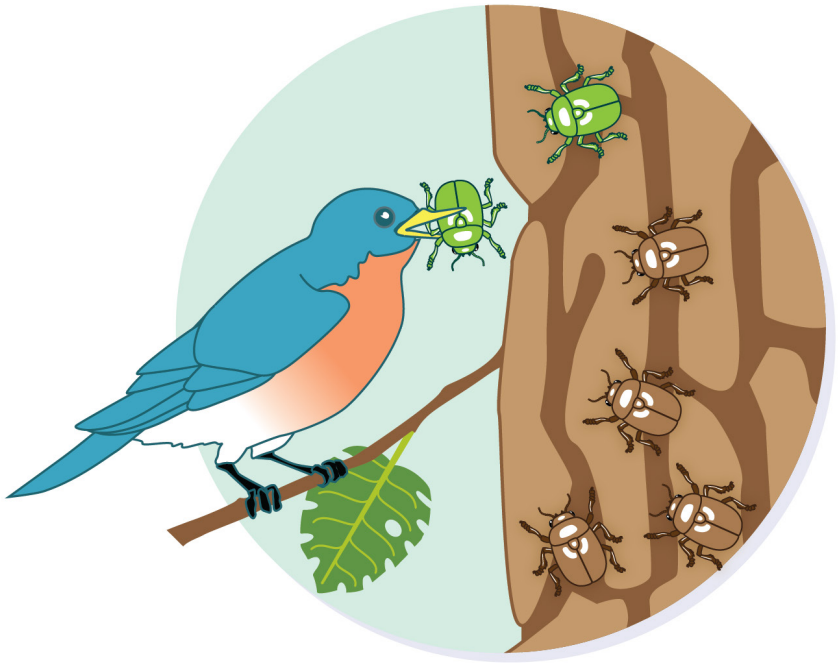
Mechanisms: the processes of evolution

Evolution is the process by which modern organisms have descended from ancient ancestors. Evolution is responsible for both the remarkable similarities we see across all life and the amazing diversity of that life — but exactly how does it work? Here, we'll find out.



Descent with modification

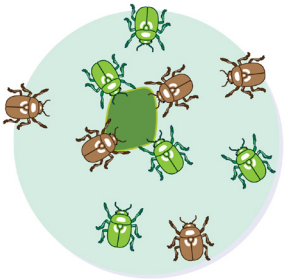
We've defined evolution as descent with modification from a common ancestor, but exactly what has been modified? Evolution occurs when there is a change in the heritable information passed from one generation to the next. Typically, we think of biological evolution as changes in gene frequency within a population over time – if, say, birds with genes that produce wide beaks went from being rare to being common over multiple generations. But biological evolution also includes changes in DNA that does not code for genes and changes in heritable information not encoded in DNA at all. In all of these cases, the modifications are heritable and can be passed on to the next generation — which is what really matters in evolution: long term change. Here, we'll focus on changes in genes and other genetic elements (e.g., in non-coding DNA) as they relate to evolution.

Compare these two examples of change in beetle populations. Which one is an example of evolution?

1. Beetles on a diet

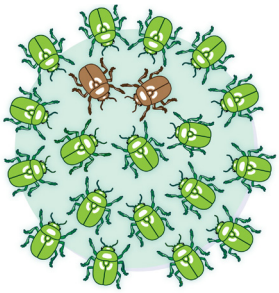
Imagine the population experiences many years of drought in which there are few plants that the beetles can eat.

All the beetles have the same chances of survival and reproduction, but because of food restrictions, the beetles in later generations are smaller than the pre-drought generations of beetles.

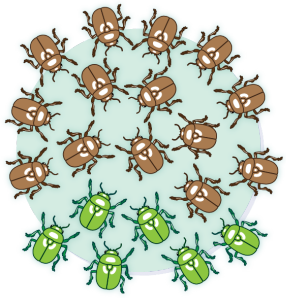


2. Beetles of a different color

Most of the beetles in the population (say 90%) have a gene for green coloration and a few of them (10%) have a gene that makes them brown.



Several generations later, things have changed: brown beetles are more common and make up 70% of the population.



Which example illustrates descent with modification? The difference in size in example 1 came about because of environmental influences — the low food supply — not because of a change in heritable information. Therefore,

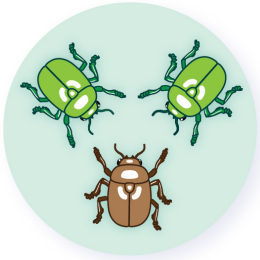
example 1 is not evolution. Because the small body size in this population was not genetically determined, it is temporary: if the beetles' food supply is restored, this generation of small-bodied beetles will produce normal size offspring.

The shift in color frequency in example 2 is definitely evolution: these two generations of the same population are genetically different. There has been a change in heritable information, and this shift will be passed down to the next generation (possibly with further changes introduced). But how did it happen?

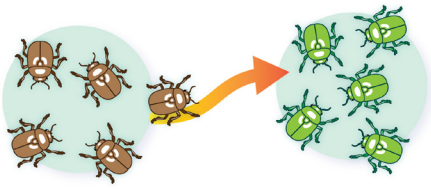
Mechanisms of change

Each of these four processes is a basic mechanism of evolutionary change and is illustrated with an example of gene frequency change in a beetle population with brown and green individuals.

Mutation: A mutation could cause beetle parents with genes for bright green coloration to have offspring with a gene for brown coloration. That would make genes for brown coloration more frequent in the population than they were before the mutation.

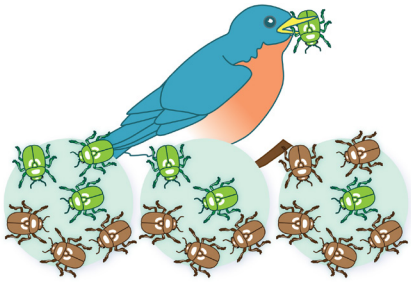


Migration: Some individuals from a population of brown beetles might have moved into a population of green beetles. That would make genes for brown coloration more frequent in the green beetle population than they were before the brown beetles migrated into it. In evolutionary biology, migration is also called “gene flow.”



Genetic drift: In one generation, brown beetles happened to have many offspring survive to reproduce. In the same generation, a number of green beetles were killed randomly when someone stepped on them and had no offspring. The next generation had more brown beetles than the previous generation — but just by chance. These chance changes from generation to generation are known as genetic drift.

Natural selection: Imagine that green beetles are easier for birds to spot (and hence, eat). Thus, brown beetles are a little more likely to survive to produce offspring. They pass their genes for brown coloration on to their offspring. So in the next generation, brown beetles are more common than they were in the previous generation.



All of these mechanisms can cause changes in the frequencies of genes and other genetic elements in populations, and so all of them are mechanisms of evolutionary change. However, natural selection and genetic drift can only change the frequency of different genes and genetic elements (e.g., making wide beaks or green beetles more or less common); they cannot introduce fundamentally new traits to a population.

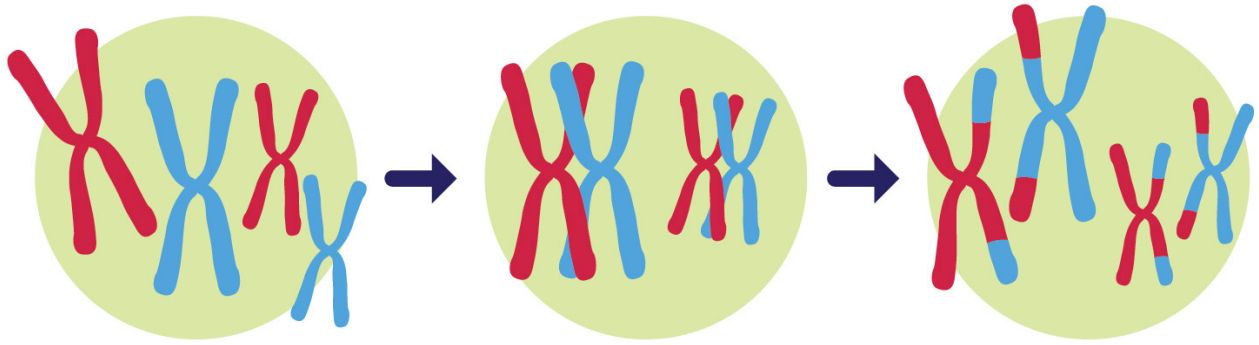
So, what are the sources of new genetic variants – i.e., genetic variation?

Genetic variation

Without genetic variation, some key mechanisms of evolutionary change like natural selection and genetic drift cannot operate.

There are three primary sources of new genetic variation:

1. **Mutations** are changes in the information contained in genetic material. (For most of life, this means a change in the sequence of DNA.) A single mutation can have a large effect, but in many cases, evolutionary change is based on the accumulation of many mutations with small effects.
2. **Gene flow** is any movement of genetic material from one population to another (e.g., through migration) and is an important source of genetic variation.
3. **Sex** can introduce new gene combinations into a population. This genetic shuffling is another important source of genetic variation.



Genetic shuffling is a source of variation.

Mutations

Mutations are changes in the information contained in genetic material. For most of life, this means a change in the sequence of DNA, the hereditary material of life. An organism's DNA affects how it looks, how it behaves, its physiology — all aspects of its life. So a change in an organism's DNA can cause changes in all aspects of its life.

Mutations are random

Mutations can be beneficial, neutral, or harmful for the organism, but mutations do not "try" to supply what the organism "needs." In this respect, mutations are random — whether a particular mutation happens or not is unrelated to how useful that mutation would be.

Not all mutations matter to evolution

Since all cells in our body contain DNA, there are lots of places for mutations to occur; however, not all mutations matter for evolution. Somatic mutations occur in non-reproductive cells and so won't be passed on to offspring.

For example, the yellow color on half of a petal on this red tulip was caused by a somatic mutation. The seeds of the tulip do not carry the mutation. Cancer is also caused by somatic mutations that cause a particular cell lineage (e.g., in the breast or brain) to multiply out of control. Such mutations affect the individual carrying them but are not passed directly on to offspring.

