

Introduction to Biological Anthropology: Notes 7  
**Population genetics and the modern synthesis**

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- We have seen that:
  - Mendel’s model explains some variations in certain traits, like seed color in peas
  - molecular biology and cell biology show us the mechanisms by which it actually works
- Mendel's model is fine for either-or characteristics like green vs. yellow peas, but what about the vast number of traits that don't work that way, like height or body proportions?
  - **dichotomous traits** are either-or characteristics; they have only two possibilities, like “blood that clots normally” versus “blood that does not clot”
  - **discrete** variation refers to traits that can any number of separate, distinct variants
    - like red, white, or pink snapdragon flowers
    - we won't treat these as a separate case, but they follow the same general principles we are covering
  - **continuous variation** (or **continuously variable traits**) refers to traits that have an unbroken range of variation, rather than distinct types
    - like height, beak depth in finches, etc.
    - there are many, many continuously variable traits that are interesting and important, probably a lot more than discrete or dichotomous traits
- Mendel’s model can explain continuously variable traits, too
- The key idea:
  - dichotomous traits are controlled by just one pair of loci
    - like the pair of loci for pea seed color
  - but continuously variable traits are controlled by *many* pairs of loci
    - each of the many pairs of loci influences the trait somewhat
    - the principles are the same
    - the details are more complicated, because there are many more combinations of alleles to consider
  - traits that are controlled by multiple pairs of loci are called **polygenic**
    - this is very common, and what we will consider today
- Example: Let's use the beak depth measured on the finches studied by Peter and Rosemary Grant on Daphne Major in the Galapagos
  - Imagine that beak depth is controlled by one pair of loci with codominant alleles
    - we could do this example imagining simple dominant and recessive alleles, too, but it would get slightly more complicated
    - say the alleles control the amount of a beak-stimulating hormone
      - H+ (more hormone), contributes to deeper beaks
      - H- (less hormone), contributes to shallower beaks
    - so the possible genotypes are
      - H+H+ (deep beak)
      - H+H- (intermediate beak)

- there are two ways to get this (H+H-, or H-H+), so there are twice as many of these
  - H-H- (shallow beak)
- This only gives three kinds of beaks, not a continuous distribution
- it works just like any other codominant Mendelian trait
- but now, imagine that alleles at another pair of loci also affect beak depth
  - say, by controlling the amount of calcium available for beak formation
    - C+ leads to deeper beaks
    - C- leads to smaller beaks
  - so now the genotypes are
    - H+H+C+C+: biggest beak
    - H+H+C+C-: slightly smaller
    - H+H+C-C-: slightly smaller yet
    - H+H-C+C+: smaller than the biggest, probably not exactly the same as either of the other two, either
      - since the effect of slightly less calcium is probably not quite the same as the effect of slightly less of the hormone
    - H-H-C+C+: smaller than above, probably not exactly the same as any of the others
    - and so on down to H-H-C-C-, which results in the smallest beak
    - there are 9 potentially different combinations with just two pairs of alleles
- now imagine a third pair that also affects beak depth... there would be 27 (3 x 3 x 3) different combinations
  - a fourth pair would allow for 81 different combinations... and so on
- a relatively small number of pairs of loci results in a large number of combinations of alleles
  - each combination will probably result in a slightly different total effect on the trait
  - in theory, there should be a large number of different beak sizes, but still with a distinctly different size for each combination of alleles
- however, each genotype does not always produce exactly the same size of beak
  - there will be a range of phenotypes (beak sizes) for each genotype, since the beak size is also affected somewhat by the bird's environment
    - its diet, air temperature, etc.
  - so each genotype actually produces not one precise beak size, but a range of beak sizes
  - this causes the different combinations of alleles to produce overlapping ranges of beak size
  - so there are no steps or gaps in beak size: the variation looks continuous
  - even though the underlying genotypes are still distinct combinations of alleles
- Cool result: this explains how matings can *usually* produce offspring that look "blended" or intermediate between the parents, but *sometimes* exceed the parents on certain traits: just like what we really observe
  - both parents will generally have a mix of alleles that affect a given trait, like beak depth
    - each will have some alleles that promote deeper beaks, and others that promote shallower beaks stature
  - the offspring each get a random selection of these alleles from each parent
    - usually, the offspring will get some alleles for deep beaks, some for shallow beaks, and will come out with a moderate height

