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Evolutionary Change

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Synonyms

Descent with modification

Definition

Evolutionary change is the heritable change in populations and species over time, due to mechanisms such as natural selection, random genetic drift, and sexual selection.

Introduction

Evolution is heritable change over time, through which species change, diverge, and sometimes create new lineages. "Evolution" and "natural selection" are often used interchangeably, but the two are distinct: evolution is the pattern, and natural selection is one of many mechanisms that *cause* evolution. Under natural selection, organisms which are better adapted to their environment have more (or healthier) offspring, so their traits are more often passed on to future generations. In this manner, natural selection drives evolution. However, other mechanisms can also drive evolution, such as random genetic "drift," or random changes in genes and traits over time, artificial selection by humans for human-desired traits in other organisms, and sexual selection for traits that increase an individual's chance of mating. All of these mechanisms operate by impacting heritable variation to drive evolution.

Evolutionary change requires that individuals vary in ways that are passed on (in whole or in part) to their offspring. Although the observation of heritability in nature ("like begets like") is sufficient for some evolutionary inquiry, scientists now have a rich understanding of the primary mechanism of heritability: genetics. All life on earth is based on genetic code that is carried within cells and translated into observable traits. Humans, sea horses, mushrooms, the common cold virus, and more all reproduce by passing genetic material in the form of deoxyribonucleic acid (DNA) or ribonucleic acid (RNA) on to their descendants. Every organism is the sum of interactions of their genes (functional stretches of DNA or RNA) with their environment, resulting in their phenotype, i.e., the set of all traits that are expressed in a given environment and are seen by other individuals. In addition to genetics, "epigenetic" modifications (changes to the ways genes are expressed, such as conditional switches that turn genes "on" or "off") can also be heritable and thus can play a role in evolution.

Mutation generates variation in the genetic code due to (i) random errors in our cellular machinery or (ii) environmental influences

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(induced errors). Genetic mutation can translate into heritable differences in an organism's observable traits, or phenotype, with neutral, positive, or negative effect on the organism's fitness. While mutation, and thus differences between organisms' genetic code, is the basis of variation, several higher-level factors contribute to variation. Gene flow between separated populations of the same species can also induce variation, and recombination during sexual reproduction reshuffles genes and their associated traits.

Scientists research evolutionary change by examining the fossil record, interpreting genetic evidence in the present, inferring genetic pathways over time, modeling change, and performing real-time evolutionary experiments with model organisms such as flies, yeast, and mice. This wealth of approaches has contributed greatly to a scientific understanding of the causes and persistence of variable life on earth.

Evolution is popularly conceived as a stairway of progress – from simple to complex, from bad to good, and from animals to man. This is completely untrue. Rather, it is more like a tangled bush with connections, splits, divergences, and interlocking parts. Humans are merely a tiny tip at the end of a tiny branch on the gigantic "Bush of Life."

Heritability

Genetics

Genes control heritable traits and are the units of heredity. "Genotype" refers to the genetic material in an organism, while "phenotype" refers to observable characteristics whether inherited or not. Phenotypes result from genes, from environment/behavior, or from the interaction of genes with the environment (often popularly written as "nature plus nurture"). Selection generally acts on phenotypes, but phenotypes are not directly transmitted from parents to offspring – genes are. A single human's overall phenotype could include tall height, brown hair, brown eyes, interest in and success at computer science, preference for neutral-toned clothing, no memory for details, and great storytelling ability. As this example suggests, not all phenotypic characteristics are heritable! Eye color is fully heritable from known genetic variants, but interest in computer science is determined in part by inherited talent but also in large part by one's environment growing up. This is one complication of evolutionary inquiry: there is not a one-to-one correspondence between traits we can observe and measure (phenotypes) and heritable units (genes) coded in DNA.

DNA, deoxyribonucleic acid, is a long thin molecule that contains the "code" which is translated into proteins, the building blocks of cells, tissues, organs, and ultimately our bodies (some viruses use RNA, rather than DNA, as their unit of heredity; it is debated whether viruses are truly alive). DNA has an iconic "double helix" structure, with chemical bases in the center surrounded by phosphate–sugar chains. The chemical bases are of four kinds, represented by the four letters A, T, C, and G (which refer to adenine, thymine, cytosine, and guanine, respectively). DNA is shaped roughly like a spiral ladder, and these four bases pair up with one another (A with T, C with G) to form the rungs while the outer bars are formed by phosphate and sugar. DNA is "transcribed," i.e., copied by the enzyme RNA polymerase into messenger RNA, a molecule similar to DNA, which is then "translated" into protein. DNA base pairs are grouped into sequences of three called "codons" which correspond to specific amino acids (or in some cases, "stop" codons signal a stop in translation). The third position of a codon is called the "wobble" position because base pairs in that slot do not always obey the pairing rules described above (A with T, G with C); further investigation of this important phenomenon is outside the scope of this article.

Genes are stretches of DNA, ranging between less than one hundred base pairs to over two million, that in essence code for a specific string of amino acids which in turn folds into a specific protein. Almost all human DNA is the same in every individual human (over 99%); thus, only a very small proportion of the genome comprises individual variation.

