Notes for an Undergraduate Course on Behavioral Genetics and Evolution

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My course at Penn covered, first, Behavioral Genetics, and, second, some facets of evolution and evolutionary psychology. These notes derive from concise handouts posted online in connection with each lecture. The handouts were intended to reinforce most of the main points. The original handouts contained cuttings from books and articles that have been removed from this edition.

The distinctive feature of the course (and the notes) is the heavy emphasis on equation-based modeling in both Behavioral Genetic and evolution. The course had no formal mathematical or statistical prerequisites, and the main tool used was high school algebra, but there was a steady diet of modeling and the course would not have been appropriate for students who disliked mathematics or statistics. In fact, about half of the students had had a college statistics course, and all students were asked to read a survey of elementary statistical concepts at the very beginning of the course.

This is certainly not an "online textbook," and I doubt that anyone will be tempted to base a course on these notes, but I do hope that some of the material will make its way into more conventional courses in these areas.

Some of the material is novel, e.g., the use of data from studies with both twins reared apart and twins reared together to assess the (in)accuracy of $2(r_{mz} - r_{dz})$ for estimating heritability from twins reared together. Use of this formula is universally deprecated, but it is surprising to see just how inaccurate it is. See Lecture 9.

My approach to estimation of heritability and environmentality is based solely on correlation coefficients since covariances are seldom reported in journal articles and raw data are seldom publicly available. In addition, I think that the old-fashioned formula-based approach using correlations has considerable pedagogical value.

These notes are only lightly edited, and citations are informal and incomplete, preserving the flavor of the lectures.

Enjoy!

Lecture 1

Basic Statistics—Correlation

See the reading assignment for definitions of mean, variance, standard deviation, z-score, scatter plot, and correlation. Correlation is perhaps the most important statistical concept for the first half of this course, and we will need to focus on certain features of correlations that are not emphasized in standard courses. Everything you need to know will be presented in the reading and lectures, but you will have to take a lot of it "on faith." I'm just "giving you the facts," not proving them.

The correlation (or correlation coefficient), usually denoted r or R, is a numeric measure of the relationship between two variables, like height and weight or math and verbal SAT, in some sample or population. These variables are often generically denoted X and Y or x and y. Correlation measures the extent to which larger values of X tend to be paired with larger values of Y, and smaller values of X tend to be paired with smaller values of Y in the given sample or population. This is certainly true for height and weight as well as for math and verbal SAT in typical samples, and results in positive correlations in these two cases.

Remember, correlation (like mean, variance, and standard deviation) is a characteristic of a sample or a population, not a characteristic of a particular pair of numbers. We don't say that two tall men are "positively correlated for height."

The largest possible positive correlation is 1. A correlation of 0 indicates no simple relation between X and Y (e.g., between height and math SAT). Negative correlations (between 0 and -1) indicate that larger Xs tend to be paired with smaller Ys and vice versa.

If the correlation is nearly 1 or -1, it means that Y is nearly <u>linearly</u> related to X, $Y = c + dX$, where d has the same sign as r.

An important feature of the correlation coefficient is that it is unaffected by linear transformations of X or Y or both. For example, if X is height measured in feet, then 12X is height measured in inches and 12(X-6) is number of inches above 6 feet. All of these variables have the same correlation with any variable Y. Since height-in-inches is much larger numerically than height-in-feet, it follows that the absolute size of the variables tells us <u>nothing</u> about the expected size of correlations.

Similarly, if X and Y are monthly incomes of randomly chosen people, we expect r to be nearly zero in wealthly neighborhoods as well as in middle-class and poor neighborhoods, provided that all people were sampled randomly from the <u>entire</u> neighborhood. A good case to think about is an unusual neighborhood where 98% of the residents are extremely rich, but the remaining 2% are extremely poor. Even though the overwhelming proportion of pairs would have two rich people, the income correlation would <u>still</u> be nearly zero, <u>provided that everyone was sampled randomly from the entire neighborhood</u>.

If, on the other hand, we sample pairs of close neighbors, r would be positive in all of these examples, since incomes of close neighbors tend to be closer than incomes of people sampled completely randomly (because of relatively wealthy areas and relative poor areas within the larger neighborhood). Also, IQ test scores for married couples are positively correlated, since there is a tendency of smarter people to choose smarter mates, and less smart people to choose less smart mates.

Here are some basic principles: Measurements on unrelated ("randomly sampled") individuals will be uncorrelated (zero or nearly zero correlation), just as the faces of two dice are uncorrelated. More generally, you should expect X and Y to be uncorrelated unless there is some fairly specific reason to expect otherwise. Factors that make X and Y large (or small) for <u>all</u> members of a sample or population

will not produce a positive correlation. But factors that make paired X and Y values (e.g., height and weight of the same person) more similar than unpaired X and Y values will produce a positive correlation.

Behavioral Genetics is interested in genetic and environmental factors that make relatives more similar than non-relatives. These factors are revealed by certain correlations. That's why correlation is a basic tool in Behavioral Genetics.

Even if all values of X and Y are enormous positive numbers, it's still possible for larger values of X to be associated with smaller values of Y and vice versa, which would produce a negative correlation (e.g., X = number of cigarettes smoked per year and Y = number of hours per year not under medical treatment for lung cancer, in a population of heavy smokers.). The correlation of enormous positive numbers can also be zero. Correlation depends only on the co-variation of X and Y—the tendency of especially large or especially small values of X to be associated with especially large or especially small values of Y.

Most of the correlations that occur in this course will be correlations between relatives, especially twin correlations and parent-child correlations, and you should think of such correlations as measures of similarity of the relatives on whatever dimension is under discussion. Thus the .69 correlation of monozygotic twins reared apart for IQ (Bouchard, Lykken, McGue, Segal, and Tellegen, *Science*, 1990, cited frequently below) is an indication that such twins have similar IQs. By the way, I will often express correlations as percentages, "69%" in this case.

Components of trait variance

T = variable under consideration

(e.g., IQ; T for "Trait" or "Test" or "Total")

= true score + measurement error

= genetic part + environmental part + measurement error

= G + E + M.

E = common (or shared) part + unique (or non-shared) part

= C + U

so, finally,

$$T = G + C + U + M$$

The fundamental objective of classical Behavioral Genetics is to tell us something about the average sizes of the components G, C, U, and M for different trait measurements T in various populations. But the way of measuring "size" is not what you might expect. Instead of measuring average size by the mean, Behavioral Genetics uses the variance. In other words, a "big component" is one with large variability. Behavioral Genetics is only concerned with trait variability, and the proportion of that variability that is associated with genes, shared environment, unshared environment, and measurement error. In particular, the proportion of total trait variability associated with genes is of fundamental interest. This proportion is called the heritability of the trait.

G, C, U, and M are assumed to be uncorrelated (r = correlation coefficient = 0) in the population under consideration. This implies that

$$Var(T) = Var(G) + Var(C) + Var(U) + Var(M) ,$$

where Var(T) is the population variance of T.

Dividing by Var(T),

$$1 = Var(G)/Var(T) + Var(C)/Var(T) + Var(U)/Var(T) + Var(M)/Var(T)$$

= (broad sense) heritability + common environmentality + unique environmentality + proportion of error variance ("unreliability")

$$1 = h_b^2 + c^2 + u^2 + m^2$$

The boxed formula relates to the variance of test scores. We can estimate these variances from certain correlations between relatives using formulas for correlations that we will develop.

Intuitive principle: For MZ twins (MZA, reared apart, and MZT, reared together), correlations between twins can be obtained by summing those terms on the right hand side of the boxed formula that correspond to components shared by the twins. Thus

(1) $r(MZA) = h_b^2$ (since these twins share only G, if we ignore the contribution of common pre-natal environment)

(2) $r(MZT) = h_b^2 + c^2$ (since these twins share G and C).

(3) r(test,retest) = reliability $= h_b^2 + c^2 + u^2$

(4) $1 = r(T,T) = h_b^2 + c^2 + u^2 + m^2$ (the equation from which we started!)

The MZ Twin Method (my terminology) of estimating h_b^2, c^2, u^2, and m^2:

r(MZA) $= h_b^2$ = heritability

r(MZT) - r(MZA) $= c^2$ = common, family, or shared environment

reliability - r(MZT) $= u^2$ = unique environment

1 - reliability $= m^2$ = proportion of measurement error variance = unreliability

These equations hold exactly with population correlations, and, with sample correlations, the quantities on the left define estimators of the parameters on the right.

Illustration, using IQ data from Bouchard, et al, 1990

		WAIS FSIQ	Raven-Mill-Hill
$h_b^2 = r(MZA)$	= .69	69%	78%
$c^2 = r(MZT)-r(MZA)$	= .88 -.69	19%	-2% (effectively 0)
u^2 = rel. - r(MZT)	= .90 - .88	2%	14%? (using WAIS reliability)
m^2 = 1 - reliability	= 1 - .90	10%	10%? (from WAIS)

Note: Large heritability is not the same as "biological determinism." Near-sightedness is highly heritable, but it is usually easily correctable by glasses, a specially adapted environmental intervention. One of the basic missions of medical science is to furnish environmental interventions to counteract genetic deficiencies. It's "no big deal."

Correlation Exercise: (a) Use the web site given in the syllabus to draw a scatter plot of the following data, and compute summary statistics.

X	Y
5	4
2	2
3	1
6	3
4	3
1	1

Study the summary statistics, especially the correlation coefficient. Print your results.

(b) Repeat (a) using only the three data points with $X \geq 4$. Print your results.

(c) Repeat (a) adding 100 to all X values and 200 to all Y values. How does this affect the means, variances, and correlation? Print your results.

(d) Repeat (a) multiplying all Xs by 10 and all Ys by 20. How does this affect the means, variances, and correlation? Print results.

Lecture 3

Observation: Gene regulation, or "genetics isn't just genes". Every cell in the body carries the same DNA, hence the same genes, which are regions of chromosomes that provide templates for making ("transcribing" or "expressing") proteins. But every cell does not blindly output all these proteins. Instead, transcription is regulated by other genes in such a way that each protein is only expressed in certain tissues at certain times. The proteins produced by the regulating genes are called *transcription factors*, and they control their target genes by attaching themselves to *promoter regions* ("promoters") on the chromosome near the target. These promoters consist of DNA that is not part of any gene, but promoters, like genes, are subject to the evolutionary processes of mutation and natural selection. Moreover, they are "better" targets for these processes than genes, since a mutation in a gene often (but not always) renders the corresponding protein useless, whereas mutation in promoters will more often have incremental effects on the amount and timing of gene expression. These matters are discussed in greater detail in Matt Ridley's *Nature via Nurture* (HarperCollins, 2003), Chapter 1. [A number of chapters from this fine book were assigned, and there are many references to it in these notes.]

Hans Eysenck's scale for Neuroticism reflects "emotional instability." His Psychoticism might be described in common language as "recklessness." Eysenck's Extraversion and Neuroticism are similar to Positive and Negative Emotionality in the Minnesota Twin Study, and Psychoticism is similar to the reverse of Constraint. Positive Emotionality, Negative Emotionality, and Constraint are constructed from Tellegen's Primary Personality scales that you read about in preparation for this class.

Analysis of Minnesota Twin IQ and Personality Correlations, via the MZ Twin Method

	WAIS IQ	R-M-H IQ	Pos Emo	Neg Emo	Constraint
h_b^2	69%	78%	34%	61%	57%
c^2	19%	-2%	29%	-7%	1%
u^2	2%	14%	26%	35%	31%
m^2	10%	10%	11%	11%	11%

General comments

1. The heritability values for IQ are quite large for these adults. The environmentality values are correspondingly small.

2. Many scientists argue that heritability estimates should be divided by reliability to get a truer picture of genetic contribution. This is called correction for unreliability. For example,

WAIS FSIQ heritability corrected for unreliability = 69%/.90 = 77%.

3. Common environment, estimated by r(MZT) – r(MZA), makes at most a small contribution to adult IQ according to these data. Of the three personality scales, only Positive Emotionality shows evidence of a common environmental effect.

There are two negative values, but they are very small ("not significantly different from zero"), consistent with r(MZT) = r(MZA) at the population level. There is no strong evidence of r(MZT) < r(MZA) at the population level for any of these measurements. This contradicts Ridley's claim (p. 78) that twins reared together are less alike than twins reared apart.

4. Large heritability values do NOT mean that environment never affects IQ. That's obviously silly. Instead, the large values mean that the ORDINARY range of environmental variation doesn't make much difference, on the average.

5. Heritability pertains to variation, not to means, and to populations, not individuals.

6. The Flynn Effect (increaing population mean IQ over time) shows that one has to consider genetic and environmental contributions separately for means and variances. Current variation in IQ is highly affected by genetic variation, but changes in population mean IQ over time depend on environmental changes. Ripley's "finger number" example illustrates the same point. The mean (or "normal") number of fingers (10) is completely determined by genes, but the variability is a function of (infrequent) environmental accidents, so heritability is zero.

7. Ridley presents no evidence for his claim (in the third paragraph on p. 92) that differences in attraction to intellectual pursuits are a major cause of differences in IQ, which he references to a paper by Dickens and Flynn (*Psychological Review*, 2001).

Lecture 4

Several ways that the theory behind the MZ Twin Method is oversimplified

1. The theory ignores possible inflation of r(MZA) due to common pre-adoptive environment, including intra-uterine environment. However, we know that intra-uterine variations don't account for most of MZA similarity since DZA twins, who also share the same womb, are not nearly as similar as MZAs.

2. The theory ignores possible inflation of r(MZA) due to selective placement—the deliberate attempt to match backgrounds of adoptive parents and biological mothers. (Bouchard shows that this inflation is small.)

3. The theory ignores reduction in environmental variation due to preferential placement—the deliberate attempt to place adoptees in "good" homes. (Thus very high IQ heritability estimates from adoption studies may not apply to the most disadvantaged.)

Both selective and preferential placement tend to inflate heritability estimates.

4. The theory ignores G-E correlation.

(a) Passive G-E Correlation. Perhaps high-G parents not only tend to have high-G children, but also tend to provide these children with high Es.

(b) Reactive (or Evocative) G-E Correlation. Perhaps parents or teachers of brighter kids notice that they enjoy intellectually engaging activities, and react by making an effort to provide them.

(c) Active G-E Correlation. Finally, brighter kids may actively seek out environments that are especially conducive to intellectual development.

5. They ignore G-E interaction. The point here is that the formula $T = G + E + M$ involves only the sum of G and E. This situation is called additivity. G and E are said to act additively. Any departure from additivity is called interaction. The quintessential example of interaction is some kind of a product term

$$T = G + E + G \times E + M.$$

Realistic formulas for departures from additivity will not be simple products. However, it is customary to denote these departures by "G×E" anyway. You are just supposed to know that this expression is "some kind of non-additive term," not literally a product.

Examples: PKU, and the Caspi and Turkheimer papers we have discussed.

Ridley does not distinguish between correlation and interaction. He call both of them interaction. But he strongly objects to neglecting these factors, whatever they are called. That's what his book is about! I assign chapters from it because I want to remind you of what is being neglected in the lectures! But ever Ridley admits (at the end of the second paragraph on p. 80) that interaction "is less powerful than many believe."

Lecture 5

Observation: Ridley's central theme is that genes function under environmental control. However, he does not emphasize the important distinction between such control during development, when organs (including the brain) are built, and day-to-day control of the fully developed organism. Some of his examples concern the latter, but only the former is relevant to the Nature-Nurture Problem.

There are two kinds of cells in the body: germ cell (sperm, eggs) and somatic cells. The former are responsible for heredity, and the latter are responsible for running the body (including servicing the mind). Traditional Behavior Genetics (including my lectures) have been 100% concerned with heredity, but Ridley's book is equally concerned with heredity and running the body. Thus use of Ridley's book (including Chapters 1 and 2 which will be assigned later) has significantly broadened the scope of the course. However, it is important for students reading Ridley to try to discern whether or not various topics are concerned with heredity.

Path diagrams and path analysis

Unlike ordinary multiple regression analysis, the "independent" variables in path analysis are typically "latent," that is, not directly observable. For example, in T = G + E + M, none of the independent variables on the right hand side are directly observable.

Path diagrams illustrate path analysis. If an independent variable affects a dependent variable, one draws an arrow from one to the other. The arrow has a numerical path coefficient attached to it. This is sometimes an actual number, but, more usually, a parameter to be estimated, denoted by a letter .

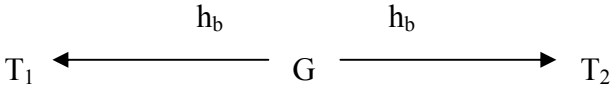
The independent variables may be correlated. This is indicated by connection by a curved double-headed arrow. The value of the correlation, or a letter denoting it, is attached to the line.

A path is defined to be a string of arrows with (a) no loops, (b) no going forward, then backward, and (c) no more than one curved arrow. ((b) means that association is not transmitted through common effects, e.g. temperature and wind are unrelated, even though they both affect wind-chill.)

The purpose of path analysis is to calculate the correlation of two dependent variables. This correlation is the sum of the weights of the paths connecting the variables. The weight of a path is the product of the path coefficients and correlations on it.

The simplest cases

MZA



If h_b is the coefficient of the path from G to T, then the diagram shows that

$r(MZA) = h_b \times h_b = h_b^2$ = broad heritability

A more complete version of the path diagram would add unique environment and measurement error. However, these additions would not affect r(MZA) since U_1 and U_2 are uncorrelated, as are M_1 and M_2. Generally, I will omit latent variables from path diagrams if they are irrelevant to calculation of correlations.

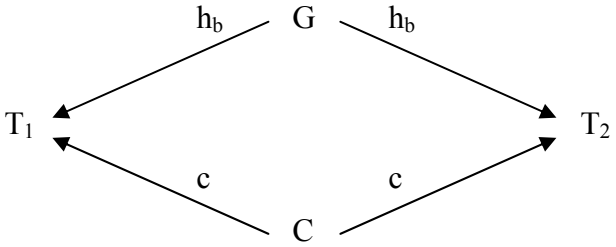
Focusing on just one side of the MZA path diagram and omitting the subscript, we see that

$r(T,G) = h_b$.

This provides the simplest answer to the question “What is h_b?”.

More generally, if two variables are connected by a straight arrow, and this is the only path between the variables, then the correlation of the variables is the path coefficient.

MZT



The environmental path from T_1 to T_2 via C has weight c^2, so

$r(MZT) = h_b^2 + c^2$.

MZA with dominance

Terminology: A_1 and A_2 are <u>alleles</u> (or genes) at some genetic locus, A_1A_1 , A_1A_2 , and A_2A_2 are <u>genotypes</u>, A_1A_1 and A_2A_2 are <u>homozygotes</u>, A_1A_2 is a <u>heterozygote</u>. The <u>maternal allele</u> is inherited from the individual's mother. The <u>paternal allele</u> is inherited from the individual's father. A <u>quantitative trait locus</u> (QTL) is a genetic locus that makes a contribution to a trait (e.g., IQ, Extraversion) that depends on many locii.

Consider, for a moment, a trait that depends on only one genetic locus. It is usually impossible to express G as a simple sum of maternal and paternal components, since some combinations of maternal and paternal genes have particularly strong effects. Such effects are called dominance, and they represent interactions of maternal and paternal genes.

More precisely, the deviation of G from a simple sum is called the <u>dominance deviation</u> and denoted D. We shall shortly see how D extends the simpler concept of dominance that you may have encountered in high school biology courses.

