PROFESSOR: Did anybody have any questions about this path analysis, which is where we ended the class last time? It's a way to find the probability for two individuals when you know they're related in some way. But if you have their-- you can draw the family tree, you're able to then, using path analysis, you can find the probability that the two alleles at one locus are identical, or how many of them are going to be identical.

It's called the inbreeding coefficient, or the coefficient of kinship. Both terms are used. But you can use the same thing to compute just how many genes do you get from your great grandparent, for example.

So this is the only slide from the first class I didn't get to. We were talking about notes from EO Wilson's book. I read the abridged addition for making those notes. But he talks about the possibility of inbreeding taboos. But when he introduces it he uses some more terms that are interesting.

He introduces by defining the effective population number. And that, I've defined it at the bottom there, the number of individuals in an ideal randomly breeding population, with one to one sex ratio, that would have the same rate of heterozygocity decrease as the total population under consideration. So that's what makes it the effective population.

It's usually the smallest number you can have and not have much inbreeding. At least you would not have anymore inbreeding than you would if they were bred randomly in the larger population.

And the numbers aren't really big. They get as low as 10 in some species. But they often are closer to 100.

And then he talks about Wright's Island Model. Now what that is he has a model of a population that's divided up into smaller groups. And we know that it's probably, at least very roughly, many populations are divided that way-- certainly humans were throughout much of our evolution.

That's why he calls it the island model, because he's got the population divided into these different groups that are-- he computed how big would those groups have to be in order to meet this effective population number definition. And then what are the advantages of a population being divided that way?

And there very clear advantages because when you have smaller groups the genetic differences that appear in the different groups will differ. And so you will have small genetic differences appearing in these different groups. And they'll be maintained if they're not interbreeding. Of course they would share the genes as soon as they-- mating involved movement from one group to the other. But if they're truly isolated that wouldn't be happening.

What would be the advantage to that? What would be the advantage if a population were divided that way? Well think if there were some big change in the environment, or new predators appeared, affecting all the groups.

Well if there's genetic differences in the groups then you're going to get better survival in some groups than in others. And that's what Wright, what his model was able to show. And that's one of the consequences of that model.

[? Menia ?] points out these, what he calls opposed selection tendencies that affects sociality, two very different effects in small groups-- that you have a greater chance of inbreeding, and inbreeding generally lowers individual fitness, depresses performance, lowers genetic adaptability. So that is more likely to happen especially if you're well below that effective population number.

But in the small groups of related individuals there would be very clear advantages because of greater amounts of altruistic behavior in close cooperation and groups. And then he talks about a somewhat different concept, assortative mating, also

known as homogamy. And this is the tendency you find in all animal groups, and certainly in humans. A tendency to choose a mate that looks similar to oneself or one's close relatives.

What does that mean? Well it probably means that the group-- I was talking to the TA's about my worries that the second project will go out, too. It's a small chance it's in the connection, but I don't-- so far, so good.

If you're going to always choose a mate, someone that looks similar to yourself, then the chances of the two shared genes, of course, might be greater. But that's only in a relatively small number of genes that affect the superficial appearance.

That was the end of that class. Then I asked you to read the chapter on genes, genetic influences on social behavior. We want to know the difference between genetic determinants of behavioral traits, genetic determination, and what sociobiologists-- what their view concerning genes and behavior is. Because you know that they've been accused a lot of basically saying genes are us, major trends are genetic.

Do the genes determine the development of specific social behaviors? First of all, I want you to think back to our discussion of ethology. We talked about Connor Lawrence and fixed action patterns. Because ethology was founded on the basis of the heriditability of fixed action patterns, and many of them are, of course, social behavior.

But we also knew that a fixed action pattern's not totally determined by the genes. It depends on motivational levels. And the motivational intensity is influenced by many environmental variables.

But the underlying action patterns, especially on the motor side, the fixed motor pattern, is considered genetic. And there's plenty of evidence for that. You can even get mouse populations that are genetically different that show differences in details of grooming behavior.

And all the members that have one gene, one genetic variant, will show one type.

And all of the individuals are almost identical. And the other groups are a little bit different. Certainly pretty strong evidence for genetic influences on that behavior.