Genes sit in a specific place on "chromosomes," which are the dense threadlike packages

of genetic information that cluster inside the nuclei of our cells. However it is worth noting that our nuclear DNA – the DNA on chromosomes in the nucleus – is not the only DNA we have in our bodies. Most notably, mitochondria have their own DNA that is inherited from mother to child (since mothers' ova contain the cell organelles). Mitochondria have DNA because they were independent organisms which our ancestors "domesticated" and put to work inside our cells. Chromosomes consist of DNA coiled around "histones," a special type of protein that allows the supercoiled structure of DNA. Humans have 23 pairs of chromosomes (since we inherit 23 from our mother and 23 from our father). Twenty-two pairs are "autosomal" chromosomes, which conventionally are numbered from 1 to 22 from largest to smallest, while the 23rd pair consists of our sex chromosomes. These are the X and Y chromosomes that determine our biological sex (Other animals have different mechanisms of sex determination, such as temperature-dependent sex determination in some reptiles (Warner and Shine 2008).). Female humans have XX sex chromosomes, while males have XY chromosomes. Some autosomes may be familiar to you; for example, if an individual inherits three copies of chromosome 21 instead of the usual two copies, this is referred to as "trisomy 21" or "Down syndrome."

The place of a certain gene on a chromosome is called its locus. Genes can have multiple slightly varying forms known as alleles; these allelic differences code for many of the differences that separate humans from one another. For most genes, we inherit two copies (one from each parent); if you have two copies of the same allele, you are *homozygous* for that allele. If you have two different alleles, you are *heterozygous*. The ways in which the two different alleles interact determine your phenotype. Different alleles can be codominant, where both are expressed to some extent; they can be in a dominant-recessive relationship where only one copy of an allele is necessary for expression; or different alleles can be related in more nuanced interactions. For an artificial, simplified example, imagine that eye color is a trait coded by a single eye color gene. Assume that you can inherit either a blue or brown allele from your parents and that brown is dominant to blue. If you have two blue alleles, you have blue eyes; similarly, two brown alleles produce brown eyes. But if you have blue and brown alleles, since brown is dominant to blue in this imaginary simplified scenario, you will have brown eyes. However, you could have a child with blue eyes, as long as (i) you pass your blue allele on to the child and (ii) that child inherits a blue allele from their other parent as well! This is an important principle: traits that are not expressed in a parent can nonetheless be genetically hidden only to be expressed in later generations (Most traits, including eye color, are much more complex than described here).

Crucially, DNA can replicate itself. This is the basis of life. When our cells divide, DNA is copied so that each cell contains a complete copy of our genetic code. During cell division, which is essential for growth, maintenance, and reproduction, DNA is "unzipped" and copied by molecules and enzymes dedicated to these tasks. Sometimes during this process of copying, our copying mechanisms make errors. A stretch of DNA that read AATC might get accidentally copied as AATG. If this mistake is not noticed and corrected by our cellular mechanisms, it will be preserved in that cell's genetic code as a mutation. If that cell is a reproductive cell, the mistake could replicate to offspring. There are many kinds of mutations, which we will turn to below; this is one source of the variation that can drive evolution.

Note that in many cases, genetic mechanisms and inheritance are more complicated than described above. The full complexity of genetics is beyond the scope of this entry, but it is important to note several caveats. "Selfish" genes act as self-interested agents that seek to further their *own* reproduction (Dawkins 2016). Indeed, sometimes the conflicting "agendas" of genes result in ingenious mechanisms for violating traditional modes of inheritance (Burt and Trivers 2006). Genes can be "sex-linked," expressed only in the phenotypes of one sex, or "incompletely penetrant," whereby, for example, a gene is not activated unless certain environmental conditions are met. Further, the connection between complex traits and genes is

an ongoing mystery that geneticists are solving on a case-by-case basis with large data sets and statistical inference.

Epigenetics

We used to think that the DNA code that is passed from parents to child is the only mechanism of heritability. However, we now realize that this is not the whole story. "Epigenetics" describes changes to our gene *expression*, prompted by life experience. Remarkably, some of these changes are heritable. Consider the following landmark study on the inheritance of learned fear (Dias and Ressler 2014). Mice were trained to associate a certain specific cherry-like scent – acetophenone – with fear. Every time the scientists flooded the mice's cage with the scent, they delivered small mild shocks to the mice. When the mice reproduced, the babies knew to fear the scent of acetophenone *without ever having undergone training.* Somehow, the learned experience – fearing acetophenone – was passed on to the mice's offspring. This was not associated with *genetic* changes. While the specific mechanism in this case is still a mystery, we know it must be due to epigenetic inheritance.

So what exactly is epigenetics? While scientists define this term in many different ways, epigenetics broadly refers to modifications that control how genes are *expressed*. A very large part of our genome is not genes but rather is specific stretches of DNA that regulate expression of genes. Recall that genes are expressed by coding for proteins which perform a variety of functions; "silenced" genes are no longer expressed as proteins, while "upregulated" genes are expressed at higher levels than usual (and "downregulated" genes do the opposite). In many cases, protein-coding genes are regulated by associated stretches of regulatory DNA or regulatory regions in the genome usually (but not always) near a particular gene. In other cases, stretches of DNA pick up "tags" or reconfigure their shape in ways that silence, upregulate, or otherwise affect expression. Epigenetics refers to external influences (e.g., environment or behavior) that modify expression of genes, leading, for example, to turning genes on or off or affecting the timing and levels of expression of genes.

For our purposes, we are most interested in heritable epigenetic changes. Two of the most important such mechanisms are DNA methylation and histone modification.

DNA methylation silences genes through chemical binding. Specifically, methyl groups are added to the chemical structure of DNA; where methyl groups bind, genes tend to be *less expressed* or turned off. Methylation is very important in genetic imprinting, which is the way in which genes are "tagged" as coming from one's mother or father (and have corresponding expression modifications). A notable example of imprinting in humans is seen in Prader-Willi and Angelman syndromes – clinically distinct disorders associated with multiple anomalies and mental dysfunction, both usually due to deletions in part of chromosome 15. Whether an individual has Prader-Willi versus Angelman syndrome depends on which parent transmitted the faulty chromosome 15 to the child.