- most offspring will tend to have a mix of alleles that is somewhere intermediate between the parents
  - this looks like blending inheritance
- but occasionally, just by the luck of the draw, an offspring will happen to get mostly alleles for deep beaks, or mostly alleles for shallow beaks
  - an offspring might happen to get almost all the father's “deep beak” alleles and few of his "shallow beak" alleles, and the same from the mother, resulting in a higher percentage of "deep beak" alleles than either parent
- this could produce individuals who have traits that exceed those of their parents
  - just as we occasionally see in real offspring
- this interaction of many loci affecting single traits is how Mendelian genetics produces offspring that
  - *usually* look like blends of most of their parents' traits
  - but *sometimes* have traits that are *not* intermediate between their parents
- This is an important way in which "new" variants are produced for natural selection to act on
  - they are not new *alleles*, but rather new *combinations* of alleles
  - they may not even really be new combinations, just combinations that are so unlikely by chance that they don't happen very often
  - of course, there is no guarantee that the next generation of offspring will get the same combination
    - in fact, it is very unlikely
  - but if the combination leads to that individual having more offspring, the alleles that make up the combination will become more common
    - the more common those alleles become, the more often this combination will turn up...
- consider the corn breeding experiment again
  - selection for high oil content in corn pushed oil content from around 5% to 18% in 80 generations
  - this is far too fast to be due to accumulating random mutations (which we'll cover in a moment) that happened to increase oil content
  - instead, it implies that many loci influence oil content
  - the starting plants were probably a mix of heterozygous and homozygous at many loci that affected oil content
  - the experimenters were selecting plants that had more of the desired alleles at the many different loci
  - since they picked parents that had more of the high-oil alleles, the offspring were more likely to get combinations with many high-oil alleles
  - even combinations with more high-oil alleles than either parent
- OK, so where do the *really* new variants come from - not new combinations of alleles, but new alleles themselves?
  - **Mutation**: a change in the genetic code
    - usually a change in the sequence of nucleotide bases in a stretch of DNA
    - usually caused by an error in copying the DNA during replication

- since most DNA is unused “junk DNA”, most mutations have no effect
- mutations that do affect a working gene are usually harmful
- just as randomly changing a part in a complex machine is much more likely to prevent it from working than to make it work better
- Kinds of mutations:
  - **point mutation**: change of a single nucleotide base
    - probably the most common kind of mutation
    - a point mutation in a gene changes one nucleotide base in a codon
      - that results in a different codon
      - Original DNA: CAT
      - DNA after a point mutation: CAA
    - the usual result: this usually changes one amino acid at that point in the protein
      - effect: often, changing one amino acid has little or no effect on the function of a huge protein
      - effect: sometimes, it changes the protein’s shape and chemical behavior
        - usually for the worse
    - a less common result: if the new codon happens to code for the same amino acid as the original one, there is no change in the resulting protein
      - say GCT, which codes for alanine, changes to GCC
      - GCC happens to code for alanine, too
      - effect: none at all
    - a rare result: the change creates a “stop” codon, which cuts off production of the rest of the protein
      - effect: this will usually have a drastic effect on the protein’s function, probably not for the better
  - **insertion or deletion**: one or more nucleotides get dropped or added to the DNA sequence
    - if the insertion or deletion happens to be of a multiple of three nucleotides, then whole codons are added to, or removed from, the sequence
      - Original DNA: CAT CAT
      - After an insertion mutation: CAT **AGA** CAT
    - result: the protein is missing one or more amino acids, or has one or more extra amino acids
    - effect: maybe very little, maybe significant
      - almost never an improvement!
  - **frame-shift mutation**: due to an insertion or deletion of one, two, four, etc. nucleotide bases: any number except a multiple of three
    - this is much more drastic
    - The boundary between all the subsequent codons gets shifted; the reading of the code changes completely
    - Original DNA: CAT CAT CAT...
    - After insertion of one base causes a frame shift: CAT **GCA** TCA T...
    - result: a completely, unpredictably different protein is produced from that point on