G = maternal gene effect + paternal gene effect + dominance deviation

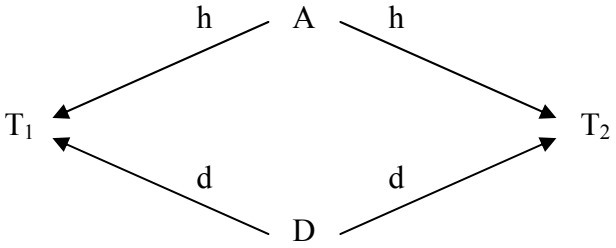
= additive genetic effects + dominance deviation

or, introducing standard notation,

G = A + D

Dominance is the interaction of the two additive gene effects.

The path diagram for MZAs with dominance is



Consequently $r(MZA) = h^2 + d^2$. Consistency with our previous formula for r(MZA) requires $h_b^2 = h^2 + d^2$.

The term heritability is ambiguous. Sometimes it refers to broad heritability, h_b^2 , and sometimes it refers to h^2 , which I will call narrow heritability. In articles in scientific journals, "heritability" usually refers to narrow heritability.

More on dominance

We have just seen that G = A + D, where A is the sum of maternal and paternal gene effects, and D is the deviation of G from such a sum. Suppose, for example, that the effect of the A_1 allele is +1 and the effect of A_2 is -1. I will illustrate various possibilities for D.

Genotype	A_1A_1	A_1A_2	A_2A_2
A	1 + 1 = 2	1 - 1 = 0	-1 - 1 = -2
D	0	0	0
G	2	0	-2

Here there is no dominance, and G for the *heterozygote*, A_1A_2 , is precisely intermediate between its values for the two *homozygotes*, A_1A_1 and A_2A_2 . It turns out that these conditions are equivalent: intermediate G for the heterozygote occurs if and only if dominance is zero for all genotypes. Dominance is present in the remaining examples.

A	2	0	-2
D	-.5	.5	-.5
G	1.5	.5	-2.5

D	-1	1	-1
G	1	1	-3

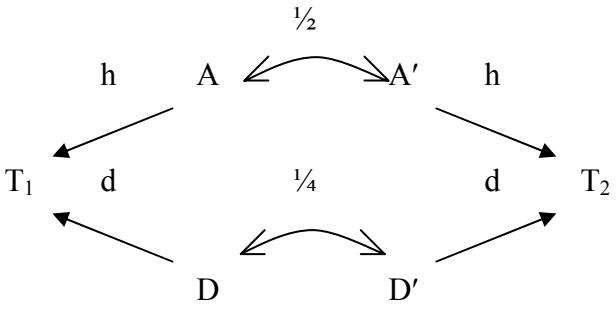
This last example is like those in high school—A_1A_1 and A_1A_2 have identical G-values. I will call this complete dominance, and I will call the prior example partial dominance, since the heterozygote is closer to one homozygote than to the other.

Let me reiterate that it is usually impossible to express G as a simple sum of maternal and paternal effects. You do as well as you can—that's A, and the error, D = G - A, is called dominance.

Lecture 6

DZ twins (or ordinary siblings) with dominance

The path diagram for DZA twins with dominance is



Therefore,

$r(DZA) = ½\ h^2 + ¼\ d^2$ (taking account of dominance).

Epistasis (discussed later) does <u>not</u> enter into DZ twin correlations.

The ½ additive factor correlation reflects the fact that the twins share half their genes, on the average.

The dominance correlation ¼ = (1/2) × (1/2) represents the probability that the twins get copies of the same gene from the mother <u>and</u> get copies of the same gene from the father. In this case the twins are copies of each other at the locus in question, and one would expect the "configural" information represented by dominance to come into play.

Similarly,

$r(DZT) = ½\ h^2 + ¼\ d^2 + c^2$.

There is no difference genetically between DZ twins and ordinary siblings, so the genetic parts of our formulas would apply just as well to siblings. However, the common environment terms would presumably be smaller for siblings, if for no other reason than their difference in age. And, by the way, c^2 may be different for MZ and DZ twins. We will consider this possibility in the next lecture.

Epistasis

Suppose now that G depends on <u>several</u> quantitative trait locii. You might try to approximate it by summing A and D terms from all these QTLs, but there will usually be an error, called <u>epistasis</u> and denoted I,

$I = G - \Sigma A - \Sigma D,$

where the sums are over all the locii on which G depends. People are too lazy to write sums all the time, so they just write A for ΣA and D for $\Sigma\ D$. Then

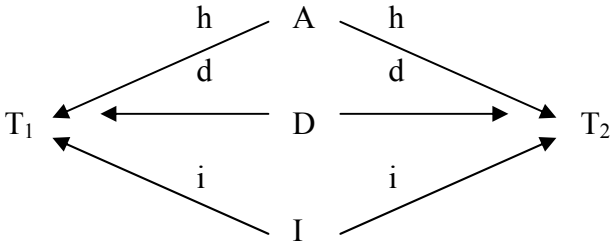
G = A + D + I = "additive part" + "dominance" + epistasis.

When this is done carefully, A, D, and I are uncorrelated.

Dominance and epistasis are referred to as ***non-additive effects***.

More path diagrams

MZA with dominance and epistasis



Thus

$r(MZA) = h^2 + d^2 + i^2$.

But $r(MZA) = h_b^2$, so $h_b^2 = h^2 + d^2 + i^2$.

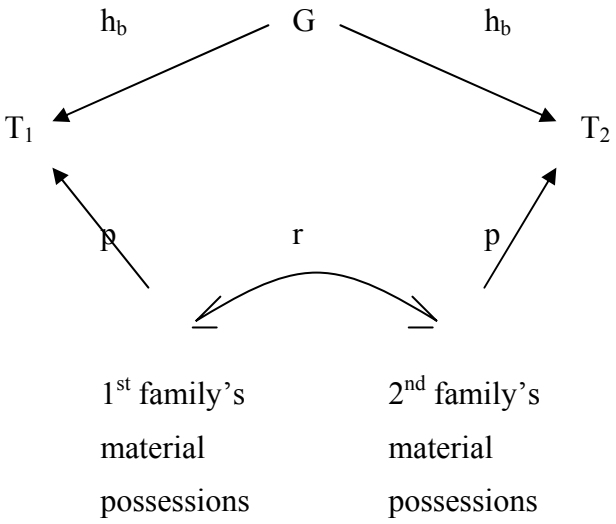
For MZTs there is an additional path through C, so,

$r(MZT) = h^2 + d^2 + i^2 + c^2$.

Sex is an example of a purely epistatic trait. The correlation between MZ twins reared apart is 100%, so broad heritability is $h_b^2 = 1$. But the correlation between DZ twins is zero, so both $h^2 = 0$ and $d^2 = 0$. Thus genetic variance must be entirely epistatic: $i^2 = 1$. And, indeed, sex does depend on an entire chromasome, not just a single locus!

MZA with selective placement

Adding "family's material possessions" to the MZA diagram, with path coefficient p and correlation r between families, we get



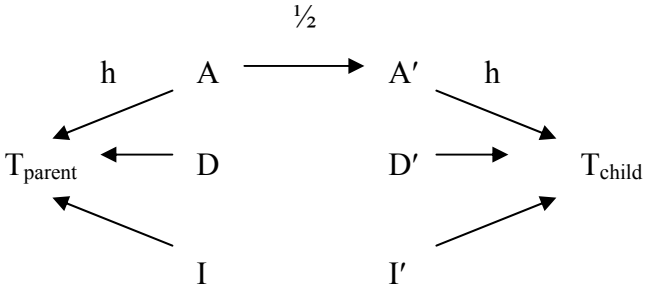
Therefore

$r(MZA) = h_b^2 + r \times p^2$.

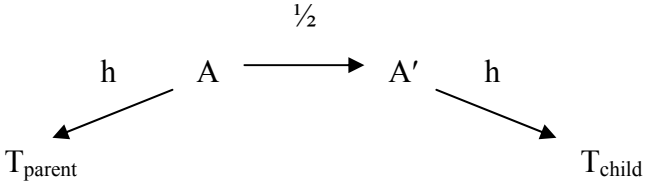
For the data of Bouchard, et al. (1990), the product term is $.402 \times .279^2 = .032$, which is not very large. Similar products for other family variables are even smaller—much smaller, in fact. So it appears that selective placement did not have a big impact on MZA correlations.

Parent and Child

If the parent and child are separated at birth, the path diagram is



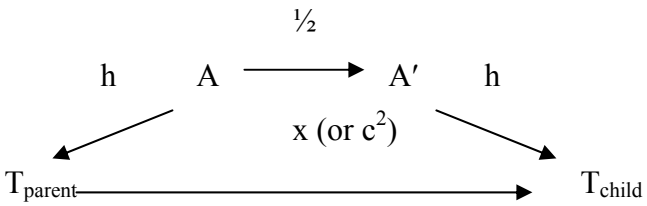
Eliminating irrelevant variables, we get



Hence

r(parent, separated child) = ½ h^2

If the parent and child are not separated, the path diagram is



so

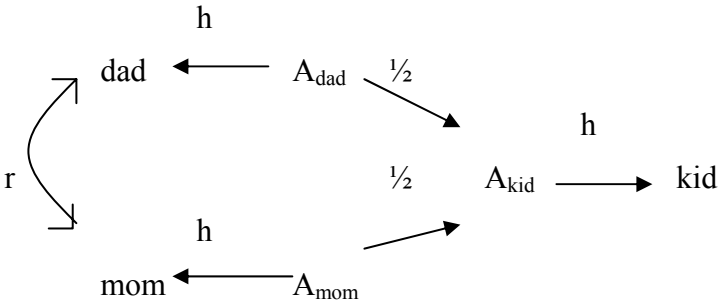
r(parent, not separated child) = ½ h^2 + x

In many cases the x is denoted c^2 to make this equation look like the twin equations. However, there is absolutely no reason to expect the x or c^2 term for parents and children to have the same magnitude as the c^2 term for twins or siblings, since the quantities are totally unrelated. The h^2 (narrow heritability) however, is, the same in all the formulas in which it appears.

Only the additive component of heritability enters these formulas. If heritability is predominantly non-additive, and x is small, then there will be little parent-child correlation. For example, the correlation of parent and child for sex is zero!

Assortative Mating

So far we have assumed that mates are chosen at random, which implies that mates are uncorrelated for all traits. If mates are correlated for some trait, as is often the case, we have assortative mating for that trait. This leads to a more complex path diagram for calculating the correlation between parent and a separated child.



Taking account of the two relevant paths, the correlation formula is

r(mom, separated child) = ½ h^2 + ½ h^2 r(mom,dad)

= ½ (1 + r(mom,dad)) h^2

and, adding an environmental path,

r(mom, not separated child) = ½ (1 + r(mom,dad)) h^2 + x.

So assortative mating increases the correlation of parent and child. This makes sense—to the extent that dad is similar to mom, the kid is getting an extra dose of mom through dad, which should increase the kid's resemblance to mom.

Correlations of Spouses for IQ and Eysenck's Personality Variables

Scale	IQ	Psycoticism	Extroversion	Neuroticism	Lie
r	.33	.16	.06	.13	.28

Spousal Correlations for Social Attitudes

Scale	Traditionalism	Radicalism	Tender-mindedness
r	.26	.54	.57

DZAs with assortative mating but no dominance

I omit the complex path diagram, but the correlation formula is

$r(DZA) = ½ (1 + r(mom,dad)\ h^2)\ h^2$

Add c^2 for DZTs. Unlike the comparable parent-child formula, the mom-dad term is deflated by h^2. Thus the effect of assortative mating on the DZ twin correlation is smaller than its effect on the parent-child correlation.

Assortative mating has no effect on MZ twin correlations.

Lecture 7

Bouchard's (*Current Direction in Psychological Science*, 2004) table estimates the heritability of Conservatism at 45-65% in people over 20 (and 0% for under 20). Data from a source discussed next week permits heritability of political party affiliation in the USA to be very crudely estimated at just under 50% by doubling the difference between MZT and DZT correlations. The estimate for Abortion attitudes is 64%. We will discuss this kind of heritability estimation in considerable detail next week.

Studies of MZ and DZ twins reared apart and together

The MZ Twin Method involved only correlations of MZ twins. I now want to extend this method by taking account of data from DZ twins. I will call the extended method the Full Twin Method. This terminology is strictly my own—you won't find it in the literature. Incorporating data from DZ twins permits us to estimate an overall measure of non-additive effects (dominance and epistasis).

Full Twin Method

$h_b^2 \sim$ r(MZA) (This means: "Use sample r(MZA) to estimate h_b^2 .")

(1) $c_{mz}^2 \sim$ r(MZT) - r(MZA)

These two formulas are carried over from the MZ Twin Method, and a formula like the last one can be applied to DZs.

(2) $c_{dz}^2 \sim$ r(DZT) - r(DZA)

Define overall common environmentality, c_o^2 , as the average, $(c_{mz}^2 + c_{dz}^2)/2$, of the left hand sides of (1) and (2). Estimate it by the average of the right hand sides. The "o" subscript stands for "overall."

Define overall unique environmentality, u_o^2 , in such a way as to preserve the equation $h_b^2 + c^2 + u^2 + m^2 = 1$, i.e.,

(3) $u_o^2 + m^2 = 1 - h_b^2 - c_o^2$

and estimate $u_o^2 + m^2$ by plugging in the above described estimators of h_b^2 and c_o^2 on the right. If m^2 is known from other sources, it can be subtracted to obtain an estimate of u_o^2 .

Define nonadditivity, n^2 , by

(4) $n^2 = ½\, d^2 + i^2$, (recall that our nonadditive effects are dominance and epistasis, indexed by d^2 and i^2 , respectively)

and estimate it via

(5) $n^2 \sim$ r(MZA) – 2r(DZA) .

The origin of these formulas is as follows. Compare the formulas

r(MZA) = $h_b^2 = h^2 + d^2 + i^2$

r(DZA) = $½\, h^2 + ¼\, d^2$,

(The correlations in these formulas are population correlations.) It follows that

(6) r(MZA) – 2r(DZA) = $½\, d^2 + i^2$,

which is our measure of non-additivity, n^2 . The estimation formula (5) arises by replacing the population correlations in (6) by the corresponding sample correlations.

Note that non-additivity is part of broad heritability. In fact,

$h_b^2 = h^2 + d^2 + i^2 = h^2 + ½\, d^2 + n^2$.

If n^2 is zero, then d^2 is zero too, and h_b^2 equals h^2 . In fact h_b^2 equals h^2 if and only if n^2 is zero.

Example. Let's consider some real data. Here are the correlations for Extraversion from the Swedish Adoption/Twin Study of Aging (SATSA):

twinship type	MZA	MZT	DZA	DZT
r	.30	.54	.04	.06

And here are the estimates obtained from our formulas:

h_b^2	c_o^2	$u_o^2 + m^2$	n^2
.30	.13	.57	.22

It is important to stress the difference between sample and population quantities. The numbers in the second row are sample estimates of the population parameters in the first row.

The sample-population distinction leads us to the to the question of whether each of these sample values is significantly different from zero, meaning that they are sufficiently large to effectively rule out the possibility that the underlying population values are zero. I have decided to downplay such questions in this course. Instead, I will simply introduce some ad hoc terminology for describing the size of the estimates themselves:

size	terminology
0.00-0.20	"small" or "inconsequential" or "trivial"
0.21-0.40	"medium" or "moderate"
0.41-0.60	"large"
0.61-1.00	"very large"

According to this terminology, the overall common environmentality in the example is small, broad heritability and nonadditivity are moderate, and unique environmentality plus measurement error is large (consistent with Judith Rich Harris (*The Nurture Assumption*, 1998)—peer influence is part of the unique environment, whereas parents are part of the common or shared environment.)

For a more statistical approach to this material, and a bibliography of possible additional readings, see my review of Genetic Influences on Behaviour in the 2002 Encyclopedia of Cognitive Science. This is an optional reading for students with good statistical backgrounds, and/or students who want to know the sources of some of the data I will be discussing. It is definitely not an assignment. The results in the Bouchard (2004) review article that I assigned for this week are also based on more more refined methods than the Full Twin Method. But the results using all methods are similar. In particular, Bouchard finds nonadditivity but no common environmentality for Extraversion.

Here is a table of correlations of monozygotic and dygotic twins reared apart and together for twenty-nine important psychological variables from two major studies. This table is presented to give you a feeling of what the data in behavioral genetics look like. I will tell you a little bit about the two studies and the 30 variables, after which we will move on to tables of parameter estimates derived from these correlations by the Full Twin Method. I have added Bouchard's (1990) IQ data for completeness, even though comparable DZ correlations have never, to my knowledge, been published.

Twin Correlations for 30 Variables

Trait	Study	r(mza)	r(mzt)	r(dza)	r(dzt)
Extraversion	SATSA-5	30%	54%	4%	6%
Neuroticism	SATSA-5	25%	41%	28%	24%
Impulsivity	SATSA	40%	45%	15%	9%
Monotony Avoid	SATSA	20%	26%	14%	16%
Openness	SATSA-5	43%	51%	23%	14%
Agreeableness	SATSA-5	15%	41%	-3%	23%
Conscientiousness	SATSA-5	19%	47%	10%	11%
Ability	SATSA	78%	80%	32%	22%
Type A	SATSA	23%	37%	19%	23%
F-Cohesion	SATSA	41%	60%	32%	43%
F-Control	SATSA	-3%	52%	21%	29%
BMI-Male	SATSA	70%	74%	15%	33%
BMI-Female	SATSA	66%	66%	25%	27%
Positive Emo	MISTRA-3	34%	63%	-7%	18%
Negative Emo	MISTRA-3	61%	54%	29%	41%
Constraint	MISTRA-3	57%	58%	4%	25%
Well-Being	MISTRA-11	48%	58%	18%	23%
Social Potency	MISTRA-11	56%	65%	27%	8%
Achievement	MISTRA-11	36%	51%	7%	13%
Social Closeness	MISTRA-11	29%	57%	30%	24%
Stress Reaction	MISTRA-11	61%	52%	27%	24%
Alienation	MISTRA-11	48%	55%	18%	38%
Aggression	MISTRA-11	46%	43%	6%	14%
Control	MISTRA-11	50%	41%	3%	-6%
Harm Avoidance	MISTRA-11	49%	55%	24%	17%
Traditionalism	MISTRA-11	53%	50%	39%	47%
Absorption	MISTRA-11	61%	49%	21%	41%
Rel.-Leisure Ints.	MISTRA	39%	60%	4%	30%
Rel.-Occupa. Ints.	MISTRA	59%	41%	20%	19%
IQ (WAIS-FS)	MISTRA	69%	88%		
Average		44%	54%	17%	23%

MISTRA is the Minnesota Study of Twins Reared Apart, led by Thomas Bouchard. We discussed this study earlier in the course

SATSA is the Swedish Adoption/Twin Study of aging, a comprehensive study of senior citizens in the Swedish twin registry, led by Nancy Pedersen.

The five SATSA-5 variables are sometimes called the Big Five personality dimensions. Ridley discusses them. Some modern theorists (e.g., P. T. Costa, R. R. McCrae, and L. K. Goldberg) consider them the central dimensions of personality, though this is rather controversial. You should think of the SATSA Ability measure as a kind of IQ. Type A is derived from the famous Framingham Type A scale, which measures the degree to which an individual is hard driving, ambitious, and feels as if he or she is under pressure. BMI is Body Mass Index, defined as weight divided by height squared. Overweight people have high BMI, as do very muscular people. F-Cohesion and F-Control are ratings by the twins of the warmth and strictness of the families that reared them, which, in some cases, were adoptive families.

The MISTRA-11 variables are the primary personality dimensions that were discussed earlier in the course. These variables are combined in different ways to yield the three higher-order MISTRA-3 personality variables that were also discussed earlier. Religious leisure time interests include attending religious services, doing work for a church or synagogue, or pursuing religious studies. Religious occupational interests include being a minister, priest, rabbi, missionary, or writer on religious subjects.