Photo from Wikipedia, by LepoRello under CC BY-SA 3.0.

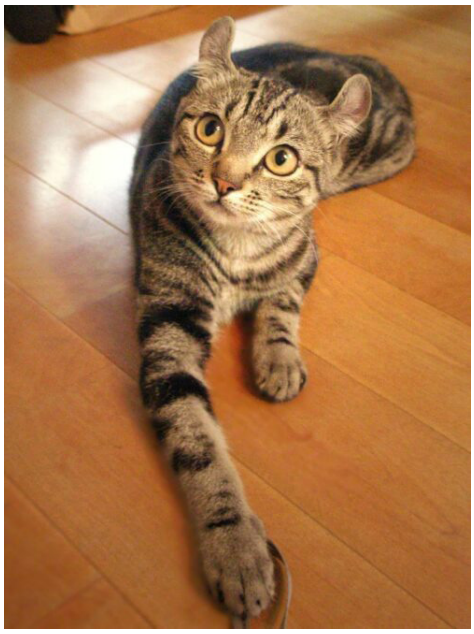
The only mutations that matter for the evolution of life's diversity are those that can be passed on to offspring. These occur in reproductive cells like eggs and sperm and are called germline mutations.

The effects of mutations

A single germline mutation can have a range of effects:

1. No effect or neutral effect

Some mutations don't have any noticeable effect on the organism. This can happen in many situations: perhaps the mutation occurs in a stretch of DNA with no function, or perhaps the mutation occurs in a protein-coding region, but does not affect the amino acid sequence of the protein. Other mutations have a noticeable effect, but one that doesn't seem to help or hurt. For example, a single mutation caused this cat's ears to curl backwards slightly, a trait that doesn't seem to affect its health.



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2. Detrimental effect

Some mutations harm an organism's ability to survive and reproduce. For example, in humans, Marfan syndrome is caused by a mutation affecting a protein that forms part of connective tissue, leading to heart problems and other health challenges. Detrimental mutations known as lethals disrupt DNA critical to survival and cause the death of the organism.

3. Beneficial effect

Other mutations are helpful to the organisms that carry them. For example, DDT resistance in insects is sometimes caused by a single mutation. While resistant insects might be downer for us, they are undoubtedly helpful for bugs trying to survive on pesticide-laden crops.

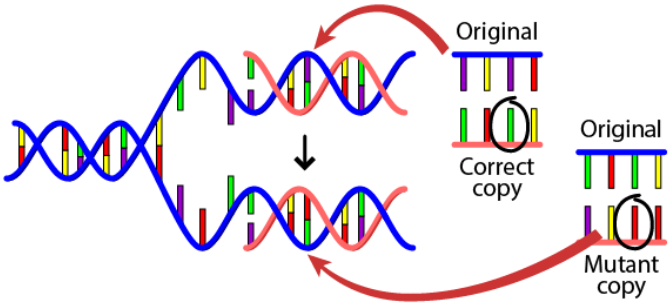
According to popular culture, it seems that mutations mainly cause either cancer or superpowers. Of course, the cancer is true enough. But in the real world, beneficial mutations are rare. Most mutations have no effect or a detrimental effect. And major evolutionary change (e.g., the "superpower" of flight in bats!) generally involves the accumulation of many, many mutations over many, many generations.

The causes of mutations

Mutations happen for several reasons.

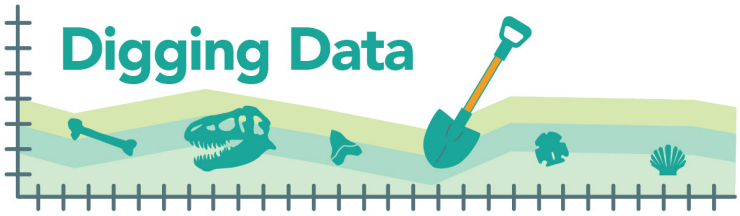
1. DNA spontaneously breaks down or is not copied accurately

Most of the mutations that we think matter to evolution are "naturally-occurring." For example, when a cell divides, it makes a copy of its DNA — and sometimes the copy is not quite perfect. That small difference from the original DNA sequence is a mutation. Spontaneous breakdown of DNA can also cause mutations.



2. External influences can cause mutations

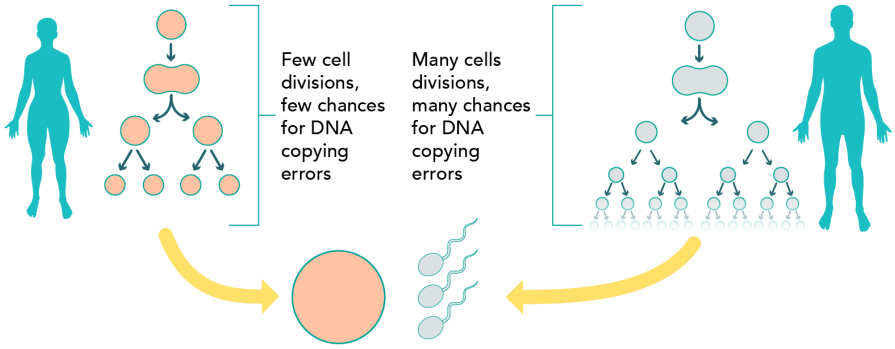
Mutations can also be caused by exposure to specific chemicals or radiation that cause the DNA to break down. Cells do have mechanisms to repair damaged or altered DNA molecules, but they aren't perfect. Whatever the cause, mutations occur any time a cell ends up carrying a DNA sequence slightly different than the original.



The causes of mutation

Mutations – changes in the genetic sequence of DNA or RNA – are the raw material for evolution. Natural selection, genetic drift, and other evolutionary processes act on genetic variation – and that genetic variation starts with mutation. Even if a genetic variant is introduced to a population through migration, ultimately, that variant got its start as a mutation. So understanding where, when, why, and how often mutations occur is key in understanding how evolution happens. Today, quick and inexpensive DNA sequencing means biologists can take a closer look than ever before at the process of mutation. What they are learning is sometimes surprising.

In humans, each baby has around 70 brand new or "de novo" mutations. De novo mutations occur in the reproductive cells of parents and are passed on to the child. Evidence suggests that most de novo mutations in a child come from the sperm that helped create that child, and relatively few mutations come from the egg. Biologists thought this made sense. In humans, beginning at puberty, the cells that produce sperm divide (and copy their DNA) throughout adulthood, leading to vast numbers of sperm. In contrast, in a person with ovaries, all the DNA copying leading up to egg production is completed before that person is even born.[1] The cells that produce sperm just go through many more cycles of DNA replication and cell division than do the cells that produce eggs.[2] If most mutations happen because a cell makes an error when it copies its DNA, producing a new DNA strand that differs slightly in sequence compared to the original, then we'd expect sperm to be the source of most new mutations. All those cell divisions in the cells that eventually lead to sperm provide many opportunities for copying mistakes to occur.

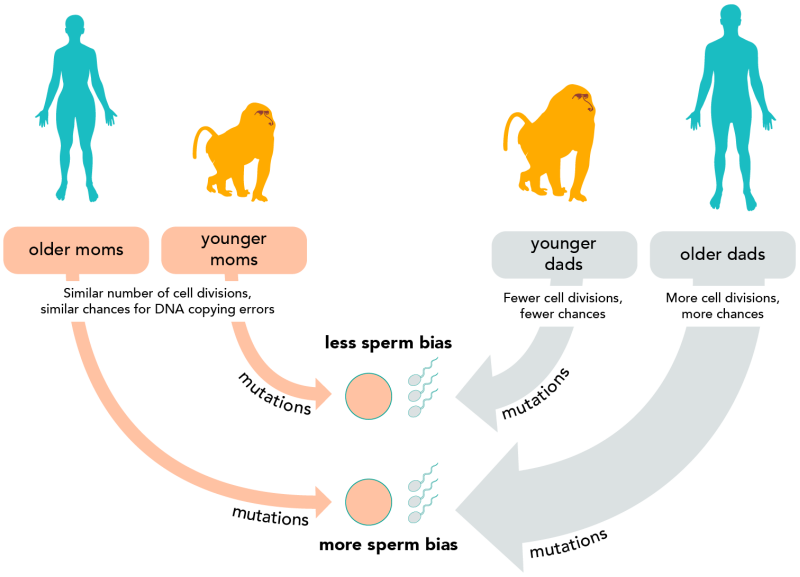


Biologists Felix Wu, Alva Strand, Laura Cox, Carole Ober, Jeffrey Wall, Priya Moorjani, and Molly Przeworski work at different universities and research centers, but they all wanted to know, **is it** *really* **the case that most new mutations in humans are caused by copying errors when cells divide?** Some evidence had already suggested that copying errors are not the whole story when it comes to heritable mutations.

The research team. Top row: Felix Wu, Alva Strand, Laura Cox, Carole Ober. Bottom row: Jeff Wall, Priya Moorjani, Molly Przeworski. Photos reproduced with permission from the individuals.