- effect: drastic, radical change in the protein; almost certainly prevents the original function, very unlikely to produce anything useful
- And others:
  - **repeat mutations:** a portion of DNA gets repeated
    - Original: CAT TAG ACT
    - After repeat mutation: CAT TAG **TAG TAG** ACT
    - If the repeat is not a multiple of 3 bases, it also causes a frame-shift mutation
    - After: CAT TAT **ATA** GAC T...
  - **inversion mutations:** a portion of the DNA gets flipped around backwards
    - Original: CAT TAG ACT
    - After repeat mutation: CAT **AGA TCT**
    - sometimes combined with repeat mutations, creating inverted repeats and often frame-shifts
    - After: CAT **AGA TAG ATC** T...
- The point: mutations produce new alleles in various ways
  - usually harmful
  - but on very rare occasions, a change is beneficial
- if a mutation occurs in a normal body cell, it usually has little impact
  - if the mutation is seriously harmful, the daughter cell dies
    - and the mutation disappears with it
  - if the mutation does not cause a serious problem for the cell's survival, the cell will go on dividing normally (by mitosis)
    - and eventually, a small number of cells in that part of the body “inherit” the mutation
    - when the organism dies, the mutation is eliminated, too
  - if the mutation causes the cell to grow and divide wildly - as in a cancer - then the abnormal growth of tissue may be harmful to the organism, even kill it
    - still, when the organism dies, the mutation is eliminated, too
  - either way, a mutation in a body cell cannot be passed on to offspring
    - so it does not add a new allele to the population
    - and it has little impact on evolution
- but mutations in the cells that produce gametes are important
  - because the mutation may be copied into a gamete, which may end up producing an offspring
    - every single cell in the offspring will then carry the new, mutated bit of DNA
    - and it might get passed onto the next generation, too
    - so: only mutations in sex cells create new alleles
    - only these mutations can directly affect evolution
- mutations may be caused by many things
  - radiation, like X-rays, may increase the likelihood of a mutation
    - this is why doctors may X-ray your torso, but they put a lead shield over your testes or ovaries

- an occasional mutation in an irradiated body cell is no big deal, but a mutation in a cell that eventually produces a gamete might be catastrophic for a future offspring
- some chemicals may increase the likelihood of mutations
- Mutations are rare
  - DNA replication is amazingly accurate
  - but there is such a vast amount of DNA in every cell that the odds of a mutation occurring *somewhere* in the functional portion the DNA are actually fairly high
  - Human gametes are estimated to usually carry at least one mutation in an allele (that is, not in junk DNA)
  - any given individual probably got at least one mutation from his or her parents' gametes
- so mutation constantly adds new alleles to the population
  - mostly neutral mutations
    - since many point mutations have little effect on a huge protein
  - the rest are mostly harmful recessive alleles
    - because a change in a protein that actually affects its function usually prevents it from working properly
    - since the offspring that got the mutated allele probably got a correct copy of the allele from the other parent, the offspring still produces the normal protein
    - so the harmful recessive mutation hides invisibly in this **carrier**
    - natural selection does not weed out the allele, because it has no effect
    - unless the carrier mates with another carrier
    - but since any given mutation is rare, the chances of a carrier mating with another carrier of the same recessive allele are very low
    - so the recessive trait is very rarely expressed, and natural selection cannot weed it out
  - rare dominant or codominant mutations
    - dominant or codominant mutations are expressed even when there is just the one mutated allele in the offspring
    - if it causes the individual to die or not reproduce, it is immediately removed from the population
    - so dominant or codominant mutations that last more than a single generation are generally not very harmful
  - and very rarely, new mutated alleles with beneficial results
- the nature vs. nurture question
  - we have been talking as though the main influence on phenotypes was the genotype
  - But what about the conditions in which the organism develops and lives?
  - "Nature": inherited genes
  - "Nurture": environment
  - the "nature versus nurture" question is: which is the main determinant of how individuals turn out (their phenotype)?
  - Answer: both, because they are inseparable; you can't have one without the other
    - An individual's phenotype is the result of the *interaction* of both genes and environment

