

# **An Introduction to Evolutionary Biology**

**Prof. Sutirth Dey**

**Biology Department, Population Biology Lab**

**Indian Institute of Science Education and Research (IISER) Pune**

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## **Examples of selection**

During our last discussion, we looked at some of the insights that you get from the 1-locus, 2-allele model of viability selection. Our discussion was primarily on the theoretical insights that we gained. And I promised that in the next discussion, which is this one, We are going to talk about some of the features of selection derived from experiments. However, I later realized that before we go there, it will be important for us to get a more real-world sense. Through examples of some of the concepts that we discussed during those theoretical discussions. So, I am slightly changing the plan.

In this discussion, we are going to look at some empirical examples of two or three of the concepts. That we dealt with in the past two discussions. And the first thing we will look at an example of is what is known as overdominance. So, if you remember, overdominance is the situation when the heterozygotes have maximum fitness compared to both homozygotes.

And the example I have chosen is, you know, a very famous example, an all-textbook example. I am sure you have read about it in your 10th or 12th class, whichever. And this is in the context of sickle cell anemia. So, you know that in our blood, we have these red blood cells, and inside the red blood cells, We have the protein called hemoglobin, which helps in the transportation of oxygen. Now, in hemoglobin, there are multiple components, one of which is called beta hemoglobin.

And when it comes to the gene for beta hemoglobin, there can be multiple forms; one of them is known as the S form. So, basically, the way it differs from the regular form of hemoglobin is that it has a point mutation at a single nucleotide. Because of which the protein, I am sorry, one amino acid changes, and because of the change in that particular amino acid, What happens is that individuals who are capital AA end up being normal phenotype. Whereas individuals who are homozygous for the mutant end up suffering from a disease called sickle cell anemia. Now, what exactly happens in sickle cell anemia? So, here what I am showing you are the red blood cells of humans, and these ones.

These are the normal ones, the circular ones, and this one over here; this is the one that is abnormal. So, you can see that it looks like a sickle, right? Now, because of this, this particular cell is not able to carry as much oxygen as normal cells can, and because of that, The organisms or individuals that are homozygous for the S allele end up having Lots and lots of physiological complications to the extent that most of them typically die by about age 5 or so. Very few of them actually attain adulthood. So, this tells you that there is a very strong selective pressure against this allele. And therefore, you expect the frequency of this allele to be very low in the population.

However, that is not entirely correct, and frankly speaking, the S allele is rather widespread across the globe. So, just to show you, the historical distribution of the sickle cell trait is over a lot of Africa, quite a bit of the Middle East, and also India. So, obviously, people ask the question, why is it that an allele which is so harmful in its homozygous form? Why is it that it is so widespread? And as you know the answer, the answer is malaria. So, it turns out that there is a very good correspondence between where you find the sickle cell allele and where you find malaria. And why is that? That is because individuals who are homozygous for SS are obviously in trouble, but individuals who are heterozygous for the S allele, that is, basically the AS genotype, Those individuals actually have an advantage. What is an advantage? Turns out that the malarial parasite is not able to invade the RBCs of these AS individuals. Because of this, these guys have a very good amount of protection against malaria. So, the way the whole thing

is happening is that if you are a capital AA genotype, then you are normal. But in places like these, you know you are going to have a very high rate of death due to malaria.

On the other hand, if you are an SS individual, you have sickle cell anemia; anyway, your fitness is going to be very low. In these malaria-prone areas, the heterozygotes actually end up having much higher fitness. Because they are being protected from malaria. So, this is a well-known example. I will give you one more example, which is again very interesting.

This is related to a flower known as the Black Vanilla Orchid, *Gymnadenia rhollicani*. Quite a tongue twister, but we will just call it the Black Vanilla Orchid. So, it turns out that this particular orchid, or rather the flower of this orchid, can be found in all kinds of shades. But three major shades are, you know, what I am showing you over here: so-called black, so-called red, and so-called white. Now, it is very interesting why exactly we have this.

So, it turns out that the red color morph, you know, this one, this middle one, This one is heterozygous again for a single nucleotide polymorphism, which basically means a change at only one base pair. which introduces a premature stop codon in a R2R3-MYB transcription factor. It does not matter what it is, something, some stuff in the genome. Because of this, it leads to reduced expression of the anthocyanidine synthase gene. And this anthocyanidine synthase gene is what is responsible for a protein known as a red cyanidine pigment.

So, the moment you have the mutation, the amount of red cyanidin pigment that is being produced goes down. And this is the interesting part: this, as far as the scientists can make out, does not affect any other phenotypic trait. So, those individuals who are so-called black have both alleles normal. They produce lots of cyanidin, and therefore the flower is extremely dark red; we call it black. The ones that are heterozygous have one copy of the original or normal allele and one copy of the mutated allele.

Therefore, their color level is somewhere in the middle; we call it red. And the ones that

are homozygous for the mutation have both alleles, both copies of the allele as faulty ones. And therefore, they produce the least amount of red cyanidine pigment, and therefore, you know, it is a very light color. Now, until this point, there is no difference between the three phenotypes except for color. As I said, this does not affect any other phenotypic traits.