Histone modification affects DNA packaging, which determines which regions are "exposed" to the cellular machinery of gene expression. Recall that DNA is wrapped around histone proteins – it is packaged in a particular way that, with the help of these proteins, influences transcription and translation (the steps of gene activation). The shape of the histone proteins and the way in which DNA interacts with and wraps around histones can change based upon environmental influences and can be inherited.

Scientists do not yet fully understand how histone modifications are heritable; however, several studies have begun to unpack the ways in which methylation patterns are preserved from parent to child. For example, methylation patterns reveal whether a chromosome originated from the mother or father. On a maternally derived chromosome, genes that represent paternal interests are methylated and thus silenced. This is an intriguing and tricky concept and a famous category of "genetic conflict"; in short, in many mammalian species, mothers and fathers have different evolutionary interests, which are expressed through epigenetic "imprints" on their child's

genome. Genomic imprintings, as it is called, is known to occur in fungi, plants, and animals–including humans. For example, in humans, mothers bear the child and thus have an evolutionary interest in keeping the baby's body at a reasonable size. Fathers, however, have an evolutionary interest in making babies as large and well-nourished as possible, thus extracting maximum resources from the mother's body. In our genomes, we see many patterns of methylation around genes related to infant growth and maternal provisioning that clearly demonstrate these opposing genetic interests a parental tug-of-war (Moore and Haig 1991, Haig 1993).

Thus, epigenetic inheritance is an additional component of heritability. It is counterintuitive to people familiar with strict genetic heritability, but there is no doubt that epigenetic inheritance plays a role in fitness, variability, and reproduction; therefore, it is important to consider as part of evolutionary change.

Variation

For evolution to occur, organisms must have heritable variation. That is, individuals must differ from others in ways that can be passed on to their offspring. Variation in genes and thus in individuals comes about through three primary mechanisms: mutation, recombination, and gene flow. In all of these cases, differences in the genetic code are the basic level at which variation occurs; these differences can be encountered and arise through mutation in an existing population, sexual recombination, described below, or gene flow (the movement of individuals and genes between populations that have been genetically diverging).

Mutation

Mutations are changes in the genetic code of an organism. Mutations can be categorized in many ways, but the most important categories for our purposes are "germline" versus "somatic" mutations, because these categories determine whether or not a mutation is passed on to offspring. In order for mutations to be passed on to offspring, they must be present in the germline, such as a mother's ova or father's sperm in humans. Mutations in somatic cells, or cells of the body that do not pass on to offspring, therefore do not affect an offspring's fitness.

Alleles are different *versions* of one gene. For example, ABO blood type is determined, very simply, by which alleles you have of the "ABO blood type" gene. You get one copy of the gene from your mother and one copy from your father. The possible alleles you could have are A, B, and O. If you have the "A" allele and the "B" allele, you are blood type AB. Alleles A and A or A and O produce blood type A and likewise for B. Finally, if you have alleles O and O, you are blood type O. These alleles vary among individuals, just as do all other alleles. Species tend to have allelic variation; that, in combination with environmental variation, is why we do not all look and behave the same! Identical twins have very little allelic variation, because they inherited the exact same copies of the same alleles from their biological parents. Thus, in general, differences between identical twins are driven by the environment and the ways in which the environment impacts gene expression. Studies of identical twins (Boomsma et al. 2002) have found a variety of intriguing and peculiar findings about which traits are inherited and which are primarily driven by environmental variation.

Different alleles are generated by mutation, which occurs for two reasons: first, as DNA undergoes the process of replication, it can make a mistake. Each time a cell divides, there is a chance for the DNA to be copied incorrectly – this difference is a mutation. Second, environmental factors can directly cause mutation. Chemicals, or certain types of radiation, can break down DNA to such an extent that our DNA repair processes cannot adequately fix the damage.

Mutations can be "silent," "deleterious," "beneficial," or "lethal." Silent mutations in the genetic code, such as redundant base pair substitutions, have no effect on the gene's function. Proteins are built from amino acids, and DNA codes for amino acids – however, each type of amino acid can be generated by multiple DNA templates. A mutation can also be silent if it occurs in a stretch of DNA with no function (But have

caution! For many years, we thought long stretches of DNA that were known as "junk" DNA did nothing. Really, they served important, if complicated, regulatory purposes.). Mutations without strong effects, and ideally without any effects at all, can be used to calibrate "molecular clocks"(Ayala 1986); that is, when we are trying to understand how long ago a species diverged from another species, we can compare how many mutations each species has in nonfunctional regions (Ho and Larson 2006). This is better than looking at functional mutations, since selection can act on those and interfere with our perception of how much time must have passed. A fundamental assumption, required to use the idea of molecular clocks, is that DNA not under selection accumulates mutations at a constant rate.

Of all functional mutations, the majority are deleterious, or harmful to the fitness of the organism in which they occur. Studies of the model fly species *Drosophila melanogaster* indicate that about 70% of functional mutations – that is, mutations that change the protein coding of a gene – are deleterious (Sawyer et al. 2007).

Beneficial mutations confer some fitness advantage to their host organism. Sometimes, of course, we might not understand their benefit in today's world! Once we are removed from the evolutionary context in which a mutation was selected for, it can begin to have negative consequences.

Many beneficial mutations come about when a stretch of genome is duplicated and passed on to offspring (but see Lynch and Conery 2000). In this case, there is genetic "redundancy" that gives a buffer for mutation. If something changes over the generations in one copy of the gene, it is less likely to cause trouble since the individual has another copy. A famous example of this is the evolution of trichromatic color vision in humans. Our ancestors used to have only two types of cones – long wavelength (red) and short wavelength (blue). There was a duplication in the gene coding for long-wavelength opsins, or light-sensitive proteins. Over time, the duplication allowed one set of long-wavelength opsins to diverge slightly and differentiate enough such that one set became specifically sensitive to green light and one to red light. Thus, we and two other primate groups independently evolved trichromatic color vision with our familiar sensitivity to red, green, and blue light through gene duplications (Dulai et al. 1999).