That is only social behavior, of course, if there's grooming of each other in a group. But I want you, next, to remember that the nature/nurture problem, the way that controversy is normally dealt with now, especially in sociobiology, is to remember that-- where are we here?-- that both nature and nurture are always involved. You never have a purely genetic determination.

And the other thing that Alcott points out here is that the genetic studies most relevant to sociobiology are not developmental genetics at all, but population genetics. They deal most directly with the consequences of population changes and the frequency of different variants, or alleles, of given genes, not with the means by which these alleles shape or influence the biochemical pathways of developing individuals.

And so a lot of times the criticisms that sociobiologists are genetic determinists about social behavior is because they don't distinguish between ultimate and proximate research in biology. And he considers that the main problem with these criticisms, especially the critics that argue in this extreme fashion. They say sociobiologists are talking about traits that are hereditary, fixed, inevitable, unchangeable except by future selection for hereditary alternatives. Well we know that that can't be true because of the nature of what we know about how genes influence behavior.

But let me ask you about-- just leaving these slides for a minute-- what about these studies of the ethology, where they're dealing with, like the cat mating behavior, for example? And you can find relationships across species, and the behavior is almost identical in different groups. Is that not social behavior? And isn't it genetically determined?

And I would argue that it certainly is. But there are many learned aspects as well. So you will find individual differences in a group, especially on the stimulus side. They develop different preferences and so forth, even when the motor side is fairly fixed.

Then Alcott talks about this review done in 1998. So that was a long time after the books appeared in 1975. He called it the study of genetically determined social behavior. That was the main theme of the review.

But Wilson actually devotes an entire chapter in his book to explain that although genes are essential for the development of behavior, they don't determine it by themselves. Of course, I think most people who look at genes in behavior now would say that that would have to be true.

You can go to an extreme, like Richard Alexander did here. He just points out that genetic determinism is a ridiculous argument if you exclude environment completely, environment outside the DNA. Because it's always there, and there's always variables, which of course makes us wonder why the claim has been so persistent. So I'll deal with that next.

But this is the way Alcock summarizes it, basically about, at least the chemical environment of the DNA is always involved. And that is [INAUDIBLE].

But I want to point out that a lot of people who argue against the influence of genes, they just feel that environment and learning are a lot more important, that you can discount a lot of the genetic factors.

They also get upset by thinking about behavior in a totally deterministic way. They basically object to the basic assumption of a scientist, who's trying to explain physical causes of the effects of whatever they're investigating, whether its behavior or anything else. I mean psychologists can't do their work without assuming that there's specific effects on behavior, including genetic effects.

And so there are people like that. They don't like, in general, the deterministic approach that scientists take. So a lot of times the criticisms of those people is much broader than just talking about sociobiology. But sociobiology is what triggers their ire more than anything.

So this is an enduring myth, according to Alcock, despite the fact that all biologists

know that every trait of every organism develops through this interaction of genes and environment. But I think the myth persists. They don't like this approach, as I just pointed out, for much more general reasons that have to do just with sociobiology. So this is a convenient straw man to set it up like that.

I think it's the nature of human psychology. We believe in our ability to change our behavior, and other people's behavior.

And for many people, the flexibility of human behavior is mistakenly taken as evidence that cultural factors are the only real determinants of our actions. In fact, in some of the meetings where these arguments have come up, people usually outside of sociobiology, of course, or behavioral biology, will argue that. Culture is the only real determinant of human behavior.

And then I also feel that championing free will and freedom of action is pretty understandably very popular. In fact, I think humans have evolved an enthusiasm for freedom of action and a belief in free will. So I think we should be thinking like sociobiologists about that. Why does everybody feel that?

I mean, yes, there are some intellectuals that take a very deterministic approach, and say, we think we have free will but we don't. And they're being philosophical. But in their own behavior, do they believe in free will? You bet. We all do.

I think it's inherited. And I think it's adaptive to be that way.

Can a difference in one allele change a behavior? And here's this interesting figure, 3.1, to explain how a difference in one allele-- and he has them represented by capital and small b-- could result in a behavioral difference between two adults. And it's a kind of multiplier effect. This is the way I've reproduced it.