Hypothesis

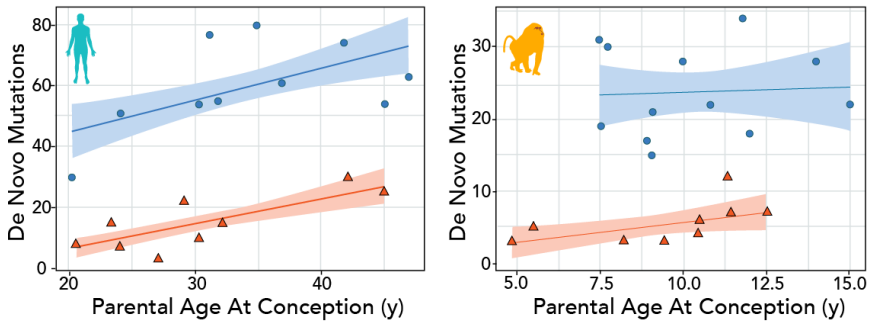
The research team set out to test the hypothesis that **most de novo mutations occur because of copying errors.** They looked at what percent of new mutations in a baby come from DNA carried by sperm, as opposed to from DNA carried by the egg; we'll call this "sperm bias" in new mutations. If new mutations mostly come from copying errors, then the number of mutations contributed by eggs should not be much affected by the age of the person contributing the egg (since, in primates, all the DNA copying that leads up to egg production occurs before that future parent is even born). However, the number of mutations contributed by the sperm *should* increase with the age of the genetic father (since DNA copying and cell divisions leading to sperm are ongoing from puberty throughout adulthood). Overall, this means that **if the hypothesis were true, we'd expect to see that species with younger genetic fathers (and a shorter interval between puberty and fatherhood) should have less sperm bias in new mutations than species with older genetic fathers (and a longer interval between puberty and fatherhood).**



Data

The team decided to compare mutations in humans to mutations in olive baboons (*Papio anubis*). The two primates are closely related, but male baboons go through puberty at age 6 and reproduce at age 10 on average, while humans that produce sperm typically start puberty around age 13 and don't reproduce until age 32 on average. This means that the cells that produce baboon sperm go through about 4 years' worth of DNA replication and cell division before a sperm leads to offspring, while the cells that produce human sperm go through about 19 years' worth of DNA replication and cell division before a sperm leads to a child! That's a big difference, providing lots more opportunity for mutations caused by DNA copying errors to accumulate in the sperm-producing cells of humans compared to those of baboons. To get data on mutations, the team sequenced the genomes of three generations of three different human families (26 people total) and three generations of two baboon families (29 baboons total).

They focused on tallying up selected spots in the genome where the DNA sequences of the parents were the same, but their child's genetic sequence was different from that of both parents.[3] The only way to explain this outcome is if one of the parents contributed a brand new mutation to the child. Then the researchers used additional sequence information to determine which parent (mother or father) had contributed each mutation.[4] This is what they found:

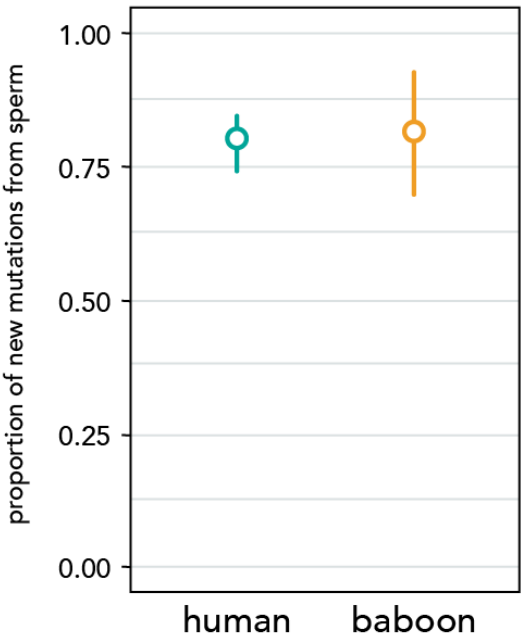


In these graphs, each offspring in which new mutations were identified is represented by a circle and a triangle.

The circle indicates the number of mutations contributed by the sperm and the triangle indicates the number contributed by the egg. The lines show the overall relationship between age at conception (on the *x*-axis) and mutations (on the *y*-axis). The shading around these regression lines indicates the 95% confidence intervals.

Right away, we can observe a few things. First, the blue circles are always positioned above the red triangles, indicating that sperm contribute more mutations than do eggs. That's just as we'd expect based on other research. Second, in humans, older genetic parents do seem to pass on more mutations to offspring, as shown by the upward slope of the regression lines. In baboons, the mothers' age is associated with more mutations (the line slopes upwards), while the regression line for fathers is nearly flat – suggesting that older fathers *don't* contribute more mutations. This result goes against previous findings about mutations and age. But if you look at the male baboon data closely, you'll notice that the blue confidence interval is wider than the others. This means that the data vary a lot, making it hard to detect patterns without a really large sample. So it is possible that baboon fathers, like humans, do pass on more mutations to their offspring as they age, but there were not enough data in this experiment to detect the pattern.

The researchers used their data to calculate the proportion of all new mutations that came from the father (i.e., were paternal), as shown in the graph below. The vertical lines represent the 95% confidence intervals for each point. A chi-squared test showed that the two values are not significantly different ($p = 0.91$). **Even though the baboons were much younger than the humans when they reproduced (and the cells that produce their sperm have been through many fewer rounds of DNA replication and cell division), the degree of sperm bias in the two species is similar! That does not match the expectation generated by the hypothesis that most new mutations come from copying errors.**



If not copying errors, what is the source of the new mutations that primate offspring are born with? The research team suspects that sloppy DNA fixes play a role. DNA, including the DNA within sperm and eggs, is easily damaged. And when it is, a cell does its best to put the strand back together perfectly. But sometimes it mistakenly substitutes one genetic letter for another, generating a mutation. The idea that errors in DNA repair are an important source of mutations that matter for evolution fits with many lines of evidence – including the evidence described here that the number of new mutations increases with the age of both genetic parents. After all, we might expect the chances of DNA damage and sloppy repair to be fairly constant over time, so as the cells that lead to eggs and sperm get older, we'd expect them to accumulate more mutations, which are passed on to daughter cells and ultimately, to the eggs and sperm themselves. But exactly how important this process is, as well as how it might vary between the sexes and across species, has yet to be worked out. Furthermore, the idea that DNA damage and poor repair are the main source of de novo mutations does not help us understand why we observe sperm bias in the first place. As our genetic techniques advance further, biologists like Felix, Molly, and their colleagues will have even more data to home in on the processes that lead to mutations, tracing genetic variation back to its original source.

Stepping into science

The research team was led by Felix Wu, a graduate student at Columbia University in New York City, and Molly Przeworski, a professor there. Molly did grow up in a large city and spent most of her leisure time reading novels. She was utterly uninterested in math or science—her least favorite topic in school was biology. But a great teacher drew her into math in college and after a series of false starts, she discovered genetics and evolutionary biology.

Footnotes:
[1]The initial stages of oogenesis (egg production) occur during fetal development before birth. During this stage, the DNA that will wind up in eggs

is copied in preparation for cell division – and it is during this stage that mutations caused by copying errors might occur. However, this process is then halted before any cell division takes place. The final cell divisions that produce eggs will actually occur during the reproductive years, during the menstrual cycle and fertilization – but no DNA replication occurs at this time.

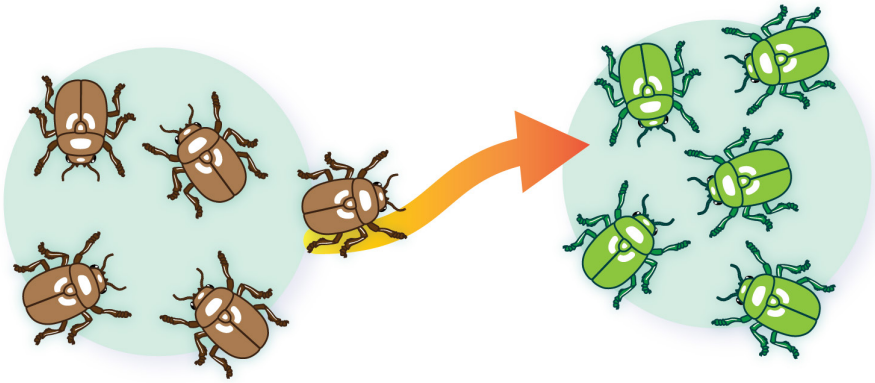
[2]Technically, we are focused on DNA replication in the stem cells that produce eggs and sperm. The stem cells that eventually lead to sperm go through more cycles of DNA replication than do the stem cells that produce eggs, providing more opportunities for DNA replication errors to occur.

[3]The researchers focused on single nucleotide polymorphisms (SNPs) – single base pair differences. To be extra certain that they were detecting brand new mutations, they focused on genome positions where the child was a heterozygote and both parents were homozygotes. They then checked their data using several different methods.

[4]This is trickier to do than it might sound. After all, primates are diploid; if a primate offspring shows up with a mutation on one chromosome out of a pair, how do we tell which chromosome, the one from the egg or the one from the sperm, contributed that mutation? Figuring it out requires looking at nearby positions on the chromosome – i.e., figuring out the genetic background on which the mutation most likely occurred. If the new mutation is located on a chromosome near a signature sequence present only in the genetic father, we know that person must have contributed the mutation.

Gene flow

Gene flow — also called migration — is any movement of individuals, and/or the genetic material they carry, from one population to another. Gene flow includes lots of different kinds of events, such as pollen being blown to a new destination or people moving to new cities or countries. If genetic variants are carried to a population where they previously did not exist, gene flow can be an important source of genetic variation. In the graphic below, a beetle carries the gene version for brown coloration from one population to another.