Indeed, *de novo* (new) gene duplications account for the largest family of genetic differences between humans and chimpanzees (Cheng et al. 2005). They are not always good – some are associated with human-specific diseases!

Finally, lethal mutations are simple and aptly named. They kill you.

Gene Flow/Migration

Mutations can arise in an existing population. However, another source of variation can come from interaction between populations. When individuals move from one population to another, they carry their genes with them. Thus, spatial migration is accompanied by genetic flow. Plants sending pollen far and wide, animals leaving their native ranges due to human pressures, and people migrating from place to place are all mechanisms of gene flow.

A related phenomenon is that of a "genetic bottleneck." In this scenario, a population of a single species encounters a "squeezing" effect on their genetic diversity, as if they are flooding from a wide bottle into a narrow bottleneck, typically due to a very small population size. This can be caused by environmental disasters, such as an asteroid strike, flood, fire, disease that sweeps through most but not all of a population, or geographic separation (due to a small group populating, say, an island). If a small subset of a population becomes reproductively isolated and "founds" a new population, biologists call the resulting lack of genetic diversity the "founder effect." Indeed, it is well understood that modern humans evolved in Africa and then spread to the rest of the world, encountering a genetic bottleneck on their way out of the continent. This is one reason why populations in (or descended from those in) Africa are far more genetically diverse than the rest of humanity.

Human impacts have had catastrophic effects on many species by decreasing their population size; overfishing, overhunting, and environmental

destruction have led to mass extinctions and severe drops in genetic diversity.

Sexual Recombination

Children may look like a blend of their biological parents, but sexual reproduction does not actually *blend* traits. Instead, genes "reshuffle" during the process of sexual reproduction. You are not a copy of either of your parents, nor are you perfectly in between. You wind up with combinations of traits that are not found together in either of your parents.

In sexually reproducing organisms (which are most animals, including humans), each parent passes on 50% of their genes to their child. In humans, for example, each individual sperm and each individual egg have been produced through a process that splits the parent's genome in two. Most of our cells have 23 pairs of chromosomes – two chromosome 1s, two chromosome 14s, etc. and either two X chromosomes or one X and one Y chromosome. This process of creating eggs and sperm – which is known as meiosis – involves stages whereby these pairs of chromosomes pair up, exchange genetic material through "crossing over," and then separate to be sequestered into eggs or sperm. Each sperm and each egg have 23 individual chromosomes, and these individual chromosomes are "reshuffled" versions of their parent's chromosome pairs. Fascinatingly, some loci in a genome can manipulate meiosis to enhance their own transmission, such that they are found in more than 50% of germ cells. This is known as meiotic drive, and it is not yet known to what extent meiotic drive has influenced our evolutionary history.

Mechanisms That Drive Evolution

Natural Selection

Natural selection is the most commonly discussed mechanism of evolution, although it acts in concert with the other mechanisms described below to drive evolution. In short, natural selection requires variation, differential reproduction, and heritability. Organisms compete for resources and to reproduce; individuals vary in a population and pass on much of this variation to offspring; and individuals that are better adapted to local conditions pass on more copies of their genes to future generations. This is popularly termed "survival of the fittest."

The building blocks of natural selection are variation within a population, differential reproductive success, and heritability. We have already seen that DNA makes up genes which are passed from parents to offspring; clearly, heredity is present. Further, variation is present in natural populations; this is clear from simple inspection, and the many studies linking genes to observable traits have demonstrated that variation is in large part heritable. Finally, "differential reproduction" refers to the fact that different individuals leave behind more or less offspring depending on how "fit" they are. Fitness is variably defined, but can be summarized as "an organism's ability to survive and reproduce". If populations were unlimited and every individual had equal chance to mate and leave behind equally fit offspring, the criteria of "differential reproduction" would not be met.

Artificial selection, or selection conducted by humans, is closely related to natural selection. Animal and plant breeding over the generations produces evolutionary change along metrics that humans care about – larger plants we want to eat, purebred dogs with arbitrarily preferable traits, and docile fat cattle for the slaughterhouse. It is the same exact mechanism as natural selection, except humans choose who gets to reproduce, rather than a harsh environment selecting for reproductive fitness.

Sexual selection is a special case of natural selection whereby individuals evolve traits that increase their chances of copulating with the opposite sex. This will be discussed in greater detail in the subsequent section "sexual selection."

Since life has heritable variation and differential reproduction, natural selection is in effect. We see overwhelming direct evidence of natural selection in real time, in the fossil record, and in our genes.

Convergent Evolution: Strong Evidence of Natural Selection

The most powerful evidence for evolution by natural selection is the remarkable "convergence" of distantly related species who face similar selective pressures. That is, many species independently evolve similar traits because they are useful and confer a fitness benefit within a specific environment. Sharks and dolphins share a similar body form even though one is an elasmobranch fish and one is a mammal the pressures of aquatic life led to selection for a torpedo-like body shape with fins. Vertebrates evolved wings for flight at least three times in birds, bats, and pterosaurs; further, mammals have evolved "gliding" body forms, with flaps of skin stretched between legs, in both the flying squirrels and the distantly related marsupial sugar gliders. While these examples are common, they barely scratched the near-unbelievable surface of convergent evolution over time and space.