- You can't have a phenotype without genes, and you can't have a phenotype that did not develop in some environment
- both are necessarily part of the process
- So far, we have been looking at inheritance at the individual level
  - asking questions like, given parents of such-and-such genotypes or phenotypes, what will their offspring be like?
- But remember that evolution is about changes in *populations*, not individuals
  - like a certain color of moth becoming more common in the population
  - or the average beak depth of all the birds in the population changing over generations
  - so, to understand evolution, we have to look at inheritance in a lot of individuals at once
- we usually think of species in terms of what is typical for individuals of that species
  - we think of the Platonic ideal, or "form", of a species: the ideal or typical example
  - like: "a human is a two-legged, mostly hairless creature with harmless little teeth"
  - of course, some humans are extremely hairy, yet they are still humans
  - this view emphasizing what is typical glosses over the variation between individuals
- in the **population point of view**, we think of species or groups of organisms in terms of the frequency of traits throughout the population
  - **population** (or **breeding population**) = a group of individuals that mate mostly with others in the same group, and little with outsiders
    - in reality, populations usually have fuzzy boundaries
      - individuals migrate into and out of groups
      - individuals mate with others from outside the group
    - but the breeding population concept is a useful first approximation
  - a population view of a species is in terms of how common certain traits are in that population
    - like: "a human is a member of a population among whom 98% have two legs, 99% have mostly hairless bodies, and 99% have small teeth"
  - in terms of *phenotypes*, we might say "91% of the population has straight thumbs and 9% have hitchhiker's thumbs"
  - in terms of *genotypes*: "49% are SS, 42% are Sh, and 9% are hh"
  - or in terms of *alleles*: "70% of the thumb alleles are S, and 30% are h"
  - we can think of a population as a **gene pool**:
    - the collection of alleles, lurking in individuals, from which alleles are randomly drawn to create gametes, which in turn randomly combine to form the next generation of offspring
  - **population genetics**: the quantitative study of the frequency and distribution of alleles, genotypes, and phenotypes in populations
- This allows us to make a more precise definition of evolution, in population terms:
  - recall our "preliminary definition" from an earlier class:
  - PRELIMINARY DEFINITION: **Evolution** = change in the frequency or magnitude of heritable features of a population from one generation to the next.
  - this was, sensibly enough, focused on changes in phenotypes, which are what we can directly observe. Now we can do better:

- **Evolution** = change in allele frequencies in a population from one generation to the next
- Why focus on allele frequencies?
  - because when the next generation of zygotes is created, allele frequency controls genotype frequency
  - genotype frequency (and environment) in turn controls phenotype frequency
  - and phenotypes are what affect natural selection
- When zygotes are created,
  - the frequency of each combination (genotype) is determined solely by
    - the frequency of the alleles in the gene pool
    - and the laws of probability
  - so zygotes are created with predictable proportions of genotypes
    - then natural selection starts weeding some out
- we can illustrate this by using a Punnett square in a new way
  - so far, we have used Punnett squares to represent the mating of a single pair of parents
  - but we can also use them to represent all the matings of a whole population
    - instead of representing a particular father, we describe the likelihood of any father producing a certain kind of gamete
    - instead of a particular mother, we show the likelihood that any mother's gamete will be of a given kind
    - for example, say there are two alleles for the thumb joint trait, and they are equally common (exactly 50% are S and 50% are h)
      - SS and Sh result in straight thumbs
      - hh results in "hitchhiker's thumb"

	Father's gametes: .50 S	Father's gametes: .50 h
Mother's gametes: .50 S	SS (straight): .50 x .50 = .25	Sh (straight): .50 x .50 = .25
Mother's gametes: .50 h	hS (straight): .50 x .50 = .25	hh (hitchhiker's): .50 x .50 = .25

- we have assumed that 50% of all males' gametes have the S allele, and the other 50% have the h allele
  - same for the females' gametes
- so the chances of an offspring getting, for example, a h from the mother is 50%
  - of those that got a h from the mother, 50% will also get a h from the father
  - so 50% of 50%, or 25%, (.5 x .5 = .25) will be hh (have hitchhiker's thumbs)
- and the remaining 75% will be Sh, hS, or SS
  - they all have straight thumbs
- now say that one allele is very common, for example, 90% of all the alleles are h and only 10% are S

	Father's gametes: .10 S	Father's gametes: .90 h
Mother's gametes: .10 S	SS (straight): .10 x .10 = .01	Sh (straight): .10 x .90 = .09
Mother's gametes: .90 h	hS (straight): .10 x .90 = .09	hh (hitchhiker's): .90 x .90 = .81

- now, 90% of gametes produced by males in the population have the h allele, and only 10% have the S allele
  - same for the females' gametes
  - so the chance of an offspring getting a h from the father *and* a h from the mother are
    - .90 x .90 = .81; 81% are hh (have hitchhiker's thumb)
    - note that this is the recessive trait, but 81% of the population express it, because the allele is so common
    - that is, a recessive trait may be common (or it may be rare)
      - dominance and recessiveness are completely independent of allele frequency
  - there will be a few heterozygotes, since occasionally one parent will have the rare allele
    - 10% x 90% = 9% will be Sh, getting the S from the father
    - 90% x 10% = 9% will be hS, getting the S from the mother
    - adding these together, 18% will be heterozygous, with straight thumbs
  - and there will be very few homozygotes for the rare allele, since the odds of two people with the rare allele mating are low
    - 10% x 10% = 1% will be SS, homozygous straight thumbs
  - in total, 18% + 1% = 19% will have straight thumbs (Sh, hS, or SS).
- we can generalize this relationship between allele frequencies and genotype
  - if the frequency of, say, the S allele in the whole population is called p
  - and the frequency of the h allele in the whole population is called q