Basic Parameter Estimates, Unsorted

Trait	h_b^2	c_o^2	$u_o^2 + m^2$	n^2
Extraversion	30%	13%	57%	22%
Neuroticism	25%	6%	69%	-31%
Impulsivity	40%	-1%	61%	10%
Monotony Avoid	20%	4%	76%	-8%
Openness	43%	0%	58%	-3%
Agreeableness	15%	26%	59%	21%
Conscientiousness	19%	15%	67%	-1%
Ability	78%	-4%	26%	14%
Type A	23%	9%	68%	-15%
F-Cohesion	41%	15%	44%	-23%
F-Control	-3%	32%	72%	-45%
BMI-Male	70%	11%	19%	40%
BMI-Female	66%	1%	33%	16%
Positive Emo	34%	27%	39%	48%
Negative Emo	61%	3%	37%	3%
Constraint	57%	11%	32%	49%
Well-Being	***48%***	***8%***	***45%***	***12%***
Social Potency	***56%***	***-5%***	***49%***	***2%***
Achievement	***36%***	***11%***	***54%***	***22%***
Social Closeness	***29%***	***11%***	***60%***	***-31%***
Stress Reaction	***61%***	***-6%***	***45%***	***7%***
Alienation	***48%***	***14%***	***39%***	***12%***
Aggression	***46%***	***3%***	***52%***	***34%***
Control	***50%***	***-9%***	***59%***	***44%***
Harm Avoidance	***49%***	***0%***	***52%***	***1%***
Traditionalism	***53%***	***3%***	***45%***	***-25%***
Absorption	***61%***	***4%***	***35%***	***19%***
Rel.-Leisure	39%	24%	38%	31%
Rel.-Occupa.	59%	-10%	51%	19%
Average	43%	7%	49%	8%

The first thing that catches your eye is that heritabilities and unique environmentalities plus measurement errors tend to be much larger than common environmentalities. This impression is confirmed by the averages at the bottom of the table. So environment is very important, but unique environment rather than shared environment.

I have highlighted the eleven Minnesota primary personality scales to call your attention to the surprising consistency of the heritabilities, common environmentalities, and unique environmentalities acorss scales. There is, however, considerable variation in the amount of nonadditivity. Let me repeat once more my warning that heritability of Extraversion is ***not*** the proportion of your Extraversion due to genes, and common environmentality of Extraversion is ***not*** the proportion of your extraversion that is due to parents and other environmental features shared with twins and siblings. Heritability and common environmentality pertain to population variation, not individuals.

Bear in mind that underlying all our estimates are the questionable assumptions of no genotype-environment correlation or interaction, and no assortative mating. This leads to inaccuracies of unknown magnitude in our estimates, and the magnitude will be different for different psychological variables. I suggest that we simply "go about our business" with our present estimates, unless there is strong evidence of error. We will see some examples of such evidence in a few moments.

On the next page there is a table of parameter estimates sorted by h_b^2, with moderate (21%-40%) and very large (> 60%) values shaded. The shaded and unshaded groups have very large, large, medium, and small values, according to the terminology introduced in the last lecture.

Note the very large heritability for Ability (IQ) and BMI (fatness). One is tempted to discount the very large heritability of Negative Emotionality, since the closely related Neuroticism variable has a much smaller value. However, Bouchard's (2004) survey gives a medium 48% value for Neuroticism.

The 59% heritability estimate for Religious Occupational Interests seems "far out." More refined estimation techniques cut this to 41%, but confirm the finding of essentially zero common environmentality.

Note also the 48% heritability of the Minnesota Well-Being scale. This scale is a fairly direct measure of happiness! Happiness is highly heritable!

Basic Parameter Estimates Sorted by h_b^2

Moderate (21%-40%) and Very Large (> 60%) values are shaded.

Trait	h_b^2	c_o^2	$u_o^2 + m^2$	n^2
Ability	78%	-4%	26%	14%
BMI-Male	70%	11%	19%	40%
BMI-Female	66%	1%	33%	16%
Negative Emo	61%	3%	37%	3%
Stress Reaction	61%	-6%	45%	7%
Absorption	61%	4%	35%	19%
Rel.-Occupa. Ints.	59%	-10%	51%	19%
Constraint	57%	11%	32%	49%
Social Potency	56%	-5%	49%	2%
Traditionalism	53%	3%	45%	-25%
Control	50%	-9%	59%	44%
Harm Avoidance	49%	0%	52%	1%
Well-Being	48%	8%	45%	12%
Alienation	48%	14%	39%	12%
Aggression	46%	3%	52%	34%
Openness	43%	0%	58%	-3%
F-Cohesion	41%	15%	44%	-23%
Impulsivity	40%	-1%	61%	10%
Rel.-Leisure Ints.	39%	24%	38%	31%
Achievement	36%	11%	54%	22%
Positive Emo	34%	27%	39%	48%
Extraversion	30%	13%	57%	22%
Social Closeness	29%	11%	60%	-31%
Neuroticism	25%	6%	69%	-31%
Type A	23%	9%	68%	-15%
Monotony Avoid	20%	4%	76%	-8%
Conscientiousness	19%	15%	67%	-1%
Agreeableness	15%	26%	59%	21%
F-Control	-3%	32%	72%	-45%
Average	43%	7%	49%	8%

Basic Parameter Estimates Sorted by c_o^2
Moderate (21%-40%) values are shaded.

Trait	h_b^2	c_o^2	$u_o^2 + m^2$	n^2
F-Control	-3%	32%	72%	-45%
Positive Emo	34%	27%	39%	48%
Agreeableness	15%	26%	59%	21%
Rel.-Leisure	39%	24%	38%	31%
Conscientiousness	19%	15%	67%	-1%
F-Cohesion	41%	15%	44%	-23%
Alienation	48%	14%	39%	12%
Extraversion	30%	13%	57%	22%
BMI-Male	70%	11%	19%	40%
Constraint	57%	11%	32%	49%
Achievement	36%	11%	54%	22%
Social Closeness	29%	11%	60%	-31%
Type A	23%	9%	68%	-15%
Well-Being	48%	8%	45%	12%
Neuroticism	25%	6%	69%	-31%
Monotony Avoid	20%	4%	76%	-8%
Absorption	61%	4%	35%	19%
Negative Emo	61%	3%	37%	3%
Aggression	46%	3%	52%	34%
Traditionalism	53%	3%	45%	-25%
BMI-Female	66%	1%	33%	16%
Openness	43%	0%	58%	-3%
Harm Avoidance	49%	0%	52%	1%
Impulsivity	40%	-1%	61%	10%
Ability	78%	-4%	26%	14%
Social Potency	56%	-5%	49%	2%
Stress Reaction	61%	-6%	45%	7%
Control	50%	-9%	59%	44%
Rel.-Occupa.	59%	-10%	51%	19%
Average	43%	7%	49%	8%

There are only four moderately large common environmentalities. All the rest are small. It's not too surprising that perceived family strictness (F-control) is at least moderately affected by family. The wonder is that the effect is not larger. And it is also interesting that family warmth (F-cohesion) shows so little family effect. Note also the very large effect of unique environment (plus measurement error) on family strictness, and the lack of heritability. We shall see momentarily that there is reason to doubt the appropriateness of this type of analysis for family strictness.

Basic Parameter Estimates Sorted by $u_o^2 + m^2$
Moderate (21%-40%) and Very Large (> 60%) values are shaded.

Trait	h_b^2	c_o^2	$u_o^2 + m^2$	n^2
Monotony Avoid	20%	4%	76%	-8%
F-Control	-3%	32%	72%	-45%
Neuroticism	25%	6%	69%	-31%
Type A	23%	9%	68%	-15%
Conscientiousness	19%	15%	67%	-1%
Impulsivity	40%	-1%	61%	10%
Social Closeness	29%	11%	60%	-31%
Agreeableness	15%	26%	59%	21%
Control	50%	-9%	59%	44%
Openness	43%	0%	58%	-3%
Extraversion	30%	13%	57%	22%
Achievement	36%	11%	54%	22%
Aggression	46%	3%	52%	34%
Harm Avoidance	49%	0%	52%	1%
Rel.-Occupa.	59%	-10%	51%	19%
Social Potency	56%	-5%	49%	2%
Well-Being	48%	8%	45%	12%
Stress Reaction	61%	-6%	45%	7%
Traditionalism	53%	3%	45%	-25%
F-Cohesion	41%	15%	44%	-23%
Positive Emo	34%	27%	39%	48%
Alienation	48%	14%	39%	12%
Rel.-Leisure	39%	24%	38%	31%
Negative Emo	61%	3%	37%	3%
Absorption	61%	4%	35%	19%
BMI-Female	66%	1%	33%	16%
Constraint	57%	11%	32%	49%
Ability	78%	-4%	26%	14%
BMI-Male	70%	11%	19%	40%
Average	43%	7%	49%	8%

The range of $u_o^2 + m^2$ values is comparable to that of h_b^2 . All six of the very large values arose in the Swedish study, which used shorter questionnaires leading to greater measurement error. Even discounting the $u_o^2 + m^2$ values by 10% (or 20% for SATSA), we conclude that non-shared environment is an enormously important factor in most psychological traits. Perhaps the reason that non-shared environment appears to be so much more important than shared family and neighborhood environment is that two twins may react differently to the same nominally shared environment. These different reactions are then implicitly tallied with non-shared environment in our accounting.

One way to keep this straight in your mind is this: shared (or common) environment is the (not well understood) part of the environment that makes twins similar, while non-shared (or unique) environment is the (not well understood) part of the environment that makes twins different.

Basic Parameter Estimates Sorted by n^2
Moderate (21%-40%) values are shaded.

Trait	h_b^2	c_o^2	$u_o^2 + m^2$	n^2
Constraint	57%	11%	32%	49%
Positive Emo	34%	27%	39%	48%
Control	50%	-9%	59%	44%
BMI-Male	70%	11%	19%	40%
Aggression	46%	3%	52%	34%
Rel.-Leisure	39%	24%	38%	31%
Extraversion	30%	13%	57%	22%
Achievement	36%	11%	54%	22%
Agreeableness	15%	26%	59%	21%
Absorption	61%	4%	35%	19%
Rel.-Occupa.	59%	-10%	51%	19%
BMI-Female	66%	1%	33%	16%
Ability	78%	-4%	26%	14%
Well-Being	48%	8%	45%	12%
Alienation	48%	14%	39%	12%
Impulsivity	40%	-1%	61%	10%
Stress Reaction	61%	-6%	45%	7%
Negative Emo	61%	3%	37%	3%
Social Potency	56%	-5%	49%	2%
Harm Avoidance	49%	0%	52%	1%
Conscientiousness	19%	15%	67%	-1%
Openness	43%	0%	58%	-3%
Monotony Avoid	20%	4%	76%	-8%
Type A	23%	9%	68%	-15%
F-Cohesion	41%	15%	44%	-23%
Traditionalism	53%	3%	45%	-25%
Neuroticism	25%	6%	69%	-31%
Social Closeness	29%	11%	60%	-31%
F-Control	-3%	32%	72%	-45%
Average	43%	7%	49%	8%

We have seen that no traits show large common environmental influence on variability. But there are three (or perhaps four) that show large non-additive influences. Of particular interest is Positive Emotionality, which is often cited in connection with non-additivity. Extraversion, which is closely related, shows moderate non-additivity.

The five moderate and large negative values at the bottom of the n^2 column may result from violations of one or more of the assumptions underlying our work. Something is definitely wrong for F-Control, and I have already mentioned that there is a 26% spousal correlation for Traditionalism, which contradicts our assumption of no assortative mating.

Lecture 9

Comparing MZTs and DZTs—the Standard Twin Method

Twins reared apart are rare, but twins reared together are plentiful. Following Galton, most behavioral genetic studies attempt to estimate heritability and common environmentality using just data from monozygotic and dizygotic twins reared together. I call this approach the Standard Twin Method.

For most traits, r(MZT) > r(DZT). The Standard Twin Method ascribes this to the greater genetic similarity of the MZTs, assuming that trait-relevant MZT and DZT environmental similarity is the same (the "equal environments assumption").

In spite of its popularity, this approach is prone to serious estimation biases. It tends to overestimate heritability and underestimate common environmentality, as we will see. I'm not saying that it is "worthless," just that you have to take account of potential biases when interpreting results.

Table 1 from an article by Alford, et. al. in the 2005 volume of the American Political Science Review gives MZT and DZT correlations for 28 political attitudes (School Prayer, Divorce, Abortion, etc), and Heritability and Environmentality estimates derived from these correlations. The formulas for the estimates are given at the tops of the columns. I'll show you where these formulas come from after we have studied the table. Note the very large numbers of twin pairs that contribute to the correlations--50 times more twin pairs than contribute to correlations in the Bouchard, et al. (1990) study. Larger sample sizes produce estimates that should be closer, on the average, to the corresponding population correlations. The term "polychoric correlation" suggests that subjects expressed how favorable they were to each idea on a scale with a small number of values (e.g., strongly disapprove, weakly disapprove, neutral, weakly approve, strongly approve), and that a certain advanced technique was used to calculate a correlation from these ratings.

As we might expect from our experience with MISTRA personality variables, most of the heritabilities are rather similar, and most of them are moderate (between .2 and .4). However there is a fair amount of variability in the shared environmentalities. Nine are moderate and the rest are small.

Applying the same formulas to twin correlations for Conservatism vs age from a 1997 by Eaves, et al. (in the journal Behavior Genetics), we would conclude that Conservatism is not heritable during childhood (when children's political opinions tend to reflect those of their parents), but becomes highly heritable in adulthood.

Where do the estimation formulas come from? Recall that

$r(MZT) = h^2 + d^2 + i^2 + c_{mz}^2$ and $r(DZT) = \frac{1}{2} h^2 + \frac{1}{4} d^2 + c_{dz}^2$.

It follows that

$$2(r(MZT) - r(DZT)) = h^2 + (3/2)d^2 + 2i^2 + 2(c_{mz}^2 - c_{dz}^2)$$
$$= h_b^2 + n^2 + 2(c_{mz}^2 - c_{dz}^2) = h_b^2 + \text{trouble} .$$

A similar calculation shows that

$2r(DZT) - r(MZT) = c_{dz}^2 - n^2 - (c_{mz}^2 - c_{dz}^2) = c_{dz}^2$ - more trouble.

The "trouble" terms are positive or zero, the latter if and only if the following assumptions are met:

Equal Environments Assumption (EEA): $c_{mz}^2 = c_{dz}^2$ (The common value is denoted c^2.)

No Non-additive Effects (NNE): $d^2 = 0$ and $i^2 = 0$ (or, equivalently, $n^2 = 0$, where $n^2 = \frac{1}{2} d^2 + i^2$)

There is some evidence in favor of EEA for some traits from studies of misclassified twins, but, in general, it is highly suspicious.

If EEA and NNE are valid,

$2(r(MZT) - r(DZT)) = h_b^2 = h^2$.

Similarly,

$2\, r(DZT) - r(MZT) = c^2$.

Equality holds for population correlations. For sample correlations, the quantities on the left are used to estimate the quantities on the right. The first equation is sometimes called Falconer's Formula or Falconer's Estimator. I call this method of estimating h_b^2 (or h^2) and c^2 the Traditional Standard Twin Method (TSTM). EEA and NNE are still assumed in Standard Twin Method studies with more sophisticated estimation techniques.

If either EEA or NNE is wrong, "trouble" is greater than zero and TSTM overestimates h_b^2 and h^2 , and underestimates c_{dz}^2 . Because of these potenital biases, modern writers sometimes describe Falconer's Formula as a "rough approximation" to heritability. D. S. Falconer himself was certainly aware of the problem. (If assortative mating is present, it will increase r(DZT), which may reduce the overestimation errors, if they are present. However, if both EEA and NNE are correct, assortative mating will introduce estimation biases in the opposite direction.)

How can we get information about the extent of these estimation biases? I will describe two methods. Method 1 requires only the MZT and DZT correlations used to compute the estimators. Method 2 uses MZA data from the Minnesota study and the Swedish study.

Method 1. According to the second formula, 2 r(DZT) - r(MZT) should tend to be positive if EEA and NNE are both true. Hence, if this quantity is found to be significantly negative, one should conclude that one of these assumptions is incorrect, so that TSTM should not be used. If one recklessly uses it anyway, one would end up with a significantly negative estimate of c^2. Amazingly, I have seen such significantly negative estimates of c^2 in the published work of at least one leading behavioral geneticist.

In addition, the heritability estimate 2(r(MZT) - r(DZT)) should tend to be less than 1. If it isn't, that strongly suggests that TSTM should not be used.

Sex, coded as female = 1 and male = 0 provides an excellent illustration of Method 1. MZ twins have the same sex, so r(MZT) = 1, and DZ twins are uncorrelated for sex so r(DZT) = 0. TSTM thus yields a heritability estimate of 2, which is impossibly large, and a common environmentality estimate of -1, which is impossibly small. What's wrong? NNE is violated, as we discussed earlier. Sex is purely epistatic. But EEA is correct for sex: $c_{mz}^2 = c_{dz}^2 = 0$.

Because of the possibility of bias, one would not normally use the TSTM formulas if all four twin correlations were available so that the Full Twin Method or something like it could be used instead. This is because the Full Twin Method provides a simple, direct estimate of nonadditivity and even separate estimates of common environmentality for MZ and DZ twins. As a consequence of this, you should never use Method 1 as an indirect way of assessing the correctness of NNE and EEA if MZT, MZA, DZT, and DZA correlations are all available (e.g., on exams). However, we can compute the TSTM estimate of heritability in such circumstances and compare it to the unbiased Full Twin Method estimate in order to illustrate the bias in the TSTM estimate. That is precisely what Method 2 does.

Method 2. Large negative c^2s suggest that the TSTM is inappropriate for the variable in question. But there may be serious biases even if c^2 is positive, and there is, unfortunately, no way to determine whether or not such biases are present using only data from twins reared together. However, we can get illustrations of the bias in the TSTM by applying it to studies like MISTRA and SATSA where a good estimator of heritability, namely r(MZA), is available, and seeing how much smaller it is than Falconer's estimate. That is done in the following table.

Falconer's Estimate of h_b^2 and Estimation Bias, Sorted by Estimation Bias, with moderate and very large bias shaded.

Trait	r(mza)	r(mzt)	r(dzt)	Falconer's estimate of h_b^2	estimate - r(mza) (= "estimation bias")
Extraversion	30%	54%	6%	96%	66%
Social Potency	56%	65%	8%	114%	58%
Positive Emo	34%	63%	18%	90%	56%
Conscientiousness	19%	47%	11%	72%	53%
F-Control	-3%	52%	29%	46%	49%
Control	50%	41%	-6%	94%	44%
Achievement	36%	51%	13%	76%	40%
Ability	78%	80%	22%	116%	38%
Social Closeness	29%	57%	24%	66%	37%
Impulsivity	40%	45%	9%	72%	32%
Openness	43%	51%	14%	74%	31%
Harm Avoidance	49%	55%	17%	76%	27%
Well-Being	48%	58%	23%	70%	22%
Rel.-Leisure	39%	60%	30%	60%	21%
Agreeableness	15%	41%	23%	36%	21%
BMI-Male	70%	74%	33%	82%	12%
BMI-Female	66%	66%	27%	78%	12%
Aggression	46%	43%	14%	58%	12%
Neuroticism	25%	41%	24%	34%	9%
Constraint	57%	58%	25%	66%	9%
Type A	23%	37%	23%	28%	5%
Monotony Avoid	20%	26%	16%	20%	0%
Stress Reaction	61%	52%	24%	56%	-5%
F-Cohesion	41%	60%	43%	34%	-7%
Alienation	48%	55%	38%	34%	-14%
Rel.-Occupa.	59%	41%	19%	44%	-15%
Negative Emo	61%	54%	41%	26%	-35%
Absorption	61%	49%	41%	16%	-45%
Traditionalism	53%	50%	47%	6%	-47%
Average	43%	53%	23%	60%	17%

According to our usual magnitude criteria, 15 of the 29 estimates show moderate, large, or very large bias. The average bias is 17%. The three large negative biases suggest the influence of assortative mating, or some other factor not discussed.