Consider, for example, the phenomenon of "mammalian woodpeckers" (Cartmill 1974). In some areas of the world, there are no naturally occurring woodpeckers, and instead, mammal species evolved to fill the woodpecker "niche." ("Niche" is a catch-all term describing an organism's or species' ecological role in the environment: e.g., what they eat and how they eat it). Today, there are two living mammal species who evolved to fill the woodpecker niche: the striped possum in Australia (*Dactylopsila trivirgata*) and the aye-aye from Madagascar (*Daubentonia madagascariensis*). These mammals are distantly related, and evolved completely independently on their respective islands. However, both have massive protruding incisors that they use to bore through and tear at wood, finely tuned ears that listen for grubs squirming within the wood, extremely elongate single fingers to reach into the wood and pull out grubs, and a uniquely reinforced skull to deal with the pressures of wood-boring (Cartmill 1974; McCoy and Norris 2012)! There are two additional proposed "mammalian woodpeckers" from the fossil record: *Hegetotherium mirabile*, an enigmatic notoungulate from South America, and *Yalkaparidon*, a marsupial from Australia. All of these species are only distantly related and yet evolved remarkably similar morphological adaptations to the specific niche of wood-boring; in other words, these animals evolved similar traits long after the spit from their comment ancestor. This is extremely strong evidence for the mechanism of natural selection (although the aye-aye and striped possum are extremely similar, I am sorry to say that the aye-aye looks like a frightening demon while the striped possum looks like an adorable pet).

Selection at What Level?

The history of evolutionary biology is filled with arguments about the level at which selection operates (Okasha 2006). Does it operate on cells, genes, individuals, and species? An overview of this debate is beyond the scope of this entry. To briefly summarize, life is composed of many replicating "selfish" elements, perhaps most notably genes (Dawkins 2016) and individuals. Selection acts on phenotypes, and genes are the unit of heritability that are passed from parent to offspring. The correlation between gene and phenotype is not perfect, since the environment exerts an influence as well. Individuals who share a high proportion of their genes may be evolutionarily motivated to "help" each other; this is known as "kin selection theory" or "inclusive fitness" (Hamilton 1964). Over geologic time, paleontologists often talk about species-level selection, by which species diverge and emerge in response to fluctuating niches (Gould 2002). Some biologists will hear nothing of selection at a level higher than the gene.

To all levels of selection, there is some truth (Okasha 2006).

Genetic Drift

Genetic drift, unlike natural selection, is completely random and not determined by fitness. In any population, whether of people or parakeets, some individuals will be more successful by chance. They will leave behind more offspring – not because they are measurably "fitter" due to selected adaptations but because that's the way the cookie crumbled. Without selective pressure on certain alleles, alleles drift in a random walk until

one allele becomes "fixed" in the population – that is, until one allele is present in every individual in a population. If a population is small, such as in the case where a small subset gets cut off from the main population, or where a few members of a species populate an island, there is a higher chance that a certain allele will drift to fixation despite having a neutral or even negative effect.

There is much debate about whether selection or drift exerts stronger influence on evolution in a population. Are most traits there by chance or because they were selected for? Proponents of the "neutral theory" of evolution argue that much of evolution is caused by genetic drift and the random fixation of alleles. It is difficult to quantify the relative contributions of random drift and natural selection (for reasons such as "genetic draft," see below), but both have demonstrably played a role in evolutionary change over time.

Genetic Hitchhiking ("Genetic Draft")

Genetic "draft" is closely related to the phenomena of drift and selection. It is also called genetic "hitchhiking," because it can be conceptualized as certain neutral or even deleterious alleles "hitching a ride" with beneficial alleles.

Genes recombine during sexual reproduction; however, the rate of recombination between genes varies depending on the distance between them. Further, the position of genes on a chromosome influences the likelihood that they will recombine and be separated from each other. If genes are close to each other, they are said to be "linked." The nonrandom association of alleles at different loci is known as their "linkage disequilibrium."

When two genes are linked, specific combinations of their alleles are almost always passed on and inherited together – as a unit. A few implications are obvious. First, if two genes are functionally related and become linked by chance, then there will be strong pressure for them to remain linked; for example, what if the gene for purple hair gets linked to a gene for preferring purple hair?

Second, a highly useful gene variant can be linked to a neutral or even deleterious gene. If they happen to be close together on the chromosome, that neutral or negative allele might sweep to high frequencies in the population just by "hitchhiking" along in the "selective advantage bus" driven by its neighbor gene. So when looking at genes in a population, it is not always safe to assume that a very common gene variant has a selective advantage. It may have "drifted" to high frequencies or been "drafted" by a neighbor.

Interpreting Evolutionary Evidence: Notes of Caution

Evolutionary "Spandrels" and Exaptations

In two landmark papers, Stephen J Gould, Elisabeth Vrba, and Richard Lewontin provided a fundamental note of caution when interpreting evolutionary evidence. It is tempting to look at a current organism with its suite of traits and think about why each trait is evolutionarily beneficial. However, scientists must be aware of "exaptations" or evolutionary "spandrels," traits that either (1) evolved for a past purpose before being co-opted or (2) are just incidental by-products of adaptive traits.

Vrba and Gould coined the word "exaptation" to describe the following evolutionary truth: the current function of a trait does not always correspond with its historical function and origin (Gould and Vrba 1982). For example, we see that birds fly using feathered wings and think "Aha! Feathers evolved so that birds could fly." However, a wealth of paleontological evidence calls this into serious doubt. Feathers evolved in dinosaurs long before flight evolved; they almost certainly aided in insulation and may have played a role in display, waterproofing, or defense (Prum 1999). Only later were feathers *co-opted* for the purpose of flight.

