transfer is an example of horizontal transfer.

7. Bacterial DNA exchange

Bacteria can exchange DNA directly. They often do this in response to environmental stress.

Effects of bacterial DNA exchange: Exchange is often fatal to one or both of the bacteria involved. Sometimes, however, one or both of the partners acquires genes which are essential for the current environment.

8. Higher level transfer

Some parasites can pick up genetic material from one organism and carry it to the next. This has been observed in fruit flies in the wild.

Effects of higher level transfer: When this happens novel alleles can spread much more rapidly through a species than they would for ordinary gene flow.

9. Symbiotic transfer

When two organisms exist in a close symbiotic relationship one may "steal" genes from the other. The most notable example of this are mitochondria. In most organisms with mitochondria most of the original mitochondrial genes have moved from the mitochondria to the nuclear genome.

Effects of symbiotic transfer: A major effect is that the symbiotic relationship changes from being optional to be obligatory.

10. Transposons

Transposons are genes that can move from one place in the genome to another.

Effects of transposons: Depending on the position of insertion, transposons can disrupt or alter the expression of host genes. In some species most mutations due to transposon insertion. For example, in *Drosophila*, 50-85% of mutations are due to transposon insertions.

Mutation Studies

by Joe Boxhorn

The material in this section is by Joe Boxhorn. It goes into greater depth than the material in the rest of the FAQ. It gives a good picture of how experiments are actually run. It also gives some examples that aren't usually seen in the popular literature.

Experimental work with bacteria, eukaryotic micro-organisms and very small animals can tell us much about the occurrence and properties of mutations, including beneficial mutations. Over the last fifty years or so beneficial mutations have been observed to occur in a number of studies.

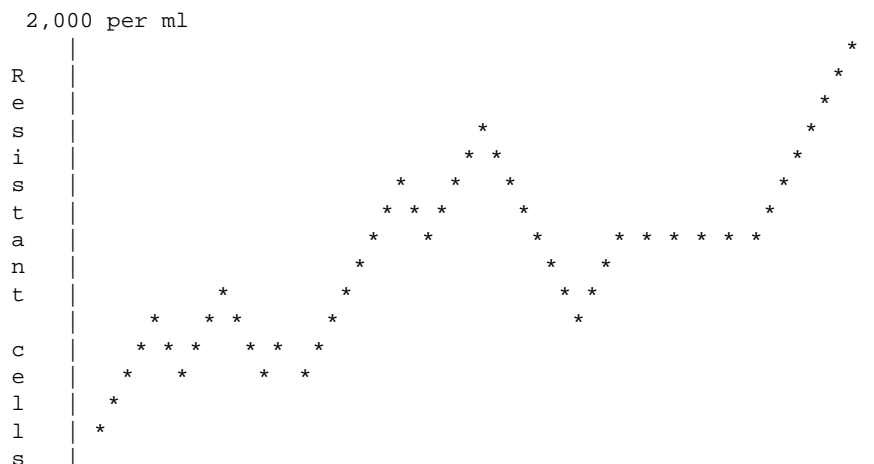
Most of these experiments were done in a continuous culture system called a chemostat. Chemostats have been used for the last fifty years in the study of the physiology, population biology and ecology of bacteria and a variety of other small organisms. They are also used widely in the commercial production of microbe produced substances. A chemostat consists of a bottle in which the organisms grow. Growth medium (i.e. food) is continuously pumped into the bottle and waste products, residual medium and organisms flow out. The contents of the bottle are well mixed so that each critter in the chemostat has an equal chance of getting at each bit of food. Factors that affect the growth of the organisms such as temperature are controlled, sometimes quite rigourously. Several variations of chemostats have been developed. They will be described as they become relevant.