However, what happens is in the context in which these flowers are found. So, these flowers are pollinated by two major groups of pollinators: the flies and the bees. Now, it turns out that these flies and bees have very contrasting preferences for flower colors. So, the flies prefer the white flowers over the black ones. So, that basically means they go more to the white flowers than to the black ones.

The bees do exactly the opposite; they prefer the black flowers over the white ones. Now, what about the reds? What about the heterozygotes? Turns out that in both cases, the heterozygotes, both pollinators have an intermediate preference for the heterozygotes. But it turns out that if you integrate over everything, then the red flowers, Because they are intermediate for both of them, overall they end up getting a relatively high number of visits. And because of this, if you actually calculate their fitness, which in this particular case is calculated in terms of How many seeds are being set? It turns out that, on average, it is the red flowers that have the highest number of seed sets. which is directly correlated with, you know, pollination and pollinated visits.

Now, because of this, the fitness of the heterozygotes is higher than that of both homozygotes. And as a result of this, you expect that in this population, This polymorphism between the two forms, between the two alleles, is going to be maintained. Note, this is the important one. As I said, there is no difference between the two alleles except in terms of the color they produce. They do not affect any other physiological features of the orchids.

However, the entire thing is being mediated by the preferences of the insects, the particular preferences of the two insects. Now, what will happen if all the insects say that

all the flies, all of them, start preferring one color? For example, the example that I have taken here is, let us assume that all the flies are killed. So, you only have the bees. What will happen? As I already told you, the bees prefer the black flowers over the white ones. So, now they are going to primarily go to the black ones; they will go to the red ones intermittently.

And there will be very few, you know, visitations to the white. So, suddenly from an overdominance situation, this entire thing will flip. Now, the black flowers will have the highest fitness; the red flowers will have intermediate fitness. The white flowers will have very little fitness, and from an overdominance case, it will become one of those other cases. That we were talking about, where you have the heterozygote, you know, where you have a proper gradation in terms of fitness: homozygote one, heterozygote, homozygote two. If that happens, then what will happen? Then the allele that is leading to the black form in the homozygous form, That is going to be preferred, and therefore, the variation from the population will be lost. So, the basic point that I am trying to make over here is two. One, overdominance maintains allelic diversity in a population; we already saw that. The second point is that the entire content is context-specific.

What is the fitness of a type depends entirely on the context in which that genotype finds itself. Under certain contexts, the system might be overdominant; in other contexts, it might not be. it might have a very different kind of a fitness. So, after overdominance, we come to underdominance. What is underdominance? Remember, this is a scenario in which the heterozygotes are less fit than both homozygotes.

So, there are not too many well-documented cases of underdominance in the literature; this is one of them. So, this is a butterfly; as you see, it is called the false wanderer, and you generally find it in Africa. So, it now turns out that in this butterfly, there are two alleles. And each allele duplicates the appearance of a different local species of butterfly. So, basically, you know there are two different species: other species, not this one, not the false wanderer, and one allele, you know, it makes the false wanderer blue and makes it resemble one species. And the other allele makes this orange and causes the false

wanderer to resemble another species. Now, it turns out that both of these other species are toxic. Which basically means that the predators normally leave them alone. So, this is a case of mimicry, where the false wanderer is mimicking two different species.

Depending on the allele, it either mimics the blue species or the orange species. Now, what happens to the heterozygotes? It turns out that the heterozygotes have an intermediate appearance. And this intermediate appearance actually belongs to neither species. And therefore, the predators actually attack these heterozygotes at a very high rate. because of which the fitness of the heterozygotes go down and it becomes a case of underdominance, Where, as I said, the fitness of the heterozygote is less than both homozygotes.

So, that is about underdominance. The third thing that we looked at was frequency-dependent selection. Now, if you remember, I told you that frequency dependence can be of many, many types and depends on How exactly are the genotypic frequency and the genotypic fitnesses related to each other? So, I will talk about two major cases of frequency dependence that have been studied empirically. One is called negative frequency dependence, and the other, of course, is called positive frequency dependence. So, in negative frequency dependence, the rare morph has an advantage. So, as the frequency of a genotype goes down, its fitness goes up; negative correlation.

So, again, this is something; there are quite a few examples of this in the literature. The one that I have chosen is in the context of these fish, known as *Perissodus microlepis*. So, this fish is found in Lake Tanganyika in Africa, and the specialty of this fish—well, there are many specialties, but— One major specialty of this fish is that it is a scale-eating parasite. So, basically, what it does is swim around, and then whenever there is a fish that is, let us say, not very attentive, It just goes and bites away a scale from, you know, one part of its body and then goes away. So, obviously, the fish from which it bites those scales do not really like this fish.

Now, the other feature of this particular fish is that its mouth is not straight. The mouth of

this fish is actually twisted, and it turns out that in the population You have one set whose mouths are twisted to the left and another set whose mouths are twisted to the right. Now what happens? So obviously, the one whose mouth is twisted towards the right, So what I am showing you here is this one; this guy ends up attacking its prey from the left side. Whereas this one, the other one has its mouth twisted towards the left. It ends up attacking the prey from the right side, which I am showing you here in this particular diagram.