	Father's gametes: Frequency of S = p	Father's gametes: Frequency of h = q
Mother's gametes: Frequency of S = p	SS (straight): p x p	Sh (straight): p x q
Mother's gametes: Frequency of h = q	hS (straight): q x p	hh (hitchhiker's): q x q

- the frequency of the SS genotype should be  $p^2$
- the frequency of the Sh genotype should be  $2pq$
- the frequency of the hh genotype should be  $q^2$
- for example, say that 70% of all the thumb alleles are the dominant, straight allele (S)
  - $p=.70$
  - 30% of the alleles are the recessive, hitchhiker's allele (h)
    - $q=.30$
  - since there are only two alleles, the two frequencies have to add up to 100%, or 1
    - $70\% + 30\% = 100\%$
    - $.70 + .30 = 1.00$
- say that individuals in this population mate at random, producing many offspring

- what fraction of the offspring will have the each genotype?
  - Frequency of SS =  $p^2 = .70^2 = .49$
  - Frequency of Sh =  $2pq = 2 \times .70 \times .30 = .42$
  - Frequency of hh =  $q^2 = .30^2 = .09$
- These general predicted proportions of genotypes are called **Hardy-Weinberg Equilibrium**
  - Hardy-Weinberg equilibrium is usually expressed as  $p^2+2pq+q^2=1$
  - The frequencies of the three genotypes have to add up to 1, or 100%, because they are the only possibilities
  - The result is called an “equilibrium” because the proportions are stable
    - if the frequencies of the alleles do not change, the frequencies of the three genotypes always come out the same
    - they are determined by the allele frequencies, and nothing else
    - if you tell me the allele frequencies of a population, I can tell you how many of each genotype there should be
- These predictions of genotype frequencies only work if the process really is completely random: every gamete is equally likely to form a zygote
  - specifically, there are five assumptions hidden in our Punnett squares and in the Hardy-Weinberg equilibrium equation
  - these are often called the **Hardy-Weinberg assumptions**
    - 1. no alleles are changed into other ones during the process
    - 2. the allele frequencies are not changed by individuals moving into or out of the population, or by mating with outsiders (which brings their alleles into the population)
    - 3. mating is random with respect to the given trait
    - 4. we assume a large enough population that the rules of probability make deviations from the predictions unlikely
      - we are calculating proportions that we expect to see if there is a large number of matings
      - but it is always possible that just by extremely bad luck, in some given case they could turn out differently
        - maybe, just by chance, every single offspring will happen to get two S alleles
        - this is very unlikely, but not impossible
      - that is, we are ignoring the possibility of getting very unlikely outcomes
      - this is reasonable if the population is large
        - out of 1000 coin flips, it is very unlikely that you will get far from 50% heads
        - but out of 4 coin flips, getting all heads is not too unlikely
    - 5. all individuals are equally likely to survive and mate (there is no selection in which some parents get to have more offspring)
- *IF* the assumptions are met
  - then the alleles of all the parents in the population will be randomly distributed in the next generation of zygotes
    - in the proportions predicted by the Hardy-Weinberg equilibrium equation
  - the frequency of each genotype will be determined only by the frequencies of the alleles
  - the offspring will have exactly the same allele frequencies as the parents