Chemostats have several properties that make them useful for biological research. Over time, the organisms in the system reach a steady state in which organism growth equals the amount of organism flowing out of the bottle. At this steady state the concentration of organisms, measured as biomass, remains quite stable as does the concentration of residual (unused) nutrient. Numbers can change somewhat due to changes in the size of individuals. It is important to note that when the system is in steady state, the critters are growing exponentially with their growth rate being the dilution rate (in flow of medium/bottle volume) of the system. The average time an organism remains in the chemostat is the reciprocal of the dilution rate. These organisms are also in a steady state physiologically. The population densities of organisms grown in these continuous culture systems can be quite high. For a fast growing bacterium like *E. coli* densities of 3×10^8 per ml are readily attainable. Similarly small eukaryotic algae such as *Chlorella vulgaris* can be easily be grown at densities of 3×10^7 per ml. Densities on the order of $10^5 - 10^6$ per ml are attainable for many larger unicellular and colonial eukaryotes. The implications of this for the study of mutations are important. Assuming reasonable mutation rates and genome sizes it is virtually certain that a culture of this sort that has been run at steady state for any length of time will contain some mutant individuals. This holds even when the culture is innoculated with a strain derived from one individual in an obligately asexual species. If, for example, we assume the following characteristics for an *E. coli* chemostat containing identical cells:

Culture volume	500 ml
Dilution rate	1.0 per day
Genome size	5,000 genes
Population density	3×10^8 cells per ml
Mutation rate	10^{-8} mutations per gene per individual per generation

We should see 7.5×10^6 mutant genes produced in one day. I would note that *E. coli* chemostats are generally run at dilution rates far faster than this. Another property of this type of system is that when organisms in a chemostat vary in their growth rates the proportion of the faster growing forms in the population tends to increase at the expense of the slower growing ones. Finally, the mathematical models describing growth of bacteria and unicellular algae in these systems are reasonably well understood and work fairly well when compared to data (see, for example, Herbert et al. 1956, Kubitschek 1970, Pirt 1975). These models do not do as well predicting the dynamics when larger organisms with more complicated life cycles are grown in chemostats.

A reinterpretation by Kubitschek (1974) of work by Novick and Szilard (1956) suggests that the argument above was reasonable. In this study resistance to a bacterial virus was used as a marker to follow the appearance of some mutations in a chemostat culture. Novick and Szilard grew *E. coli* in a chemostat at a steady-state density of about 3×10^8 cells per ml. Periodically they assayed cells sampled from the chemostat for resistance to infection by bacteriophage T5 and calculated the density of T5 resistant cells in the culture. At no time was phage T5 present in the chemostat nor had the cells in the chemostat been exposed to phage T5. They found that there was always a fraction of cells in the culture that was resistant to T5. The density of resistant cells fluctuated between 10^2 and 10^3 per ml. It followed a pattern like the one drawn below:



(Note: this is not the actual graph. It shows the pattern of changes that the system went through. For the actual graph see Kubitschek 1974.) The increases and decreases reflect the occurrence of mutations within strains in the chemostat. The initial increase in the frequency of resistant cells occurs because a mutation occurs within a T5 resistant strain that makes it (and its descendents) the fastest growing cells in the culture. As long as this strain remains the fastest growing one its representation in the population will increase. Eventually different favorable mutation occurs in a cell that is sensitive to T5 that makes it (and its descendents) the fastest growing cells in the culture. This causes the frequency of T5 resistance to decline. Later a different mutation occurs in a T5 resistant strain that makes it the fastest growing strain. Its frequency increases, and so on.

It is important to note here that in this environment sensitivity and resistance to infection by T5 is a neutral trait here. Because there is no T5 in the environment, resistance does not provide an advantage. But it doesn't seem to provide much disadvantage either. If it provided a disadvantage, the resistant cells would washout of the chemostat. In this environment, it is selectively neutral. Mutations in other genes cause some cells to have a higher growth rate. It is just a matter of whether these mutations occur first in resistant or sensitive cells that determines whether the frequency of T5 resistant cells increases or decreases. It's a hitchhiking effect - the T5 resistance gene just goes along for the ride with the genes causing the fluctuations.

Now in a different environment, the value of the mutation producing resistance to infection by a virus might have a totally different value. Chao et al. (1977) grew wild type *E. coli* B in a chemostat. Once the vessel reached steady state they inoculated it with bacteriophage T7. The bacteria are sensitive to infection by T7. Needless to say, T7 grew like mad on the bacteria. After a short time, though, a mutation attributable to a single gene appeared in a cell surface receptor site which gave the bacteria complete resistance to T7. This bacterial strain was designated B1. Shortly after this a mutation occurred in the virus which allowed it to infect strain B1 (strain T7.1). A second mutation occurred in B1 which made it resistant to this second virus strain as well as to the original virus strain (strain B2). All five of these critters happily coexisted in the same chemostat.

