Heritability and Behavior Genetics

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Outline

Informal Definition

The fraction of variance in the phenotype that is due to genetic factors. Several notions of heritability, including:

- Broad heritability (h^2_G): fraction of variance explained by the genetic factor $G(\boldsymbol{x_i})$.
- Narrow heritability (h^2_A): fraction of variance explained by the additive component $A(\boldsymbol{x_i})$.

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Broad Heritability (h^2_G)

Recall the causal model from Dan's lecture:

$$\tilde{y}_i = \underbrace{G(\boldsymbol{x_i})}_{\equiv A(\boldsymbol{x_i}) + N(\boldsymbol{x_i})} + \boldsymbol{z_i}\gamma + \vartheta_i$$

where $\boldsymbol{z_i}, G(\boldsymbol{x_i})$ and $\boldsymbol{z_i}$ are random variables that vary across individuals in our population.

Broad heritability is the fraction of variance in $\tilde{y}$ explained by the genetic factor $G(\boldsymbol{x_i})$:

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Narrow Heritability (h^2_A)

An alternatively way to formulate the causal model is to absorb the non-additive component, $N(\boldsymbol{x_i})$, into the residual:

$$\begin{aligned}\tilde{y}_i = & \underbrace{G(\boldsymbol{x_i})}_{=A(\boldsymbol{x_i})+N(\boldsymbol{x_i})} + \boldsymbol{z_i}\gamma + \vartheta_i \\ = & \underbrace{A(\boldsymbol{x_i})}_{\equiv \boldsymbol{x_i}\beta} + \boldsymbol{z_i}\gamma + \underbrace{\varepsilon_i}_{\equiv \vartheta_i + N(\boldsymbol{x_i})}\end{aligned}$$

where $A(\boldsymbol{x_i})$ is the best linear predictor of $\tilde{y}_i$ given $\boldsymbol{x_i}$.

Narrow heritability is the fraction of variance in $\tilde{y}$ explained by the additive factor $A(\boldsymbol{x_i})$:

$$h^2_A \equiv \frac{V(A(\boldsymbol{x_i}))}{V(\tilde{y}_i)} = \frac{V(\boldsymbol{x_i}\beta)}{V(\tilde{y}_i)}.$$

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Remarks

- Heritabilities are, of course, population-specific.
- h^2_A is the R^2 from a population regression of $\tilde{y}_i$ on $\boldsymbol{x_i}$.
- For several reasons, more common to focus on h^2_A than h^2_G
- Tractability: $G(\boldsymbol{x_i})$ could be an incredibly complicated object.
  - In some applications (e.g. animal breeding) h^2_A is the more relevant parameter.
  - For many phenotypes, there is evidence that magnitude of $V(N(\boldsymbol{x_i}))$ is at most modest relative to $V(A(\boldsymbol{x_i}))$.

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Some Intuition

- **Why do we often observe** $V(A(\boldsymbol{x_i})) \gg V(N(\boldsymbol{x_i}))$?
- For some intuition, consider the dominance component of the non-additive factor. It can be shown that for genetic variant j:

$$\lim_{MAF \to 0+} \frac{V(D(x_{ij}))}{V(\tilde{y}_i)} = 0$$

- Most variants have low minor allele frequencies (MAF) and therefore...
- Similar arguments apply to other higher-order interactions.

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Dominance Deviation and Dominance Variance

Recall the simple single-locus model from the problem set:

Table: Genotype Value Parameters in Single Locus Model

Genotype	-,-	-,+	+,+
x_i	0	1	2
$\mathbb{E}(y_i \mid x_i)$	-a	d	a
Freq	q^2	$2pq$	p^2

Varying d

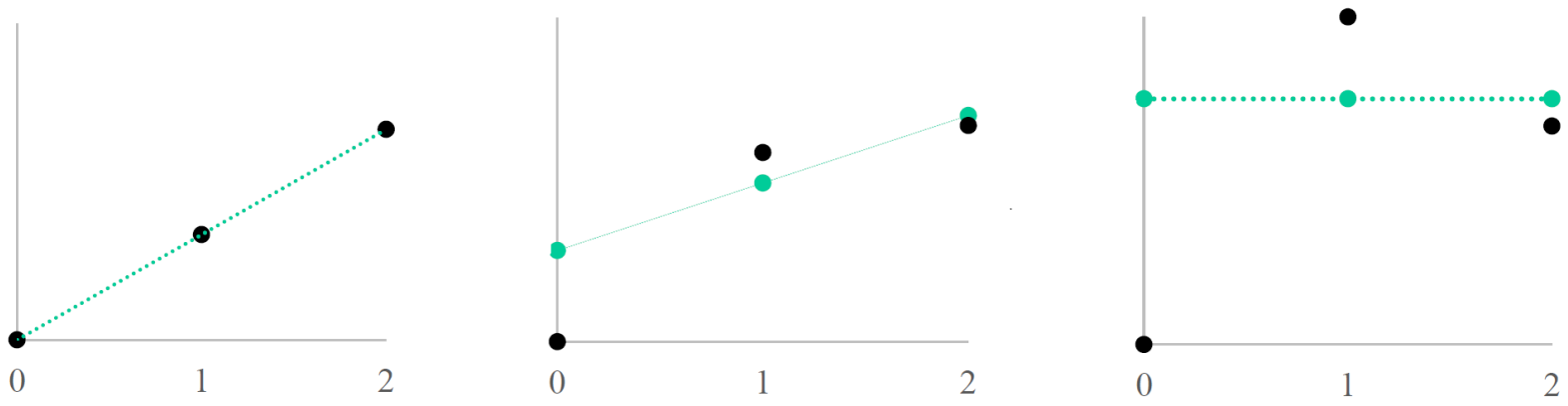

Figure shows how the BLP changes relative to the conditional mean as we vary d from 0 (left panel, zero dominance) to 0.5 (middle panel, partial dominance) to 2 (right panel, overdominance). In all calculations, $a = 1$ and $p = 0.75$.