- so *their* offspring will have the same frequencies of the three genotypes, and the same frequencies of alleles
- *IF* the assumptions are met, then the allele frequencies and genotype frequencies will continue unchanged, generation after generation
  - nothing about reproduction makes dominant alleles become more common
  - nor recessive alleles become less common
- every generation, the alleles just get remixed
  - each time recreating the genotypes in the proportions required by their frequencies and chance:  $p^2$ ,  $2pq$ , and  $q^2$
- Recall that evolution is a change in allele frequencies
  - allele frequencies only change (that is, evolution only happens) when one or more of the assumptions of Hardy-Weinberg equilibrium is *not* met
  - These violations of the Hardy-Weinberg assumptions are the "forces" that could cause evolution
  - Let's look at each in turn
- A force of evolution?: *Mutation*
  - Mutation violates assumption 1: "no alleles are changed into other ones during the process"
    - naturally, adding or taking away alleles would change the allele frequency
    - this would be evolution
  - Mutation does indeed change allele frequencies by occasionally replacing an existing allele with a new one
  - but mutations are so rare that they have a miniscule effect on allele frequencies
    - among humans, any given allele is likely to get converted to a new variant by mutation in about 1 out of 50,000 births
    - in a population of 1000 humans, that is *one* allele changed for that locus every 50 generations
  - it would take a *very* long time for mutation by itself to add a significant number of any particular new allele to the population
  - it is true that every allele was initially created by mutation
  - but when we see that an allele has become common (say, 1% or more of the alleles), we cannot attribute that to mutation alone
    - because mutation just can't produce a lot of copies of an allele in any reasonable period of time, even on a geological time scale
  - Some other force of evolution must have made it common
  - so mutation produces new alleles, but it does not directly cause evolution
- A force of evolution: *Gene Flow*
  - Gene flow violates assumption 2: "the allele frequencies are not changed by individuals moving into or out of the population, or by mating with outsiders (which brings their alleles into the population)"
  - Gene flow can change allele frequencies by removing alleles or adding alleles to the gene pool as individuals leave or join the population
    - individuals move into or out of the population, bringing their different allele frequencies with them

- or individuals mate with others from outside the population, bringing in the different allele frequencies of the outsiders in the form of gametes that contribute to the next generation
- Gene flow definitely happens in some cases, and can cause evolutionary changes in those populations
  - but it generally cannot cause speciation (cladogenesis or anagenesis) in itself
  - because gene flow is within a single species
  - the alleles moving into or out of the population are in about the same proportions as in the other populations of the species
  - since we are usually interested in evolution that leads to speciation, gene flow is usually not an important factor
- A force of evolution?: *Non-random mating*
  - Non-random mating violates assumption 3: “mating is random with respect to the given trait”
  - Non-random mating can indeed change *genotype* frequencies in a population
    - if people with hitchhiker’s thumb (hh) did not choose mates randomly, but instead preferred to mate with other who have hitchhiker’s thumb
    - then their h gametes would be more likely to pair up with other h gametes, and would rarely encounter S gametes
    - so there would be more hh offspring than predicted, and fewer Sh offspring than predicted
  - but this does not affect *allele* frequencies
  - so it does not result in any long-term change
  - if this non-random mating went on for 500 generations, the allele frequencies would still be the same, so if mating became random again, the frequencies of each genotype would return to the Hardy-Weinberg predictions in a single generation
  - so non-random mating does violate one of the Hardy-Weinberg assumptions, but it does not cause evolution
- A force of evolution: *Genetic drift*
  - Genetic drift violates assumption 4: “we assume a large enough population that the rules of probability make deviations from the predictions unlikely”
  - **Genetic drift**, or the effect of unlikely chance outcomes in reproduction, does change allele frequencies in some cases
  - Say a population has 50% S and 50% h alleles
    - we predict 25% SS, 50% Sh, and 25% hh offspring
    - in a large population, we can be confident of this
      - just as you can predict around 50% heads if you flip a coin 1000 times
    - but in a tiny population, by sheer bad luck, the actual offspring might differ from the prediction significantly
      - maybe they produce just 3 SS and 1 Sh offspring
      - now the S allele is much more common in this generation
        - when they have offspring, the S allele is likely to be common among them, too
      - the change is long-term: all future generations are descended from this gene pool
      - the chance of another bout of sheer bad luck switching the change back is very low
      - what if that one Sh offspring does not reproduce?

- then ONLY S alleles are left
- now, no amount of chance events, natural selection, or anything else can change the frequencies of the thumb alleles
- they are permanently fixed at 100% S, 0% h
- this is called **fixation**: the permanent elimination of variation when an allele frequency falls to zero
  - fixation is much more likely to happen in small populations
  - it is the most extreme form of genetic drift (it can also be caused by directional selection)
- so genetic drift can cause evolution
  - note that drift does not improve adaptation; it is purely random change
- A force of evolution: *Selection*
  - Selection violates assumption 5: “all individuals are equally likely to survive and mate (there is no selection in which some parents get to have more offspring)”
  - **Selection** (natural or artificial) can change allele frequencies in the next generation by causing individuals with some genotypes to leave more offspring, and those with other genotypes to leave fewer
  - Say a newly mutated allele is beneficial
    - maybe individuals who have one copy have more offspring than those who don't
    - then the allele will become more common
      - that is evolution
- so selection can cause evolution