Now whether these mutations were favorable or detrimental depends on which environment the critters were put in. In an environment containing T7, *E. coli* B1 or *E. coli* B2 could survive while *E. coli* B suffered tremendous mortality. But the mutant strains paid a cost. They were not as fast at taking up nutrient as the wild type and, consequently, could not grow as quickly. In competition experiments in phage-free environments, *E. coli* B outcompeted every time. So whether a mutation conferring resistance to T7 is beneficial depends on:

1. whether there is T7 in the environment, and
2. if there isn't, whether sensitive conspecifics are present.

There has been a considerable amount of work on resistance of bacteria to bacteriophage that supports this. Some of it is reviewed in Lenski (1987).

The presence of a predator in a continuous culture system can place strong selection upon the critters being grown. When mutations appear in the prey that confer resistance to predations, they can spread through the chemostat quite rapidly -- in real time! This has been seen in many of the studies whose results are reported in the continuous culture literature.

Shikano et al. (1990) observed a major morphological change in an unidentified gram negative bacterium when it was grown in semicontinuous culture with a predator. Semicontinuous culture is a culture technique where critters are grown in a mixed flask. Periodically, a set volume of medium and organism are removed and replaced by fresh medium. This type of system reaches a pseudosteady state similar to the steady state found in a chemostat. In this study, an amotile, short (1.5 micrometer) rod-shaped bacterium was grown with the ciliate predator *Cyclidium*. Medium transfers occurred every seventh day. After 8 to 10 transfers long bacterial cells (up to 20 micrometers) appeared in cultures which had the ciliate. These cells lacked crosswalls. They coexisted with a shorter morph. After appearance of the long form, the density of ciliates in the experimental flasks declined. Feeding experiments showed that the ciliates fed preferentially on the shorter cells.

To test whether the change in the bacterium was a genetic change, Shikano et al. (1990) examined size distributions of cell in 30 colonies derived from an experimental flask. The frequency distribution of sizes of the short cells in the flasks were indistinguishable from those in the controls and the parental strain. The frequency distribution of sizes for the long cells was considerably broader. The fact that daughter colonies derived from colonies of the long cells show the same distribution of cell lengths as the long cell colonies from the experimental flasks suggests that this change in morphology reflects a genetic change.

Selection for filamentous by a phagotrophic predator seems to be common with bacteria. Pernthaler et al. (1997) reported the appearance of a filamentous form of an unidentified member of the beta-proteobacteria when the predatory flagellate *Bodo saltans* was added to a chemostat growing a mixed bacterial assemblage. Similar filaments have been seen to appear when *E. coli* is grown in a chemostat with the predatory flagellate *Poteroiochromonas malhamensis* (Gillott et al. 1993). Within about 5 days nonseptate filaments as long as 100 micrometers appeared. Many were so long that the flagellate could not completely ingest them. (Note: I have done some feeding study work with this strain. I have videotape of flagellates trying to engulf a long filament, pushing the filament through itself until the whole mess looks like a gall on a goldenrod stem and finally pushing the filament out of itself like an arrow shooting out of a bow.) *E. coli* has been known to produce filaments like this as a result of exposure to radiation or chemical agents for a long time (Deering 1958, Curry and Greenberg 1962, Hoffman and Frank 1963, Adler and Hardigree 1964). The mechanism appears to be a mutation in crosswall formation (Begg and Donachie 1985).

Nakajima and Kurihara (1994) produced a different favorable mutation in *E. coli*. They grew the bacteria in a chemostat with the predatory ciliate

Tetrahymena thermophila. Within 15 days of inoculation of the ciliates, chains of normal-sized *E. coli* cells appeared. This morphological change lasted through several platings on agar. Again, the new form provide protection against predation.

Van den Ende (1973) introduce the ciliate predator *Tetrahymena pyriformis* into a chemostat containing the bacterium *Klebsiella aerogenes* growing in steady state. During the period 140 hours to 200 hours following inoculation with the predator, the bacterium's colony morphology changed from normal mucoid appearance to a glassy appearance. This reflected a loss of the bacterium's mucoid capsule. Bacteria began to adhere to the walls of the culture vessel at this time. No wall growth was seen in the controls. The morphological change seems to be an adaptation that allows the bacteria to utilize the wall as a refuge from predation. This is supported by a change van den Ende saw in the size distribution of the ciliates. At inoculation the ciliates showed a distribution of lengths ranging from 40 - 200 micrometers. After 800 hours in the chemostat, few ciliates exceeded 60 micrometers in length. This appears to be due to starvation.