Related to "exaptations" are Gould and Lewontin's evolutionary "spandrels," a term inspired by architectural phenomena at the San Marco Cathedral in Venice (Gould and Lewontin 1979). This cathedral has a beautiful dome resting upon arches, and "spandrels" are the triangular windows between the intersecting arches. Usually spandrels are decorated

ingeniously, as is the case at San Marco. If one just looks at the spandrels, they might be tempted to conclude that the spandrels were carefully and intentionally shaped and designed, and perhaps even that the rest of the building exists only to frame them. But in reality, spandrels are a necessary result of mounting a sphere on a dome; they are only an incidental by-product of the grander architectural scheme.

Similarly, it is tempting to look at an evolved creature, focus on a certain trait, and try to come up with its evolutionary purpose. But sometimes, that trait is just a "spandrel" – an insignificant by-product of a trait that was truly evolutionarily significant. Indeed, Gould and Lewontin give as a primary example of spandrels the many peculiar processes in the human brain that we cannot fully understand (Gould and Lewontin 1979).

An Incomplete, Mysterious Fossil Record

Often in evolutionary history, we have incomplete evidence. Fossils give us clues about the past, and we can only see current life as a mere snapshot of the continuously changing spectrum of life and diversity.

The vast majority of life on earth is available to us only through fossil evidence. Some fossils are remarkably well-preserved, which allowed us to see, for example, the imprint of soft tissue, the outlines of dinosaur feathers, and more. Other fossils are frustratingly incomplete.

But even when scientists have a wealth of evidence, it is common to initially misinterpret fossils. For example, one of the most famous fossils of all time, the Tully monster (*Tullimonstrum gregarium*), was thought to be a mollusk or worm until it was recently found to be a vertebrate (McCoy et al. 2016).

This example illustrates the difficulty of correctly interpreting fossil evidence, even in the case of the relatively abundant Tully Monster. The history of human evolutionary biology is replete with sparse fossil finds, angry feuds, and misinterpretation. Many species of our hominin relatives were initially thought to be "deformed" examples of normal *Homo sapiens*, including Neanderthals and *Homo floresiensis*. Further, many of our hominin relatives are known by only extremely tiny scraps of bone and fossil. As Gould once wrote, "an old paleontological in joke proclaims that mammalian evolution is a tale told by teeth mating to produce slightly altered descendant teeth."

Evolutionary Change over Life's History

> The complete history of life ranges over four billion years, through alien worlds of sulfurous oceans beneath asphyxiating air, past iron-breathing bacteria and microscopic chimera, to arrive at last at our familiar world of oxygen and ozone, forested valleys, and animals that swim, walk, and fly. Scheherezade could hardly have invented a more engaging tale. – Andy Knoll, Life on a Young Planet

A History of Life, Skewed Toward Vertebrates

The origins of life are uncertain, but scientists have generated some likely conditions and circumstances (Knoll 2015).

Life began in the ocean about 4 billion years ago, and it stayed there for some time. The last universal common ancestor (LUCA), the most recent type of organism from which all living things commonly evolved, was likely a thermophilic, anaerobic microbe (Weiss et al. 2016).

Life requires replicating molecules. The earliest such molecules were probably RNA, which is a nucleic acid similar to DNA that is capable of self-replicating. In short, certain RNA molecules can act as a polymerase which can replicate template RNA molecules by joining together individual RNA monomers, much like the current protein RNA polymerase. This process, self-replication, is crucial for life.

From the initial, murky moment that self-replication first came to be, three major branches of life evolved. The three domains of life are Bacteria, Archaea, and Eukarya. The first two, Bacteria and Archaea, lack a nuclear membrane; some members of Archaea are known to thrive in the most extreme environments, such as in volcanoes or at the bottom of the ocean. Eukarya are most familiar to us: we belong to Eukarya, as do fungi, plants, all animals, and some single-celled

organisms such as amoeba. We eukaryotes are united by our possession of a nucleus within our cells – a tightly packed center of genetic code.

Millions of years ago, ancestral eukaryotes domesticated other single-celled organisms by enveloping them; these became our mitochondria. Mitochondria take care of certain respiratory functions of our cells. This phenomenon is known as endosymbiosis and has played many important roles in large evolutionary transitions. For example, endosymbiosis involving photosynthetic cyanobacteria and other organisms allowed photosynthesis to spread across the globe.

About 600 million years ago, the first animal species evolved. These mysterious early species are called the Ediacaran biota and are poorly understood. Some look like jellyfish, others look like worms, and some look distinctly different from anything living today; much of what we know about the enigmatic Ediacarans comes from fossil molds of the original bodies, so we have very little information about their ecology and evolutionary relationships. During the so-called Cambrian explosion, which actually lasted many years, most of the modern phyla of animals came to be (Briggs et al. 1992). The most famous fossil record of these species is the Burgess Shale, a rich deposit in Canada.

Four hundred million years ago, plants evolved from "green algae" (which had already domesticated cyanobacteria to enable photosynthesis). Then, 380 million years ago, vertebrates clawed their way onto land (in the form of amphibious critters). Arthropods were the first terrestrial animals by many millions of years, but we do not know exactly when they first crawled ashore.

Two hundred and fifty million years ago, the earth saw the most catastrophic extinction event in its history: the Permian Extinction, also known as the "Great Dying." Somewhere between 81% and 96% of all life went extinct during this period; it took 30 million years for Earth to recover a comparable diversity of life. Both catastrophic and gradual processes led to the Great Dying, including volcanic eruptions, methane release from the sea floor, and increased anoxia and aridity worldwide (Sahney and Benton 2008).

Around 200 million years ago, in the wake of the Permian Extinction, sauropsids, archosaurs, and dinosaurs came to dominate land vertebrates. At this time, mammals were mostly small and insectivorous- but not for long.