So you start with the fertilized egg. And here's one individual. Notice most of the genes are the same except this gene here. And this could be big B, little b. But it's different from this individual, who has the two recessive genes at the same locus.

So his behavior, if it's going to be affected, will be any effect of that one allele. And

this person doesn't even have that allele. So he's got to be affected by the other one. It's just one gene, so one protein. And that genetic difference, of course, will interact with the environment. And the environment can be different.

So the effects, if the environment's different for these two individuals, you're going to get differences that affects the adult. But then as they develop, of course, and neurons develop, we know that there are many effects of environment on that that are independent of these. But that, in fact, this allele could make a difference in the way these things happen during development.

So the effect here could be present throughout development with environments, as long as they're slightly different. It could lead to pretty big differences into adults, in spite of that allele.

And yet, the differences developed specifically because of that one genetic difference. So that's all that we we're talking about here.

So let's talk exactly how and why the relative frequency of the two alleles could change over multiple generations. Let's raise the possibilities from what we know. And these are the things I can think of.

Why B could increase and the recessive trait could decrease. First of all, if the recessive trait resulted in death before the age of reproduction, more often than the dominant trait. I'm just going to refer to them that way because that's a common way of expressing for one allele, the recessive and dominant allele affecting that gene.

A second thing could be B could result in behavioral difference, reduce the probability of successful reproduction in a particular environment, but not in all environments. So at least in that particular environment, the genes would change in their frequency.

All we need is probability differences. And if the recessive trait resulted in no change in probability of reproduction, but it did reduce parental care, that alone, you see, would result-- because you would have, statistically anyway, less survival the

offspring. And that would be enough then to lead to changes over multiple generations in the frequency of those two genes.

So let's talk about breeding for behavioral traits. We know that animals are bred for physical traits. You know how dogs have been bred to look different. As we see especially in farm animals, we've bred beef cattle, so they're meatier.

Well what about behavior? It's been done in the laboratory for a number of particular behavioral traits, where you ignore any physical differences and you only have a measure of behavior. For example, how loud crickets sing, and the ones that sing louder you can breed those with each other. The ones that sing less.

And at the beginning, they're probably all singing. But some of them don't sing as loud as others. And if you keep breeding, you will eventually end up with a loud singing group and a pretty silent group.

It's also been done with fruit flies. This has been the most common. And probably the most famous experiments with fruit flies are the production of learning mutants first produced by Chip Quinn of this department. Most of that work he did when he was at Princeton.

And he produced strains of fruit flies that in a particular learning test that he had set up to screen the fruit flies, they were particularly stupid. And he named them after vegetables. So he has rutabaga, he has turnip, and such individuals. And he became well known for that work-- the learning mutants in fruit flies.

It's also been done in mice and in rats. In mice they've produced spatial learning differences in different groups, and groups that differ in nest building activity. You just select the ones that are most vigorous about building big nests, and the one's that don't build such big nest, and don't nest in such a vigorous way. And if you breed them selectively for those behavioral traits, you'll eventually get groups that are very, very different in the way they build nests.

And you can do the same thing for spatial learning problems. And there, the best known is the rat experiments, where they had what's called the Hebb-Williams

maze. A fairly complex maze that they could change easily. They could change the problems so the rat would be trained on one, and then they'd change the barriers around and test them again, and do that repeatedly. And they'd come up with a measure of how intelligent-- it was called a rat intelligence test, how smart are they in learning mazes.

And of course, some of them learned much better than others. And so again, we can selectively breed them for how well they learn the maze. We can have the ones to learn better. We'll interbreed those ones that learn more poorly.

And you end up with a maze bright, maze dull strain. And then you can test for other differences, and what else happens to this animal? What happens in the brain?

It was the fruit fly work that led most directly to neuroscience studies. In fact Chip, after he came here, eventually did zero in on a particular gene for a particular factor in the brain that was found by other groups also to be involved in learning.

And when we deal with humans, the best studies probably that support genetic influences on behavior, are the twin studies. Do you all know the experiments studying concordance rates?