Varying Frequency of + allele, p

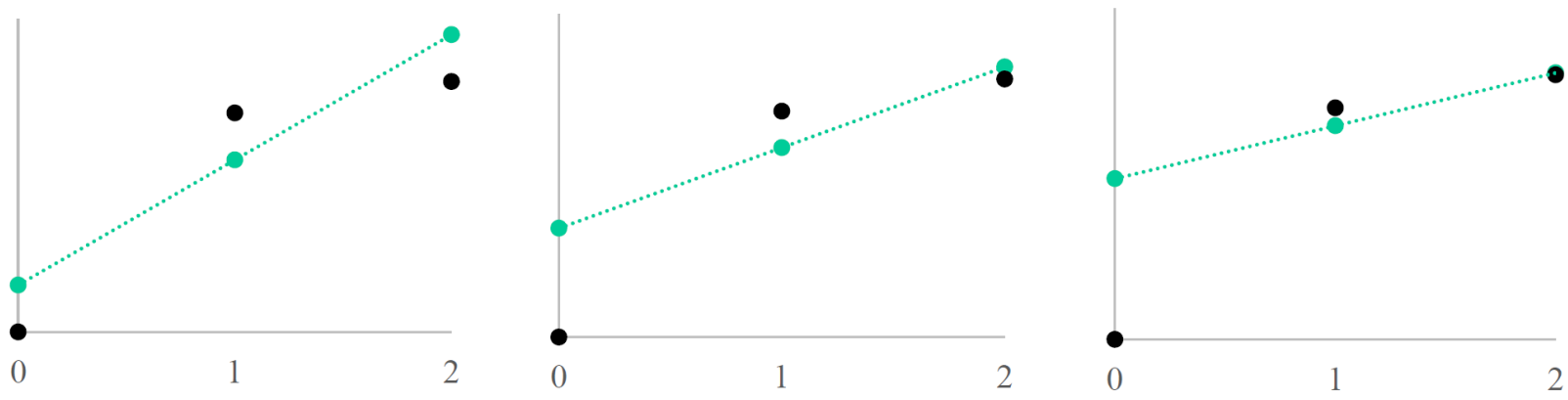

Figure shows how the BLP changes as we vary the frequency of the trait-increasing allele from 0.5 (left panel) to 0.75 (middle panel) to 0.95 (right panel). In all calculations, $a = 1$ and $d = 0.5$.

Additive and Dominance Variance

Common misconceptions:

- Increasing d reduces $V(A(\boldsymbol{x_i}))$.
- If d is large, $V(D(\boldsymbol{x_i}))$ must be large relative to $V(A(\boldsymbol{x_i}))$.

However:

You showed on the homework that under HWE:

$$V(A(\boldsymbol{x_i})) = 2pq\underbrace{[a+d(q-p)]^2}_{\beta^2}$$

$$V(D(\boldsymbol{x_i})) = (2pqd)^2$$

Easy to come up with examples that show the common intuitions are mistaken.

- For example, $\frac{\partial V(A(\boldsymbol{x_i}))}{\partial d} > 0$ whenever $(q-p) > 0$.

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Why Not to Care About Heritability

Fallacy. If the heritability of an outcome is high, there is little scope for policy to change it.

E.g. when Taubman (1976) found that income has a heritability of $\sim 40\%$, Hans Eysenck offered the following interpretation of the findings:

> *"[These results] really tell the [Royal] Commission [on the Distribution of Income and Wealth] that they might as well pack up."*

The Goldberger Critique

Goldberger's (1979) not-so-subtle retort:

> *"A powerful intellect was at work. In the same vein, if it were shown that a large proportion of the variance in eyesight were due to genetic causes, then the Royal Commission on the Distribution of Eyeglasses might as well pack up. And if it were shown that most of the variation in rainfall is due to natural causes, then the Royal Commission on the Distribution of Umbrellas could pack up too."*

The Jencks Critique

Genes may influence outcomes through mechanisms that are modifiable.

- By evoking environmental responses.
- By causing people to select into environments.

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The Jencks Critique: Toy Example

1. Educational attainment (EA, y_i) is determined by the number of books I read, b_i, and other environmental factors, η_i: $y_i = b_i\gamma + \eta_i$. Fraction of variance in EA due to environmental factors: 100%.

2. Suppose that number of books read is determined by additive genetic factors:

$$b_i = \boldsymbol{x_i}\beta + \varepsilon_i$$

with η_i and ε_i independent.

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The Jencks Critique: Toy Example (cont'd)

3. Then:

$$\begin{aligned} y_i &= b_i\gamma + \eta_i \\ &= \boldsymbol{x_i}\beta\gamma + (\gamma\varepsilon_i + \eta_i) \end{aligned}$$

and $h^2_A = \frac{\gamma^2 V(\boldsymbol{x_i}\beta)}{V(y_i)}$.

4. Fraction of variance in EA due to environmental plus genetic factors is therefore $100\% + h^2_A > 100$.

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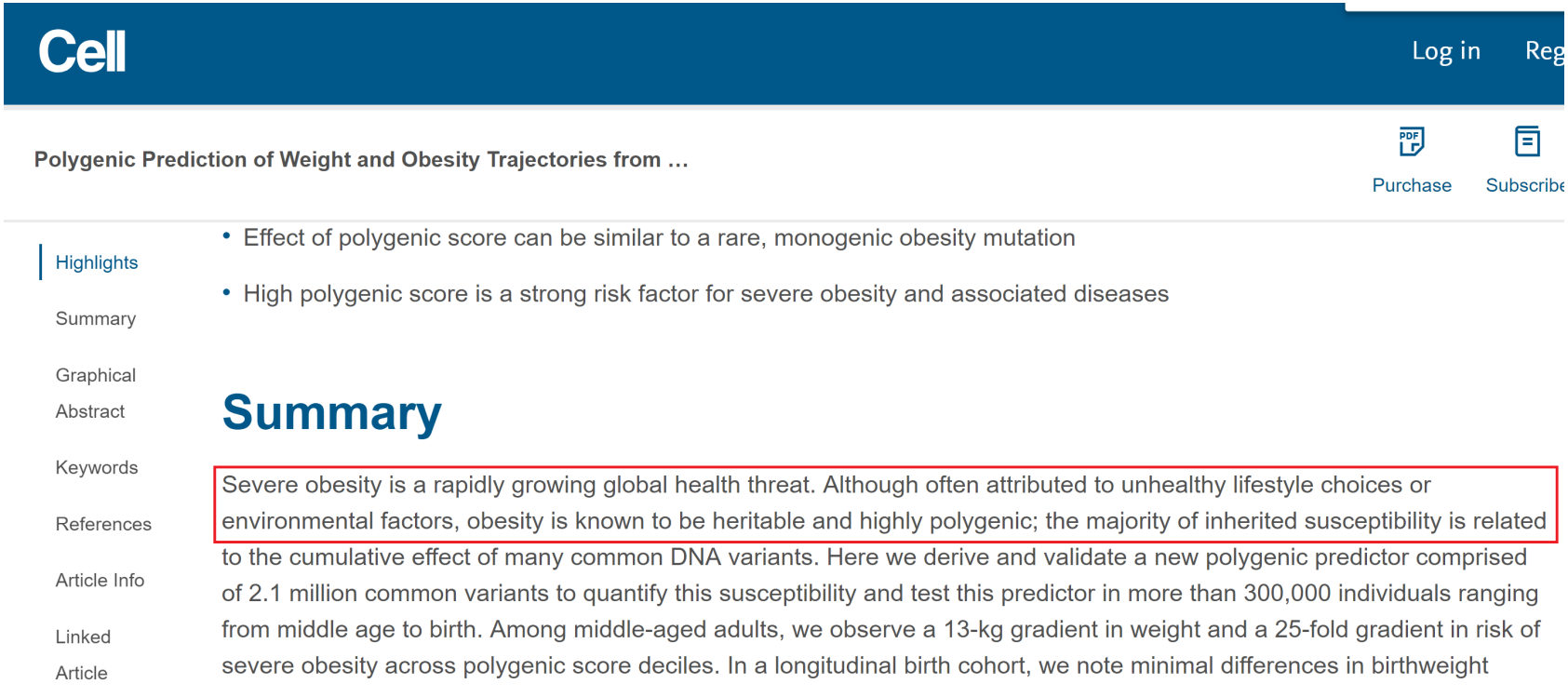
Jencks' Fallacy in Action (1)