Sixty-six million years ago, the Cretaceous period ended with a comparably minor mass extinction: the extinction of the dinosaurs (and many other animal groups). An asteroid collided with the earth which, in combination with other factors, wreaked havoc on the reptilian population. However, their descendants and relatives, birds, still cover the earth today. Indeed, from a formal phylogenetic perspective, birds are dinosaurs. The extinction of the nonavian dinosaurs paved the way for mammals to stride forth from their nocturnal, small-bodied forms. Humans have recently become a particularly notorious ape.

Now, human actions are causing a global mass extinction greater and swifter than any before on record.

Human Evolution: Early Fossils, Close Relatives, and Genetic Traces

> Four out of five animals on Earth are nematode worms – if all solid materials except nematode worms were to be eliminated, you could still see the ghostly outline of [the continents]. . .in nematode worms. – E. O. Wilson

And yet, here is a section dedicated to the evolution of man.

Humans evolved from apes. From fossils, we know that primates came to be about 55 million years ago, while genetic evidence (using molecular clocks) indicates that they first evolved 85 million years ago. Many, many species of Homo and relative genera flourished in Africa while our direct ancestors were evolving. Just a few of them emerged from Africa before our own migration out: *Homo floresiensis* (the hobbits), *Homo neanderthalensis* (the Neanderthals), and *Homo sapiens denisova* (the "Denisovans," status as species or subspecies currently debated).

East Africa is rich with fossils of early hominins, particularly in the regions of Olduvai Gorge and Lake Turkana. Many of these fossils were found by the famous Kamoya Kimeu as well

as Leakey family: Mary and her husband Louis, as well as their son Richard and daughter-in-law Meave. They were assisted by extremely capable fossil hunters and paleontologists.

Three of the most famous and important early fossils in this region are Lucy, Ardi, and the Nariokotome Boy. They are important because they are relatively complete, early relatives of ours who have human-like traits in combination with apelike traits.

Donald Johansen discovered "Lucy" (so named for the Beatles song "Lucy in the Sky With Diamonds"), the most famous hominid fossil of all time: Lucy lived about 3.2 million years ago as a member of the small-brained, bipedal species *Australopithecus afarensis*. Lucy is also known as Dinkinesh, a translation of "you are marvelous" in the Amharic language. Lucy is famous for being a biped from so long ago and for indicating that bipedalism apparently preceded brain enlargement.

"Ardi" that is a hominid fossil more complete and older than Lucy was found first by a college student named Yohannes Haile-Selassie and uncovered and examined fully by Tim D. White. This was Ardi, a female individual of the species *Ardipithecus ramidus*, featuring opposable thumbs and *opposable big toes*, as well as an apparently bipedal gait. The species name comes from the words Ardi ("ground floor") and the word ramid ("root") in the Afar language – the point being that Ardi lived on the ground and lies at the root of all humanity.

Kamoya Kimeu, found the Nariokotome Boy: this fossil, formerly known as the Turkana Boy, is a young *Homo erectus* dating to 1.5–1.6 million years ago. It is the most complete early human skeleton known and had an extremely large brain and tall stature.

The East African fossils are incredible, but a recent, remarkable find brought Southern Africa back onto the map: *Homo naledi* was found in South Africa in the "Cradle of Humankind," where 15 individuals were found in an extraordinary cave deposit, seemingly placed there intentionally. These fossils have not been dated due to the unique circumstance of their deposition, but the morphological characteristics make them strange chimera of near-modern features and highly "primitive" apelike features: human feet but apelike shoulders, human-like hands with apelike curved fingers, and a tiny brain. The full significance of this fossil find will be unraveled in years to come.

Our direct ancestors, archaic *Homo sapiens*, evolved sometime between 400,000 and 250,000 years ago. The "out of Africa" model describes how humans evolved to our modern form about 200,000 years ago, then left Africa, and displaced, interbred with, and perhaps outcompeted the other species of *Homo* already spread across the globe. It is certainly possible that Homo sapiens left Africa multiple times. There may have even been a coastal dispersal of humans from the horn of Africa, leading to populations in Oceania and Southeast Asia.

Our closest living relative is the chimpanzee. We share between 95% and 99% of our genomes with chimps. However, we see genetic persistence of Neanderthal and Denisovan DNA in many populations of *Homo sapiens* today. This is because our ancestors interbred with these related subspecies of human; most human lineages of non-African origin have 1–3% Neanderthal DNA in their genomes (while a jawbone from an early modern human from Romania had 6-9% Neanderthal DNA (Fu et al. 2015)); many modern humans also have Denisovan DNA (Reich et al. 2010). For example, genes conferring resistance to high altitudes in Tibetans were derived from gene flow with Denisovans (Huerta-Sánchez et al. 2014).

Interestingly, human culture has driven genetic evolution (This gene-culture coevolution was recently demonstrated to exist in orcas as well, the first nonhuman species for which this has been conclusively demonstrated Foote et al. 2016). For example, a turn toward cultivating cattle for milk in Europe led to the evolution of human lactase persistence genes, genes that facilitate lactose digestion and allow adults to drink milk without adverse digestive upsets (Beja-Pereira et al. 2003).

The Evolution of Psychological Traits

> When you light a candle, you also cast a shadow. – Ursula K. Le Guin
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> All we ever see of stars are their old photographs. – Alan Moore, Watchmen

Much of evolutionary inference comes from looking at shadows. This is true perhaps most of all for cognitive and behavioral traits, which tend to disappear in the fossil record (except for leaving "traces" such as footprints and stone tools) (In Chauvet Cave in Southern France, the footprints of a boy from 20 to 30,000 years ago appear alongside the footprints of a wolf with an apparently shortened middle digit, a characteristic of dogs (Garcia 1999). Was the wolf domesticated? Did the boy and the wolf explore the cave together? Or are the footprint sets merely aligned by chance, separated by hundreds or thousands of years in real time?). Luckily for evolutionary psychologists, certain psychological phenomena have evident molecular, morphological, and genetic correlates which can be tracked by the fossil record and our own genomes.

