

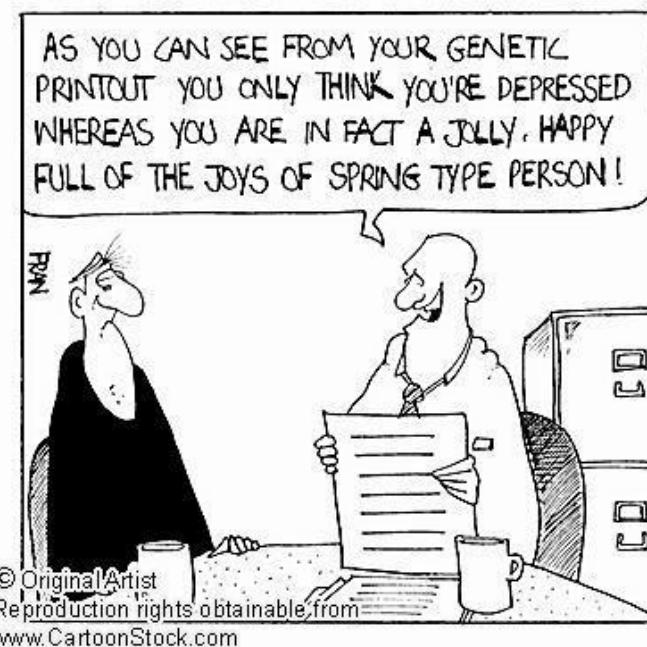
PSYC3016: Developmental Psychology

Lectures 3 & 4: Behavioural Genetics

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Behavioural Genetics

Overview

Lecture 3:

- Heritability estimates
- Twin studies
- Types of environment

Lecture 4:

- The missing heritability problem!
- Interactions
- Why, as psychologists, do we care?

Behavioural Genetics

Learning Outcomes

Lecture 3:

- LO1 – how genetic and environmental variables can shape development
- LO2 – understand the benefits and limitations of twin study designs
- LO3 – understand how to interpret results from behavioural genetic twin studies
- LO4 – understand how data obtained from behavioural genetic twin studies can inform understanding of child psychopathology

Lecture 4:

- LO1 – understand the missing heritability problem and what challenges this creates for understanding the mechanisms to explain child development.
- LO2 – be able to describe the theoretical and methodological reasons for missing heritability
- LO2 – be able to describe mediation and moderation interactions in the context of child development
- LO3 – understand the differential susceptibility hypothesis and how it can be demonstrated through gene-environment interaction analyses.
- LO4 – be able to critically evaluate the importance of understanding the relative roles of genes, the environment, and their interaction in public policy and psychological research.

Behavioural genetics

Recap – all psychological traits are heritable

- **Heritability (h^2):** how much of the variation of a trait (phenotype) in a population is due to genetic differences in that population
- Phenotype (P) = genetic effects (G) + environmental effects (E)
- Let's look at something simple...
- Is height genetically determined?