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Polygenic Prediction of Weight and Obesity Trajectories from …

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Highlights

Summary

Graphical Abstract

Keywords

References

Article Info

Linked Article

- Effect of polygenic score can be similar to a rare, monogenic obesity mutation
- High polygenic score is a strong risk factor for severe obesity and associated diseases

Summary

Severe obesity is a rapidly growing global health threat. Although often attributed to unhealthy lifestyle choices or environmental factors, obesity is known to be heritable and highly polygenic; the majority of inherited susceptibility is related to the cumulative effect of many common DNA variants. Here we derive and validate a new polygenic predictor comprised of 2.1 million common variants to quantify this susceptibility and test this predictor in more than 300,000 individuals ranging from middle age to birth. Among middle-aged adults, we observe a 13-kg gradient in weight and a 25-fold gradient in risk of severe obesity across polygenic score deciles. In a longitudinal birth cohort, we note minimal differences in birthweight

The Jencks Critique: BMI

1. Suppose my BMI (BMI_i) is determined by the quality of my diet,d_i, and other lifestyle factors, η_i: $y_i = d_i\gamma + \eta_i$. Fraction of variance in BMI due to environmental factors: 100%.

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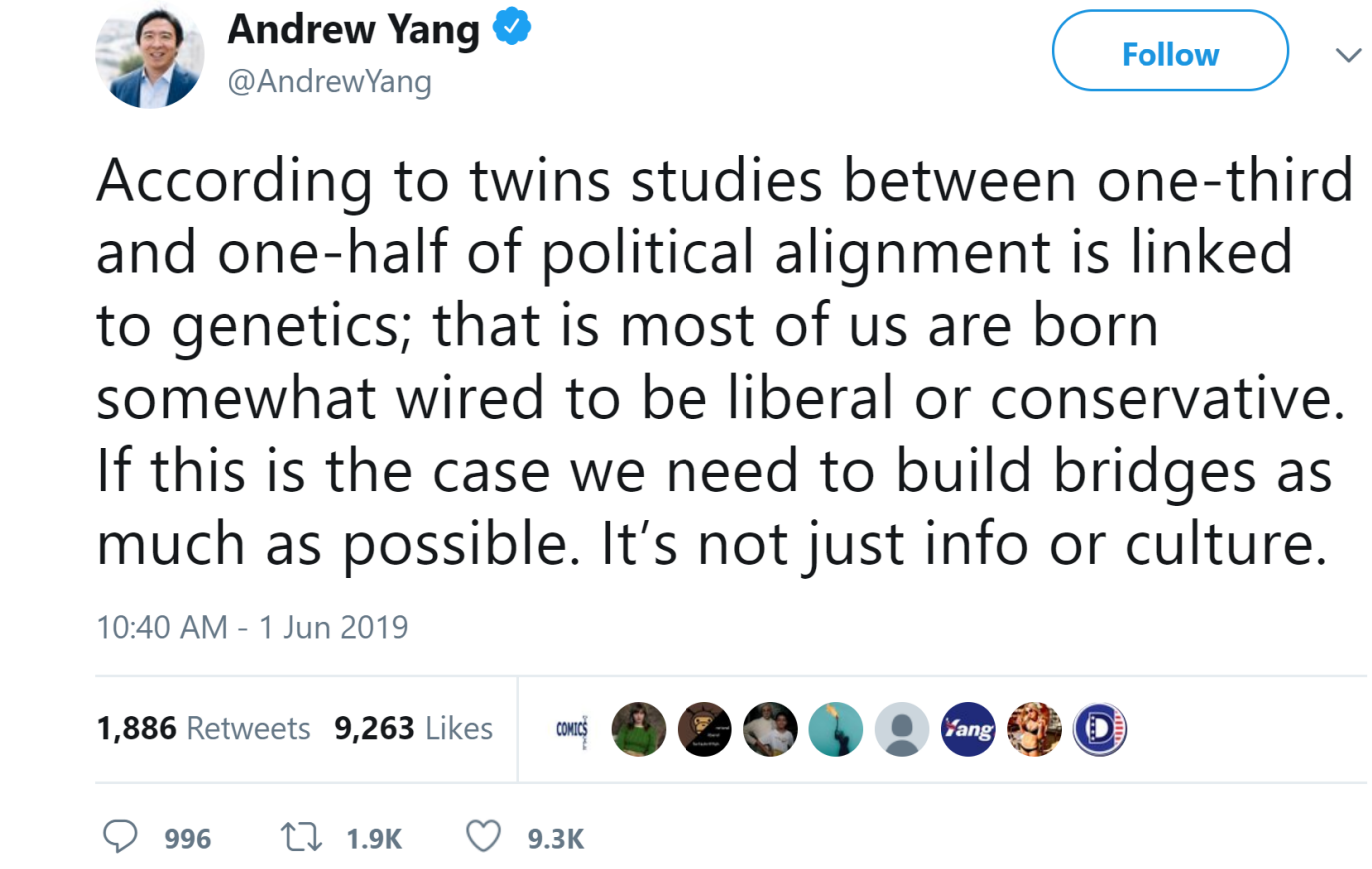

Why Care?