We can sum up the status of heritability estimation as follows: r(MZA) does not have serious bias problems, but it is often unavailable because of the difficulty of procuring MZAs. In contrast, Falconer's estimate is much easier to obtain, but often seriously biased. (Please bear in mind that questions of "bias" and "availability" may have different answers for different psychological variables.)

Once again, let me warn you against reading undue pessimism into this lecture. I don't dismiss results obtained by the Traditional Standard Twin Method; I just try to mentally correct for possible biases.

Lecture 10

Digression on interaction, variation and multiple causation.

Consider the sound emitted when a hand hits the blackboard. You can say that the hand causes the sound, or you can say that the blackboard causes sound, but it is most correct to say that sound results from the hand hitting the blackboard, which is an interaction of the hand and the blackboard. Analogy: hand = genes, blackboard = environment, sound = IQ.

Now consider hitting the board harder and weaker. Sound variation is now correlated with hand force variation. So the hand, not the board, is the source of sound variation, in the same way that genetic variation is mainly responsible for IQ variation under normal socioeconomic conditions in our society.

On Summers and Baumeister

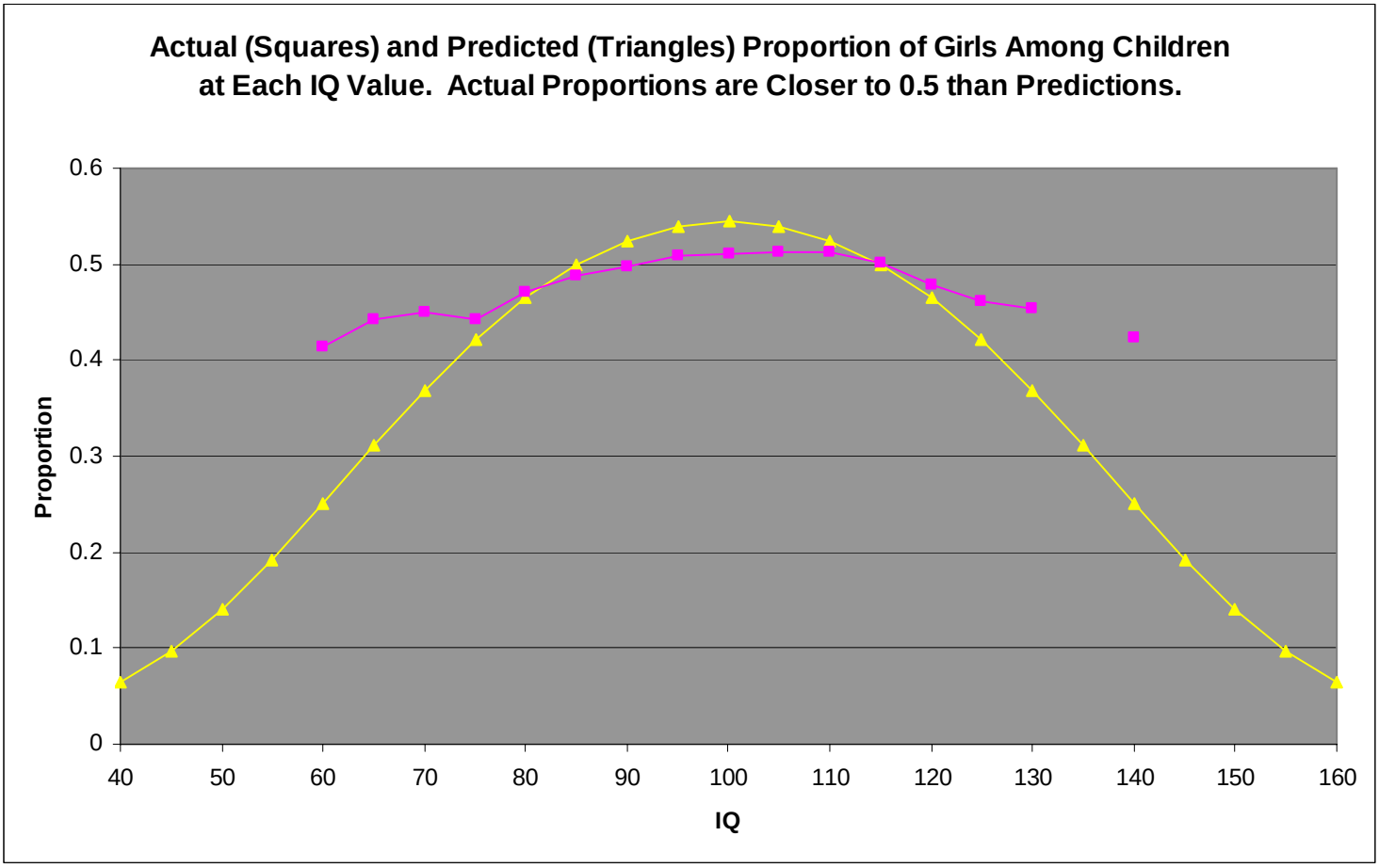
Several students noted the best objection to Summers' (Remarks at NBER Conference on Diversifying the Science & Engineering Workforce, 1/14/2005) presenting his young daughters' behavior as evidence of innate female tendencies toward maternal behavior: They surely saw sex-typical doll play at friends' (peers) homes even if their parents never gave them sex-typical playthings. However, even though this is poor evidence for innateness, it would, in my opinion, be astounding from an evolutionary viewpoint if innate female tendencies toward maternal behavior did not exist. And it seems to me obvious that family responsibilities stemming in part from these tendencies interfere with high-powered careers for many women in today's society in most countries. However, one could rearrange job requirements (and home life) to lessen this interference (as has allegedly been done in Europe), and Summers actually suggested that he would view such changes favorably. For example, one could weigh "quality of five best publications" much more heavily that "number of publications" in evaluating academic job performance. I think that such changes would be resisted by many of the same professors who demanded Summers' resignation.

In my opinion, there is an element of truth in many of Summers' and Baumeister's (Is There Anything Good About Men?, American Psychological Association Invited Address, 2007) arguments, but the magnitude and significance of these effects may be exaggerated in some cases. For example, both speakers note greater ability test score variation in men than in women (without, by the way, firmly committing to genetic or environmental origin of this variation), but, in the following example, the effect of this difference in variation is not great.

In 2003, Ian Deary and a team of Scottish researchers published an analysis of a massive survey of IQs in 11 year old Scottish school children in 1932 (*Intelligence*). 79,376 kids were included, almost all the 11 year olds in Scotland. Putting aside their age, this is surely one of the best data sets available for looking for the kinds of variability effects that Summers and Baumeister discuss. And, just as in their talks, means for boys and girls are practically identical, but the standard deviation of the distribution of girls' scores is only 83.7% of the standard deviation for boys. This means that the distributions of girls' scores and boys' scores would probably be bell-shaped curves with peaks in the same place, but the boys' bell would be a bit flatter. The article does not draw these curves, probably because they would be hard to distinguish because they are so close. Instead, the article presents a graph, Fig. 1, that emphasizes the difference in the distributions, and this graph is frequently cited.

I have constructed below another graph that focuses on the differences, and compares the differences with predictions of the theory that I think Summers had in mind. The actual proportion of girls at each IQ is represented by pink squares, and the predicted proportion is represented by yellow triangles. Both curves deviate from the flat 50% (0.5) line, and, in both cases, there is an overrepresentation of girls in the mid-range, and an underrepresentation of girls at the high and low extremes. However, the girls underrepresentation is not great—more that 40% of kids with IQ of 140 are girls, and this is a rather high IQ, the highest measured in the study. (The actual data curve is comparable to the girls' curve in Deary's Fig. 1.)

I said that the score distributions for boys and girls are probably bell-shaped curves, but there are lots of different bell-shaped curves. There is, however, a particular bell-shaped curve that is favored by IQ theoreticians. It is called the normal distribution, and that is the one that I used to derive predictions. In accordance with Summers, there is predicted to be a drastic underrepresentation of girls at astronomically high (and low) IQs. However this theory overrepresents sexual disparities for IQ 140, so we have no reason to trust its predictions for astronomical IQs. Furthermore, no one has ever shown that astronomical IQs are closely linked to scientific or any other kind of achievement. IQ tests were just not designed for the purpose of predicting spectacular achievement.



More on Sex Differences in Psychological Traits

A study by McGue, Pickens, and Svikis (*Journal of Abnormal Psychology*, 1992, discussed further in Lecture 11) finds heritability of alcoholism for men but not for women. Bouchard's (2004) article found the evidence for sex differences in heritability of liability for alcoholism "mixed," but suppose, for the moment, that McGue is correct. Would McGue's finding justify a statement that "there is a genetic basis for higher rates of alcoholism in men than in women." No, it would not. "Higher rates of alcoholism in men than in women" is a statement about mean differences between men and women. Sex differences in heritability, such as those reported by McGue, Bouchard, and others for certain traits, do not imply sex differences in means. More generally, twin studies like those considered in this course do not imply anything about mean differences between men and women.

Here is a clearer example. Consider the all-or-none trait "employed as a physical scientist (chemist, physicist, mathematician) or engineer". There are certainly large mean differences in this trait. Suppose (contrary to fact) that twin experiments had established that this trait is 100% heritable within both sexes. Would this prove that the mean difference between men and women in this trait is due to genetic differences? No! Here's that standard (Psychology 1) example that illustrates this. Suppose that one has two types of grass, S and T. At any given altitude (environment), T grows taller than S, however both grow shorter at higher altitudes, and T grown at very high altitude is shorter than S grown at sea level. Thus the statement "the mean difference between T and S is due to genes ('type')" is unjustified unless one specifies that they are grown at the same altitude. Now it is simple to understand and prescribe the growth altitude of grass, but very difficult to understand or prescribe environmental factors relevant to eventual employment as a physical scientist. For example, one doesn't go very far in this direction by supplying only typically-male toys to little girls.

Although within sex heritability results don't ***prove*** that mean sex differences are due to genes, one often hear that they ***suggest*** that mean sex differences are due to genes. This is a controversial point. Some people are more susceptible to such "suggestions" than others, based on prior data, prior intuition, or prior prejudices. The "politically correct" position is to unconditionally reject such suggestions, but such an attitude strikes some observers as dogmatic. As I have said many times, conclusions can be correct even if they are based on faulty arguments. But, as a scholar and citizen, one is obliged to try to articulate the bases for one's conclusions, so that others can make their own judgments about the validity of the conclusions. E.g., someone might say "There are no behavioral genetic data supporting a genetic contribution to mean sex differences on this trait, but I feel in my bones that there is a genetic contribution" and someone hearing this might way, "my bones are different."

Psychopathology

Please study the diagnostic criteria for Schizophrenia at

http://www.mentalhealth.com/dis1/p21-ps01.html .

A. Family studies of Schizophrenia. Here are the lifetime risks of schizophrenia for general population and various categories of relatives, averaged over several studies conducted before 1980:

Category	Lifetime risk (in percent)
General population	1.0
First-degree relatives of schizophrenics	
Parents	5.6
Siblings	10.1
Children	12.8
Second-degree relatives	3.3
Third-degree relatives	2.4

(I can't, at the moment, recall the source of this table.)

So Schizophrenia is definitely "familial," but that fact, *by itself*, does not imply that it has a genetic component, since families share environment as well as genes.

B. Models for Schizophrenia (and other conditions, such as Depression)

1. Single Major Gene Model. Only one genetic locus influences whether an individual becomes schizophrenic. There are two alleles at this locus, which I will call A_1 and A_2. Thus there are three genotypes, A_1A_1, A_1A_2, and A_2A_2.

A_1A_1 individuals won't become schizophrenic, no matter what.

A_2A_2 individuals will become schizophrenic, no matter what.

A_1A_2 individuals may or may not become schizophrenic, depending on experiences (environment, e.g. birth complications).

The probability that A_1A_2 becomes schizophrenic is called the penetrance of the "schizophrenic gene" A_2 and denoted by p. p is a parameter of the model. If $p = 1$, the schizophrenic gene is dominant, if $p = 0$, it is recessive. But p can have any value between 0 and 1 (.3 or .5, for example).

If a condition is caused by a dominant allele, and neither parent is affected, then none of their offspring will be affected. If a condition is caused by a recessive allele, and both parents are affected, then all of their offspring will be affected. In either the dominant or the recessive case, MZ twin concordance would be 100%. (Be sure that you understand why.)

Single major gene models don't work well for schizophrenia or for most other forms of psychopathology. Multi-locus models, like the liability-threshold model described next, work better.

2. Liability-threshold model. Liability, L = G + E, has uncorrelated and non-interactive genetic and environmental components. Schizophrenia is manifested if liability, L, exceeds a certain threshold, T (L > T, e.g., L > 2.2). It is assumed that many genetic loci contribute to liability, which will give it a continuous range of possible values. The multi-locus assumption makes this a polygenic model, in sharp contrast to the single major gene model. Both models can be applied to any disease, not just to Schizophrenia.

G, E, and L vary from individual to individual, but T is the same for everyone in a given population. It is assumed that L has a normal distribution with variance 1, with higher means for groups at greater risk, like relatives of affected individuals.

When the liability-threshold model is applied to twin studies, it is assumed that the threshold T is the same for both twins. The twins will have different liabilities, L and L', and these liabilities will be correlated. The threshold and correlation are population parameters that can be estimated, as we shall see.

Neither model allows for G-E interaction.

C. Twin studies of psychopathology. The standard way of describing twin similarities for "all-or-none" variables (like "sick or well") is concordance. Concordance is, roughly speaking, the likelihood that the second twin is affected, given that the first is affected. There are two popular variations of this idea, pairwise and probandwise concordance, which I will explain in a moment.

Concordances are not correlations, but we will see that concordances can be transformed into correlations of underlying liabilities, which can, in turn, be analyzed by conventional behavioral genetic methods.

Pairwise and probandwise concordance. Let c = number of concordant pairs and d = number of discordant pairs.

Then pairwise concordance = $c/(c+d) = \frac{\text{number of concordant pairs}}{\text{number of pairs with at least one twin affected}}$,

probandwise concordance = $2c/(2c+d) = \frac{\text{number of affected co-twins}}{\text{number of affected individuals ("probands")}}$,

Try calculating both for c=10 and d=14. Probandwise, which is based on individuals rather than pairs, is preferred. It is always larger than pairwise.

D. Heritability estimates from twin studies. Concordances are not correlations. However, given data on the base rate of schizophrenia and probandwise concordance, your friendly computer (running, for example, the LISREL program) can estimate the corresponding threshold and liability correlation in the model. The estimated correlation is, as usual, denoted r, but it has a special name: tetrachoric correlation coefficient. In summary: we can infer liability thresholds and correlations, even though we can't directly observe liabilities!!!!

Here is an example from a 1983 study by Kendler and Robinette.

concordance:	base rate	T	r
MZ = 60/194 = 31%	1.63%	2.14	.708
DZ = 18/277 = 6%.	1.83%	2.09	.253

Falconer's formula then yields the estimate

heritability (h^2 or h_b^2) = 2*(.708 - .253) = .91

The .91 heritability estimate seems suspiciously high, which should not surprise us, since we know that Falconer's formula has a pronounced tendency to overestimate heritability. The corresponding c^2 estimate is 2*.253 - .708 = -.202.

Kenneth Kendler (in Dunner, et al., *Relatives at Risk for Mental Disorders*, Raven Press, 1988, Table 2) gives a table of heritability of liability estimates from older studies. The Kendler and Robinette study is in the last line.

Large variability of heritability estimates. We have just seen that these heritability estimates are probably biased. That's not their only problem. They are also highly variable, as can be seen by scanning down the last column. The width of the intervals given must be DOUBLED to get what statisticians call 95% confidence intervals for population heritability—an interval around the sample value that has a 95% chance of covering the true (but unknown) population value. Part of this variability is doubtless due to real differences in study populations and diagnostic criteria, but part is also due simply to sampling variability. This sampling variablity, indexed by the ± SE column, reflects the small numbers of cases in most of these studies, and also the large intrinsic variability of tetrachoric correlations.

Sullivan, Kendler, and Neale (*Archives of General Psychiatry*, 2003) attempted to deal with the variability problem. They used a technique called Meta Analysis to combine data from all "good" studies available in 2002. Thus they included data from five studies that appeared after 1988. They also excluded the earlier Rosanoff and Inouye studies, on the grounds that the method of recruiting subjects was haphazard, leading to possible ascertainment bias. In this way, they obtained overall tetrachoric correlations of MZ = 0.92 and DZ = 0.52. Falconer's formula then yields the heritability estimate 2*0.40 = 80%. The corresponding shared environmentality estimate is 1.04 - .92 = 8%. However, unlike the 1988 paper, the recent paper uses a more sophisticated estimation method that yields the estimates heritability = 81% and common environmentality 11%. As expected, the 95% confidence inverals for these overall values are much smaller than for individual studies: 73% to 90% for heritability, and 3% to 19% for shared environmentality.

The 81% heritability estimate should be regarded as our current "best guess" concerning the heritability of Schizophrenia. And this value is VERY high, especially for a condition that was thought to be of 100% environmental origin in the early years of the last century. The 11% shared environmentality figure is small in absolute terms, but large enough that it is unlikely to have arisen by chance (peculiar properties of the samples in the various studies). So there does seem to be SOME parental contribution to the development of Schizophrenia, but the genetic contribution is much larger.

E. Adoption studies of schizophrenia. Table 3 of Kendler (1988) summarizes data on adoption studies of Schizophrenia. The "bottom line" on the adoption studies is that they support twin studies in showing that there is a genetic component of schizophrenia. But these studies do not provide a quantitative measure of this component. The pioneering Heston study is discussed in Ridley's Chapter 4. Psychiatrists were extremely surprised at the outcome.

F. Diathesis-stress vs liability-threshold. Diathesis-stress theories of schizophrenia say that genetic predispositions and environmental features (stressors and protectors) contribute to development or non-development of schizophrenia in some way. G-E interaction is allowed.

The liability-threshold model is a particular theory of this type. It says that genetic predispositions and environmental features can be represented as numerical variables, G and E, that add together to produce an overall liability (no G-E interaction), and that overt schizophrenia occurs if and only if overall liability exceeds a threshold. Symbolically: L = G+E > T.

G. Alcoholism . We have previously mentioned that McGue, Pickens, and Svikis (*Journal of Abnormal Psychology*, 1992) found heritability of alcoholism for men but not for women, but the Bouchard (2004) survey noted that some studies have found evidence of heritability for women also.

McGue's study found an especially high risk of alcoholism in male relatives of female alcoholics. According to the liability threshold model women have a lower mean liability for alcoholism than men. A woman who is alcoholic thus has an exceptionally large departure from this mean. Since there is a substantial liability correlation between relatives (due to common genes, common environment, or both) this implies a tendency of her male relatives to have large departures from the male mean, which is higher than the female mean. Thus her male relatives are at exceptional risk of exceeding threshold and being alcoholic. (This is a complex story, requiring careful study.)

If we apply Falconer's formulas to the tetrachoric liability correlations in the first three lines of Table 5, be get 0.724, 0.382, and 0.662. The first of these agrees with the corresponding heritability estimate in the table, but the second and third are much larger. From this we conclude that McGue is not using Falconer's estimation procedure. Moreover, since two of his three estimates are lower, we conclude that whatever method he is using may not suffer from Falconer's tendency to overestimate heritability.