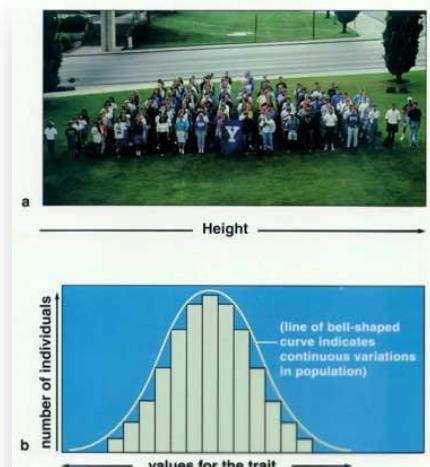
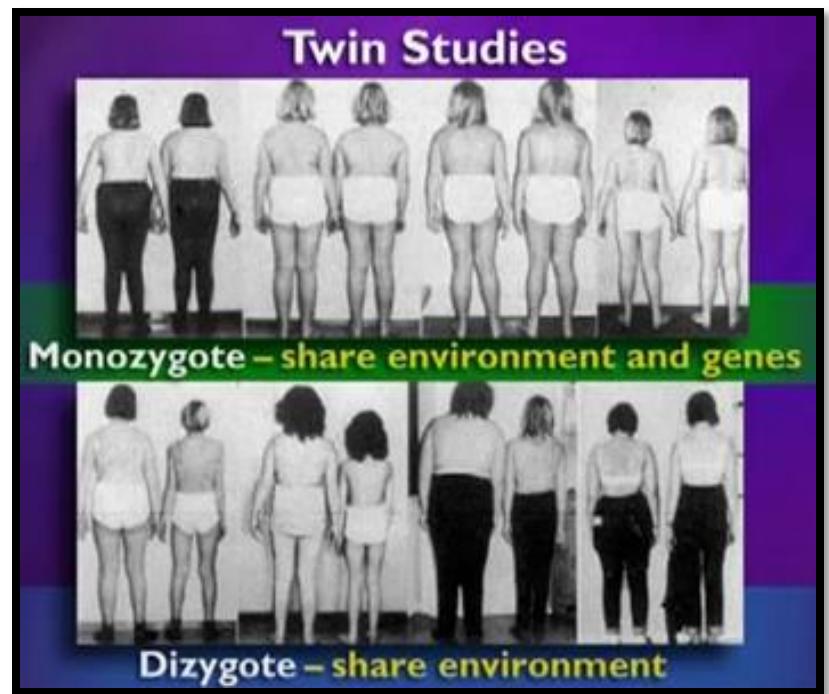


Fig. 9.18 (a) Continuous variation in human height.
(b) Bell-shaped curve typical of continuous variation in a trait.

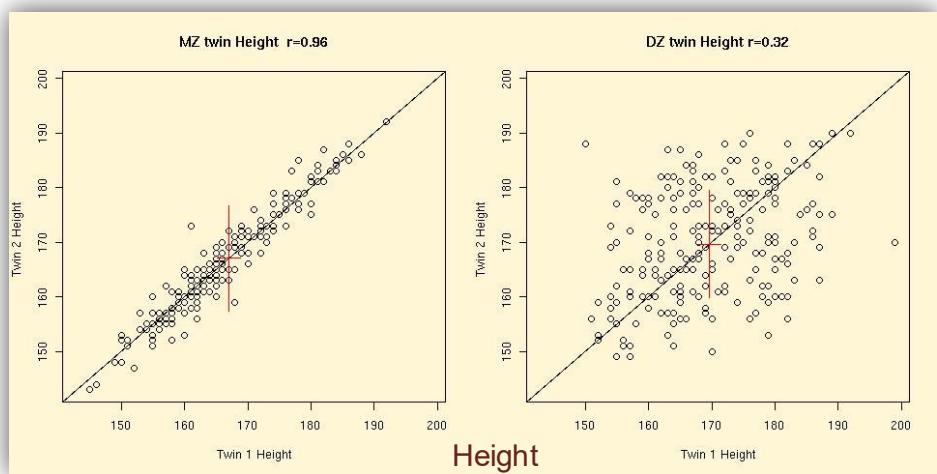
Where does the heritability index come from?

- By and large, we estimate the extent to which something is heritable by using a ‘naturalistic experiment’, the existence of twins
- Monozygotic = identical
- Dizygotic = non-identical (siblings)



Where does the heritability index come from?

- The power of twin studies, as noted, is grounded in the appealing naturalistic experimental design they so obligingly provide us with:
 - MZ: Monozygotic, all genetic information is shared
 - DZ: Dizygotic, an average of 50% of genetic information is shared
- You need to grapple with two important statistical constructs to understand the basic logic of twin studies
 - Correlation
 - Variation