- Heritabilities of various traits, etc., are facts that may constrain the set of theories regarding heterogeneity that should be considered plausible.
- All else equal, higher heritabilities imply greater potential for genetic factors to confound estimates of environmental effects.
  - E.g., parental income on children's outcomes.
- Heritability quantifies how accurately one could, in principle, predict people's outcomes from their x_{ij}s.
  - Predictive power of polygenic scores based on genetic data is increasingly practical and feasible.
  - Provides guidance regarding which outcomes are more promising targets for gene discovery.

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- Jencks, C. (1980). "Heredity, Environment, and Public Policy Reconsidered." *American Sociological Review,* 45(5), 723-736.
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Key Readings for Tomorrow's Lecture (cont'd)

- Goldberger, AS. (2005). "Structural Equation Models in Human Behavior Genetics." Chapter 2 in Identication and Inference for Econometric Models, DW Andrews and JS Stock.. Cambridge: Cambridge University Press.
- Polderman *et al.* (2015). "Meta-analysis of the heritability of human traits based on fifty year of twin studies," *Nature Genetics* 47 (7), 702–709.
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Basic Approach

If we define $(Y_i, G_i, U_i) = (\frac{\tilde{y}_i}{\sqrt{V(\tilde{y}_i)}}, \frac{G(\boldsymbol{x_i})}{\sqrt{V(\tilde{y}_i)}}, \frac{\vartheta_i}{\sqrt{V(\tilde{y}_i)}})$ our causal model without covariates can be written as:

$$Y_i = G_i + U_i$$

This is a convenient parameterization because $h^2_G = V(G_i)$.

The problem: we only observe Y_i.

BG solution: try to infer h^2_G from phenotypic resemblance of pairs of relatives who differ in their environmental and genetic relatedness.

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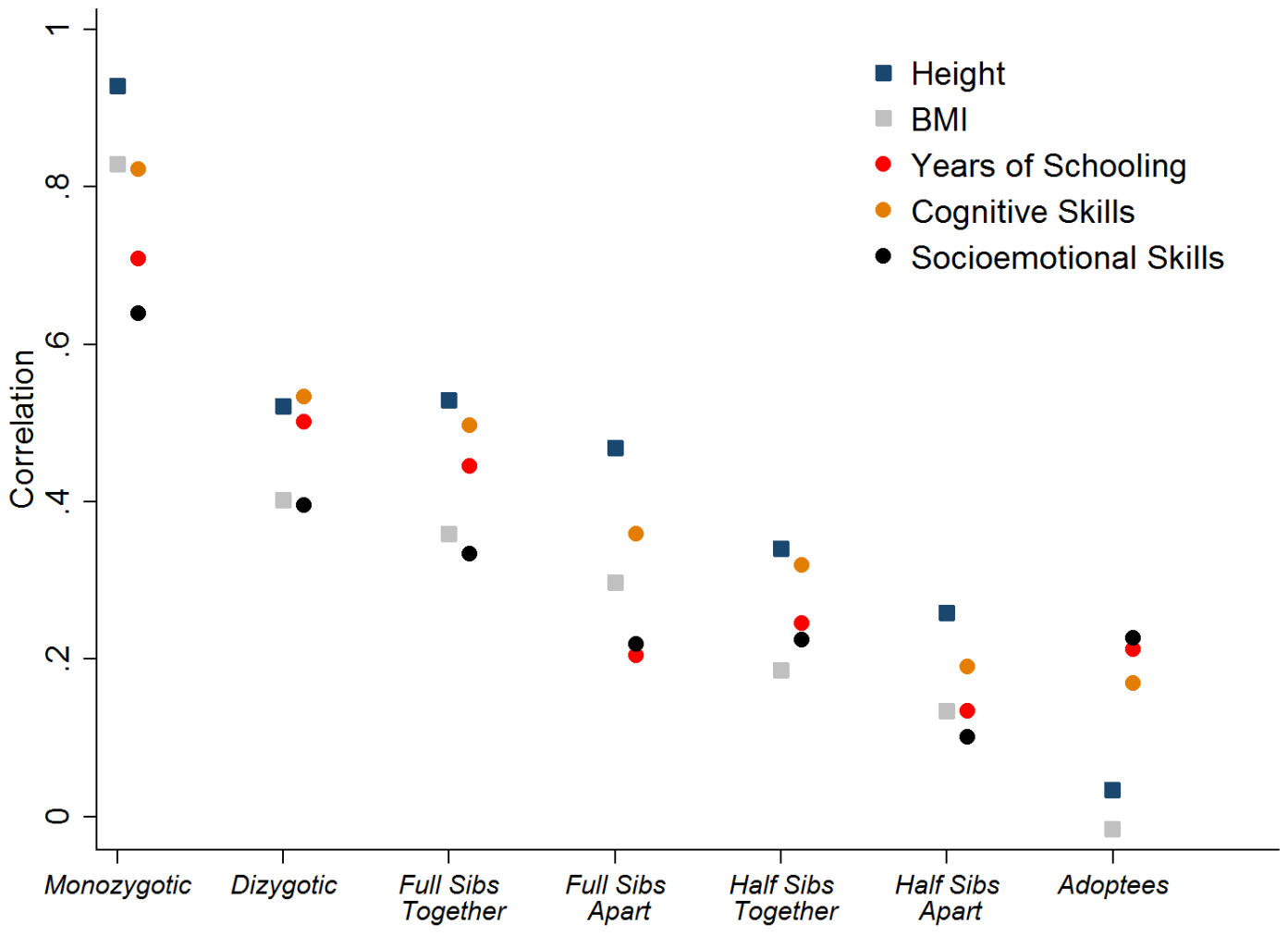
Sibling Correlations for Behavioral Traits



See Cesarini and Visscher (2017). This figure displays sibling correlations for five traits measured in a large sample of Swedish brother pairs born 1951–1970.