First of all, you have to collect large numbers of identical twins so that you can either test them or find out a lot about them. For example, do they become schizophrenic? And so that's certainly a big behavioral difference, the ones that become schizophrenic and the one's that don't.

So you find out, if one of the twins, you find out, is schizophrenic, what is the probability that his twin will have schizophrenia? Do you know difference what the probability is? It's about 2/3.

So it's not totally genetic. And it's interesting that that 2/3 for schizophrenia also applies to the kind of diabetes I have. If I had a twin brother-- it would have to be a brother because it would have to be monozygotic-- the brother would have a 2/3 chance of having the same thing. The concordance rate is not 100%.

So that just means there has to be environmental factors that also have a strong influence. And the degree to which the genetics and environment play roles can be estimated from just studying concordance rates. And that has been done for a number of different things.

About the selected reading for behavioral traits, I like many of the examples in this book, by Temple Grandin. She's a very interesting lady. She's actually, I believe, come here to give a talk once, at least I've heard her. I think when I heard her she was promoting one of her books, probably this one, at the bookstore in Porter Square, where they get authors to come every week, and sometimes twice a week, to give talks on their new books.

And I met her. I had a copy of her book, she signed it and talked to me about the teaching I was doing at MIT and so forth. Gave me permission, carte blanche, to use anything in her book in the class.

So she points out various things the behavioral traits that dogs have been bred for. They've been bred, as you know, for herding ability-- for example, the sheep dog. If you want to herd sheep, and you really do need dogs to do it efficiently, you don't want to choose just any dog. No matter how smart he is, how loyal, they are genetically different. And they have been bred specifically for herding. So the sheep dog is very, very good.

You're enhancing certain fixed action patterns and decreasing the influence of other fixed action patterns. In a way, you're enhancing detecting and stalking behavior. But you're inhibiting the later parts of the predatory behavior. They never attack the sheep. Predatory attack is also determined by multiple genes, or multiple components. It's not a single fixed action pattern, really, at least on the motor side.

Then retrievers, same kind of thing. Some dogs are terrible retrievers. But if you get a golden retriever, or a Labrador retriever, very easy because it's part of their genetics. And of course their trained for hunting, pointing.

I remember my father, who hunted pheasants, he had a pointer when I was a little

kid. And I still remember that dog, because he was so incredibly-- not just so incredibly loyal to my father, and not to his kids-- he was so good at what he did. He would stay right at my father's side.

I mean they're used for they're incredibly good senses, but also for their ability to detect the pheasant, and then stop and withhold their tendency to try to grab it and attack it themselves. Pheasants are mostly on the ground. They can fly to get away. So he would become rigid and go into this posture. It was very obvious.

And then, little signal from my father, then the dog would rush forward. Not to try to kill the pheasant, but to flush him so he would fly up. Because people that hunt like this usually wait, if they can't shoot them in air-- they're not good enough shots for that-- then they don't succeed in getting the pheasant.

And I remember the situation where there were two pheasants pulled up. And my father had a double barreled shotgun. And he got both pheasants. And then another little signal, out the dog went to retrieve them. So he could do both of these things, retrieving, pointing, hunting.

And yet many, many breeds of animals, you cannot train them. In fact, they weren't very good with children. So as his kids started growing older he gave the pointer to a farmer who had a lot of space.

We were now living in a town. When he did that hunting we were still outside of the town. But then he moved into the town so his kids could go to school, and got a dachshund, much better family dog.

So that's the dog I became most familiar with as I was growing up. Farm animals we know have been bred very specifically not just for their physical nature, but you can't help but when you're breeding farm animals, you'll tend to choose the ones that aren't as aggressive. So aggression has been reduced in the cattle, for example. And the amount of meat they produce has been increased.

For chickens, of course, it's egg laying ability. And that's been enhanced. They also breed chickens, of course, for meat.

Bulls it's a little different. And you find bulls that are breeders at cattle firms in he US, where they breed cattle. Again, they choose bulls they have to limit their aggression. But you want big, meaty bulls because that's what you're trying to breed.