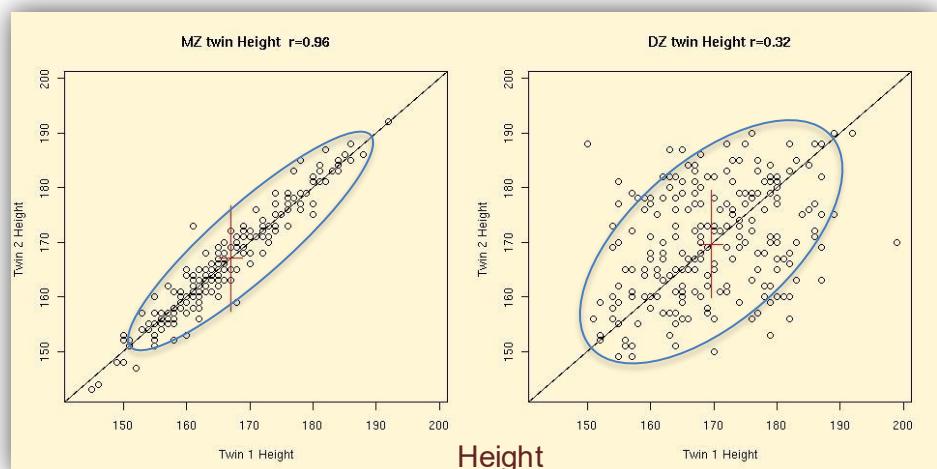
Where does the heritability index come from?

Correlation:

Correlation refers to the fact that there is a systematic relationship between Twin 1 and Twin 2, here seen in the cluster of points around the straight line summarizing the data in each image (which is a line of *best fit* or a *regression line*)

The correlation is stronger for MZ twins than DZ twins, see how the points cluster more tightly around the line – we often refer to this as the ‘cloud’

(Another important feature is that the relation between Twin 1 and Twin 2 is linear. Practically, that means that your co-twin is likely to be the same height as you irrespective of whether you are short or tall)



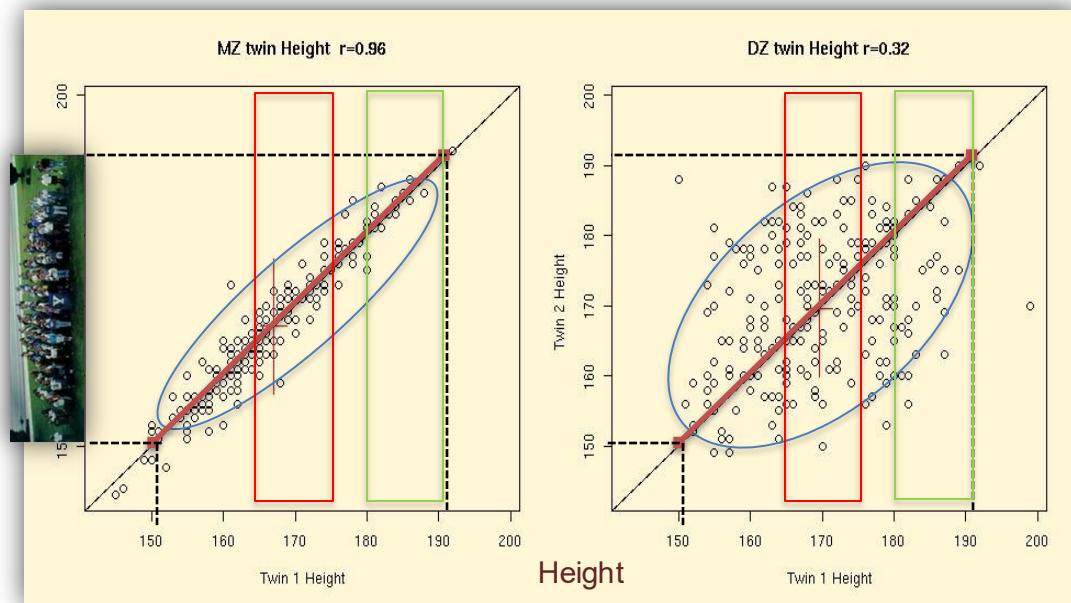
Where does the heritability index come from?

Variation:

Variation refers to the way the points are distributed over the measurement scale, or spread out around the line in these Figures

In both of these Figures you can see that the *Range* (smallest to largest observation) is approximately the same for both the MZ twins and the DZ twins. However, if you think back to the normal curve, they are not distributed quite the same way over that Range.