General Approach

Let unprimed and primed variables denote the two members of all pairs of relatives of a certain type, k. Then:

$$Cov_k(Y_i, Y_i') = Cov_k(G_i, G_i') + Cov_k(U_i, U_i') + 2Cov_k(G_i, U_i')$$

If relatives of type k are representative of population as a whole, we have:

$$\rho_Y^k = \rho_G^k h_G^2 + \rho_U^k u^2 + 2Cov_k(G_i, U_i')$$

The BG Approach:

Use genetic theory to justify choice of ρ_G^k (▸ Deriving Genetic Covariances).

Use information about whether a pair of individuals were reared together to justify restrictions on ρ_U^k and covariance term.

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MZ Twins Reared Apart (MZA)

Thought experiment: take a large representative sample of twins and randomly assign to environments at birth.

- Assuming pre-birth conditions similar, random assignment implies $Cov_{MZA}(G_i, U_i') = 0$ and $\rho_U^{MZA} = 0$.
- Since monozygotic, $\rho_G^{MZA} = 1$. Implied moment condition:

$$\rho_Y^{MZA} = h_G^2$$

- Given suitable data, sample analogue of ρ_Y^{MZA}, $\hat{\rho}_Y^{MZA}$, is a consistent estimator of h_G^2.
- Cannot identify h_A^2 unless we are prepared to make the strong assumption that $h_G^2 = h_A^2$.

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- Assuming pre-birth conditions similar, random assignment implies $Cov_{MZA}(G_i, U_i') = 0$ and $\rho_U^{MZA} = 0$.
- Since monozygotic, $\rho_G^{MZA} = 1$. Implied moment condition:

$$\rho_Y^{MZA} = h_G^2$$

- Given suitable data, sample analogue of ρ_Y^{MZA}, $\hat{\rho}_Y^{MZA}$, is a consistent estimator of h_G^2.
- Cannot identify h_A^2 unless we are prepared to make the strong assumption that $h_G^2 = h_A^2$.

Full or Half Sibs Reared Apart (FSA, HSA)

Same thought experiment, except with full or half sibs this time.

- Due to random assignment, set $\rho_U^{FSA} = \rho_U^{HSA} = 0$ and covariance term to zero.
- ρ_G^{FSA} and ρ_G^{HSA} harder to pin down without strong assumptions. If HWE holds and $h_G^2 = h_A^2 + h_D^2$:

$$\rho_Y^{FSA} = \frac{1}{2} h_A^2 + \frac{1}{4} h_D^2$$

- Hence, $\hat{\rho}_Y^{FSA}$ is an estimator of $\frac{1}{2} h_A^2 + \frac{1}{4} h_D^2 \rightarrow$ cannot identify h_A^2 from $\hat{\rho}_Y^{FSA}$ alone.
- For half sibs:

$$\rho_Y^{HSA} = \frac{1}{4} h_A^2$$

$\rightarrow$Half sibs can potentially help with identification.

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Adoptees (ADO)

Thought experiment: randomly assign a representative sample of children to households that are representative of the full population.

- Under random assignment, we expect $Cov_{ADO}(G_i, U_i') = 0$ and $\rho_G^{ADO} = 0$. Consequently:

$$\rho_Y^{ADO} = \rho_U^{ADO} u^2$$

  and $\hat{\rho}_Y^{ADO}$ is an estimator of $\rho_U^{ADO} u^2$.

- As we shall see, $\rho_U^{ADO} u^2$ interpretable as the proportion of variance attributable to nongenetic factors shared by siblings reared together.

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Multiple Sibling Types

Jointly analyzing data on multiple kinships is often a useful way to probe robustness. Each kinship provides a moment condition that

$$\hat{\theta} = \text{argmin} \sum_k w_k (\hat{\rho}_Y^k - \rho_Y^k(\theta))^2$$

where w_k is the inverse variance of the sample correlation for sibling pairs of type k.

Illustration

Example: For $\theta = \left(h_A^2, h_D^2, \rho_U^{ADO} u^2\right)$, consider the moment conditions:

$$\underbrace{\begin{bmatrix} \rho_Y^{MZA}(\theta) \\ \rho_Y^{FSA}(\theta) \\ \rho_Y^{HSA}(\theta) \\ \rho_Y^{ADO}(\theta) \end{bmatrix}}_{\rho} = \underbrace{\begin{bmatrix} 1 & 1 & 0 \\ 1/2 & 1/4 & 0 \\ 1/4 & 0 & 0 \\ 0 & 0 & 1 \end{bmatrix}}_{\boldsymbol{X} = [\boldsymbol{x_1}, \boldsymbol{x_2}, \boldsymbol{x_3}]} \times \theta' \tag{1}$$

With four independent moment conditions and three parameters, model is (over)identified. Can then estimate θ by a weighted least-square regression of $\hat{\rho}$ on $\boldsymbol{x_1}, \boldsymbol{x_2}$ and $\boldsymbol{x_3}$ (▸ Technical Details).

Twin Studies

- Without random assignment to environments, genetic similarity is generally confounded by environmental similarity.
- Basic idea: MZ twins and DZ twins differ in genetic similarity but are both raised in the same family, so we can difference out factors that siblings share.

Assumptions

1. All genetic variance is additive, so $h^2_A = h^2_G$.
2. No assortative mating at genetic level, so $\rho_G^{DZ} = 1/2$.
3. No gene-environment correlation: $Cov_k(G_i, U_i') = 0$ for $k = MZ, DZ$.
4. Equal environments: $\rho_U^{MZ} = \rho_U^{DZ} \equiv \rho_U$.

What do these assumptions mean? Are they plausible?

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Moment Conditions

The moment conditions are:

$$\begin{bmatrix} \rho_Y^{MZ}(h_A^2, \rho_U u^2) \\ \rho_Y^{DZ}(h_A^2, \rho_U, u^2) \end{bmatrix} = \begin{bmatrix} 1 & 1 \\ 1/2 & 1 \end{bmatrix} \times \begin{bmatrix} h_A^2 \\ \rho_U u^2 \end{bmatrix}$$

and the sample analogoue of the moment condition $2 \times (\rho_Y^{MZ} - \rho_Y^{DZ}) = h_A^2$ will be familiar to many of you.

ACE Parameterization

Usually these equations are presented after a re-parameterization.

- Additive genetic component: $h_A^2 \equiv a^2$
- Common environmental component: $\rho_U u^2 = c^2$
- Unshared environment: $e^2 = 1 - h_A^2 - \rho_U u^2$.