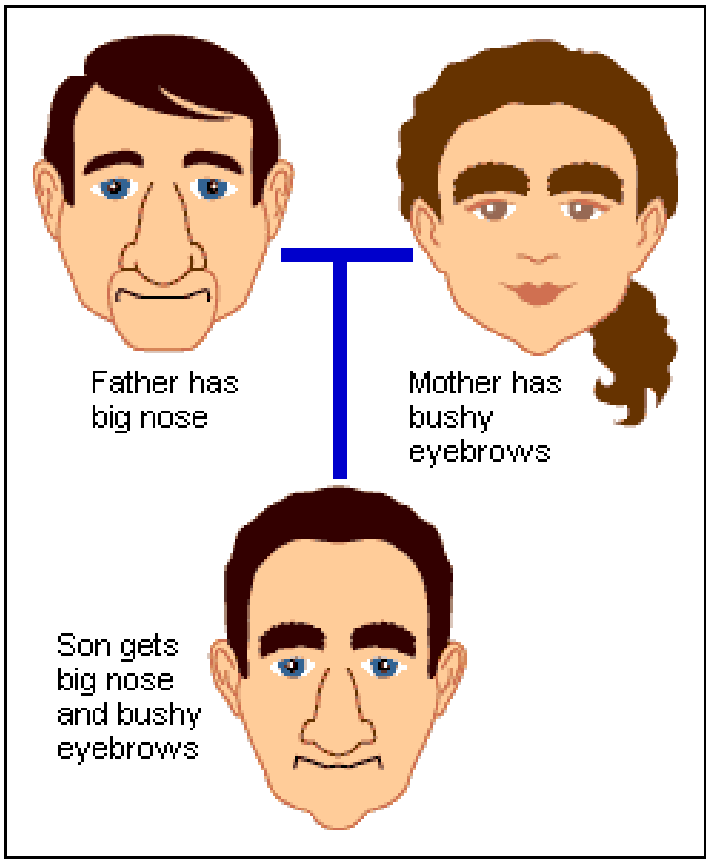
The genetic variation in modern human populations has been critically shaped by gene flow. For example, by sequencing ancient DNA, researchers have reconstructed the entire Neanderthal genome – and they've found that many snippets of these archaic sequences live on in modern humans. It's clear that ancient humans and Neanderthals interbred, and that this gene flow introduced new genetic variation to the human population. Furthermore, this ancient gene flow seems to affect who we are today. Neanderthal gene versions have been linked to immune functions, metabolic functions (e.g., affecting one's risk of developing diabetes), and even skin color.

Sex and genetic shuffling

Sex can introduce new gene combinations into a population and is an important source of genetic variation.

You probably know from experience that siblings are not genetically identical to their parents or to each other (except, of course, for identical twins). That's because when organisms reproduce sexually, some genetic "shuffling" occurs, bringing together new combinations of genes. For example, you might have bushy eyebrows and a big nose since your mom had genes associated with bushy eyebrows and your dad had genes associated with a big nose. These combinations can be good, bad, or neutral. If your spouse is wild about the bushy eyebrows/ big nose combination, you were lucky and hit on a winning combination!

This shuffling is important for evolution because it can introduce new combinations of genes every generation. For example, in a particular population, plants with reddish flowers and plants with longer more tubular flowers might each do fine on their own – but if sex and genetic shuffling produced a plant with both traits (red tubular flowers), the combination might attract a new pollinator (hummingbirds) and alter the evolutionary trajectory of the lineage. Of course, sex and genetic shuffling can also break up good combinations of genes and form bad ones.



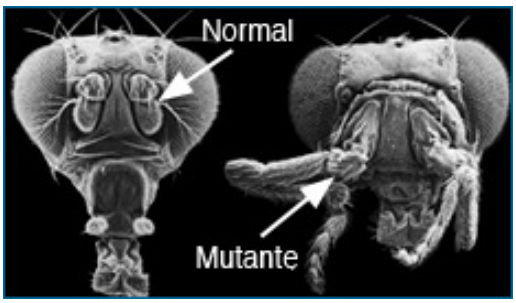
Development

Development is the process through which a fertilized egg, the earliest stage of an embryo, becomes an adult organism. Throughout development, an organism's genotype is expressed as a phenotype, exposing genes and the genetic elements that control their expression to the action of natural selection. Genetic variation in genes affecting development seems to have played an important role in evolution.

Explaining major evolutionary change

Changes in the genes controlling development can have major effects on the morphology of the adult organism. Because these effects are so significant, scientists suspect that changes in the expression of developmental genes have helped bring about large-scale evolutionary transformations. Developmental changes, as well as new genes, may help explain, for example, how some hoofed mammals evolved into ocean-dwellers, how water plants invaded the land, and how small, armored invertebrates evolved wings.

Mutations in the genes that control fruit fly development can cause major morphology changes, such as two pairs of wings instead of one.

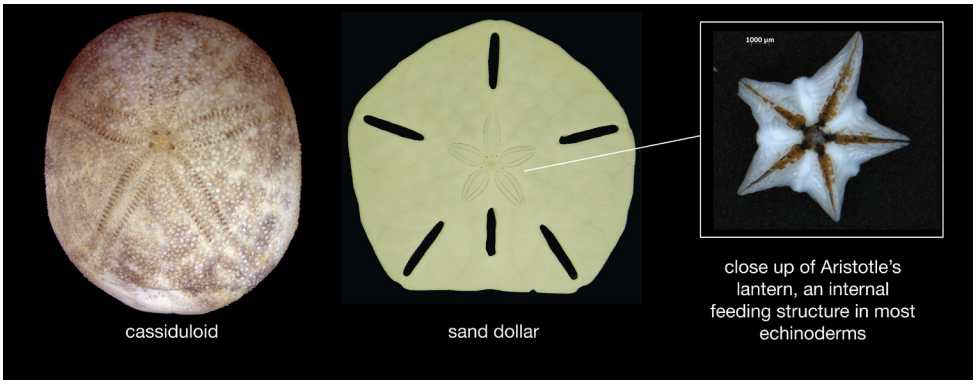


Another developmental gene mutation can cause fruit flies to have legs where the antennae normally are, as shown in the fly on the right. Fruit fly images courtesy of Jean-Michel Muratet, Syndicat National des Ophtalmologistes de France (SNOF).

Developmental processes may also constrain the sorts of phenotypes that genetic variation can lead to, and so might prevent certain characters from evolving in certain lineages. For example, development may help explain why there are no truly six-fingered tetrapods among living species.

Learning about evolutionary history

In addition, an organism's development may contain clues about its history that biologists can use to help build evolutionary trees. For example, the relationship between sand dollars and an unusual group of sea urchins called cassiduloids was once a conundrum. The two groups strongly resemble each other and so seemed closely related; however, sand dollars have a complicated jaw structure (called the lantern) that cassiduloids lack. Could they really be close relatives with such a big difference? The answer, it turns out, was yes. Scientists discovered that developing cassiduloid embryos pass through a stage where they have a lantern; it was merely lost in the adult stage through evolution. Sand dollars and cassiduloids have more in common than one might think from their adult forms alone, and they do occupy the same branch of the sea urchin family tree after all.



All photos courtesy of Camilla Souto, PhD.

Genetic drift

Genetic drift is one of the basic mechanisms of evolution.

In each generation, some individuals may, just by chance, leave behind a few more descendants (and genes, of course!) than other individuals. The genes and other genetic elements of the next generation will be those of the "lucky" individuals, not necessarily the healthier or "better" individuals. That, in a nutshell, is genetic drift. It happens to ALL populations — there's no avoiding the vagaries of chance.

Earlier we used this hypothetical cartoon. Genetic drift affects the genetic makeup of the population, but unlike natural selection, through an entirely random process. So although genetic drift is a mechanism of evolution, it doesn't work to produce adaptations.

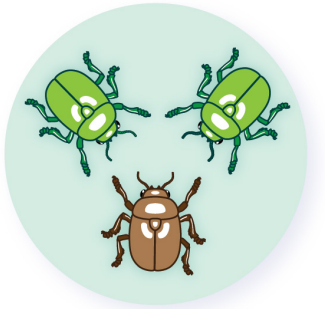
Natural Selection

Natural selection is one of the basic mechanisms of evolution, along with mutation, migration, and genetic drift.

Darwin's grand idea of evolution by natural selection is relatively simple but often misunderstood. To see how it works, imagine a population of beetles:

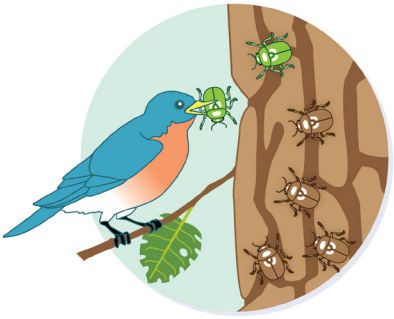
1. There is variation in traits.

For example, some beetles are green and some are brown.



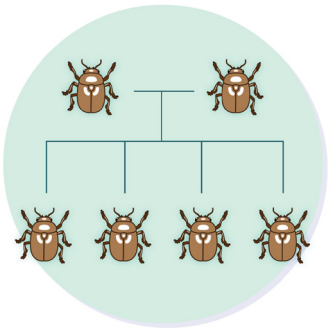
2. There is differential reproduction.

Since the environment can't support unlimited population growth, not all individuals get to reproduce to their full potential. In this example, green beetles tend to get eaten by birds and survive to reproduce less often than brown beetles do.

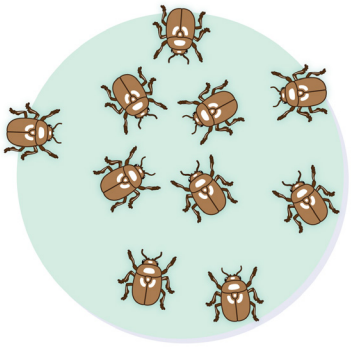


3. There is heredity.

The surviving beetles (more of which are brown) have offspring of the same color because this trait has a genetic basis.



4. End result: The more advantageous trait, brown coloration, which allows the beetle to have more offspring, becomes more common in the population.



If this process continues, eventually, all individuals in the population will be brown If you have variation, differential reproduction, and heredity, you will have evolution by natural selection as an outcome. It is as simple as that.

Natural selection at work

Scientists have worked out many examples of natural selection, one of the basic mechanisms of evolution.

Natural selection can produce impressive adaptations that help organisms survive and reproduce. A few examples are shown below.

Orchids fool wasps into "mating" with them. Orchid and wasp image courtesy of Colin Bower.

Katydids have camouflage to look like leaves. Image © Greg Neise, , GE Neise Digital Communication.

Non-venomous king snakes mimic venomous coral snakes. Images courtesy of Neurotoxin.

Behavior can also be shaped by natural selection. Behaviors such as birds' mating rituals, bees' wiggle dance, and humans' capacity to learn language have genetic components and are subject to natural selection. The male blue-footed booby, for example, exaggerates his foot movements, an adaptation that helps him attract a mate.