In the above cases, mutations appeared which gave resistance to predation. Mutations which confer resistance to parasites have also been seen in studies of bacteria growing in chemostats. Varon (1979) introduced the parasitic bacterium *Bdellovibrio* into a chemostat with the luminescent bacterium *Photobacterium leiognathi* growing in steady state. Within six days a new strain of the host appeared which was resistant to attack by the parasite. This mutant coexisted in the culture with a form similar to the original strain. Normally *P. leiognathi* grows as pairs of rod-shaped cells and forms translucent colonies. The mutant strain grows as chains of oval cells and forms opaque colonies. Plaque assays showed that the efficiency of plating of *Bdellovibrio* suspension on lawns of the mutant was at least 10^7 times lower than on the original strain or on the wild-type cells from the culture. Examination of mixed suspension of parasite and host using phase-contrast microscopy showed that wild type cells were attacked immediately upon mixing by *Bdellovibrio*, while mutant cells remained untouched. Batch-culture studies showed that under similar culture conditions, the mutant strain has a much lower growth rate than the wild-type bacterium.

This concludes what I'm going to discuss about prokaryotes. Several conclusions seem to emerge from these studies. First, given exponential growth and large population sizes, lots of mutations seem to occur in bacterial populations. When bacteria under these conditions are placed under strong selection, by means such as the introduction of a predator or a parasite, adaptations countering the effect of the selective agent rapidly appear and spread through the population. This could not happen unless mutations which confer these benefits were appearing. This must be the case when we consider that standard practice in microbiology is to start cultures from single colonies on agar plates - colonies which represent the descendants of a single cell. Whether the mutation is beneficial depends on the environment that the mutant is in. In the presence of the selective agent (e.g. a predator), the mutation is beneficial. In a different environment the mutation may be detrimental. A common effect of mutations conferring resistance to predators and parasites seems to be a lowering of the maximum growth rate of the mutant bacteria. In at least some cases (e.g. *E. coli* and T phages), this results from the same mutation producing resistance and reducing the ability to take up nutrients. In any case, the appearance of beneficial mutations seems to occur in continuous culture in a number of bacterial species and is probably a general phenomenon.

It is my opinion that these conclusions also apply to eukaryotes. I'll discuss a few examples from work here in the Counter Culture Lab to support this assertion.

Chlorella vulgaris is a common unicellular green alga that is used as a "lab rat" in labs throughout the world. We've grown the same strain of it for thousands of generations on agar and in liquid culture without it losing its unicellular morphology. Dozens to hundreds of labs have done this. Steady-state unicellular *C. vulgaris* cultures were inoculated with the predator *Ochromonas vellesiaca*, a phagotrophic flagellate. Within less than 100 generations a multicellular form of the *Chlorella* became dominant in the culture. (Boraas 1983b, Boraas et al. 1998). The alga first formed globose clusters of tens to hundreds of cells. After 10-20 generations in the presence of the flagellate, eight-celled colonies predominated. These colonies retained the eight-celled morphology indefinitely in continuous culture and when plated onto agar. The basis of the change appears to be a change in the cell wall. Cell division in normal *Chlorella* occurs within the cell wall of the maternal cell. The cell undergoes 1-4 divisions to form 2-16 daughter cell. This is followed by a split in the mother cell wall and dispersal of the neonatal cells. In a culture, empty mother cell walls are interspersed with whole cells at a ratio of about 1:4. Empty mother cell walls are not found in cultures of the multicellular form. The colonies are enclosed in a "membrane" which appears to be modified cell wall material.

As was seen in the bacterial cases, this mutation provided *Chlorella* with resistance to predation at the cost of growth rate. Neonatal colonies are barely small enough for *Ochromonas* to engulf. After they have grown slightly they are too big to be eaten. In the presence of the predator, the colonial form of *Chlorella* displaces the unicellular form and persists. When the predator is not present, the unicellular form displaces the colonial form. This makes sense as the colonial form has less surface area exposed to the environment available for nutrient uptake than the unicellular form has.