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Canonical Results

Swedish Sibling Results

	Twins & Adoptees			Full Sibs		Half Sibs	
	MZ	DZ	ADO	Same	Apart	Same	Apart
Height	0.928	0.521	0.034	0.529	0.468	0.341	0.259
Edu	0.709	0.502	0.213	0.445	0.205	0.246	0.134
BMI	0.829	0.402	-0.016	0.359	0.298	0.186	0.134
Cog Skill	0.822	0.534	0.170	0.497	0.359	0.320	0.191
Noncog	0.640	0.396	0.227	0.334	0.219	0.225	0.101
N_{min}	1,154	1,601	643	151,789	1,033	4,088	11,566

Sibling correlations for five traits measured in a large sample of Swedish brother pairs born 1951–1970 (See Cesarini and Visscher (2017) for further details). N_{min} is the smallest number of sibling pairs used to calculate a correlation in the column.

Stylized Fact #1

Similarity of Siblings Raised Together Increasing in Relatedness

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Turkheimer's Three Laws of BG

1. All human behavioral traits are heritable.
2. The effect of being raised in the same family is smaller than the effect of genes.
3. A substantial portion of the variation in complex human behavioral traits is not accounted for by genes or families.

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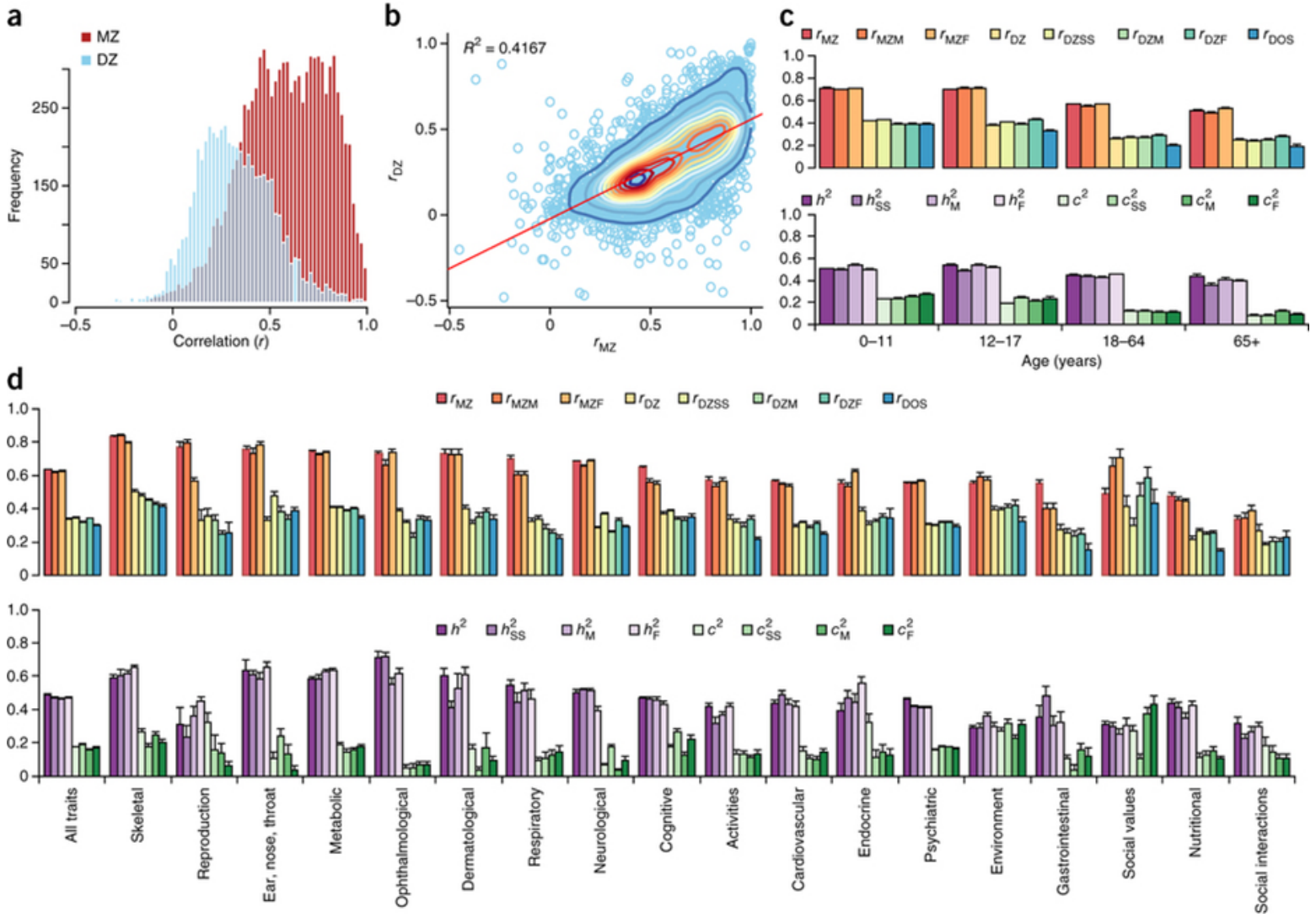
Polderman (2015)



Adoption Studies

- Sacerdote (2007) analyzes survey data on and Korean-born American adoptees, their adoptive parents and any biological children of the adoptive parents.
- Typically, adoption studies need to assume random assignment, even though unlikely.
- Unusual features of these data:
  - Adoptee assignment plausibly random.
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Analyses

1. Perform ACE decompositions based on comparisons of biological and adoptee sibling correlations.
2. Also estimates parent-child transmission coefficients, separately for biological and adopted children.
3. Estimates treatment effect of being assigned to a particular family type:
   - Type 1: small families, both parents completed college (27%)
   - Type 3: large families, neither parent completed college (12%)
   - Type 2: neither type 1 or 3.