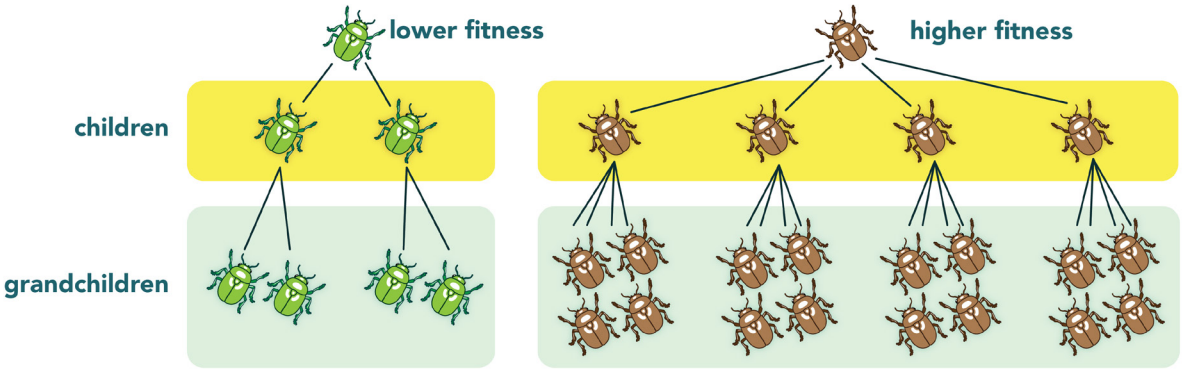
In some cases, we can directly observe natural selection occurring. Very convincing data show that the shape of finches' beaks on the Galapagos Islands has tracked weather patterns: after droughts, the finch population has deeper, stronger beaks that let them eat tougher seeds.

In other cases, human activity has led to environmental changes that have caused populations to evolve through natural selection. A striking example is that of the peppered moth, which may have either light or dark coloration. During the Industrial Revolution, when air pollution darkened tree trunks, dark-colored forms were favored because they were better camouflaged and so became more common. When pollution was later reduced, light-colored forms rebounded and became more common. Natural selection triggered by human activity can often be observed and documented.

Blue-footed booby image courtesy of Ian Skipworth.

Evolutionary fitness

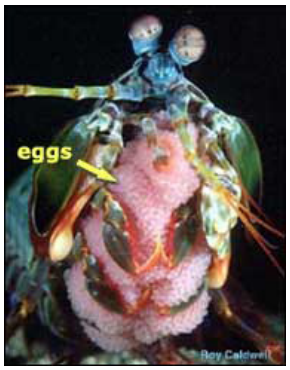
Evolutionary biologists use the word fitness to describe how good a particular genotype is at leaving offspring in the next generation relative to other genotypes. So if brown beetles consistently leave more offspring than green beetles because of their color, you'd say that the brown beetles had a higher fitness. In evolution, fitness is about success at surviving and reproducing, not about exercise and strength.



Of course, fitness is a relative thing. A genotype's fitness depends on the environment in which the organism lives. The fittest genotype during an ice age, for example, is probably not the fittest genotype once the ice age is over.

Fitness is a handy concept because it lumps everything that matters to natural selection (survival, mate-finding, reproduction) into one idea. The fittest individual is not necessarily the strongest, fastest, or biggest. A genotype's fitness includes its ability to survive, find a mate, produce offspring — and ultimately leave its genes in the next generation.

A penguin and its two offspring. Photo by Jeff Abbas © California Academy of Sciences.



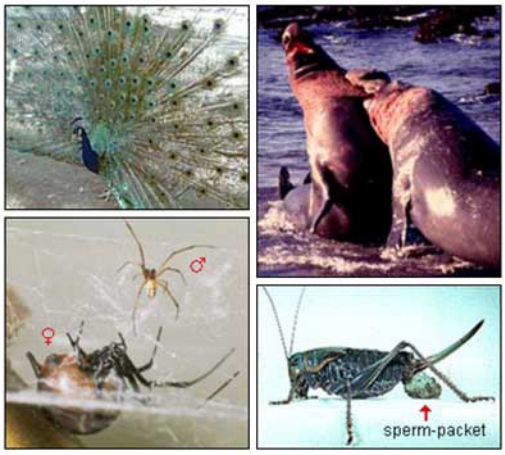
A stomatopod (or mantis shrimp) with thousands of small, light pink eggs. Photo by Roy Caldwell.

A male peacock with its tail feathers displayed. 1Photo courtesy of Rock Maple Farm.

Caring for your offspring (above left), producing thousands of young — many of whom won't survive (above right) — and sporting fancy feathers that attract females (left) are a burden to the health and survival of the parent. These strategies do, however, increase fitness because they help the parents get more of their offspring into the next generation.

We tend to think of natural selection acting on survival ability — but, as the concept of fitness shows, that's only half the story. When natural selection acts on mate-finding and reproductive behavior, biologists call it sexual selection.

Sexual Selection



Peacock image courtesy of Rock Maple Farm; Elephant seals image courtesy of Craig's Homepage; Mormon cricket image courtesy of Grasshoppers of Wyoming and the West; Redback spiders image courtesy of Andrew Mason, University of Toronto at Scarborough

Sexual selection is a "special case" of natural selection. Sexual selection acts on an organism's ability to obtain (often by any means necessary!) or successfully copulate with a mate.

Sexual selection has shaped many extreme adaptations that help organisms find mates: peacocks (top left) maintain elaborate tails, elephant seals (top right) fight over territories, fruit flies perform dances, and some species deliver persuasive gifts. After all, what female Mormon cricket (bottom right) could resist the gift of a juicy sperm-packet? Going to even more extreme lengths, the male redback spider (bottom left) literally flings itself into the jaws of death in order to mate successfully.

Sexual selection is even powerful enough to produce features that are harmful to the individual's survival. For example, extravagant and colorful tail feathers or fins are likely to attract predators as well as interested members of the opposite sex.

Sexual selection

Maydianne Andrade, evolutionary biologist and Professor and Vice Dean at the University of Toronto Scarborough

The venomous female redback spider – also known as the Australian black widow – poses a danger to humans … and to male redback spiders, which are often eaten by their mates. Males seem to go out of their way to make this happen, flipping themselves over and presenting their abdomens to the female while mating. This behavior might at first seem like one that selection would act against. After all, how could risking one's life be adaptive? Remember that evolutionary fitness is about getting genes into the next generation, not just survival. **Perhaps this extreme behavior is favored by sexual selection because it gives males a fitness boost. But what advantage could it offer?** Biologist Maydianne Andrade made observations and designed a set of experiments to find out

Background

Male redback spiders deliver their sperm to females using specialized mouthparts. If the female is hungry, she will eat the male during the mating process. In the wild, this happens about 65% of the time. Females often mate with more than one male and can store sperm (sometimes for years!) to use later. Females produce multiple egg sacs throughout their lives, each of which can contain hundreds of eggs. Different eggs in a single egg sac may be fertilized by sperm from different fathers.

Female redback spider with egg sac at right. Much smaller male (circled) at left. Photo courtesy of Wikimedia.

Hypotheses

There are several explanations that could lead to the evolution of males' risky mating behavior:

1. The nutrients provided by eating the male are passed on to the eggs/offspring. In this scenario, sexual selection would favor males that offer themselves up as a meal because those males would leave behind more or perhaps more robust eggs that are more likely to hatch into live spiderlings.

2. Eating one's mate decreases the likelihood that a female will mate again with another male. In this scenario, sexual selection would favor the risky behavior because males that allow themselves to be eaten would prevent later matings and, thus, would father more of a female's brood.

3. Males that are eaten mate for longer and so fertilize more of a female's eggs. Perhaps eating a mate takes time, or perhaps females simply allow mates that offer up their abdomens to mate longer. In either case, evolution would favor the risky behavior if it allows a male to father more of the female's offspring than do males that do not offer up a snack.

Maydianne made observations and carried out experiments to test each of these hypotheses.

Data

Hypothesis 1 – Does a female's "snack" give a boost to her eggs? In captive redbacks in the lab, Maydianne compared the number of eggs in and weight of egg sacs from matings where the male was eaten to those from matings in which he was not:

Factor	Cannibalistic matings			Noncannibalistic matings			Mann-Whitney
	*N**	95% CL	Median	*N**	95% CL	Median	*(P)*
Egg sac mass (mg)	9	170 to 230	198	13	155 to 198	179	0.082
Number of eggs per sac	9	223 to 361	256	13	199 to 321	249	0.526

The 95% CL (confidence level) is the range within which the true value is likely to fall (i.e., in 100 cases with similar data, the true value is within this range in 95 of those cases). The Mann-Whitney test looks at whether two samples are likely to come from sources with the same median. The p value of this test indicates the probability that the two samples come from sources with the same median (i.e. are *not* different).

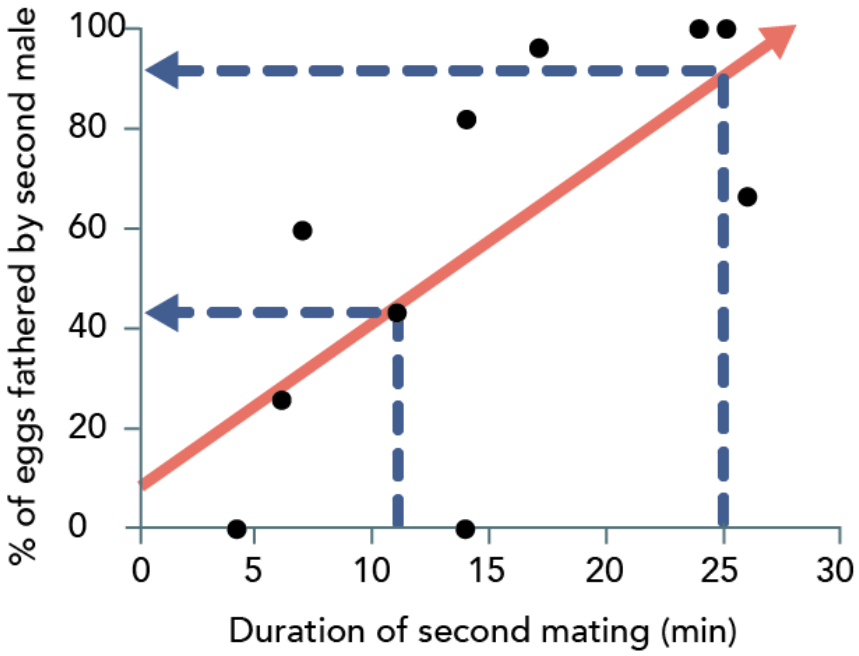
There is a very slight trend towards more eggs and heavier egg sacs resulting from cannibalistic matings (as seen by comparing the pink highlighted medians), but this difference is not significant (blue highlighted box). This contradicts hypothesis 1.