There is also evidence that mutations occur and are selected for in animals grown in chemostats and related systems. Boraas (1983a) observed several changes the rotifer *Brachionus calyciflorus* when it was grown for 24 months in a chemostat. The mean adult body size of the animal declined steadily over time. Rotifers ceased production of males and resting eggs after 1-2 months in continuous culture, suggesting that the chemostat environment selected against sexual reproduction. After 2-3 months in the sexuality could not be induced in animals removed from the culture. They appear to have lost the ability to undergo sexual reproduction.

Bennett and Boraas (1988, 1989) saw even more striking changes in the same rotifer species when it was grown in a turbidostat. A turbidostat is a variation on the chemostat. While a chemostat is designed for constant input of medium, a turbidostat is designed to keep the organisms at a constant concentration. A turbidity sensor measures the concentration of organisms in the culture. When it exceeds a preset value, additional medium is added. In Bennett and Boraas' study, the sensor measured the concentration of residual food (algae) in the culture (Boraas and Bennett 1988). When it dropped below a certain level, more was added. This type of culture system allows the organisms to grow at the maximum rate physiologically possible in a given environment and selects for rapid growth rate. In a chemostat, the investigator chooses the growth rate that the critters grow at and the population density is a response variable, in a turbidostat the investigator chooses the population density and the critters grow as quickly as they can.

Bennett and Boraas saw the rotifers undergo several changes. The result of the changes was a fast-growing strain of the rotifer. Over 8 months in the chemostat the maximum growth rate of the rotifers increased from 0.053 h^{-1} to 0.080 h^{-1} . This change persisted even when the animals were grown for over 100 generations in a chemostat at the slow growth rate of 0.009 h^{-1} . There was a shift in fecundity to younger age classes in the fast-growing strain. Longevity of the fast-growing strain was 28% shorter than longevity in the parental strain. Egg development time was shorter, and egg volume was considerably smaller in the fast-growing strain. As seen in Boraas' (1983a) study, the adults were smaller and sexuality was lost.

The rotifer example show changes in life history characters which are under genetic control. These examples suggest that the argument made for prokaryotes can be extended to eukaryotes.

Notes

[1] There are actually two different varieties of dark peppered moths, with the darkness being determined by different genes.

[2] There is no note [2]. See [2] for details.

[3] Proteins are the workhorse chemicals in the cell. There are two major kinds of proteins, structural proteins and enzymes. Typically an enzyme is optimized to perform a simple chemical operation on another chemical (the substrate). However it can also perform operations with much less efficiency on other substrates. A change in an enzyme often changes its efficiency on alternate substrates; it also may change the optimal conditions in which the reaction occurs, e.g. the temperature or the pH.

Diploid organisms have two copies of each gene. When a mutation in one copy occurs the organism can have alternate alleles with different properties. In some environments organisms with copies of both alleles (they are said to be heterozygous for the gene) will have an advantage.

[4] The human genome has 3 billion base pairs. The average rate of point mutations is about 20-30 in a billion per individual. Almost all point mutations in multi-cellular organisms are strictly neutral. In human beings 90-97% of the DNA is "junk DNA" that does nothing (as best as can be determined.) One third of the changes to codons (sections of DNA that code for proteins) are silent; that is, the DNA changes, but the amino acid coded for remains the same. Thus 93-98% of all point mutations in humans are strictly neutral.

Of the remaining 2-7% almost all of them are also neutral. A typical protein is a sequence of about 1,000 amino acids which folds up around a reaction site consisting of about 50 amino acids. Changes in the reaction site have a strong effect on the properties of the protein; changes elsewhere often do not unless they affect the folding pattern. As a result, less than 1% of the point mutations are subject to selection. [7]

[5] Johnathan Wells has written an excellent summary [article](#) on the peppered moth which should not be taken as being definitive. The topic is the subject of considerable controversy. For dissenting commentary see: <http://www.calvin.edu/archive/evolution/199904/0100.html> and <http://www.calvin.edu/archive/evolution/199904/0103.html>

[6] A trait is a physical feature of an organism. An organism's traits are determined by a combination of its genes and by its responses to its environment. The effect of genes on traits is often very indirect.