Reviewing, the Standard Twin Method uses just twins raised together to estimate heritability and shared environmentality, assuming EEA and NNE. However, only *traditional* uses of this method make use of Falconer's formula and the companion formula for estimating shared environmentality. Some modern Standard Twin Method studies, like the political attitude study discussed earlier, are traditional in this sense, but most modern studies, especially by leaders in the field like McGue, are non-traditional. The generic terminology for advanced estimation techniques in this field is "model-building."

H. Major Affective Disorders. Table 2.2 from a 1992 study by Nurnberger and Gershon (E. S. Paykel, *Handbook of Affective Disorders, 2nd ed*,1992) shows that MZs have a substantially higher concordance for major affective disorder than DZs, suggesting genetic involvement. The Figure 9.1 in that article shows that unipolar depression may be part of a continuum of affective disorders. The following table describes Gershon's (*Archives of General Psychiatry*, 1982) multi-threshold model for this continuum. As usual, L = G + E.

Value of liability	Condition
$L \le T_1$	normal
$T_1 < L \le T_2$	unipolar depression
$T_2 < L \le T_3$	bipolar depression
$T_3 < L$	schizoaffective

This kind of multithreshold model can also be used to interpret the effects of stricter and looser diagnostic criteria for schizophrenia. Lower thresholds correspond to looser criteria.

I. Divorce. McGue and Lykken (*Psychological Science*, 1992) estimated heritability and common environmentality for liability to divorce at 52.5% and 0% (via model building).

J. Bulimia. In a study by Kendler, et al. (*American Journal of Psychiatry*, 1991), MZ concordance for narrowly defined Bulimia was 22.9% and DZ concordance was 8.7%. Estimated heritability (via model building): 55%, using an approach that assumes no common environmentality, which fits the data very well. However, an approach assuming no heritability fits the data nearly as well! Since these two models are incompatible, it appears that sample size was inadequate for modeling purposes.

K. A Note on Asperger's syndrome. According to a 2000 article by Simon Baron-Cohen that is posted at

http://www.geocities.com/CapitolHill/7138/lobby/disability.htm ,

> Autism is diagnosed on the basis of abnormalities in the areas of social development, communicative development, and imagination, together with marked repetitive or obsessional behaviour or unusual, narrow interests . Individuals with autism may have an IQ at any level. By convention, if an individual with autism has an IQ in the normal range (or above), they are said to have 'high-functioning autism' (HFA). If an individual meets all of the criteria for HFA except communicative abnormality/history of language delay, they are said to have Asperger's syndrome (AS).

So AS children have normal intelligence by definition. This should not be taken as an indication that most autistics have normal intelligence; they do not. Are AS children better than children in general at Folk Physics, as Ridley suggests? I don't really know. Within Baron-Cohen's theoretical framework, one might reason that IQ is a composite of Folk Physics and Folk Psychology abilities, and the latter are deficient in AS, so the former ought to be elevated, on the average, in order to achieve overall normality. But then the elevation would be an artifact of the definition of AS.

Note that Baron-Cohen's "male brain-female brain" viewpoint seems to predict that men will be better than women at IQ tests and school work, which, intuitively, depend more heavily on Folk Physics, and this prediction is simply incorrect.

A 2003 review by Bouchard and McGue (*Journal of Neurology*) estimates heritability of liability to autism, schizophrenia, Tourette syndrome ADHD at 80%. These are the most heritable psychopathologies, and their heritabilities are extremely large. The same review gives 50-60% for alcoholism and cannabis dependence, and "only" 40% for major depression.

L. Romantic Love The following table gives twin and spouse correlations for six dimensions of romantic love, from an article by Waller and Shaver (*Psychological Science*, 1994)

Love Style	MZ Correlation	DZ Correlation	Spousal Correlation
Eros	.16	.14	.36
Ludos	.18	.30	.08
Storge	.18	.12	.22
Pragma	.40	.32	.29
Mania	.35	.27	-.01
Agape	.30	.37	.28

Since the MZ - DZ correlation differences are trivial, there is no evidence for a genetic contribution to these dimensions. If we assume that there is no genetic contribution, then the MZ and DZ correlations are estimates of common environmentality, c^2.

In the Spousal Correlation column, note that there is no hint that "opposites attract."

M. Alzheimer's Disease. Allele 4 at the Apolipoprotein E locus on the 19th chromosome is much more prevalent in people with Alzheimer's Disease than in people who don't have the disease (40% vs 15%) . So this is a "real gene" QTL associated with Alzheimer's.

N. Smoking. A study published in 2003, (Li, et al., *Addiction*) synthesizes results of six twin studies of liability for smoking initiation (SI) and ten studies of liability for smoking persistence (SP). Studies of this kind are "meta-analyses". They are highly respected. The results are

	h^2	c^2	u^2
SI, men	0.37	.49	.17
SI, women	.55	.24	.16
SP, men	.59	.08	.37
SP, women	.46	.28	.24

Penn's Caryn Lerman is a leading researcher on the genetics of smoking. One of her studies (Health Psychology, 1999) found two specific locii that may be relevant to smoking. There is an interesting article about Lerman and her smoking research in the Pennsylvania Gazette: http://www.upenn.edu/gazette/0103/hughes.html.

("***Lecture 14***" was the mid-term exam, and "***Lecture 15***" was a discussion of graded exams.)

Lecture 16

The second half of the course is concerned with the general topic of evolutionary psychology (uncapitalized) which means, roughly, the application of evolutionary ideas in some way to various aspects of human cognition and behavior. There are at least five major approaches to this topic: Human Sociobiology, Human Behavioral Ecology, Evolutionary Psychology (capitalized), Gene-Culture Coevolution, and Memetics. I have no intention of defining and systematically differentiating all these these approaches, at least not at this time. My sole objective in listing the approaches is to suggest the diversity of ideas that fall under the general heading of evolutionary psychology. Gintis (Review of Buller ..., *Journal of Bioeconomics*, 2007) attempts to differentiate between evolutionary psychology and Evolutionary Psychology, but this distinction is often not as clear as he claims, and I consequently won't emphasize it in this introductory course.

My plan in the rest of the course is to give a brief general introduction to biological evolution, then an introduction to Evolutionary Psychology, then a rather long development of a standard simple mathematical model for natural selection, and then some very speculative models for the evolution of altruism and cooperative behavior that fall within the framework of Human Sociobiology.

Introduction to Evolution and Evolutionary Psychology

(based on Steve Pinker's *The Language Instinct*)

A. Evolution and adaptation <u>in general</u>.

1. Natural selection—generalities.

a. Charles Darwin (and Alfred Russel Wallace)

b. Natural selection, as Darwin understood it, means increasing frequency (over generations) of structures that are associated with superior fitness and decreasing frequency of structures that are associated with inferior fitness. Fitness here means reproductive success—number of offspring, or, more precisely, number of surviving offspring. It's not "fit" for birds to lay lots of eggs and then leave them in the nest to rot. One could go even further and specify that the survivors should be capable of attracting mates.

A structure that is associated with high fitness is said to be <u>adaptive</u>. There are many cases in which something that was once adaptive is no longer adaptive.

2. Other evolutionary forces.

a. mutation.

b. random genetic drift.

3. Complex, "well-engineered" structures can only arise by natural selection, and such structures are called adaptations in this context.

The following quote is due to the theologian William Paley (1802), and is reproduced on p. 360 of Pinker's book.

"In crossing a heath, suppose I pitched my foot against a stone, and were asked how the stone came to be there; I might possibly answer, that, for anything I knew to the contrary, it had lain there for ever: nor would it perhaps be very easy to show the absurdity of this answer. But suppose I had found a watch upon the ground, and it should be inquired how the watch happened to be in that place; I should hardly think of the answer which I had before given, that for anything I knew, the watch might have always been there."

Previous to Darwin, adaptive complexity in animals was almost always taken as evidence for the existence of God. The watch implies a watchmaker, and beautifully adapted eyes in animals implied a divine eyemaker. Darwin proposed natural selection as a non-theological alternative explanation of adaptive complexity. Other evolutionary forces, such as mutation and random genetic drift, have an important role in the evolutionary process, but they cannot, by themselves, account for the evolution of complex organs.

4. Special considerations in the study of adaptations.

a. Adaptations are expected to be highly specialized, since specialized machines are more efficient than general purpose machines, thus more likely to be victorious in the Darwinian struggle.

b. Understanding of function is the royal road to understanding adaptations.

The Adaptationist Program systematically considers the possible evolutionary origin of a wide variety of complex structures and behaviors, with special reference to their possible functions.

c. The function of a structure or behavior that is crucial to evolution is not its current function, but its function long ago in the EEA—Era of Evolutionary Adaptiveness.

d. Adaptations are expected to show little or no genetic variability.

We say that the adaptations are species typical, meaning that they are universal within a species, but may (or may not) vary from species to species.

B. Evolutionary Psychology

Evolutionary Psychology is one approach to the study of the evolution of complex human behavioral systems, and their supporting neural mechanisms.

1. According to Evolutionary Psychology, we should look for highly specialized, species-typical, neural subsystems underlying complex behaviors.

2. These subsystems will leave a distinctive and universal stamp on all humans. This stamp is what is popularly called "human nature" or "human universals". Smiles mean the same thing in all cultures. There is sexual jealousy in all cultures. The list of putative human universals presented in Language Instinct is very long.

3. Evolutionary Psychology challenges what it calls the Standard Social Science Model, or SSSM. This outlook has two main tenants:

a. The central feature of human biopsychology is our general learning capacity. Almost every behavior of any importance is learned.

b. These learned behaviors vary in a largely arbitrary way from culture to culture ("Cultural Relativism.") Psychological universals are few. "Human nature" is unimportant.

4. Evolutionary Psychology complements Behavioral Genetics. Behavioral Genetics is concerned with present levels of behavioral variability. Evolutionary Psychology deals, ***for the most part***, with evolution of species typical behavior.

Lecture 17

More on Evolutionary Psychology

From Darwin's autobiography:

> When I left the school I was for my age neither high nor low in it; and I believe that I was considered by all my masters and by my father as a very ordinary boy, rather below the common standard in intellect. To my deep mortification my father once said to me, "You care for nothing but shooting, dogs, and rat- catching, and you will be a disgrace to yourself and all your family." But my father, who was the kindest man I ever knew and whose memory I love with all my heart, must have been angry and somewhat unjust when he used such words.

Digression on proximal and distal (or ultimate) explanations. Suppose someone says "animals seek sex because sex seeking was once somewhat heritable, and those animals that sought sex left more descendents" and someone else says "animals seek sex because certain hormones are circulating in their blood" and someone else says "animals seek sex because they feel horny." These three spokesmen are not contradicting each other; they are offering explanations at different "levels," namely proximal in the latter two cases and distal (or ultimate) in the former case. Some magazine and newspaper articles on evolutionary psycholgy wrongly suggest that demonstration of proximal mechanisms disproves Evolutionary Psychology.

Non-adaptive adaptations. Adaptations need not be currently adaptive. Recall that adaptations are complex mental structures shaped by natural selection long ago. Thus they were adaptive (fitness enhancing) long ago. However, that does not imply that they are adaptive now. The human cravings for salty and sugary foods are examples of adaptations that are no longer adaptive. High IQ was probably once more adaptive than it is today (though Evolutionary Psychologists still might not consider it an adaptation, since they might not consider it "highly specialized.")

Evolutionary Psychologists do not expect adaptations to be currently adaptive and some of them are not especially interested in whether they are or are not currently adaptive. However, there is a discipline called Human Behavioral Ecology or Evolutionary Anthropology that is interested in the current adaptiveness of behavior (not mental structures), especially among "primitive" peoples. For example, an Evolutionary Anthropologist named Napoleon Chagnon has made a career of studying an extraordinarily ill-tempered tribe call the Yanamamo in South America. There is a very high murder rate among these people, and Chagnon has shown that killers tend to leave more descendents!

Adaptationist Reasoning must be used with caution. Recall that the Adaptationist Program systematically considers the possible evolutionary origin of a wide variety of complex structures and behaviors, with special reference to their possible functions. The Gintis (2007) review has some criticisms of this approach, and there is a powerful critique in

Gould, Stephen Jay, and Lewontin, Richard C. The spandrels of San Marco and the Panglossian paradigm: a critique of the Adaptationist Progrmme. *Proc. R. Soc. London*, 1978, 205, 581-598.

Spandrals are the triangular spaces between intersecting arches in cathedrals like Saint Mark's in Venice. Beautiful though they are, they are by-products of intersecting arches, not independent architectural elements. They have no "function." Gould and Lewontin think that Evolutionary Psychologists often ascribe fictitious functions to evolutionary by-products. For example, Gould and Gintis claim that female orgasm is a by-product of evolution of male orgasm, just as male nipples are a by-products of evolution of female nipples.

Often speculation about the evolutionary origin of a structure or behavior degenerates into unbridled fantasy. Critics liken such speculation to the ridiculous explanations of natural phenomena given in Rudyard Kipling's humorous *Just So Stories*.

The title of Gould's article refers to Dr. Pangloss in Voltaire's *Candide*, who taught that "Whatever is, is right." That phrase embodies the Naturalistic Fallacy. Some Evolutionary Psychologists claim that jealous violence against wives is a product of evolution, but they don't claim that it is "right", and they agree that it should be illegal. Much criticism of Evolutionary Psychology wrongly accuses it of endorsing the Naturalistic Fallacy.

Some Special Topics in (or Examples of) Evolutionary Psychology

Evolutionary Psychology of Sexual Jealousy. Both men and women are jealous of their mates. This is true worldwide. David Buss showed (e.g., Behavioral and Brain Sciences, 1989), however, that the nature of this jealousy tends to be different for men and women. Men are, on the average, more concerned about their wives' sexual infidelity, whereas women are, on the average, more concerned about their husbands' emotional infidelity. (The average sex difference is, in fact, rather small, and there is considerable individual variability, as we shall see later.)

A (distal) Evolutionary Psychology explanation of this difference goes as follows. It was evolutionarily adaptive in the EEA for men to be concerned about sexual infidelity. Otherwise they might have ended up wasting their resources on someone else's children. It was evolutionarily adaptive for women in the EEA to be concerned about emotional infidelity, since it could have led to diversion of resources from them and their children to the other woman. Such diversion is very bad news even today, but in the EEA, it would have been catastrophic. Male and female jealousy evolved as a defense against both kinds of infidelity.

Here is an alternative, non-evolutionary, proximal explanation: Men are more concerned about sexual infidelity because they assume that, for women, sex implies love. So wives' sexual infidelity implies both sexual and emotional infidelity. On the other hand, women are more concerned about emotional infidelity because they assume that, for men, love implies sex. So husbands' emotional infidelity implies both emotional and sexual infidelity. These feelings could intermingle with deeper jealousies arising from our evolutionary heritage.

Adoption. But, if men where shaped by evolution to focus their resources on their offspring, how come there are so many men who willingly, even eagerly, seek to adopt children? (See

http://www.cdc.gov/nchs/data/series/sr_23/sr23_027.pdf) This is hard to square with Evolutionary Psychology. (Widespread homosexuality is also hard to square with Evolutionary Psychology.)

Sex Difference in Spatial Abilities. Men are better than women, on the average, at complex spatial relations problems—mental rotation and mental box assembly problems, for example—so one frequently hears that "men have higher spatial ability than women." Two Evolutionary Psychologists, Irwin Silverman and Marion Eals (in Barkow, et al., *The Adapted Mind*, 1992), wondered whether standard spatial test problems might have been more relevant to men's activities (e.g., hunting) than to women's (e.g., gathering) in the EEA, and whether modern women might excel at problems more relevent to women's EEA activities. And, indeed, modern women turn out to be better than men at remembering the locations of lots of spatially placed objects, analogous to remembering the locations of lots of objects to be gathered in the EEA.

This illustrates four very important points: (1) Contrary to many magazine and newspaper articles, Evolutionary Psychology is not a male conspiracy to "put women down." (2) Speculation about the EEA is a powerful tool for generating new research ideas for testing in the current era. (3) The results (e.g., women excel at certain spatial tasks) are of interest even if one strips away the Evolutionary Psychology rationale that motivated their discovery. (4) One does not have to confirm the speculative side of Evolutionary Psychology for Evolutionary Psychology to be scientifically useful. For example, it doesn't matter very much that the definition of the EEA is imprecise.

. Sperm competition and ancestral promiscuity. (This was discussed in Ridley's Chapter 1.)

Gorilla	Orangutan	Human	Chimp
.02	.05	.08	.27
1	2.5	4	13.5

The values in the top line of the table represent testical weight, as a percentage of body weight. The second line gives comparative values, relative to Gorillas. Why do Gorillas have such small testicles, relatively speaking, and why do Chimps have such large ones?

Some Evolutionary Psychologists relate testicle size to sperm competition. They note that testical weight correlates with female promiscuity. Gorillas typically live in small groups with one male and several females, so the females typically mate with a single male. Chimpanzee females, however, are extremely promiscuous, often mating with many males in rapid succession when they are in estrus. Such promiscuity can lead to situations where the female is carrying sperm from multiple males, and, in such situations, males who put out more sperm are more likely to achieve fertilization. Thus evolution would favor larger testicles in species with more promiscuous females.

This line of argument suggest that ancestral human females were somewhat promiscuous, but not spectacularly so.

Lecture 18

Additional Examples of Evolutionary Psychology

1. Age preferences in mates. Men and women have different age preferences in mates. On the average and with many exceptions, men prefer younger women, and women prefer older men. According to Ev Psych, men prefer younger women because, during the EEA, such women enjoyed more years of fertility, thus leading to more sons who inherited their fathers' preferences for younger women. According to Ev Psych, women prefer older men because, during the EEA, older men tended to have more resources, thus permitting support of more daughters who inherited their mothers' preferences for older men. Note, that both of these are distal explanations, referring to the EEA. However, for women's preferences, there is a closely related proximal explanation. For example, a young secretary in a law firm might decide that she would like to live a life of ease (with or without children) and, with this in mind, she might attempt to attract the attention of a rich senior partner. But the Ev Psych approach appears to be more powerful because it correctly predicts the even wealthy women tend to prefer older mates, despite the fact that these men are likely to die well before they do. However, we will see shortly that there is substantial overlap between men's and women's age preferences in mates..

2. Morning Sickness in the early months of pregnancy may be an adaptation to protect the foetus from toxins during its period of greatest vulnerability. (See Margie Profet's article in Barkow, et al., *The Adapted Mind*.) For example, it has been found that women with more nausea during pregnancy are less likely to have miscarriages. This is another example of a discovery that was motivated by evolutionary ideas, but which can be appreciated apart from these ideas.

3. Infant resemblance to mothers and fathers (see abstract on the next page). There is a study that shows that mothers and their relatives tend to claim that infants resemble their husbands. This looks like an adaptation for reassuring husbands of their paternity. Husbands are equally likely to attribute resemblance to themselves and to the mother.

Objectively, infants tend to resemble their mothers more than their fathers, according to unrelated observers. This looks like an adaptation for concealing non-paternity from the husband, who might mistreat the child if he suspected it was not his. Males in many species routinely kill offspring of males that they displace. Also, sad to say, stepchildren are much more likely to be mistreated than are non-stepchildren.