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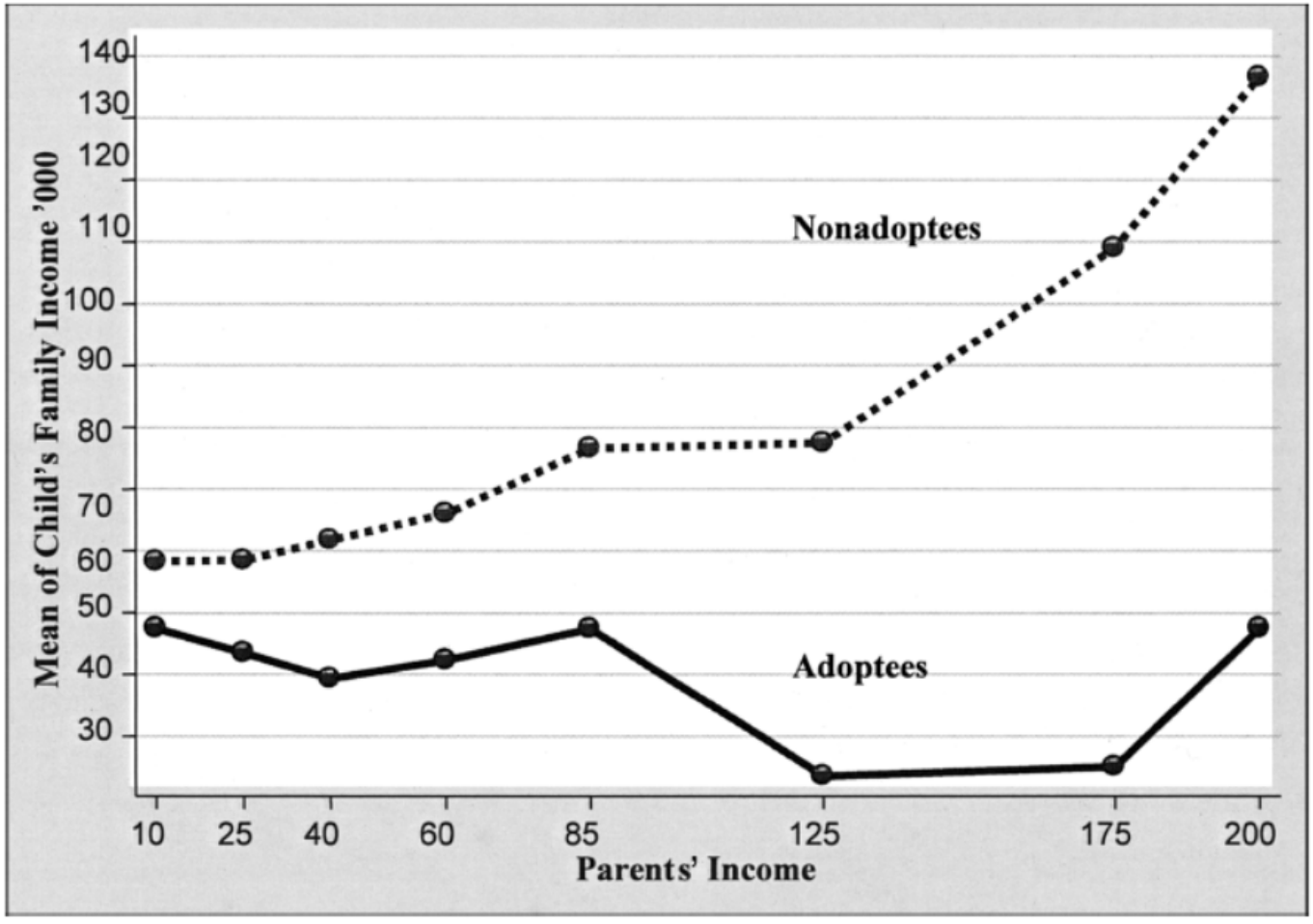
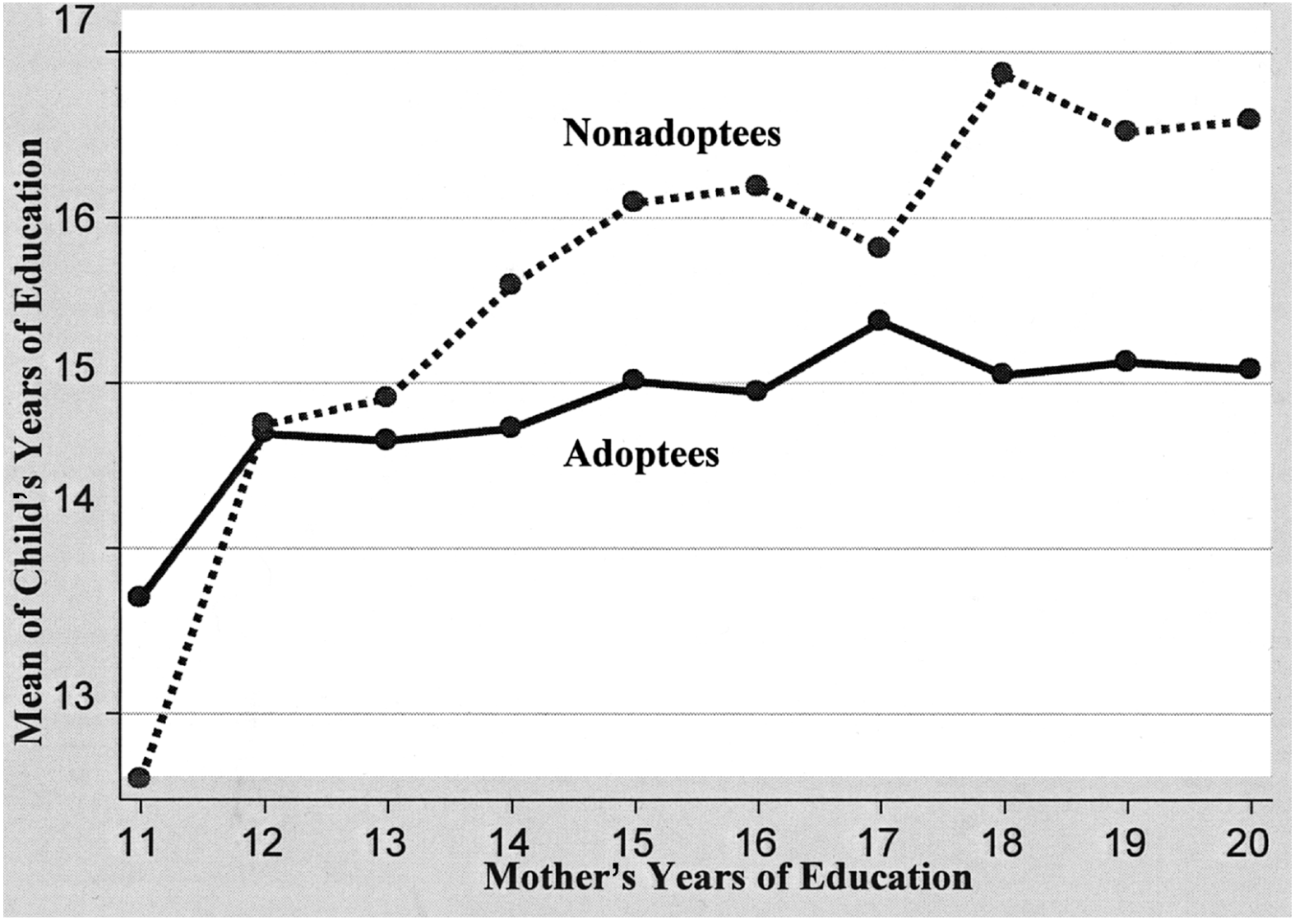
ACE Decomposition

