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PROFESSOR: Did anybody have any questions about this path analysis 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 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. All right.

So this is the only slide from the first class I didn't get to. We were talking about Wilson's notes from E.O. Wilson's book. I actually got these. I read the abridged edition for making those notes. But he talks about the possibility of inbreeding taboos.

When he introduces it, he uses some more terms that are interesting. He introduces by defining the effective population number, and I've defined it at the bottom there. The number of individuals in an ideal randomly breeding population with 1-to-1 sex ratio that would have the same rate of heterozygosity 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 any more inbreeding than you would if they bred randomly in the larger population. And the numbers aren't really big. They get as low as 10 in some animal-- some species, but they often are more 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 calls it the island model because he's got the population divided into these different groups that he computed how big would those groups have to be in order to be this effective population number definition.

And then what are the advantages of a population being divided that way? And there are 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-- assumes mating involved movement from one group to the other.

But if they're truly isolated, that wouldn't be happening. What would be 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.

And then he points out these what he calls supposed 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 and close cooperation in the 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.

And what does that mean? Well, it probably means that the group-- I was talking to the TAs about my worries that the second projector will go out too. There'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 that you share genes, of course, might be greater. But that's only in a relatively small number of genes that affect the superficial appearance.

All right. That was the end of that class, and 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. And we talked about Konrad Lorenz and fixed action patterns, because ethology was founded on the basis of the heritability 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's influenced by many environmental variables. But the underlying action pattern, 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 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 but 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 Alcock 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 pathways, 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 passion, they say that 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 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. OK? 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 Alcock talks about this review done in 1998-- so that was a long time after the books appeared in 1975-- about the study. 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. And of course, I think most people who look at genes and 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, 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're much more concerned-- they just feel that environment and learning are a lot more important, OK, 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 the scientist who's trying to explain physical causes of the effects of whatever they're investigating, whether it's behavior or anything else. I mean, psychologists can't do their work without assuming that there are 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 the [INAUDIBLE] 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 again, 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 adapted to be that way.

All right. Can a difference in one allele change a behavior? And he has this interesting figure 3.1 to explain how a difference in one allele-- and he has them represented by the capital and the 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. OK? And here's one individual. 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. OK. 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 [? within ?] environments, as long as they're slightly different, could lead to pretty big differences in two adults in spite of that allele. And yet the differences developed specifically because of that one genetic difference. So that's all that 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. OK? And these are the things I can think of-- why B could increase and the recessive trait could decrease. First of all, if b, 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.

The second thing, it 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 of the offspring. And that would be enough then to lead to changes over multiple

generations in the frequency of those two genes.

All right. So let's talk about breeding for behavioral traits. We know that animals are bred for physical traits, how dogs have been bred to look different. OK? And as we see it 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 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. OK?

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 produced spatial learning differences in different groups and groups that differed in nest-building activity. You just select the ones that are most vigorous about building big nests and the ones that don't build such big nets 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 was called the Hebb-Williams maze, a maze, a fairly complex maze, that they could change easily.

They can change the problem so the rat would be trained on one, and then they'd change the barriers around, 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 learn much better than others. And so again, we can selectively breed them for how well they learn the maze. The ones that learn better we'll interbreed with 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. OK.

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 ones that don't. So if one of the twins, you find out, is schizophrenic, what is the probability that his twin will have schizophrenia? Do you know what the probability is? It's about 2/3. OK? 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-- OK, it would have to be a brother because I'd have to be monozygotic OK-- 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, OK, 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 class.

OK. So she points out various things, 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 smarter 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. I mean, this 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, they're 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 their 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. You know, 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 a 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 the 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 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, a much better family dog. OK. 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 farms in the US where they breed cattle. Again, they choose bulls that are-- 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 is 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. OK. 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 bullfighting.

And we know, of course, we're more familiar with the 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 the Thoroughbred for a quarter of a mile, but they can't keep up with the Thoroughbred in a longer race.

And so there's 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. Few of them now, but 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 liked was Collie dogs, because when I was growing up, I remember-- do you remember Lassie? I don't know if you even who Lassie is, but that was a popular dog in Hollywood movies.

And Lassie grew up before this intense breeding in this period started to change the

appearance of Collies to make them have narrower and narrower skulls and emphasize 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. Cocker Spaniels, for example, many of them get a type of epilepsy. That's certainly a major behavioral problem, behavioral disease, that 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 the Harvard professor Lewontin, 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 absence 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 [? aren't ?] being found that are correlated with various social behaviors and especially behavioral problems, they're not-- genes code for proteins, OK?

And if you 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, and environmental effects are still strong. OK?

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 at age 24. What's this gene doing around? Should 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, so during the age of reproduction. Many effects do occur later. It may have effects only in certain environments. If I weren't in an environment, I wasn't exposed to the same viruses, I may never have 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 can find it also on the web.

And then I'm posting these homework questions. It's already at the end of the class here. 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.