Mistaken paternity is not at all rare: "Averaging across a handful of studies of paternal discrepancy from Europe, Africa, North America, and Oceania, Baker and Bellis estimate that roughly 9 percent of children have genetic fathers who are different from those who believe that they are the father." (Buss, *The Dangerous Passion*, p.171) In 1999, 28.2% of the tests performed by paternity testing services had negative results. (See http://www.childsupportanalysis.co.uk/papers/knowledge/evidence.htm, which has a link to more data)

4. Female beauty and male attractiveness. According to Evolutionary Psychology, female beauty and male attractiveness are not "in the eye of the beholder." Instead, they are universally understood signals of reproductively important conditions. Female beauty is a signal of youth and good health. Facial and body symmetry, an indication of pathogen resistance, is a component of both male and female attractiveness. The idea is that basic developmental programs produce symmetrical body features unless they are interfered with by pathogens, which leave subtle ("fluctuating") asymmetries. One amazing study showed that women preferred t-shirts of more symmetrical men at the most fertile point of their monthly cycle, but not at other times. Another showed preference for men with more masculine faces at the most fertile point in the cycle. (See abstracts on the next page.)

Sex vs. Reproduction

Until recently, sexual behavior and reproductive behavior were almost synonymous, but that is no longer the case. Evolutionary Psychology is concerned with adaptations that evolved because they promoted sex leading to increased reproduction. Many of these adaptations still promote sex, but this no longer has much to do with reproduction. A woman can be spectacularly sexy and have a rich and varied sex life, yet choose not to have any children. So, if you are interested, as I am, in factors influencing family size in the modern world, Evolutionary Psychology doesn't have much to offer. In fact, birth rates are either rapidly declining or very low everywhere in the world except USA and sub-Saharan Africa, and this is, in my opinion, the most important bio-socio-economic phenomenon of our era. There's a bit about this phenomenon in the 2008 New York Times Magazine article

http://www.nytimes.com/2008/06/29/magazine/29Birth-t.html?ref=magazine.

Some Evolutionary Psychology Abstracts

D. Kelly McLain, Deanna Setters, Michael P. Moulton, and Ann E. Pratt. Ascription of resemblance of newborns by parents and nonrelatives, *Evolution and Human Behavior* 21(2000) 11-23.

Abstract: It has been hypothesized that human females ascribe the resemblance of their infants to the father or his relatives to promote assurance of paternity. Assurance renders fathers more likely to invest in and less likely to harm mothers and their children. Other studies have documented a bias toward the father's side of the family when mothers ascribe the resemblance of their infants. We test if this bias extends to newborns (1-3 days old) and if such bias reflects actual resemblances. This was done by noting the responses of mothers when asked whom they thought their newborns resembled and by comparing those responses to resemblances ascribed by unrelated judges. During in-hospital visits, mothers were significantly more likely to ascribe resemblance to the domestic father than to themselves. This bias was exaggerated in the presence versus absence of domestic fathers. Yet, judges matched photographs of these mothers to their newborns significantly more frequently than they matched domestic fathers to newborns. Thus, the bias in how mothers remark resemblance does not reflect actual resemblance and may be an evolved or conditioned response to assure domestic fathers of their paternity. The low rate with which newborns are matched to fathers may be biologically significant. Concealment of paternity may be favored when suspicion of cuckoldry leads fathers to abandon or harm newborns. Genomic imprinting is one mechanism by which resemblance of newborns could be biased toward mothers. We suggest that on average females benefit from concealed paternity even if cuckoldry is rare provided that their verbal ascriptions of resemblance are effective in assuring domestic fathers of their paternity.

I.S. Penton-Voak and D.I. Perrett. Female preference for male faces changes cyclically: Further evidence. *Evolution and Human Behavior* 21(2000) 39-48

Abstract: Research has failed to reach consensus on the characteristics of attractive male faces. Different studies have reported preferences for phenotypically average faces, and faces with both exaggerated and reduced sexual dimorphism. Recent studies demonstrate cyclic changes in female sexual behavior and preferences for odors and facial characteristics that may reflect conditional mating strategies. We employed computer graphic techniques to manipulate the "masculinity" or "femininity" of a composite male face by exaggerating or reducing the shape differences between female and male average faces. Five stimuli with varying levels of masculinity and femininity were presented in a national U.K. magazine, with a questionnaire. Female respondents in the follicular phase of their menstrual cycle (n = 55) were significantly more likely to choose a masculine face than those in menses and luteal phases (n = 84). This study provides further evidence that when conception is most likely, females prefer testosterone related facial characteristics that may honestly advertise immunocompetence.

Steven W. Gangestad and Randy Thornhill. Menstrual cycle variation in women's preferences for the scent of symmetrical men. *Proceedings of the Royal Society of London*, B, 265(1998), 927-933.

Abstract: Evidence suggests that female sexual preferences change across the menstrual cycle. Women's extra-pair copulations tend to occur in their most fertile period, whereas their intra-pair copulations tend to be more evenly spread out across the cycle. This pattern is consistent with women preferentially seeking men who evidence phenotypic markers of genetic benefits just before and during ovulation. This study examined whether women's olfactory preferences for men's scent would tend to favor the scent of more symmetrical men, most notably during the women's fertile period. College women sniffed and rated the attractiveness of the scent of 41 T-shirts worn over a period of two nights by different men. Results indicated, that normally cycling (non-pill using) women near the peak fertility of their cycle tended to prefer the scent of shirts worn by symmetrical men. Normally ovulating women at low fertility within their cycle, and women using a contraceptive pill, showed no significant preference for either symmetrical or asymmetrical men's scent. A separate analysis revealed that, within the set of normally cycling women, individual women's preference for symmetry correlated with their probability of conception, given the actuarial value associated with the day of the cycle they reported at the time they smelled the shirts. Potential sexual selection processes and proximate mechanisms accounting for these findings are discussed.

Buss' International Mate Preference Study

In the remainder of this lecture I would like to have a closer look at Buss' (*Behavioral and Brain Sciences*, 1989) celebrated mate preference study, which showed similar sex differences in average mate preferences over many countries. The point that I want to make is that these averages conceal huge numbers of individual reversals of the average pattern, so it cannot be claimed that we are talking about truly species general male-female preference differences. For example, lots of men care at least as much about mates' financial prospects than many women do.

Here's how the study worked. Men and women in many countries were asked to rate how strongly they valued various characteristics in mates, such as good financial prospects. Ratings were 0, 1, 2, or 3, with 0 meaning unimportant and 3 meaning indispensable. Table 2 in the article gives means and standard deviations of rankings for Good Financial Prospects for men and women in 37 countries or ethnic groups within countries. In every single case, women's mean ranking was higher than men's average ranking, and the difference was statistically significant (i.e., probably not due to peculiarities of their sample) in every country except Spain.

However, there was still considerable overlap between men and women. To see this, consider the data for China. In this country, men's mean and SD are 1.10 and 0.98 and women's are 1.56 and 0.94. Assuming (as a very, very, very rough approximation) that rankings have normal distributions, and plugging these values into the Two Normals spreadsheet linked to the syllabus (or the formula given below), you see that the distributions of rankings for the men (lower) and women (upper) overlap considerably, and that if you choose a man and woman at random, the probability is 37% that the man will care at least as much about mate's financial prospects as the woman. [If X and Y are independent and normal, with means a and b and variances c and d, then the probability that $X > Y$ is the same as the probability that $X - Y > 0$, or that the corresponding (normally distributed) z score exceeds $(b - a)/\sqrt{(c + d)}$.]

This degree of overlap is fairly typical for Buss' tables. As another example, consider ratings for Good Looks in Bulgaria. Men's mean and SD are 2.39 and 0.68, women's are 1.95 and 0.84. The probability of a preference reversal (woman cares at least as much as man) for a randomly chosen pair is 34%. Or consider Chastity in mainland USA. Men's mean and SD are 0.85 and 0.96, women are 0.52 and 0.85. The probability of a preference reversal (woman cares at least as much as man) for a randomly chosen pair is 40%.

So there isn't that much differences between men and women on most dimensions in most countries. Thus Buss' results don't demonstrate "species typicality" of sex differences in mating preferences. It's just not true that almost all men care more about mate's chastity than almost all women. Instead Buss' results demonstrate consistency of average sex differences over countries, which is something else entirely. Buss' results are interesting, but they don't relate in any obvious way to the principles of Evolutionary Psychology that I described in previous lectures.

(Statistical note: The male and female ranking distributions show considerable overlap, but the distributions of mean rankings from populations of this kind do not, since their SDs are much smaller that the SDs of the rankings themselves. That's how differences between Buss' means come to be statistically significant. The SD of a mean of n observations is smaller that the SD of the underlying observations by a factor of square root of n.)

Lecture 19

New topic: Mathematical Models for Evolution and Evolution of Behavior (An overview follows.)

A. General Micro-Evolutionary Theory

The fitness of a genotype (denoted w_{11} , w_{12} , or w_{22}) is the expected number of offspring of an individual of that genotype that survive to reproductive age. Equivalently, it is the expected number of genes contributed by an individual of that genotype to the next generation, since a child has only one gene from each parent.

Fitness examples:

Genotype	A_1A_1	A_1A_2	A_2A_2	
Example 1	+2.2	2.1	2.0	(no dominance for fitness)
Example 2	+2.2	2.2	2.0	(A_1 completely dominant)

Note that fitness is a trait, just like IQ and Extraversion, but only fitness directly affects evolution. Other traits affect evolution indirectly to the extent that they are correlated with fitness.

Natural selection leads to changes in the proportion of A_1 genes from one generation to the next. Let

p_n = "A_1 gene frequency" = proportion of A_1 genes in the n^{th} generation.

I will derive the following equation describing how p_n changes from generation to generation:

$$p_{n+1} = p_n (w_{11} p_n + w_{12} q_n)/(w_{11} p_n^2 + 2 w_{12} p_n q_n + w_{22} q_n^2) ,$$

where the w's are the fitnesses of the three genotypes, and q_n is $1-p_n$. This equation describes deterministic changes in A_1 gene frequency in a large population with random mating.

B. Sociobiological Models

This general theory is then applied to Human Sociobiology, a branch of evolutionary psychology that predates Evolutionary Psychology.

1. Evolution of altruism toward related individuals via kin selection. A central problem in Human Sociobiology is the evolution of altruistic behavior—behavior that benefits others at the expense of self. Examples are birds who risk their lives to defend their nests and animals who expose themselves to predation by giving alarm calls to alert others. According to kin selection theory, "altuistic genes" have a selective advantage if the altruistic behavior benefits relatives who share these genes. This mechanism would promote altruistic behavior toward relatives and towards individuals who cannot be easily distinguished from relatives.

2. The game theoretic approach to the evolution of competitive strategies. Game theory is a modern mathematical theory that describes the effects of using different strategies in all kinds of competitions, from informal parlor games to deadly serious economic struggles between firms. The payoffs of such competitions are usually distributions of money to various players. A theoretical biologist named John Maynard Smith has linked game theory to evolutionary theory by thinking of competitions between animals and related competitions between humans as analogs of human games. Different genotypes employ different competitive strategies, like aggressive or defensive behavior. The payoffs are increments or decrements in reproductive success instead of money. The winner achieves greater reproductive success than the losers, so competitive strategies are subject to selective pressure.

One of the central ideas of Maynard Smith's theory is the notion of an Evolutionarily Stable Strategy, which is a special case of the more general concept of a Nash Equilibrium. The Nash Equilibrium describes optimal strategies in complicated multiperson games that are often used as models for economic rivalries.

3. Group selection. One version of group selection can be illustrated by an amusing experiment involving chickens. Selection for egg production "by the coop" ultimately produces more eggs that selection "by the hen." The latter approach produces antisocial chickens that are too aggressive to be conveniently housed together. In this example of artificial selection, group selection for egg production indirectly promotes sociality. Moving to natural selection, it is not inconceivable that groups containing more cooperative individuals will in certain circumstances prosper economically and/or militarily, leading them to subjugate less cooperative groups and make greater genetic contributions to subsequent generations. In so far as cooperation is genetically based, this scenario should produce enhanced cooperation over time.

In another type of group selection, some individuals possess an altruistic trait that promotes the welfare of an entire community, even though it hinders the reproduction of the individual who possesses this trait. Think of celibate spiritual leaders who promote community-beneficial cooperative attitudes within a society. Under certain conditions, individual disadvantage is overcome by community advantage, and altruism of this type can be maintained by evolution.

A. General Micro-Evolutionary Theory

1. The Hardy-Weinberg Law

My version of the Hardy-Weinberg Law describes the relation between gene and genotype frequencies in newly conceived individuals, which are called zygotes. Let

p_i = Proportion of A_i genes in zygotes in some generation, and

$P(A_iA_j)$ = Proportion of A_iA_j genotypes among these zygotes.

Hardy-Weinberg Law. If mating is random, zygotic gene and genotype frequencies are related by

$$P(A_iA_i) = p_i^2 \text{ and } P(A_iA_j) = 2\, p_i\, p_j\ .$$

This is valid for any number of alleles, even if mutation and selection are present, provided that selection is of the "viability-fertility" type described in the next section.

The Hardy-Weinberg Law relates to genetic rearrangements at the moment of conception. It says that random mating is mathematically equivalent to "random union of gametes." (A "gamete" is a sex cell—an egg or a sperm.) It's as if all sperm and eggs are floating around outside the body, and unite at random. This is a physically accurate picture of mating in certain fish. But the Hardy-Weinberg Law says that it is a mathematically accurate picture for other species as well, if mating is random.

2. Derivation of Haldane's Selection Equation

We will be studying changes in gene frequency due to selection, mutation, migration, and random genetic drift. The technical term for this kind of study is microevolution.

Until further notice, we will make the following assumptions:

1. Large population.
2. Discrete, nonoverlapping generations.
3. Random mating among surviving adults.

4. Selection is controlled by one locus with two alleles.

The (Darwinian) fitness of a genotype is the expected number of genes contributed by an individual of that genotype to the next generation:

w_{ij} = fitness of A_iA_j for i j = 11, 12, 22 .

We shall assume that fitness has two components, called viability and fertility, and denoted s_{ij} and t_{ij}. s_{ij} is the probability that an A_iA_j individual survives to reproductive age. There is assumed to be random mating among such survivors, and the expected number of offspring of a randomly mated couple is assumed to be the product of their fertilities. It can be shown that this scheme leads to the following simple formula for fitness:

w_{ij} = k × (viability factor) × (fertility factor)

= k s_{ij} t_{ij}

where k is a numeric factor that does not depend on genotype, and which, for present purposes, can be set to 1.

(It makes no difference to evolution of gene and genotype frequencies, but k depends on gene frequency and plays an important role in the evolution of population mean fitness. For more on this topic, see the Introduction and Interactive Grapher at

http://psych.upenn.edu/~norman/Additive and Multiplicative Fertility Models.htm

and

http://psych.upenn.edu/~norman/Additive and Multiplicative Fertility Models.xls)

Aside: Natural and artificial selection are environmental processes that affect the genome over generations. Ridley's fox domestication example illustrates this, and Ridley says that this is an example of nurture affecting nature. But this has nothing to do with the standard "nature-nurture problem," which relates to the degree of environmental influence on gene expression and other processes during individual development.

We shall now see how fitness differences cause gene frequency changes from generation to generation. Let

p = proportion of A_1 genes in zygotes of the present generation (population size = N),

p′ = same for next generation.

The following display shows how to calculate p′ from p and w_{ij}. (q = 1-p)

	A_1A_1	A_1A_2	A_2A_2
zygote proportions (from H-W)	p^2	2pq	q^2
number of individuals	$N\,p^2$	$N\,2pq$	$N\,q^2$
total no. of genes contributed to zygotes of the next generation	$w_{11}\,N\,p^2$	$w_{12}\,N\,2pq$	$w_{22}\,N\,q^2$
same for A_1 genes	$w_{11}\,N\,p^2$	$w_{12}\,N\,pq$	0

It follows that

total A_1 genes in next generation = $w_{11}\,N\,p^2 + w_{12}\,N\,pq$ and

total genes in next generation = $w_{11}\,N\,p^2 + w_{12}\,N\,2pq + w_{22}\,N\,q^2$.

Therefore

$$p' = (w_{11}\,N\,p^2 + w_{12}\,N\,pq)/(w_{11}\,N\,p^2 + w_{12}\,N\,2pq + w_{22}\,N\,q^2),$$

or, canceling N and factoring out p,

$$p' = p\,(w_{11}\,p + w_{12}\,q)/(w_{11}\,p^2 + 2\,w_{12}\,pq + w_{22}\,q^2)\ .$$

I will call this important relation Haldane's Equation, in honor of J. B. S. Haldane, who discovered it in the 1920s.

If we use n and n+1 subscripts to denote one generation and the next (e.g., n = 53, n+1 = 54), Haldane's Equation can be rewritten in the equivalent form

$$p_{n+1} = p_n\,(w_{11}\,p_n + w_{12}\,q_n)/(w_{11}\,p_n^2 + 2\,w_{12}\,p_n\,q_n + w_{22}\,q_n^2)\ .$$

We could use this equation for step by step calculation of $p_1, p_2, p_3, \ldots$, but this can be done more conveniently using the alternative form of the equation derived below.

3. Alternative Forms of Haldane's Equation.

It is very instructive to do some elementary algebra on the right hand side of Haldane's equation

$$p' = p(w_{11}\,p + w_{12}\,q)/(w_{11}\,p^2 + 2\,w_{12}\,pq + w_{22}\,q^2)\ .$$

Let $W_1 = w_{11}\,p + w_{12}\,q$ = "fitness of A_1", $W_2 = w_{12}\,p + w_{22}\,q$ = "fitness of A_2" ,

and $W = w_{11} p^2 + 2 w_{12} pq + w_{22} q^2$ = mean genotype fitness = "population mean fitness" .

Then $W = W_1 p + W_2 q$ (mean genotype fitness = mean gene fitness) ,

and Haldane's Equation can be expressed in the extremely compact and easy to remember form

$p' = p W_1/W$.

It follows from this that $p' > p$

iff $W_1 > W$

iff $W_1 > W_1 p + W_2 q$

iff $W_1 - W_1 p > W_2 q$

iff $W_1 q > W_2 q$

iff $W_1 > W_2$.

The same argument applies if > is replaced by = or < . Thus

$p' > p$ if $W_1 > W_2$

$p' = p$ if $W_1 = W_2$

$p' < p$ (or $q' > q$) if $W_1 < W_2$.

4. Example of step-by-step calculation of p_1, p_2, etc.

Suppose that

p_0 = "initial value of p" = .1, and that

$w_{22} = 1$, $w_{12} = 2$, and $w_{11} = 3$.

Note that more "doses" of A_1 produce greater fitness, so A_1 is clearly the "better gene." Hence we expect A_1 gene frequency to increase from generation to generation.

We can use Haldane's Equation $p' = p W_1/W$ to calculate $p' = p_1$:

$W_1 = 3 \times .1 + 2 \times .9 = .3 + 1.8 = 2.1$

$W_2 = 2 \times .1 + 1 \times .9 = .2 + .9 = 1.1$

$W = 2.1 \times .1 + 1.1 \times .9 = 1.2$

$p' = p_1 = .1 \times 2.1/1.2 = .21/1.2 = .175$

Exercise (Do it, but don't hand it in.): Plug this value of p_1 into the right hand side of Haldane's Equation and show that $p_2 = .2819$. p_3, p_4, and so on can be calculated in the same way.

Note that $p_0 < p_1 < p_2$, as expected. This gives us a very rough indication that we have not made an egregious calculational error.

Last time we showed that

$p' > p$ if $W_1 > W_2$

$p' = p$ if $W_1 = W_2$

$p' < p$ (or $q' > q$) if $W_1 < W_2$.

So, in any case, the frequency of the fitter gene increases. We shall see shortly that the frequency of the fitter genotype does not always increase. It is as if selection were acting directly on genes rather than genotypes. The notion that genes rather than genotypes are the fundamental units of evolution was popularized in a famous 1976 book by Richard Dawkins called The Selfish Gene. The Selfish Gene perspective is one of the basic contributions of Sociobiology. We will consider its implications later. One problem with this notion is that the gene fitnesses W_1 and W_2 depend on p (they are "frequency dependent"), so the "fitter gene" may be different for different values of p. We will see the effect of this dependence shortly.