So these are the preys. Now turns out that when the frequency of any one of them say let us say the right jawed parasite When this frequency goes up, then what happens? Most of them are attacking from the left. If most of them are attacking from the left, the prey is this big fish over here, the one whose scale is being taken away. That prey is more attentive to the left side. And therefore, when it becomes more attentive towards the left side, it becomes more and more difficult for these guys to You know, snatch the scale, and at that point in time, the fitness of this fish goes down. On the other hand, when this happens, what happens? These guys get an advantage.

Now when they get an advantage, what happens is their numbers go up, and when their numbers go up, the opposite thing starts happening. The prey starts paying more attention to the other side, the right-hand side, and now it is the other morph which is at an advantage. And this sounds like a story, but it is not a story. People have actually shown this. So, it turns out that they actually ended up looking at the relative frequency of these two morphs over time.

So here on the y-axis, they are showing the frequency of the left-jawed individuals, and you can see that. Over time, the frequency is just going up, down, up, down, up, down, exactly the way you predict it should happen. So this is a situation of negative frequency-dependent selection, which ends up maintaining polymorphism in a population. Why is that so? That is because each time the fitness of a genotype becomes high, the number of that genotype goes up. The frequency goes up, the fitness comes down, now the other guy's fitness is high, and This keeps repeating itself, and therefore

you actually end up getting a polymorphism in the population in terms of the various traits.

Now this particular kind of selection, negative frequency-dependent selection, is something that you need to keep in mind. Because about 3-4 weeks down the line, we will come back to this concept when we are discussing the evolution of sex. So, as I said, you have negative frequency-dependent selection, and you have positive frequency-dependent selection. What is positive frequency dependence? It occurs when the common morph has a fitness advantage. So just to give you an example, what I am showing you here are two species of butterflies.

So whatever is on top, this is known as *Heliconius melpomene*; these ones on the top, these ones. And whatever is at the bottom is another species called *Heliconius erato*. Now, for both species, it turns out that they are found all over South America. Although it is the same species, in different areas of South America, they look very, very different. So if you just compare all the top guys—this guy, this guy, this guy—they are all the same species, okay? This one, this one.

They are all the same species, and yet you can see how different they look from each other based on where they are found. The same is true for the other species; however, this is very interesting. If you look at a particular species in a given area, there is hardly any variation. So all the variation is across areas, with no variation within the area. Now, what the hell is going on? So, it turns out that both of these kinds of butterflies, both species, are very distasteful to predators.

So, they have certain chemicals in their bodies that predators really do not like when they try to eat them. However, to figure out what is going on, scientists took both kinds of species. But we will deal with one species at a time. So, let us say we are talking about the second species, *Heliconius erato*. So, they took individuals of that species, marked them, and then released them in a particular way.

What is the way? So, for any given space, they released two kinds of butterflies: one that was of the same species and resembled the local species, Local markings and the other, which is the same species, but it is a marking from a completely different place. In other words, just to show you, for example, suppose they will introduce *Heliconius erato* to this place, which looks like this. And they will also take *Heliconius erato*, let us say, from here, and they will introduce it over here. So, two kinds of *Heliconius erato* are being introduced in the same place.

Similarly, for this place, local and from some other places. And when they did all that, they found to their amazement that it is always the introduced ones. which has the local pattern that always survives better than the one with the foreign pattern. And why is that happening? That is happening because, remember, I told you that these are being predated upon by birds. Now, the predators, when they are born, do not have an innate ability to distinguish between You know what is edible and what is not edible. They learn over time that, okay, if I eat these butterflies with this particular pattern, I am going to get a stomachache, or you know it is going to be very bitter, or something like that.

So, it turns out that these predators are not very smart, right? They also tend to forget very soon. Therefore, in order for them to know, learn, and retain that, These guys, you know, the prey has to be very common. If the prey is not common, then the predator will tend to forget that it cannot eat this kind of prey. Now, whenever you are introducing something from outside as they did in this experiment, if those people had the same pattern. As the local one, then the predator is mistaking them for the common local pattern, you know.

However, the moment the predator sees something that has a different pattern, the predator does not really know that. You know this is the one that it does not want to eat, and therefore, it tends to start killing it. and because their numbers are very low, it does not encounter them very often and therefore, It takes a much longer time to learn that I do not want to eat this particular type, this particular pattern. And therefore, these guys, the ones whose patterns do not match the local pattern, end up facing a much greater

mortality.

They end up, you know, their fitness goes down drastically. So, this is a case where you have the common morph, the common phenotype, getting an advantage. and the rare phenotype is at a disadvantage. The same results were also obtained with the other species of butterflies. So, that is why I said you should concentrate on one species; the results for the other species are exactly the same. So, why is this interesting? Because in this particular case, this is operating exactly the opposite of the negative frequency-dependent situation.

Here, instead of maintaining polymorphism, positive frequency-dependent selection removes polymorphism from a population. Okay, so this is more or less what I wanted to share with you, the examples. Now, in the next discussion, we are going to go back and look at some features of selection more from an experimental point of view. See you. Bye.