(There is an important assumption underlying the statistical models used to do twin analyses, which says that the variance should be approximately equal for different groups)



Where does the heritability index come from? A simple model and a little bit of maths...

- Phenotype (P) = genetic effects (G) + environmental effects (E)
 - Heritability estimate (h^2) = genetic influences on the phenotype
- ~~Phenotype (P) = genetic effects (G) + environmental effects (E)~~
- $1 = h^2 + E$
- So... if we can work out E we know what the heritability estimate is.
- Help us twins!
- MZ twins share 100% genes and 100% environment
- DZ twins share 50% genes and 100% environment
- Right?
- Nope...
- It is a little bit more complicated...



Where does the heritability index come from? A simple model and a little bit of maths...

- There are *different types* of environment
 - **Common environment (C)** – events that happen to both twins, influencing them in the same way
 - e.g. (uterine conditions, same family members, same house, same school etc)
 - **Unique environment (E)** – events that occur to one twin but not the other, or events that happen to both twins but influence them in different ways
 - e.g. (accidental injury, death of a parent, bullying)
- So... the *environment* comprises (C) + (E)
- Phenotype (P) = genetic effects (G) + common environment (C) + unique environment (E)
- $1 = h^2 + C + E$
- Ok, now we can use the twins...



Where does the heritability index come from? A simple model and a little bit of maths...

- MZ twins share 100% genes and 100% **common** environment
 - The correlation between identical twins (r_{mz}) provides an estimate of $h^2 + C$
 - E.g. Correlation of 0.96 (height) = $h^2 + C$
 - So, in MZ twins only 0.04 (4%) due to unique environment (E)
- DZ twins share 50% genes and 100% common environment
 - So the correlation between DZ twins (r_{dz}) is a direct estimate of $\frac{1}{2} h^2 + C$
- So, for any given phenotype...
- $r_{mz} = h^2 + C$
- $r_{dz} = \frac{1}{2} h^2 + C$

Here comes the maths... rearrange the equations...

- h^2 , therefore, is twice the difference between identical and fraternal twin correlations (Falconer's formula)
- $h^2 = 2(r_{mz} - r_{dz})$
- $C = r_{mz} - h^2$
- $E = 1 - r_{mz}$

Are we done...?

Nope, sorry!

Where does the heritability index come from? A simple model and a little bit of maths...

- Think of our height example...
- $h^2 = 2 (r_{mz} - r_{dz})$
- $h^2 = 2 (0.96 - 0.32)$
- $= ?! > 1$
- This simple model makes an assumption about the genetic effects
- Assumes **additive** genetic effects – also known as “narrow sense heritability”
 - AA, Aa, aa
 - The risk conferred by an allele is increased r -fold for heterozygotes and $2r$ -fold for homozygotes
 - $Aa = rAA$
 - $aa = 2rAA$
- What is wrong with this assumption?
 - Effects can be dominant, recessive, heterozygous...
 - Gene-gene interactions
 - Gene-environment interactions
- But, it is a nice simple model to get the idea
 - You will see additive genetic effects referred to as (A) in models...
- Phenotype (P) = additive genetic effects (A) + common environment (C) + unique environment (E)¹²

Behavioural Genetics

Criticisms of the twin model

Heritability values are strongly tied to the populations in which they were gathered (in more diverse environments, we'd expect the degree of heritability to go down)

Heritability values depend on the extent to which twin pairs actually reflect genetic and environmental variation in the population

- Most twin pairs are raised under highly similar conditions (low variation in unique environment)
- Assumes no assortative mating
 - Assumes mating is random
 - In reality, people tend to choose partners based on certain traits (e.g. similarities or familiarity of ethnicity, religious/ethical beliefs, life-style choices).

Behavioural Genetics

Criticisms of the twin model

Always remember: Heritability estimates refer to the *proportion of variation between individuals* on a trait that is due to genetic factors.