Furthermore, it can be shown that both the Hardy-Weinberg law and Haldane's equation depend critically on our assumption that a couple's expected number of offspring is a product of maternal and paternal fertility parameters. If it is, for example, a sum instead of a product, then there is no simple way to describe evolution at the "genic" level, so the "Selfish Gene perspective" is only an approximation, though an interesting and useful one.

Beware of confusing terminology. We have been implicitly assuming that genotype fitnesses are not frequency dependent. However, all our formulas are valid whether or not genotype fitnesses are frequency dependent. The term frequency dependent selection applies to cases where genotype fitnesses are frequency dependent. For example, some genotypes may be fitter when they are rare than when they are prevalent, e.g. they may compete for a limited resource. We will consider models involving frequency dependent selection later. (Once again: gene fitnesses are always frequency dependent, whether or not the model involves frequency dependent selection.)

5. The course of evolution, according to Haldane's Equation.

The precise value of A_1 gene frequency in the 77^{th} generation depends on the precise values of initial A_1 gene frequency and all three fitness values (or, at least, their ratios, as we will see). However, for many purposes, it suffices to know the general shape of the graph of p_n as a function of n, and the limiting value of p_n as n approaches infinity. It turns out that there are only a few such shapes, and they are not dependent on the precise fitness values. The objective of this lecture is to show you all such shapes, and show you the range of fitness values that produces each of them.

We want to know for which values of p $W_1 > W_2$, or, equivalently, $W_1 - W_2 > 0$, since p increases in such cases. Subtracting our formulas for W_1 and W_2, we get the useful expression

(gene) fitness difference = $W_1 - W_2 = (w_{11}-w_{12})p + (w_{12}-w_{22})q$.

The simplest case is the one where all genotype fitnesses are equal. It then follows from the formula that gene fitnesses are equal for all p. Consequently p never changes: $p_0 = p_1 = p_2 \ldots$ That's just what you would expect when there are no fitness differences.

There are several other cases to consider, depending on the value of w_{12}. We next consider cases where w_{11} and w_{22} are different, and w_{12} is between them. If $w_{11} > w_{22}$ and $w_{11} \geq w_{12} \geq w_{22}$, A_1 is always the "better gene," as we shall see. If the inequalities are reversed, A_2 is the better gene. In either case, we will see that the better gene completely displaces the other gene after a large number of generations. We say that the better gene "takes over."

a. Partial dominance or no dominance ($w_{11} > w_{12} > w_{22}$, or vice versa). Consider first the case $w_{11} > w_{12} > w_{22}$. Then the coefficients of both p and q are positive in the fitness difference. Thus fitness difference is positive, for any value of p, since it is an average of two positive quantities. Since fitness difference is positive for p_0, p_1 is bigger. Since fitness difference is positive for p_1, p_2 is bigger, etc. So each successive value of A_1 gene frequency is bigger than the previous one, $p_0 < p_1 < p_2$ …., and it can be shown that p_n converges to 1, its maximum value, as n approaches infinity. A_1 takes over, regardless of its initial frequency. If $w_{22} > w_{12} > w_{11}$, the same kind of argument shows that A_2 takes over.

Similar conclusions follow by similar arguments if one of the alleles is dominant. The only difference is in the speed of evolution, as we will see.

You should illustrate all cases for yourself by plugging corresponding fitnesses into the Evolution Grapher at http://psych.upenn.edu/~norman/Haldane3.xls.

b. A_1 is a beneficial dominant (equivalently, A_2 is a deleterious recessive; $w_{11}=w_{12} > w_{22}$). In this case, $W_1 - W_2 = (w_{12}-w_{22})q$ is always positive, as in the first case, but it is small when q is small, that is, when p is large. Thus evolution slows down when p gets large. Intuitively, this is because only the A_2 homozygote is selected against, and there aren't many A_2 homozygotes when A_1 gene frequency is large. This means that recessive genes are unlikely to be completely eliminated by natural selection, even if they cause terrible diseases.

c. A_1 is a beneficial recessive (equivalently, A_2 is a deleterious dominant; $w_{11} > w_{12}=w_{22}$). In this case, a similar argument shows that A_1 gene frequency increases very slowly when it is small. Intuitively, this is because only the A_1 homozygote has a selective advantage, and such homozygotes are very rare when A_1 gene frequency is small. This means that recessive mutants have a hard time "gaining a foothold" in a population, even if they are beneficial.

Similar conclusions apply if the beneficial dominant or beneficial recessive is A_2 instead of A_1.

We have now covered all possibilities where w_{11} and w_{22} are different,, and w_{12} is between them. In all such cases, the better gene takes over. The final two cases are very different.

d. Overdominance ($w_{12} > w_{11}$ and $w_{12} > w_{22}$). Sickle cell anemia is a famous case of this sort. Here it is useful to rewrite our fitness difference formula as $W_1 - W_2 = k(p^* - p)$ where $k = (w_{12}-w_{11}) + (w_{12}-w_{22})$ and $p^* = (w_{12}-w_{22})/k$.

Note that k is positive. Consequently the fitness difference is positive when p is less than p*, and negative when it is greater. It follows that, if p_0 is less than p*, then p_n increases from generation to generation, but if p_0 is greater than p*, then p_n decreases from generation to generation. In either case, A_1 gene frequency converges to p* as a limit. p* is greater than 0 and less than 1, so both alleles are present in appreciable frequency even after a great many generations. This is how the gene that causes sickle cell anemia is maintained. Note that the limit, p*, does not depend on p_0 . For sickle cell anemia, the heterozygote selective advantage is small and the asymptotic frequency of A_2 is very low. This is illustrated in the Evolution Grapher.

The discussion in the Evolution Grapher notes that the most prevalent genotype at asymptote may or may not be the fittest genotype in the case of overdominance. (The fittest genotype is not the most prevalent asymptotically for sickle cell anemia.) Often one can estimate the fitness of phenotypes (e.g., "blue eyes," "brown eyes," etc.), but one has no idea of the genetic structure underlying these phenotypes. In such circumstances, it is natural to try to short-circuit refined, Mendelian, evolutionary theory by simply assuming that fitter phenotypes will achieve greater prevalence after many generations. This is a respected research strategy that is called the Phenotypic Gambit. However, the sickle cell anemia example shows that the Phenotypic Gambit can be wrong. More generally, it is dangerous to infer EEA fitness from modern prevalence. (The Phenotypic Gambit fails in precisely those cases where the fitness advantage of the hetereozygote is not too great, resulting in $p^* > 2/3$.)

e. Underdominance ($w_{11} > w_{12}$ and $w_{22} > w_{12}$). Here an analysis similar to the last case shows that p_n converges to 0 if $p_0 < p^*$, but p_n converges to 1 if $p_0 > p^*$. This case, and the case of equal genotype fitnesses, are the only ones where the limit of p_n depends on the initial value p_0.

Only ratios of fitnesses are relevant to Haldane's Equation. Consequently, for calculating gene proportions, fitnesses can be rescaled so that one of the fitnesses has a prescribed value, like 1 or 100. This is often done in the population genetics literature. Such a rescaling would not be acceptable if one were concerned with the absolute numbers of different genes and genotypes, instead of with corresponding proportions.

Conclusions regarding the asymptotic value p_n of A_1 gene frequency can be summarized as follows:

If all genotypes are equally fit, then $p_\infty = p_0$. This case, and the case of underdominance, are the only ones where p_∞ depends on p_0 .

If $w_{11} > w_{22}$ and $w_{11} \geq w_{12} \geq w_{22}$, then A_1 is fitter for all p_0 and A_1 takes over ($p_\infty = 1$) for all p_0 . Evolution is slow "at the end" if A_1 is a beneficial dominant, and very slow "at the beginning" if A_1 is a beneficial recessive.

If the order of the ws is reversed, A_2 is fitter for all p_0 and A_2 takes over ($p_\infty = 0$) for all p_0.

If $w_{12} >$ both w_{11} and w_{22} (overdominance) then p_n converges to a stable equilibrium value

$$p_\infty = p^* = (w_{12} - w_{22})/(w_{12} - w_{22} + w_{12} - w_{11})$$

for all p_0 . For certain fitness values, the fitter of the two homozygotes will ultimately be more prevalent than the even fitter heterozygote, contrary to the rough-and-ready Phenotypic Gambit viewpoint, which encourages us to ignore "constraints" like Mendelian genetics and expect superior phenotypes to achieve superior frequency. In spite of this defect, the Phenotypic Gambit is a useful viewpoint.

If $w_{12} <$ both w_{11} and w_{22} (underdominance) then $p_\infty = 0$, p^*, or 1, depending on whether $p_0 <, =,$ or $> p^*$. p^* is an unstable equilibrium (a "tipping point"). A_1 is wiped out if it is initially rare and takes over if it is initially prevalent.

For overdominance and underdominance, nothing is assumed about the values of w_{11} and w_{22}. Either can be bigger, or they can be equal.

Digression on the Demographic Transition

A standard measure of demographic fertility is the Total Fertility Rate, which is the number of lifetime live births per woman. Table A.2 on p. 696 of Haines' and Steckel's *A Population History of North America* (Cambridge, 2000) shows that US TFR fell continuously from 1800 to 1940, and French data also show general decline since 1800. These are examples of the Demographic Transition. Sure, it's good for the planet, but it contradicts the expectation of high fitness that flows from the notion that evolution produces organisms that are "well adapted" to their environments. (This is illustrated by increasing population mean fitness in all output of the Evolution Grapher.) Cultural phenomena of some sort must be responsible for modern low TFR.

Note that the fertility decline began in America and France way before modern contraceptives became available. Some of that early decline is undoubtedly achieved via sexual abstinence and coitus interruptus. Anyway, there are obvious "economic" reasons for limiting family size, but humans didn't have to give economic considerations priority over the joys of large families, and, just because contraceptives are available, they don't have to use them. It says something about us that most of us want to use them.

Incidentally, the same table shows very low life expectancies in 1850, especially for Blacks (23 years, versus 39.5 for Whites).

Use Gapminder World at http://www.gapminder.org to explore worldwide fertility declines.

Lecture 23

6. Evolution in Small Populations—Random Genetic Drift.

Fitness typically has both survival ("viability") and fertility components, among others. One possible formula, discussed earlier, is

$w_{ij} = k \times$ (viability factor) $\times$ (fertility factor) $= k\, s_{ij}\, t_{ij}$

Viability is the probability of surviving to reproductive age. The product of parental fertilities is the expected number of offspring. For most purposes, it does not matter whether selection is based on viability or fertility differences, or both. In our computer runs, selection was based on fertility differences.

The random component of finite population evolution is called ***random genetic drift***. It produces jagged, irregular graphs of A_1 gene frequency as a function of generations. Random genetic drift is less and less pronounced as population size increases, and disappears entirely in the idealized infinite population cases described by Haldane's equation. Random genetic drift is a component of evolution, along with selection and mutation.

Here are the results of some systematic, large scale experiments with a small population evolution simulator. (The simulator is described at http://psych.upenn.edu/~norman/EvolSimRandomDrift.pdf, which give a link to an archive containing the simulator). For each of seven condition, five million generations were simulated. ps = population size. Fitnesses are given as percentages of the fitness of A_2A_2. A single A_1 mutant gene was introduced into a population of A_2A_2s, initiating an episode that ended when A_1 "took over," completely displacing the "wild type" A_2s, or else A_1 "died out". Then A_1 was reintroduced and the experiment was repeated again and again. The successive conditions considered were less and less favorable to the new A_1 mutant, and, as expected, A_1 takeover probability decreased. Regardless of fitness values, dieouts were much more plentiful than takeovers, but there were some A_1 takeovers even when selection was strongly against A_1. It is interesting to note that, in the three most disadvantageous conditions, the rare takeovers that do occur occur quickly, on the average. Mean takeover times are longest in the case of pure drift.

ps = 400. Fitness 22 12 11 = 100.0 101.0 102.0. A_1 advantageous, no dominance.

Number of A_1 takeovers = 4416 (1.99%). Mean of A_1 takeover times = 660.08

ps = 400. Fitness 22 12 11 = 100.0 101.0 101.0. A_1 advantageous and dominant.

Number of A_1 takeovers = 2859 (1.83%). Mean of A_1 takeover times = 1210.73

ps = 400. Fitness 22 12 11 = 100.0 100.0 102.0. A_1 advantageous and recessive.

Number of A_1 takeovers = 1995 (0.56%). Mean of A_1 takeover times = 625.08

ps = 400. Fitness 22 12 11 = 100.0 100.0 100.0. Pure drift.

Number of A_1 takeovers = 434 (0.14%). Mean of A_1 takeover times = 1589.07

ps = 400. Fitness 22 12 11 = 100.0 99.0 102.0. A_1 advantageous, underdominance.

Number of A_1 takeovers = 286 (0.06%). Mean of A_1 takeover times = 551.53

ps = 200. Fitness 22 12 11 = 100.0 99.5 99.0. A_1 slightly disadvantageous.

Number of A_1 takeovers = 79 (0.02%). Mean of A_1 takeover times = 661.81

ps = 200. Fitness 22 12 11 = 100.0 99.0 98.0. A_1 highly disadvantageous.

Number of A_1 takeovers = 2 (0.00%). Mean of A_1 takeover times = 503.50

Underdominance and "barrier busting." In underdominance in large populations, A_1 allele frequency p decreases to 0 if it starts below p*, and increases to 1 if it starts above p*. Therefore, even if A_1A_1 has greater fitness than A_2A_2 , A_1 gene frequency cannot move from small values to large values because of the "p* barrier." Thus the population cannot move from mostly A_2A_2 to mostly A_1A_1 with its attendant greater fitness. However, there is no insurmountable "p* barrier" in small populations, and one of the examples above shows that there is a small probability that introduction of an A_1 mutant can lead to an A_1 takeover in small populations with underdominance.

An analogous phenomenon is important in the more complex models for the evolution of social cooperation, in which genes that promote cooperation are disadvantageous when rare (since amiable cooperative genotypes may be taken advantage of by prevalent non-cooperators, and have little chance of finding other cooperators with whom to reap the rewards of cooperation), but advantageous when prevalent (when the odds described above are reversed). So social genes will almost surely be wiped out in very large population, but have a chance to take over a small population and, under favorable conditions, "infect" neighboring small populations.

Lecture 24

B. Sociobiological Models

1. Evolution of altruism toward related individuals via kin selection—Hamilton's Theory.

Altruism in this course means behavior that reduces one's own fitness but benefits the fitness of others. Extreme altruism produces very low personal fitness (e.g., celibate priests or suicide bombers). The evolution of altruism is somewhat mysterious, since lower fitness seems to imply extinction of relevant genes. We have to distinguish between altruism toward relatives and altruism toward unrelated individuals. The evolution of altruism toward relatives (or individuals that might be mistaken for relatives) is fairly well understood on the basis of kin selection theory, to be considered today. Evolution of altruism toward non-relatives exits on a massive scale in human societies and may have evolved via some kind of genetic or cultural group selection.

The basic idea of kin selection is that altruism sometimes evolves because altruistic behavior enhances the reproductive potential of relatives, and these relatives share genes with the altruist.

In W.D. Hamilton's (Journal of Theoretical Biology, 1964) very influential kin selection theory, evolution in a large random mating population proceeds more or less as in Haldane's Equation, but ordinary fitness is replaced by what he calls <u>inclusive fitness</u>,

R_{ij} = inclusive fitness = base value (usually taken to be 1)

+ benefit to self of the behavior in question (x_{ij})

+ benefit to relatives (y_{ij}), weighted by their relatedness, r (e.g. r = ½ for parent-child)

In summary, $R_{ij} = 1 + x_{ij} + r\, y_{ij}$.

The last term may be described as the benefit to <u>copies</u> of your genes that are carried by relatives.

<u>Interpretation of altruism, selfishness, and neutrality within Hamilton's theory</u>

description of A_iA_j	x_{ij}	y_{ij}	R_{ij} - base value = $x_{ij} + r\, y_{ij}$
neutral	$= 0$	$= 0$	$= 0$
altruistic	< 0	> 0	> 0 or < 0 or $= 0$
selfish	> 0	< 0	> 0 or < 0 or $= 0$

Clearly an additional calculation is necessary to determine whether the net effect of altruism or selfishness is positive or negative. Let

$k = y_{ij}/x_{ij}$ = change in relatives' fitness per unit change in A_iA_j's personal fitness, in consequence of the altruistic or selfish behavior under consideration

(e.g., change in offspring's fitness per unit change in parent's fitness, in consequence of parent's tenacity in defending a nest against predators).

For altruistic or selfish behavior, x and y have opposite signs, so $k < 0$. Large $|k|$ means that relatives are helped or hurt a great deal by a behavioral variation that is of little consequence to A_iA_j. Small $|k|$ means that relatives are hardly affected by a behavioral variation that is of great consequence to A_iA_j. Hamilton restricts his attention to cases where k is the same for all genotypes. That's why k has no subscripts.

Blackboard algebra shows that, <u>if $|k| > 1/r$, altruistic genotypes have above average fitness. If $|k| < 1/r$, selfish genotypes have above average fitness. I will refer to this as Hamilton's Rule.</u>

Here's an example to which we can apply Hamilton's Rule to see who has above average fitness. Assume that r = ½.

genotype	description	x_{ij}	y_{ij}	R_{ij}	asymptotic frequency
	altruistic	-1/8	3/8	17/16	
	neutral	0	0	1	
	selfish	1	-3	½	

In this example, k = (3/8)/(-1/8) = -3 = -3/1, and |k| = 3 > 2 = 1/r , so, according to Hamilton's Rule, altruistic genotypes will have above average inclusive fitness.

There is no magic in Hamilton's Rule. Once you have actually calculated inclusive fitness, you don't need Hamilton's Rule to tell you if it is above average or not!

Hamilton's Rule tells us which genotype has above average inclusive fitness, but, as we saw earlier, the genotype with largest inclusive fitness need not predominate at asymptote if it is heterozygotic.

I will illustrate this by specifying genotypes underlying the altruistic, neutral, and selfish phenotypes in my previous example. I will do this in two different ways.

Example 1: altruistic = A_1A_1 , neutral = A_1A_2 , selfish = A_2A_2 Then we are in a situation where more copies of A_1 lead to greater inclusive fitness, so our previous work with Haldane's equation shows that A_1 takes over. This implies that the altruistic A_1 A_1 type completely displaces all other types at asymptote, just as Hamilton's statement would lead us to expect.

Example 2: altruistic = A_1A_2 , neutral = A_1A_1, selfish = A_2A_2 Here we have overdominance, so A_1 gene frequency converges to

$p^* = (17/16 - 8/16)/((17/16 - 8/16) + (17/16 - 16/16)) = (9/16)/(9/16 + 1/16) = 9/10$.

The corresponding asymptotic genotype probabilities are given in the table:

genotype	description	x_{ij}	y_{ij}	R_{ij}	asymptotic frequency
A_1A_2	altruistic	-1/8	3/8	17/16	$2 \times .9 \times .1 = .18$
A_1A_1	neutral	0	0	1	$.9^2 = .81$
A_2A_2	selfish	1	-3	½	$.1^2 = .01$

The altruistic genotype is much less prevalent than the neutral genotype at asymptote.

The moral is that, to understand the course of evolution, it is not sufficient to know whether altruistic or selfish genotypes have above average inclusive fitness. Equivalently, it is not sufficient to know whether |k| > 1/r or < 1/r. To understand the course of evolution, it is necessary to know, in addition, whether the altruistic and selfish genotypes are homozygotic or heterozygotic. So Hamilton's Rule is an oversimplification.