[7] Most of the numbers relating to the size of the effective genome, the number of genes, and the average size of genes are approximate and are still being refined.

A number of genomes, both bacterial and eukaryote, have been completely sequenced. Protein sizes average about 350 amino acids (1050 base pairs).

Older estimates of the number of genes in the human genome fall in the range 50-100 thousand. More recent estimates using data from the genome project are about 60-70 thousand.

Estimates of the size of the effective genome vary. Drake gives an estimate of 80,000,000 base pairs of coding DNA. The number may be as low as 3% (Drake) or as high as 10% (older estimates). The issue is complicated by the fact that some (unknown) percentage of the non-coding DNA is not junk.

Estimating mutation rates is not simple. It should be understood that current estimates are extrapolations from sampled sections in genomes. Moreover mutation rates vary for different sites. Different techniques, however, seem to consistently produce estimates of 1 to 6 point non-silent mutations in coding DNA per individual in humans. The total number of point mutations per individual is much higher (Drake gives ~64; other estimates are of the same order) but, as discussed in [note 4](#) almost all of these are either silent or are in non-coding (junk) DNA.

[8] The percentages of occurrences of different alleles in a population is always fluctuating because different individuals have different numbers of offspring. In diploid species such as ourselves there is an additional source of randomness; each offspring gets a different combination of genes from its parents. Not only are the percentages fluctuating, but they can by chance drift from one ratio to another.

This random change is what is meant by genetic drift. When a particular allele is beneficial compared to another the fluctuation will be biased; this biased movement of the changes in ratios is called natural selection.

[9] If we use the numbers in appendix I the effective genome size (for humans) is about 80,000,000 base pairs and the average number of point mutations in the effective genome is about 4. This works out that each base pair in the effective genome will mutate about once in every 20,000,000 individuals.

This means that in species with large populations such as human beings (currently) every relevant point mutation appears in the species. On the other hand, given a small group such as a hunter/gatherer tribe, a given mutation probably will not appear in the tribe.

Appendix I - Frequency of Mutations

When we speak of the frequency of mutations we have to distinguish between the mutation rate for the entire genome and the mutation rate for the effective genome (the 10% that is not junk DNA). In Genetics 148:1667-1686, April 1998) John W. Drake et al estimate that the average human zygote has about 64 mutations, most of which occur in "junk" DNA.

From tables 4 and 5 in "Rates of Spontaneous Mutation", by JW Drake et al, Genetics 148:1667-1686 (April, 1998):

<u>Organism</u>	<u>Effective genome size (Ge)</u>	<u>Mutations per genome per replication</u>
bacteriophage M13	$6.4 * 10^3$	0.0046
bacteriophage lambda	$4.9 * 10^4$	0.0038
bacteriophages T2 & T4	$1.7 * 10^5$	0.0040
E. coli	$4.6 * 10^6$	0.0025
Saccharomyces cerevisiae	$1.2 * 10^7$	0.0027
Neurospora crassa	$4.2 * 10^7$	0.0030
C. elegans	$1.8 * 10^7$	0.004
Drosophila	$1.6 * 10^7$	0.005
Mouse	$8.0 * 10^7$	0.014
Human	$8.0 * 10^7$	0.004

Note that for humans, the number of cell divisions prior to sperm formation in a male of age 30 is about 400. This works out to about 1.6 mutations per sperm cell.

In the 28 January 1999 issue of Nature, in the article "*High genomic deleterious mutation rates in hominids*" Walker and Kneightey estimate that the mutation rate in the effective genome is a bit higher, 4.2 mutations per individual, of which 1.6 are deleterious. See [note 7](#) for further discussion.

Appendix II - A favorable mutation, journal abstract

Arterioscler Thromb Vasc Biol 1998 Apr;18(4):562-567. "PAI-1 plasma levels in a general population without clinical evidence of atherosclerosis: relation to environmental and genetic determinants," by Margaglione M, Cappucci G, d'Addetta M, Colaizzo D, Giuliani N, Vecchione G, Mascolo G, Grandone E, Di Minno G; Unita' di Trombosi e Aterosclerosi, IRCCS Casa Sollievo della Sofferenza, San Giovanni Rotondo (FG), Italy.