My grandfather knew all about this kind of breeding of the bulls because I remember he wanted to let us see how he did it. And he went in on horseback and got a bull. And here comes the bull and we were all right there in the yard. Here comes this charging bull.

You could imagine what we felt. Here's this charging bull. But the bull, well known to my grandfather, was not interested in kids at all. He was only interested in one thing, those females in the yard. So my cousin opened the gate, the bull ran through and immediately began soliciting the attentions of the females.

But of course, if you're using the bulls for other things, like bullfighting, then you breed them differently. And there are bull breeders in Spain that specifically breed bulls for their ferocity in bull fighting.

And we know, of course, we're more familiar with breeding of horses. And they've been bred for various things, for speed-- the Thoroughbred-- for herding cattle-- the quarter horse. They're actually faster than Thoroughbred for a quarter of a mile. But they can't keep up with the Thoroughbred in a longer race.

So there are several breeds of horses. We know that workhorses tend to be bigger, meatier, stronger, but slower. But they're used to pull things-- pull wagons, pull plows, and so forth. Fewer of them now, but so some of them are still maintained.

So very specific breeding for behavioral traits. So this question concerns the problems that often happen when animals are bred for a single physical trait. What happens to them?

It's common for behavioral problems to appear. And this is discussed quite a bit in Temple Grandin's book. Her example I like was collie dogs, because when I was

growing up I remember Lassie. I don't know if you even know who Lassie is, but that was a popular dog in Hollywood movies.

Lassie grew up before this intense breeding in this period started to change the appearance of collies to make them have narrower an narrower skulls and emphasized the needle nose. So she said the result was brainless ice picks. Not like Lassie at all.

And that's the danger if you just ignore behavior when you're breeding for a particular physical trait. And this is not the only example. Breeds of dogs have particular problems. Usually it's that they get certain diseases. Or Cocker Spaniels, for example, many of them get a type of epilepsy. That's certainly a major behavioral problem, behavioral disease they get because of this breeding for their appearance.

So is it true that no genes for human behavior have been found? The argument was that we can dispense with the direct evidence for a genetic basis of human social forms in a single word, no evidence. And here's another quote from this book, called *Not In Our Genes*, by a Harvard professor, Lewonton, Rose, a British professor, and Kamin, I don't remember where he's from.

He said , no one has ever been able to relate any aspect of human social behavior to any particular gene or set of genes. Is that true still? I don't think so.

First of all, in your book there, Alcock just points out the absence of evidence isn't really evidence of absent, of non involvement of genes in social development. I guess it seemed to him that way, and that was the argument he made.

But I would point out more recent evidence for specific genes we say for a social behavior. I want you to keep in mind that even though specific genes are being found, that are correlated with various social behaviors and especially behavioral problems, genes code for proteins. And if we look at these behaviors, these abnormalities in humans-- autism, schizophrenia, and other mental diseases, attention deficit hyperactivity disorder-- yes, there's genetic contribution to all of

these. There's always multiple genes involved. Environmental effects are still strong.

So how could a gene that has what appears to be maladaptive consequences be present in some animals or people today? Why wasn't it lost? It's about the first thing I asked when I got diabetes is age 24. What's this gene doing around? Shouldn't it have disappeared 1,000 years ago?

First of all, it could be a recent mutation and will be lost. Or maybe it has its bad effects after reproduction. I said I got it at age 24, during the age of reproduction.

Many effects do occur later. It may have affects only in certain environments. If I were in an environment I wasn't exposed to the same viruses, I may never gotten it. It's an autoimmune disease triggered by viruses.

It's also of course because of modern medical treatments. But it's also-- remember genetic swamping. If you've forgotten what that is, go back and read it. It was in my notes, and you could find it also on the web.

And then I'm posting these homework questions. It's already at the end of the class here. But I am willing to give you-- I want you to spend most of the time on this one.

The others should be easier for you. If you have trouble with those, I'm happy to give you hints. I'm asking you to do these. They're similar to homework I gave last year because I want you to think about these things and be clear about the type of thinking you need to do. And this is the one that causes the most controversy.

So think about that. And you can ask me about it on Friday, if you wish. And I'm happy to give you some suggestions.