The notion that genetics can be ignored in predicting the course of evolution is the Phenotypic Gambit discussed in previous lectures. According to this notion, one simply expects the fittest genotype to predominate at asymptote. This is a natural approach in situations when the genotypes underlying observed phenotypes are unknown, as is often the case. But, as we have repeatedly seen, it can lead to errors.

Changing the subject, please understand that Hamilton's rule is supposed to specify circumstances under which altruism would have evolved in the EEA. It is not supposed to specify circumstances under which animals will currently act altruistically. It does not say "Act alturistically in situations where $|k| > 1/r$." A bird with a Hamilton-type altruistic genotype will always act altruistically.

Lecture 25

2. The game theoretic approach to the evolution of competitive strategies and social cooperation ("Evolutionary game theory")

A Hawk-Bully-Dove Game

(Terminology nonwithstanding, the're all conspecifics [of the same species].) Each bird is assumed to have exactly one fitness determining "encounter" with a conspecific. The "payoff matrix" below gives the fitness ***of the birds listed at the left of the rows*** as a function of the type of bird encountered, listed above the columns.

C is the "base level" or "ordinary value" of fitness (analogous to the base level of "1" for inclusive fitness).

Two cells with the same shading represent payoffs to different participants in the same encounter.

	Hawk	Bully	Dove
Hawk	C-12 (Hawks injure each other)	***C+6*** ***(Hawks frighten off bullys and doves)***	***C+6*** ***(Hawks frighten off bullys and doves)***
Bully	***C*** ***(Hawks frighten off bullys and doves)***	C-6 (Bullys injure each other, but less than Hawks injure each other)	C+6 (Bullys frighten off doves)
Dove	***C*** ***(Hawks frighten off bullys and doves)***	C (Bullys frighten off doves)	C+F (Two Doves can cooperate in some way, e.g., share food.)

R. L. W. Brown's Analysis (from R. L. W. Brown, Evolutionary game dynamics in diploid populations, *Theoretical population biology*, 24, 1983, 313-322.) Brown, in whose article this example appears, is primarily concerned with how the frequencies of these different strategies change from generation to generation, and in their asymptotic frequencies. In particular, will one of these strategies displace the others at asymptote? These are the kinds of question we have asked about all previous models. Details will be given later.

John Maynard Smith's Analysis. Maynard Smith asks a somewhat different question: Suppose all birds are currently using a certain strategy. What strategies are good for repulsing "invaders" who use other strategies? If a strategy has this property, Maynard Smith calls it an Evolutionarily Stable Strategy, or ESS.

If F > 6, then Dove is an example of an ESS. The reasoning is as follows: Suppose that the population is currently 100% doves. If a solitary mutant or migrant Hawk appears, he is sure to encounter a Dove, in which case his fitness will be C+6. On the other hand, almost all Doves will encounter other Doves and thus have greater fitness C+F. Thus the Hawk will not be able to gain a foothold. The same is true of an invading Bully. So Dove is an ESS.

Simple ESSs (SESS)

We define the main diagonal of any square matrix as the diagonal that goes downward from the upper left to the lower right.

A strategy is an ESS when rare "invaders" using other strategies have lower fitness that prevalent "residents". A sufficient condition for a strategy to be an ESS is that the largest thing in its column is on the main diagonal. If this condition is satisfied, I will say that the strategy is a Simple ESS (SESS—my terminology).

Note that the only payoffs that are relevant to whether a strategy is a SESS are the payoffs in the column corresponding to that strategy. The entry on the main diagonal is the fitness of prevalent residents, who compete primarily with others of their kind. The other entries are fitnesses of various kinds of invaders, who also compete with these prevalent residents.

It is easy to check that, in Brown's matrix, neither Hawk nor Bully can ever be an SESS. And Dove is an SESS if and only if $F > 6$. Simple numerical examples of other evolutionary games show that there may be no SESSs, one SESS, or multiple SESSs.

Two problems with ESS theory

1. Maynard Smith's ESS analysis of evolutionary games relates only to the "invadability" issue. It does not describe the entire course of evolution. That is one problem with the theory.

Another problem with ESS theory is that it takes no account of Mendelian genetics. No indication is given of the genetic structures corresponding to the different genotypes, and the implication is thus that genetic structure is irrelevant. That is ridiculous. Suppose, in this example (which Maynard Smith does not explicitly address), that Hawk, Dove, and Bully have genotypes A_1A_1, A_1A_2, A_2A_2 . Then a population of all Doves will necessarily bring forth lots of Hawk and Bully "invaders" in the next generation, regardless of the value of F! So the ESS analysis is simply wrong. This error occurs because

2. ESS analysis implicitly assumes "like begats like" asexual genetics (the genetics of viruses!), rather than the more complex Mendelian genetics. Ignoring Mendelian genetics is a drawback to ESS theory, or at least of the most popular form of this theory.

Implicit use of asexual genetics is a defining feature of the Phenotypic Gambit, discussed earlier. As I have said many times, the Phenotypic Gambit is a respected approach, even though it can lead to errors.

Brown's model. Brown assumes the sensible genetic structure,

Strategy	Hawk	Bully	Dove
Genotype	A_1A_1	A_1A_2	A_2A_2

and the same payoff matrix as before

	Hawk	Bully	Dove
Hawk = A_1A_1	C-12	C+6	C+6
Bully = A_1A_2	C	C-6	C+6
Dove = A_2A_2	C	C	C+F

The genotype fitnesses are weighted averages of fitnesses against different opponents. The weights are the Hardy-Weinberg probabilities of these opponents. If p is the proportion of A_1 genes, we get, after a bit of algebra,

$$w_{11} = C - 12\,p^2 + 12\,pq + 6\,q^2 \qquad w_{12} = C - 12\,pq + 6\,q^2 \qquad w_{22} = C + F\,q^2 .$$

Note that this is an example of frequency dependent selection, ***but this does not prevent us from applying our usual large population analysis based on Haldane's Equation***. Skipping details, the results can be summarized as follows:

1. If $F > 6$ (in which case Dove is the only SESS), the "dovish" A_2 gene takes over, as does the Dove genotype.

2. If $F < 6$ (in which case there are no SESSs), gene frequencies converges to intermediate values, so that all three genotypes are present at asymptote.

So, in both cases, Brown's model is consistent with expectations from ESS theory, but Brown's model goes beyond ESS theory by making detailed predictions about the course and asymptote of evolution.

Review of Maynard Smith's Contributions

1. Linking behavioral evolution to game theory via fitness "payoffs".

2. The notion of Evolutionarily Stable (i.e., noninvadable) Strategies (ESSs).

3. Probabilitstic ESSs, discussed in the next lecture.

Lecture 26

Mixed strategies and probabilistic ESSs.

Maynard Smith's third major contribution is to call attention to situations where ESSs may involve probabilistic mixtures of simple strategies like Hawk and Dove. Under certain conditions, it might be advantageous for an animal to be Hawkish a third of the time and Dovish two thirds of the time. It's as if the animal flipped a biased coin and behaved Hawkishly if it came up heads. This is called a mixed strategy, to distinguish it from simple strategies, like Hawk and Dove.

This is an individual interpretation of a mixed strategy—each individual has a certain probability, say 1/3, of adopting the Hawk strategy. Alternatively, there is a population interpretation of the same strategy, according to which an individual can only be pure Hawk or pure Dove, but 1/3 of the individuals are pure Hawk. In this case, we should really use a different terminology: "distribution" or "polymorphism" instead of "mixed strategy," but it is customary to use the "mixed strategy" terminology in both cases. Evolutionary game theory applies to both individual and population interpretations of mixed strategies.

The definition of an ESS for mixed strategies is similar to the definition for simple strategies. In both cases, an ESS is a strategy (or polymorphism) that cannot be invaded. In the population interpretation, we consider a small band of invaders with a different distribution than the resident population. It can be shown that mixed strategy ESSs have the property that all participating simple strategies have the same average payoff. This property permits us to calculate mixed strategy ESSs, as we will illustrate below.

In some of Maynard Smith's papers, he illustrates mixed strategies in the context of a game involving just Hawks and Doves. Let me review this game with you, since it is often used to introduce Maynard Smith's ideas.

	Hawk	Dove
Hawk	(V-D)/2	V
Dove	0	V/2

V is the payoff for victory, and D is the cost of defeat if there is an actual fight.

Dove can never be an ESS, since V/2 is always less than V. Hawk is an ESS if $V > D$, but not if $V < D$. Thus, if $V < D$, neither Hawk nor Dove is an ESS. However, there is a mixed strategy ESS. Let P be the probability of the Hawk simple strategy in the mixed strategy. Then the average payoff to a Hawk, $P(V-D)/2 + (1-P)V$, equals average payoff to Dove, $(1-P)V/2$. This linear equation can be easily solved for P, and the solution is $P = V/D$. Animals using this ESS act Hawkish this proportion of the time and Dovish the rest of the time.

In Brown's Hawk-Bully-Dove game, we saw that there were no simple ESSs when F, the payoff for dovish cooperation, is less than 6. It can be shown that, when F is less than 6, there is a mixed strategy ESS (or an ESS polymorphism in the population interpretation). Finally, we saw that, when F is less than 6, Brown's model predicts a certain 3-strategy polymorphism at the asymptote of evolution. It is natural to ask whether the ESS polymorphism is the same as Brown's asymptote-of-evolution polymorphism. The answer is "No"—the two polymorphisms are different. I feel that this is a shortcoming of Maynard Smith's ESS approach, but not necessarily a major shortcoming.

Empirical Tests of Game Theoretic Ideas.

Game theoretic ideas have had a major impact on thinking in the evolution of behavior. For example, a standard textbook on Behavioral Ecology (Krebs and Davies, 1984) is absolutely brimming with qualitative applications.

A news article in *Science* magazine ("Putting game theory to the test," 1995) describes a few relatively recent attempts to rigorously test predictions of game theory in several areas: territorial defense in spiders, the tendency of young birds to kill their siblings, conflicts between worker and queen naked mole rats, destruction of other bird's nests by male bower birds, and tit for tat behavior in guppies.

1. The spider problem was to predict when female spiders will defend desirable web sites (!) against intruders. Good web sites are necessary for reproductive success, but fights to defend them often lead to costly injuries like loss of legs. The model considered was apparently much more complex than the Hawk—Dove model, since it took account of availability of alternative sites as well as weights of the two contestants. Defense is predicted to be less likely if alternative sites are readily available or the adversary is heavier. Predictions of the model were confirmed in areas with few alternative sites, but there was more fighting than expected in areas with many alternative sites.

2. Game theory successfully predicted the tendency of larger nestlings to kill smaller ones at precisely the point where their own survival was threatened by shortage of food.

3. Naked mole rats are ugly creatures with very distinctive life styles. The problem considered is the tendency of certain members of the worker caste to slack off and avoid the effort as well as the dangers of work. This brings them into conflict with the queen, who is the chief disciplinarian. Game theory successfully predicts which naked mole rat workers are most likely to slack off. They are the largest workers, who may someday become breeders, and also workers less related to the queen and her offspring.

The Prisoner's Dilemma, Cooperation, and Tit-for-Tat

The next two examples relate to a situation called the Prisoner's Dilemma. It was discussed by game theorists long before Maynard Smith introduced game theory to biologists. It is a convenient vehicle for illustrating how evolution might favor cooperation of unrelated individuals in a competitive situation.

The payoffs are "years off a maximum sentence of five years", which should be positively related to fitness. If one prisoner implicates and the other cooperates, the implicator goes free and the cooperator gets the maximum sentence. If both cooperate, they both get 3 years off the maximum sentence. If both implicate, they both get one year off.

	Cooperate with Accomplice	Implicate Accomplice
Cooperate with Accomplice	3	0 (max. sentence)
Implicate Accomplice	5 (goes free)	1

Note that Implicate has a higher payoff than Cooperate, regardless of the strategy used by the other player. If the second (column) player Cooperates, the first (row) player gets 5 years off for Implicating but only 3 years off for Cooperating. If the second player Implicates, then the first player gets 1 year off for Implicating but no years off for cooperating. So, from this point of view, Implicate looks like the better strategy.

It is also the preferred strategy from the point of view of Evolutionary Game Theory. Clearly Implicate is a simple ESS and Cooperate is not. Moreover, it can be shown that there are no mixed ESSs. So Maynard Smith's analysis suggests that evolution would heavily favor the Implicate strategy, even though, if everyone adopted this strategy, everyone would be worse off than if everyone always cooperated with their accomplice. Flipping from humans to birds, we find that non-cooperation predominates in bower birds. Male bower birds routinely destroy each other's nests, even though it seems at least possible that all the birds would be better off if there were no such destruction.

And yet, we know that cooperation does sometimes occur in situations of this kind. In humans, cooperation may depend on a cultural mechanism that has nothing to do with evolution. But there is an interesting extension of game theory that permits us to see how the inclination to cooperate in situations like this could have evolved in both humans and animals.

The basic idea is that we might have repeated interactions of roughly this kind with the same accomplice, and we might take his past behavior into account in deciding what to do in the present interaction. To make a long story short, Robert Axelrod proved that a Tit-for-Tat strategy is an ESS if an indefinite number of repeated interactions are permitted. Tit-for-Tat has two parts. The prisoner cooperates on the first interaction. On subsequent interactions, he does what his accomplice did on the last interaction. If the accomplice cooperated last time, he cooperates this time. If the accomplice implicated last time, he implicates this time.

As I say, this is an ESS, and so one might not be surprised to see rough analogs of Tit-for-Tat in the behavior of real animals. The Science magazine news article mentions two examples involving fish. Cooperation consists of scouting a possible predator in groups, whereas non-cooperation means leaving the group when it gets close to the possible predator. Tit-for-Tat like behavior is definitely observed.

However, another *Science* news article ("Cowardly lions confound cooperation theory," 1995) points out that these are laboratory experiments, and only one real-world example of Tit-for-tat (involving reciprocal feeding in vampire bats) has been found despite more than a decade of searching. Moreover, even that one example is disputed. The article also reports failure of Tit-for-Tat in predator defense by pairs of lions. There are certain cowardly lions that never cooperate—that is, they always run away when faced with danger—even if their defense partner previously cooperated (stood firm). So things are not going all that well for Tit-for-Tat.

Lecture 27

Technicalities regarding mixed strategy ESSs.

In the last lecture, I said that "It can be shown that mixed strategy ESSs have the property that all participating simple strategies have the same average payoff. This property permits us to calculate mixed strategy ESSs." This statement is correct, but it must be read carefully. The property in question is a necessary condition for ESSs, but not a sufficient condition—a mixed strategy can satisfy this condition without being and ESS. The standard example of this the rock, paper, scissors payoff matrix

	Rock	Paper	Scissors
Rock	0	-1	1
Paper	1	0	-1
Scissors	-1	1	0

Obviously none of these simple strategies is a SESS. All three simple strategies have zero expected payoff against the mixed strategy which uses the three simple strategies with equal probabilities (1/3), but it can be shown that this mixed strategy is not an ESS. This evolutionary game does not have any ESSs, simple or mixed.

Iterated Prisoner's Dilemma

Here's the payoff matrix from the last lecture

	Cooperate with Accomplice	Implicate Accomplice
Cooperate with Accomplice	3	0 (max. sentence)
Implicate Accomplice	5 (goes free)	1

Recall that Implicate always does better than Cooperate, regardless of what the other player does, and Cooperate is not an ESS, so it's hard to see how such unconditional cooperation would ever evolve. If the game is played repeatedly, all the payoffs are simply multiplied by n, and nothing changes. However, the situation does change if the game is played repeatedly with the same player, and if we replace unconditional cooperation with Tit for Tat (TFT), which is unconditionally cooperative only in the first play. Here's the payoff matrix for n repetitions.

	Tit for Tat	Implicate
Tit for Tat	3n	n-1
Implicate	5 + (n-1)	n

If n = 2, TFT is just as effective (payoff = 6) as Implicate against TFT, and if n is 3 or greater, TFT is more effective than Implicate against TFT, so TFT is a SESS. And the advantage over implicate increases with n. Moreover, TFT is almost as good as Implicate against Implicate when n is large (the difference is 1, but that's only 100/n percent, which is small when n is large). So the prospects for evolution of TFT conditional cooperation are much more favorable that for unconditional cooperation.

One can make this into a Mendelian genetic model by thinking of Tit for Tat as a dominant A_1 mutation in a population of A_2A_2 implicators. The resulting frequency dependant model behaves like the constant fitness model with underdominance. The cooperative gene A_1 is disadvantaged when rare but advantageous when prevalent. ***(This is a characteristic of cooperation in many different models)***. The critical frequency above which cooperation becomes advantageous becomes smaller as n becomes larger. When the critical frequency is quite small, it can be exceeded relatively easily by random drift in a small population, and could then spread to other groups by migration.

3. Group Selection and Price's Equation

Price's Equation concerns a metapopulation consisting of a number of subpopulations or "demes", e.g., USA and its constituent states or an archipelago and its constituent islands. Each deme has a certain frequency of a gene that promotes altruism. "Altruism" is defined by its effects: it make the altruists themselves less fit ***(unconditionally, regardless of their frequency)***, but it makes their demes more fit. Thus the proportion of altruistic genes is decreasing in each deme, but increase in population size is positively correlated with the proportion of altruistic genes, so that demes with more altruistic genes are contributing disproportionately to the next generation. Under certain circumstances, this disproportionate contribution can overcome the decreasing frequency of altruistic genes within each deme to produce an increase in the proportion of altruistic genes in the whole metapopulation.

Here is a highly artificial example. Suppose that there only two demes, 1 and 2, with population sizes n_1 and n_2 in this generation and n_1' and n_2' in the next generation. The comparable proportions of the altruistic gene are q_1, q_2, q_1', and q_2', and n, n′, q, and q′ are the population sizes and gene proportions in the entire metapopulation. (The qs here correspond to the ps in my earlier lectures.) Note that

$q = (n_1/n)q_1 + (n_2/n)q_2$

and that a similar equation holds for q′. Let

$n_1 = 900$, $n_1' = 100$, $n_2 = 100$, $n_2' = 900$ (hence $n = n' = 1000$)

$q_1 = .4$, $q_1' = .3$, $q_2 = .8$, $q_2' = .7$.

Then $q_1' < q_1$, $q_2' < q_2$, but

$q = .9\times.4 + .1\times.8 = .36 + .08 = .44$,

which is much less that

$q' = .1\times.3 + .9\times.7 = .03 + .56 = .59$.

This paradoxical outcome occurred because the second deme experienced an enormous increase in population, due to its greater frequency of altruistic genes. Price's equation shows precisely under what circumstances such an expansion can overcome the downward drift of altruistic gene frequency within each deme.

Note that, in the example, the increase in q was greater than the decrease in either q_1 and q_2. None the less, q′ is less than the larger of q_1' and q_2', which is, in turn, less than the larger of q_1 and q_2. This is always the case, and it implies that paradoxical increases in q cannot continue indefinitely if the demes are isolated. For continuing increase, there must be some kind of migration between demes, and numerous migration schemes have been considered. In my opinion, the most interesting and successful of these is Athena Aktipis' (*Theoretical Population Biology*, 2004) "walk away" scheme, whereby individuals tend to leave groups with an insufficient number of "cooperative" individuals who contribute to a common resource pool. Since such individuals are disadvantaged in comparison to "free riders" whatever their frequency, they qualify as "altuists" for the purpose of the present discussion. Please run some simulations using the model at

http://www.psych.upenn.edu/~aktipis/MovementGroups.htm

and observe that the number of cooperators is generally much larger than the number of free riders. This represents an evolutionary success of altruists.