Hypothesis 2 – Did eating a mate decrease the odds that a female would mate again with a different male? In the lab, Maydianne observed females' first and subsequent matings and collected the following data:

Female behavior	% of time she mated again
Ate first mate	33% (3 out 9 matings)
Did not eat first	96% (22 out of 23 matings)

Apparently, skipping breakfast (not eating one's first mate) leaves female redbacks interested in a snack (one's second mate)! These data support hypothesis 2.

Hypothesis 3 – Does self-sacrifice pay off with paternity? Maydianne also observed and timed matings in the lab, and then determined the paternity of the eggs that the female ultimately produced. Maydianne focused on the second male to mate with a female. She thought that a male allowing himself to be eaten might pay off in terms of paternity, particularly if he were able to mate for longer if cannibalized. She observed that cannibalized second males mate for much longer (a median of 25 minutes) than second males that are not eaten (and mate for a median of just 11 minutes). Here are data from 10 matings:



For each mating, she plotted a point (black dot) representing how long the second mating took (x-axis) and the fraction of the eggs in the sac fathered by that second male (y-axis). The data are pretty clear: longer mating is associated with more paternity. This is shown by the upward slope of the regression line (red arrow). The dotted lines and blue arrows show how much a second male can improve his fitness by fathering more eggs if he is eaten and mates for 25 min, as opposed to surviving and mating just 11 minutes. Based on these data, we'd expect self-sacrificers to father 92% of eggs versus just 45% for survivors. These data support hypothesis 3.

Sexual selection seems to be shaping male redback spiders' self-sacrificial mating behavior, not through the nutrition provided by eating one's mate, but through its effect on fertilization rate and female behavior. But note the small sample sizes. More data might make us more confident in this interpretation.

Stepping into science

Maydianne started doing research as an undergraduate. She got interested in studying invertebrates, since she could mimic their natural environments in the lab. She was particularly curious to learn what males contribute to their mates and offspring – so when her Master's advisor told her about the strange behavior of male redback spiders, she was intrigued. And when she realized she'd be able to escape the Canadian winter and visit sunny Australia, she was sold!

Why is sexual selection so powerful?

It's clear why sexual selection is so powerful when you consider what happens to the genes of an individual who lives to a ripe old age but never produced offspring: no offspring means no genes in the next generation, which means that all those genes for living to a ripe old age don't get passed on to anyone! That individual's fitness is zero. Compare that to an individual who does not live very long, but leaves behind children…

Selection is a two-way street

Sexual selection usually works in two ways, although in some cases we do see sex role reversals:

- **Male competition**

  Males compete for access to females, the amount of time spent mating with females, and even whose sperm gets to fertilize her eggs. For example, male damselflies scrub rival sperm out of the female reproductive tract when mating.

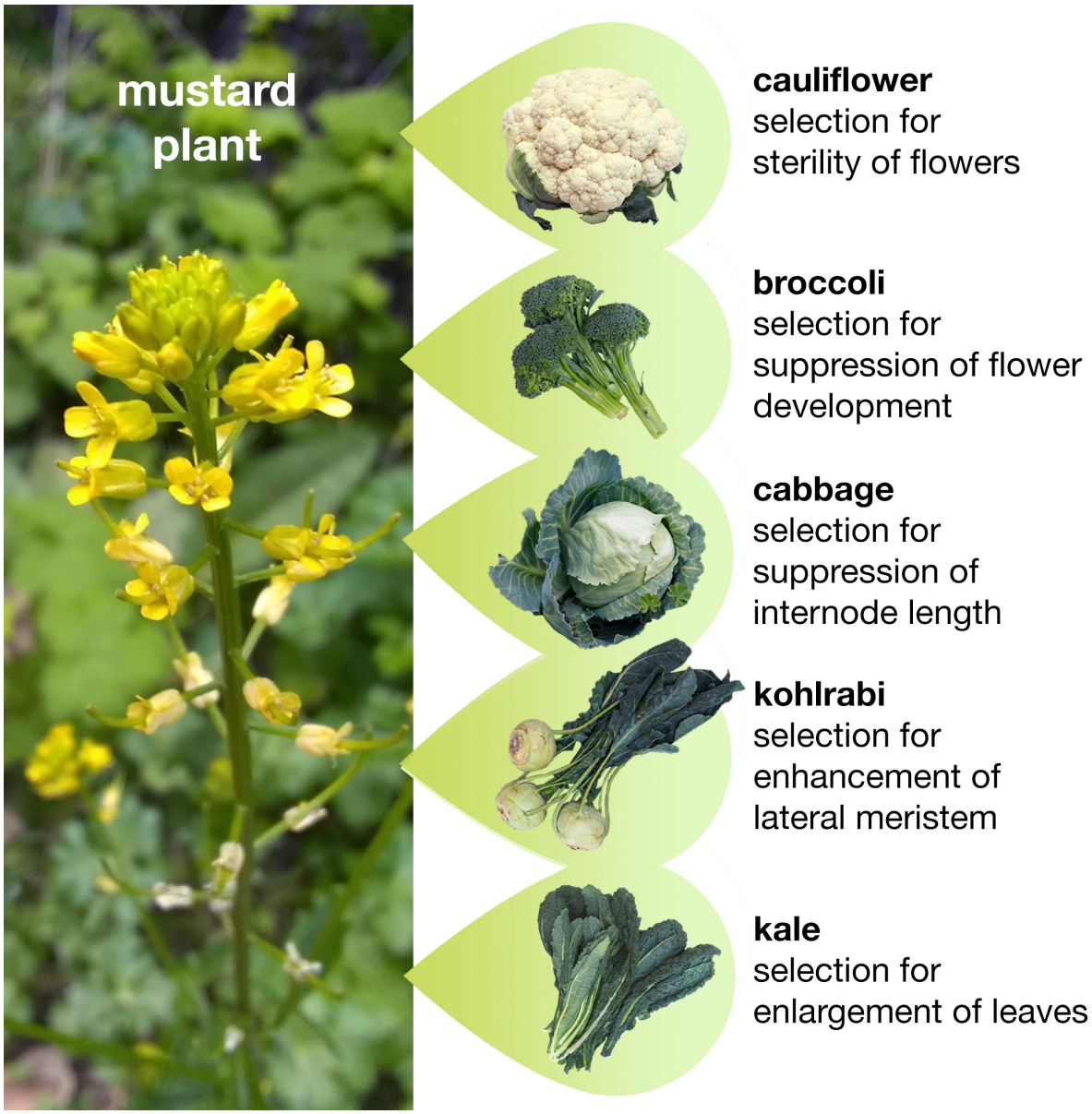
- **Female choice**

  Females choose which males to mate with, how long to mate, and even whose sperm will fertilize her eggs. Some females can eject sperm from an undesirable mate.

Artificial selection

Long before Darwin and Wallace, people were using selection to change the features of plants and animals. Farmers and breeders allowed only the plants and animals with desirable characteristics to reproduce, causing the evolution of farm stock. This process is called artificial selection because people (instead of nature) select which organisms get to reproduce.

As shown below, farmers have cultivated many crops from wild mustard by artificially selecting for certain attributes.



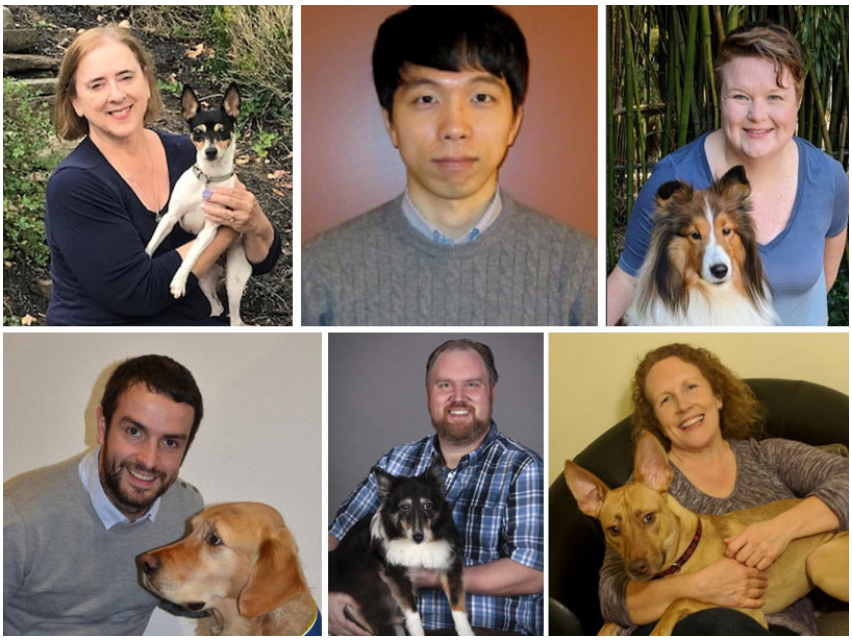
Mustard photo courtesy of Pat Holroyd, all other brassica courtesy of Helina Chin.

Artificial selection

Just as humans developed crop plants and domesticated animals from wild ancestors, we also used artificial selection to create distinct varieties and breeds of these species. Domestic dogs evolved from ancient, now-extinct wolf ancestors tens of thousands of years ago[1] – and then, over the last 200 years, humans further selected subsets of dogs, creating Great Danes, Chihuahuas, and the full gamut of more than 450 breeds.