- It does not indicate the degree of genetic influence on the development of a trait of an individual.
- E.g. It is incorrect to say that since the heritability of personality traits is about .6, that means that 60% of your personality is inherited from your parents and 40% comes from the environment
- **Recommended reading:** (Turkheimer, E. (2000). Three laws of behavior genetics and what they mean, *Current Directions in Psychological Science*, 9(5), 160-164)

Let's look at an example

- Burt (2009) Psychological Bulletin, 135, 608-637: Meta-analysis of genetic influences on child psychopathology (i.e., mental health)
 - A meta-analysis of 490 studies of internalizing and externalizing psychopathology prior to adulthood
 - Traditional wisdom (twin studies) says additive genetic effect and non-shared environment are of great importance for psychopathology – ALMOST NO influence of shared environment!
 - However, initial survey of literature suggests shared environment may be important prior to adulthood, and especially in more extreme environments

Genetic and environmental influences: child psychopathology

Burt (2009). *Psychological Bulletin*, 135, 608-637

- Areas of interest

- Externalizing Disorders (EXT)
- Conduct Problems (CP)
- Oppositional Defiant Problems (ODP)
- Attention Deficit/Hyperactivity Problems (ADHP)
- Internalizing (INT)
- Depression (DEP)
- Anxiety (ANX)

Table 4
Parameter Estimates From Better-Fitting Overall Model by Phenotype

Phenotype	a^2	c^2	d^2	e^2
EXT ($N = 16$ samples with 10,957 sibling pairs)	.590 (.552, .629)	.153 (.118, .187)		.258 (.248, .269)
CP ($N = 38$ samples with 28,709 sibling pairs)	.576 (.550, .602)	.145 (.121, .169)		.280 (.273, .287)
ODP ($N = 9$ samples with 12,692 sibling pairs)	.591 (.547, .636)	.101 (.062, .140)		.308 (.297, .319)
ADHP ($N = 26$ samples with 25,712 sibling pairs) ^a	.259 (.198, .320)	.101 (.062, .140)	.444 (.383, .505)	.297 (.289, .305)
INT ($N = 17$ samples with 13,099 sibling pairs)	.507 (.467, .547)	.164 (.129, .198)		.330 (.318, .343)
DEP ($N = 17$ samples with 21,027 sibling pairs)	.437 (.400, .474)	.139 (.110, .169)		.424 (.411, .438)
ANX ($N = 23$ samples with 20,786 sibling pairs)	.475 (.438, .512)	.122 (.091, .153)		.404 (.392, .416)

Note. a^2 = additive genetic influences; c^2 = shared environmental influences; d^2 = dominant genetic influences; e^2 = nonshared environmental influences; EXT = externalizing; CP = conduct problems; ODP = oppositional defiant problems; ADHP = attention-deficit/hyperactivity problems; INT = internalizing; DEP = depression; ANX = anxiety.

^a ADE model is presented.

Genetic and environmental influences: child psychopathology

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Conclusions

- The shared environment accounted for a modest to moderate amount of variance in psychopathology outcomes for every domain except ADHP
- It is also notable that the non-shared environment (i.e., e^2) also had a very profound impact on psychopathology, even in the case of ADHP

How does the shared environment influence child psychopathology?

Child psychopathology

- Increasing evidence shows that certain parental styles and behaviours increase children's risks for psychopathology outcomes
- Parental over-protection and over-control promotes children's anxiety
- Parental hostility and poor boundary setting increase the likelihood that children will have externalizing problems
- Externalizing problems in childhood are the best predictor of **all** of the common forms of mental health problems in adulthood

Genetic and environmental influences

General conclusions

- We have some pretty good ideas about how the shared (and non-shared) environment may actually have an influence
- That is to say, we know something about the environmental **processes** that may be important
- It matters **when** you measure something and **how** you measure it (e.g. balance between unique and shared environment)
- We find it harder to think about what the genetic influence actually means!!

Genetic and environmental influences

General conclusions

- Turkheimer: “*There is an interesting parallel between the search for individual genes that influence behavior and the failed attempt to specify the nonshared environment in terms of measured environmental variables*”

❖ PTC – why would an environmental influence on a phenotype change as a function of time?

Behavioural genetics

Next time...

- The missing heritability problem
- Interaction, interaction, interaction
- Some very cool biological processes – I will require some volunteers!