Sensory perception is the tunnel between our brain and the world, and a wealth of evolutionary, molecular, and genetic evidence has unwound the perplexing spools of sight, smell, touch, and hearing (If we were so lucky to be pit vipers or vampire bats, we could also sense infrared; likewise for many other animals and their ability to sense electromagnetic fields). For example, we know about when our ancestors switched toward a primarily visual mode of life, because we see evidence that vision improved while our sense of smell got worse. Scientists have noted genetic signatures of color-sensitive cell evolution paired with the degeneration of olfactory ability. Similarly, we can interpret certain sensory and cognitive biases by better understanding how the visual system works; phenomena such as "color correction" (by which a red apple looks red whether it is in the bright sunlight or dark blueish light) are highly conserved across many vertebrate lineages.

Sensory perception is not the only evolutionary avenue to the brain: for example, certain genes have been tightly linked to higher cognitive functions. The defining human trait of syntactical language depends at least in part on a mysterious gene called *FOXP2*, which is necessary for proper speech and language. Scientists can trace the evolutionary history of *FOXP2* in the genomes of humans around the world, our close ancestors, and our extinct human relatives. Neanderthals, for example, shared our variant of *FOXP2* (Krause et al. 2007). We have discovered this gene's function through characteristic language deficiencies in a family with many members who had an impaired copy of the gene; we have further expanded upon this knowledge with model organism experiments (Vargha-Khadem et al. 2005). Only two amino acid changes separate our human version of *FOXP2* from that of chimpanzees, our nearest living relative; despite this tiny difference, chimpanzees do not have any communication system that we would call language.

The fossil record can even tell us information about the cognitive capacity of our ancestors and sister groups. Fossil endocasts of human brains – that is, the inside of preserved skulls of our human relatives – give us information about how large their brains were and sometimes even whether or not they had enlargement of certain regions associated with language processing, such as Wernicke's Area and Broca's Area (Jerison 2012). Braincases are fragile and are rare to discover in the fossil record, but they can reveal the brain volume of an extinct human and help us understand its relative cognitive abilities.

Finally, indirect correlates in the paleontological and archeological record provide clues to the nature of human cognition. For example, cave art and artistic engravings are associated with increasing creativity and complex psychology. Even more simply, tool use is associated with intelligence. Of course, humans are not the only toolmakers and users. For example, New Caledonian Crows (who live on a peculiar South Pacific island) are sophisticated toolmakers (With New Caledonia's relative isolation, rich metal deposits, and friendly climate, one cannot help but wonder: is there an alternate world where intelligent crows dominate the globe?). But the existence of hominin-made tools in the fossil record tells us a bit about cognitive development and

technological advances over time. When these tools are found in close company with actual hominin fossils, we can learn even more. The fossil record is filled with stone tools from our ancestors and our hominin sister taxa, including stone axes, stone choppers, stone flakes for cutting, and hammerstones. The oldest stone tools were long thought to be made 2.6 million years ago, in eastern Africa, by the aptly named *Homo habilis.* However, a wealth of tools found at Lake Turkana in Kenya date back even farther to 3.3 million years (which is prior to the evolution of the genus *Homo*). Thus, scientists think that these early tools may even have been *Australopithecus afarensis* or *Kenyanthropus platyops* (Harmand et al. 2015).

Finally, evolutionary psychology can draw upon behavioral and cognitive studies of extant nonhuman animals. Most useful for these purposes are (i) our relatives, most notably great apes and other primates, and (ii) distantly related species who convergently evolved similarly enlarged brains, complex social behaviors, and sophisticated cognition. From our primate relatives, we have learned much about the evolutionary roots of primate social structure, behavior, cognition, and perception. For example, a study of monkeys who were introduced to a system of currency suggests that the roots of "loss aversion" (our exaggerated "dislike" for losses that exceeds the corresponding "like" for an equivalent gain) run deep in our evolutionary history (Chen et al. 2006). Cetaceans, elephants, corvids, and other large-brained animals have given us further information about *in what circumstances* and *along what pathways* complex cognition has arisen in evolutionary history. For example, with the notable exception of the short-lived, asocial, intelligent octopus, most animals who have convergently evolved large brains and complex cognition are social and long-lived.

Through fossil evidence (such as brain endocasts), archeological and paleontological records (such as cave paintings and tools), genetic history of genes such as the famous *FOXP2*, genetic and molecular studies of sensory perception, and comparative cognition studies of living organisms, we can begin to understand the evolution of our large, unusual brains.

Conclusion

Over time, species change and diverge under the influence of natural selection, genetic drift, and population effects such as bottlenecks. Scientists can learn much about the evolutionary history of extant organisms, and their relatedness to each other, by examining the fossil record and genetic evidence. Natural selection is a powerful force that has shaped our own evolution, and that of all living things. Natural selection does not select "good" traits; rather, it selects traits that allow organisms to survive and reproduce. Evolution is not a staircase describing the ascent of man; rather, it is a complicated bush with myriad organisms adapted to myriad environments. Interpreting evidence to make evolutionary conclusions is difficult, particularly when scientists seek to understand complicated traits such as human intelligence, which have a spotty fossil record and complex genetic and environmental underpinnings. Nonetheless, no trait can be completely understood outside of an evolutionary framework. Understanding evolution can help explain where we humans came from, where we are going, and why the way we are. As Theodosius Dobzhansky famously wrote, "nothing in biology makes sense except in the light of evolution".

Cross-References

▶ Adaptations
▶ Misconceptions In Evolutionary Psychology

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