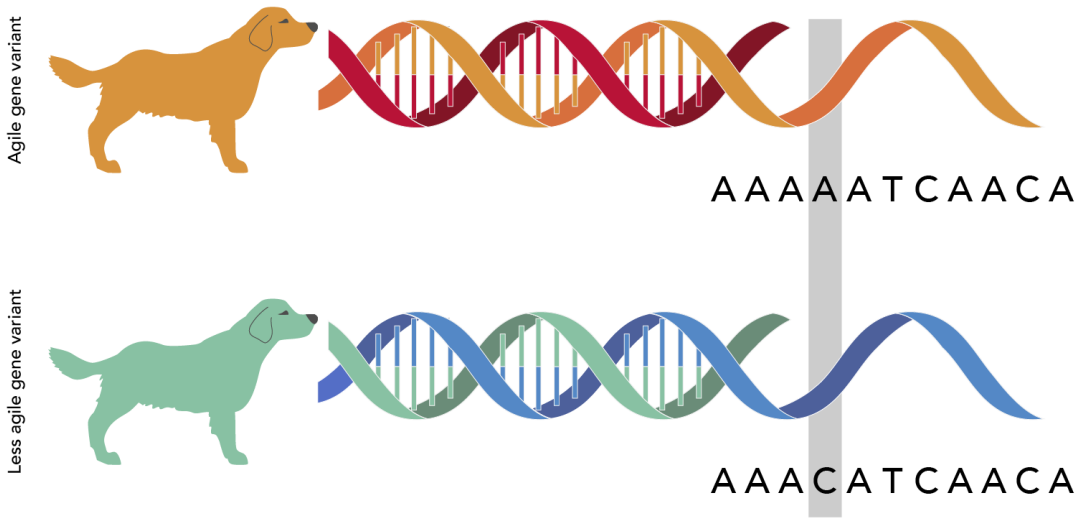
Chihuahua (left) and Chesapeake Bay Retriever (right). Photos provided by Dayna Dreger.

During the process of developing different breeds, the focus was on traits. We didn't know which genes bestowed, for example, swimming skills or rat-catching ability; we just knew that we wanted dogs that were better at pulling fishing nets (leading to Chesapeake Bay Retrievers and Portuguese Water Dogs) and clearing out pests (leading to the Rat Terrier). In recent decades, scientists have been able to track down some of the genes that we unwittingly selected for in different breeds. Elaine Ostrander, a biologist at the National Institutes of Health, is one of these scientists. She and her team study dogs in order to answer basic questions about genetics that have implications for human health.



The team of scientists behind the study: Elaine Ostrander, Jaemin Kim, Dayna Dreger (top row), Jocelyn Plassais, Brian Davis, and Heidi Parker (bottom row). Falina Williams, also an author on the study, is not pictured. Photos provided by Ostrander lab.

In one study, Elaine and a team that included Jaemin Kim, Falina Williams, Dayna Dreger, Jocelyn Plassais, Brian Davis, and Heidi Parker focused on sport hunting dogs, which are an active group of breeds that perform many different jobs. This group includes spaniels, retrievers, and hunting dogs. The team identified 59 genes that seem to have been the targets of recent selection in these dogs.[2] A particular variant (i.e., an allele) of one of these genes – called *ROBO1* – was common in the most agile breeds. These athletic breeds were unusually likely to have an A base at a location in the gene where other breeds were more likely to have a C base. **The initial data suggested that *ROBO1* was one of the genes that humans unwittingly affected when they artificially selected breeds for different sorts of athletic abilities, but the team wanted more evidence.**



Background

Broadly, agility is the ability to move quickly and easily, but for dogs, it can have a more specific meaning. Agility is a sport in which a person directs a dog to run through a complex obstacle course. For a dog to perform well in agility competitions, it must have both the athletic skills to make it through the course quickly and the mental skills to respond nimbly to commands and assess obstacles.

Australian Shepherd in an agility competition. Photo by Ron Armstrong from Helena, MT, USA, CC BY 2.0 via Wikimedia Commons

ROBO1 may be related to those mental skills. From studies of humans, we know that *ROBO1* is involved with brain development. Different versions of *ROBO1* affect how the brain works and learns. For example, having a certain version of *ROBO1* seems to contribute to dyslexia, a condition where people have trouble reading.

Hypothesis

The team had identified the A version of *ROBO1* based on a relatively small sample of breeds. This led to the hypothesis that **the A version of the gene was favored by artificial selection in breeds in which humans desired traits that lead to high agility. If this hypothesis were true across breeds, then we'd expect the A gene version to be more common in more agile breeds** – that is, we'd expect to observe a positive relationship between the frequency of the A gene version and breed agility.

Data

To assess agility, the team used records of which breeds were entered in agility competitions and which breeds actually won agility titles. For each breed, they calculated the number of titles won per dog of that breed entered in competition. They then divided breeds into eight groups according to how likely each breed was to win agility titles:

Breed groups by increasing order of winning more titles at agility event →

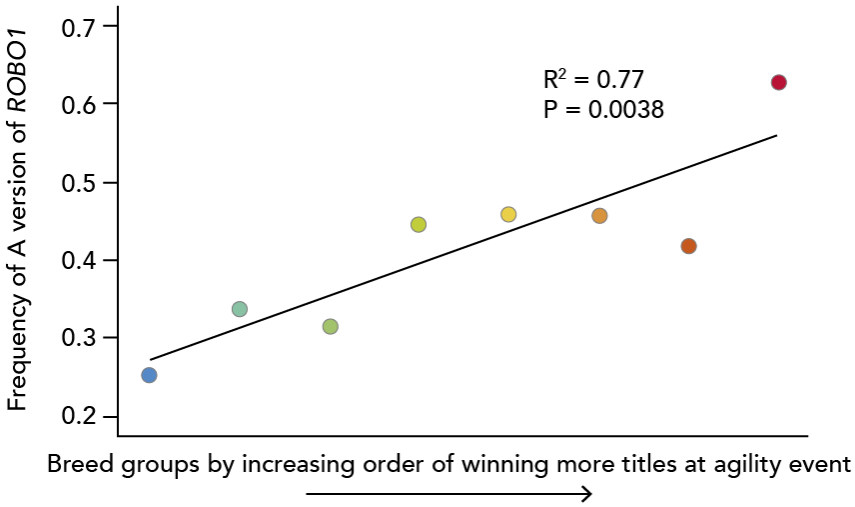
No. Breeds	20	20	20	20	20	20	20	8

Top performers (such as Border Collies and Pumis, both herding dogs) were in the red group. Breeds that never or very rarely won agility competitions (like the Greyhound and Anatolian Shepherd) were in the blue group.

To figure out how common the A gene version of *ROBO1* was in these different groups of dogs, the team turned to a database of dog DNA sequences. They found *ROBO1* data for 1243 dogs of breeds in one of the eight breed groups.

Greyhound. Photo provided by Dayna Dreger.

These were not dogs that had necessarily competed in agility trials themselves; they were just dogs that were members of the same breeds as competitors. For each of the eight breed groups, the team calculated how common the A gene version of *ROBO1* was compared to other gene versions. Here is what they found:



On this graph, the *x*-axis orders the eight breed groups from least likely to win agility titles (far left) to most likely (far right). The *y*-axis shows how common the A gene version is in that group. So, for example, a frequency of 0.4 means that the A gene version was found in 40% of the sequences from dogs of that group. **There is a clear, strong relationship between tendency to win agility titles and frequency of the A gene version:** the regression line slopes upwards, and this is significant ($p = 0.0038$). R^2 indicates how well the frequency of different gene versions is explained by the breed group. An R^2 of 0 would indicate that breed group cannot predict the frequency of the A gene version at all. An R^2 of 1 would indicate that one can precisely predict the frequency of the A gene version based on breed group. In this case, the R^2 value of 0.77 indicates that 77% of the variation in the frequency of the A gene version is explained by breed group. **These findings further support the hypothesis that the A version of *ROBO1* was under strong artificial selection by humans in breeds for which humans desired traits that lead to strong agility.** Now researchers can build on this hypothesis to learn exactly how the A version of *ROBO1* impacts dogs that carry it, why the A gene version was favored in some breeds but not others, and importantly, how this might be related to athletic performance and other traits in humans.

Stepping into science

When Elaine Ostrander learned about genetics as a sophomore in high school, she knew she'd found her calling. She wanted to be a scientist. But she didn't have any role models around her: no parent or friends were scientists. So Elaine forged her own path. Throughout high school and college, she worked all sorts of jobs – tutor, waitress, janitor, dishwasher – so she could get the training she needed for her dream career. And she made it. Elaine now leads her own lab, where she helps train the next generation of scientists and builds new knowledge about genetics.

Footnotes
[1]Scientists are still working on untangling exactly how many wolf populations were involved in this process and where and when it occurred.

[2]Identifying these genes was a big part of the study, though not the focus of this Digging Data article. The process involved comparing the genomes of many sport-hunting dogs to the genomes of terriers (breeds selected to hunt vermin). The team looked for areas in the genome where 1) sport-hunting dogs tended to have one sequence and terriers tended to have a different sequence (suggesting that the two groups have evolved genetic differences from one another) and 2) the stretch of DNA sequence common in one of the groups of dogs was unusually long – that is, many dogs in one of the two groups (but not the other) had chromosomes with the exact same long genetic sequence at a particular location. This suggests that somewhere in that sequence is a genetic variant (allele) that was so advantageous that it became common quickly – so quickly that recombination didn't have time to mix up the sequence near the useful variant. This process is known as a selective sweep. So, in short, the team looked for parts of the dog genome where there was evidence of genetic differences between sport-hunting dogs and terriers and evidence that a selective sweep caused those differences to evolve.

Adaptation

An adaptation is a feature that arose and was favored by natural selection for its current function. Adaptations help an organism survive and/or reproduce in its current environment.