Abstract:

Plasminogen activator inhibitor-1 (PAI-1) plasma levels have been consistently related to a polymorphism (4G/5G) of the PAI-1 gene. The renin-angiotensin pathway plays a role in the regulation of PAI-1 plasma levels. An insertion (I)/deletion (D) polymorphism of the angiotensin-converting enzyme (ACE) gene has been related to plasma and cellular ACE levels. In 1032 employees (446 men and 586 women; 22 to 66 years old) of a hospital in southern Italy, we investigated the association between PAI-1 4G/5G and the ACE I/D gene variants and plasma PAI-1 antigen levels. None of the individuals enrolled had clinical evidence of atherosclerosis. In univariate analysis, PAI-1 levels were significantly higher in men ($P < .001$), alcohol drinkers ($P < .001$), smokers ($P = .009$), and homozygotes for the PAI-1 gene deletion allele (4G/4G) ($P = .012$). Multivariate analysis documented the independent effect on PAI-1 plasma levels of body mass index ($P < .001$), triglycerides ($P < .001$), sex ($P < .001$), PAI-1 4G/5G polymorphism ($P = .019$), smoking habit ($P = .041$), and ACE I/D genotype ($P = .042$). Thus, in addition to the markers of insulin resistance and smoking habit, gene variants of PAI-1 and ACE account for a significant portion of the between-individual variability of circulating PAI-1 antigen concentrations in a general population without clinical evidence of atherosclerosis.

Appendix III - A favorable mutation - Journal abstract

J Biol Chem 1985 Dec 25;260(30):16321-5. "Apolipoprotein AIMilano. Accelerated binding and dissociation from lipids of a human apolipoprotein variant," by Franceschini G, Vecchio G, Gianfranceschi G, Magani D, Sirtori CR.

Abstract:

The lipid binding properties of apolipoprotein (apo) AIMilano, a molecular variant of human apolipoprotein AI, characterized by the Arg173----Cys substitution, was investigated by the use of dimyristoylphosphatidylcholine liposomes. Both the variant AIMilano and normal AI are incorporated to the same extent in stable complexes isolated by gel filtration, showing similar dimensions and stoichiometries. A higher affinity of apo-AIMilano for dimyristoylphosphatidylcholine is suggested by the faster association rate of the variant apoprotein compared to normal AI; similarly, apo-AIMilano is more readily displaced by guanidine hydrochloride from the isolated dimyristoylphosphatidylcholine- apoprotein complexes. When the secondary structure of apo-AIMilano was investigated by spectrofluoroscopy and circular dichroism, a higher fluorescence peak wavelength and a lower alpha-helical content were detected in the variant apoprotein compared to normal AI. The substitution Arg173----Cys in the AIMilano dramatically alters the amphipathic nature of the modified alpha-helical fragment of apoprotein AI. The association rate with lipids is accelerated by an increased exposure of hydrophobic residues. The reduced stability of the lipid-apoprotein particles is possibly mediated by a reduction in the number of helical segments involved in lipid association. The high flexibility of the AIMilano apolipoprotein in the interaction with lipids may explain its accelerated catabolism and the possibly improved uptake capacities for tissue lipids.

Appendix IV - Selection for HIV resistance - Journal abstract

Am J Hum Genet 1998 Jun;62(6):1507-15. by JC Stephens et al.

Abstract:

The CCR5-Delta32 deletion obliterates the CCR5 chemokine and the human immunodeficiency virus (HIV)-1 coreceptor on lymphoid cells, leading to strong resistance against HIV-1 infection and AIDS. A genotype survey of 4,166 individuals revealed a cline of CCR5-Delta32 allele frequencies of 0%-14% across Eurasia, whereas the variant is absent among native African, American Indian, and East Asian ethnic groups. Haplotype analysis of 192 Caucasian chromosomes revealed strong linkage disequilibrium between CCR5 and two microsatellite loci. By use of coalescence theory to interpret modern haplotype genealogy, we estimate the origin of the CCR5-Delta32-containing ancestral haplotype to be approximately 700 years ago, with an estimated range of 275-1,875 years. The geographic cline of CCR5-Delta32 frequencies and its recent emergence are consistent with a historic strong selective event (e.g., an epidemic of a pathogen that, like HIV-1, utilizes CCR5), driving its frequency upward in ancestral Caucasian populations.

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