Adaptations can take many forms: a behavior that allows better evasion of predators, a protein that functions better at body temperature, or an anatomical feature that allows the organism to access a valuable new resource — all of these might be adaptations. Many of the things that impress us most in nature are thought to be adaptations.

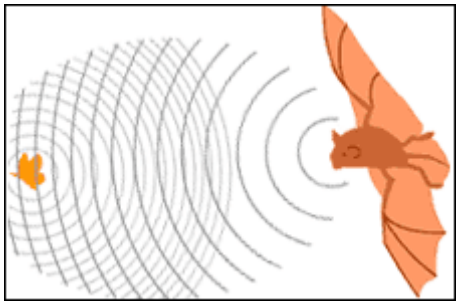
Mimicry of leaves by insects is an adaptation for evading predators. This example is a katydid from Costa Rica.

A side-facing image of a katydid, which looks like a leaf with six legs and antennae. Image © Greg Neise, GE Neise Digital Communication.

The creosote bush is a desert-dwelling plant that produces toxins that prevent other plants from growing nearby, thus reducing competition for nutrients and water.

A creosote bush. Image courtesy of US Geological Survey.

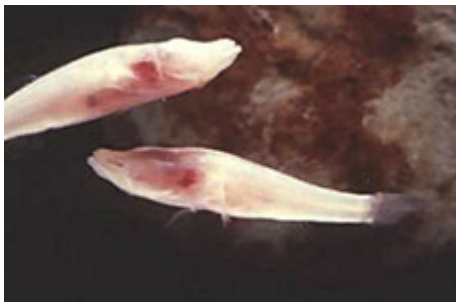
Echolocation in bats is an adaptation for catching insects.



Echolocation illustration, UCMP

So what's not an adaptation? The answer: a lot of things. One example is vestigial structures. A vestigial structure is a feature that was adaptive for the organism's ancestor, but that evolved to be non-functional because the organism's environment changed.

Fish species that live in completely dark caves have vestigial, non-functional eyes. When their sighted ancestors ended up living in caves, there was no longer any natural selection that maintained the function of the fishes' eyes. So fish with better sight no longer out-competed fish with worse sight. Today, these fish still have eyes — but they are not functional and are not an adaptation; they are just the by-products of the fishes' evolutionary history.



Milyeringa fish which has no eyes. Image courtesy of Wetland Care Australia.

Misconceptions about natural selection

Because natural selection can produce amazing adaptations, it's tempting to think of it as an all-powerful force, urging organisms on, constantly pushing them in the direction of progress — but this is not what natural selection is like at all.

First, natural selection is not all-powerful; it does not produce perfection. It's all about getting genes into the next generation, and if your genes are "good enough" to do that, you don't have to be perfect. This should be clear just by looking around us: human populations carry genes that cause disease, plants may not have the genes to survive a drought, a predator may not be quite fast enough to catch her prey every time she is hungry. No population or organism is perfectly adapted.

Second, it's more accurate to think of natural selection as a process rather than as a guiding hand. Natural selection is the simple result of variation, differential reproduction, and heredity — it is mindless and mechanistic. It has no goals; it's not striving to produce "progress" or a balanced ecosystem.

variation + differential reproduction + heredity = natural selection

Evolution does not work this way.

This is why "need," "try," and "want" are not very accurate words when it comes to explaining evolution. The population or individual does not "want" or "try" to evolve, and natural selection cannot try to supply what an organism "needs." Natural selection just selects among whatever variations exist in the population. The result is evolution.

At the opposite end of the scale, natural selection is sometimes interpreted as a completely random process. This is also a misconception. The genetic variation that occurs in a population because of mutation is random — but selection acts on that variation in a very non-random way: genetic variants that aid survival and reproduction are much more likely to become common than variants that don't. Natural selection is NOT random!

Coevolution

The term coevolution is used to describe cases where two (or more) species reciprocally affect each other's evolution. So for example, an evolutionary change in the morphology of a plant, might affect the morphology of an herbivore that eats the plant, which in turn might affect the evolution of the plant, which might affect the evolution of the herbivore… and so on.

Coevolution is likely to happen when different species have close ecological interactions with one another. These ecological relationships include:

1. Predator/prey and parasite/host
2. Competitive species
3. Mutualistic species

Acacia photo courtesy of Steve Prchal and the Sonoran Arthropod Studies Institute.

Many cases of coevolution can be found between plants and insects. For example, plants and their pollinators are so reliant on one another and their relationships are sometimes so exclusive that biologists have good reason to think that "matches" between the two are the result of a coevolutionary process.

Another example involves Central American *Acacia* species, which have hollow thorns and pores at the bases of their leaves that secrete nectar. These hollow thorns are the exclusive nest-site of some species of ant that drink the nectar. But the ants are not just taking advantage of the plant — they also defend their acacia plant against herbivores.

This system is probably the product of coevolution: the plants would not have evolved hollow thorns or nectar pores unless their evolution had been affected by the ants, and the ants would not have evolved herbivore defense behaviors unless their evolution had been affected by the plants.

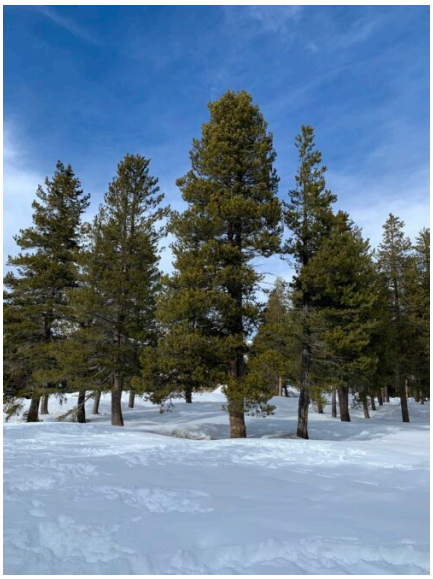
Red squirrel photo by P. Holroyd.

Crossbill photo by Noah Strycker CCBY-NC4.0

A case study of coevolution: squirrels, birds, and the pinecones they love

In most of the Rocky Mountains, red squirrels are an important predator of lodgepole pine seeds. They harvest pinecones from the trees and store them through the winter. However, the pine trees are not defenseless: squirrels have a difficult time with wide pinecones that weigh a lot but have fewer seeds. Crossbill birds live in these places and also eat pine seeds, but the squirrels get to the seeds first, so those birds don't get as many seeds.

However, in a few isolated places, there are no red squirrels, and crossbills are the most important seed predator for lodgepoles. Again, the trees are not defenseless: crossbills have more difficulty getting seeds from cones with large, thick scales. But the birds have a mode of counterattack: crossbills with deeper, shorter, less curved bills are better able to extract seeds from tough cones.



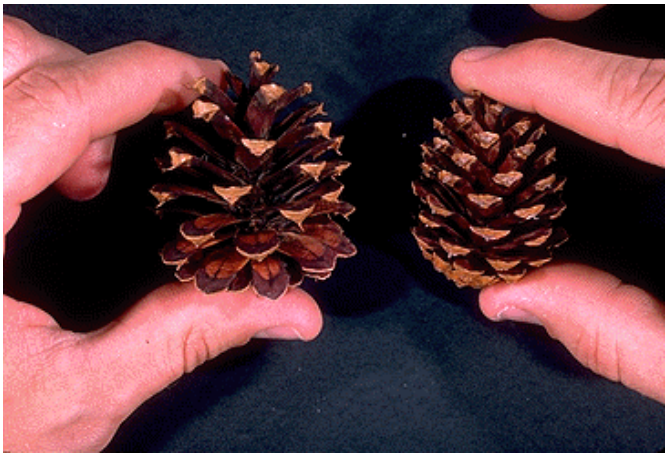
Lodgepole pine photo by kendalloei CC BY-NC 4.0

Close ecological relationships (like the predator/prey relationship described above) set the stage for coevolution to occur. But did it actually happen in this case? To figure that out, we need evidence that suggests that the prey (the trees) have evolved in response to the predator (squirrels or birds) and that the predator has evolved in response to the prey. Researchers Craig Benkman, William Holimon, and Julie Smith set out to see if their observations would support the hypothesis of coevolution.

The scientists reasoned that if coevolution had occurred they would expect to observe the following:

- **Differences between pinecones from different regions**

  If the trees have evolved in response to their seed predators, we should observe geographic differences in pinecones: where squirrels are the main seed predator, trees would have evolved stronger defenses against squirrel predation, and where birds are the main seed predator, trees would have evolved stronger defenses



Lodgepole pinecone (left) adapted to defend against squirrels – easier for crossbills to eat. Lodgepole pinecone (right) adapted to defend against crossbills – easier for squirrels to eat. Image of lodgepole cones courtesy of Ed Jensen, Oregon State University.

against bird predation. This turned out to be true. Where there are squirrels, the pinecones are heavier with fewer seeds, but have thinner scales, like the pinecone on the left. Where there are only crossbills, pinecones are lighter with more seeds, but have thick scales.

- **Geographic differences between predators that correspond to differences in prey**

  If the crossbills have evolved in response to the pine trees, we should observe geographic differences in birds: where the pinecones have thick scales, birds would have evolved deeper, less curved bills, which are better for getting seeds out of tough cones, than they have where the pinecones have thin scales. This also turns out to be true.

The bill is less curved on this female red crossbill. Image of female red crossbill courtesy of Greg Lasley, Greg Lasley Nature Photography.

The bill is more deeply curved on this male red crossbill. Image of male red crossbill courtesy of Dennis Oehmke and the Illinois Raptor Center.

So we have evidence that the trees have experienced natural selection and adapted to the birds (and the squirrels) and that the birds have adapted to the trees. (However, note that we don't have evidence that the squirrels have adapted to the trees.) It's easy to see why this is called a coevolutionary arms race: it seems possible for the evolutionary "one-upping" to go on and on…even thicker-scaled pinecones are favored by natural selection, which causes deeper-billed birds to be favored, which causes even thicker-scaled pinecones to be favored, and so on…