Outcome	Adoptive sibling correlation	Biological sibling correlation	*N* Adoptive	*N* Biological
Has 4 years of college	0.135	0.338	1360	578
Highest grade completed	0.157	0.378	1360	578
Family income	0.110	0.277	1314	554
Log (family income)	0.139	0.301	1314	554
Drinks	0.336	0.363	1903	640
Smokes	0.152	0.289	1938	654
Height	0.014	0.443	1910	646
Weight	0.044	0.273	1822	629
BMI	0.115	0.269	1821	629
Overweight	0.087	0.173	1821	629
Attended US News ranked school	0.249	0.416	1360	578
Acceptance rate of school	0.337	0.460	560	245
Married	0.076	0.048	1917	650
Number of children	0.105	0.203	1802	633

Income Transmission



EduYears Transmission



Treatment Effect Estimates

TABLE VII
TREATMENT EFFECTS FROM ASSIGNMENT TO HIGH EDUCATION, SMALL FAMILY

	Treatment effect "middle group" of families vs. large, less educated	Treatment effect high education small family vs. large, less educated	Nonadoptees: High education small family vs. large, less educated	Effect from a 1 standard deviation change in family environment index
Child's years of education	0.314 (0.226)	0.749 (0.245)**	2.157 (0.264)**	0.845
Child has 4+ years college	0.060 (0.056)	0.161 (0.057)**	0.317 (0.031)**	0.179
Log child's household income	0.071 (0.081)	0.113 (0.089)	0.210 (0.089)*	0.263
Child four-year college ranked by US News	0.082 (0.052)	0.231 (0.060)**	0.365 (0.052)**	0.224
Acceptance rate of child's college	−0.007 (0.035)	0.016 (0.036)	−0.053 (0.032)	0.098
Child drinks (yes/no)	0.099 (0.050)*	0.178 (0.049)**	0.229 (0.041)**	0.280
Child smokes (yes/no)	0.013 (0.044)	−0.006 (0.048)	−0.075 (0.024)**	0.162
Child's BMI	−0.509 (0.460)	−0.941 (0.468)*	−0.929 (0.498)	1.224
Child overweight	−0.030 (0.047)	−0.077 (0.045)	−0.088 (0.048)	0.121
Child obese	−0.020 (0.023)	−0.044 (0.018)*	−0.037 (0.018)*	0.047
Child has asthma	−0.005 (0.028)	0.013 (0.031)	−0.005 (0.034)	0.085
Number of children	−0.070 (0.099)	−0.199 (0.103)*	−0.580 (0.132)**	0.267
Child is married	0.014 (0.050)	0.000 (0.056)	−0.092 (0.053)	0.123

Outline

Estimating Behavior Genetic Models

- BG models often estimated by maximum likelihood using the software OpenMX:
  - an excellent and versatile resource that can estimate a wide range of user-specified models.
  - accomodates a range of fitting procedures, including full-information MLE, that can be applied to individual-level data.
  - easily implements standard procedures for estimation and hypothesis testing, allows users to specify non-linear constraints (e.g. non-negativity constraints for variance components) and request fit diagnostics.
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Estimating Behavior Genetic Models (cont'd)

- We will simplify many aspects of the procedures to highlight some key points about identification.
- At the end of the day, all methods amount to little more than inferring parameters by comparing empirical moments to theoretical moment conditions that are (hopefully!) derived from some set of structural equations.
- The core structural equations of a model are important!
  - Embed important assumptions about biological, psychological, sociological and economic processes.
  - Transparency about the structural assumptions forces clear thinking and can also be helpful for attempts to quantify any biases introduced by violations.
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Preliminary Issue: Fisher's Z Transformations

Define

$$z^k = \frac{1}{2} \ln \left[\left(\frac{1+\hat{\rho}^k}{1-\hat{\rho}^k} \right) \right]$$

$$\eta^k = \frac{1}{2} \ln \left[\left(\frac{1+\rho^k}{1-\rho^k} \right) \right]$$

Given a sample of n^k pairs drawn independently and randomly from a bivariate normal population, z^k is approximately $\mathcal{N}(\eta^k, 1/n^k)$.

Since n^k is the inverse variance of the sample correlation, our estimator $\hat{\theta}$ solves the non-linear least squares problem:

$$\hat{\theta} = \text{argmin} \sum_k n^k \times (z^k - \eta^k)^2.$$

We will illustrate the approach using some of the previously discussed Swedish brother correlations for BMI.

Example: Full- and Half Sibs

The z-transforms for $k = \{FST, FSA, HST, HSA\}$ are:

$$\begin{bmatrix} z^{FST} \\ z^{FSA} \\ z^{HST} \\ z^{HSA} \end{bmatrix} = \begin{bmatrix} \frac{1}{2}\ln(1+\underbrace{0.359}_{=\hat{\rho}^{FST}}/1-\underbrace{0.359}_{=\hat{\rho}^{FST}}) \\ \frac{1}{2}\ln(1+\underbrace{0.298}_{=\hat{\rho}^{FSA}}/1-\underbrace{0.298}_{=\hat{\rho}^{FSA}}) \\ \frac{1}{2}\ln(1+\underbrace{0.186}_{=\hat{\rho}^{HST}}/1-\underbrace{0.186}_{=\hat{\rho}^{HST}}) \\ \frac{1}{2}\ln(1+\underbrace{0.134}_{=\hat{\rho}^{HSA}})/1-\underbrace{0.134}_{=\hat{\rho}^{HSA}})) \end{bmatrix} = \begin{bmatrix} 0.376 \\ 0.307 \\ 0.188 \\ 0.135 \end{bmatrix}$$

Example: Full- and Half Sibs

If we assume an ACDE model, $\hat{\theta} = \left(\hat{h}_A^2, \hat{h}_D^2, \hat{c}^2\right)$ is the argmin of

$$\underbrace{151,789}_{=n^{FST}} \times \left[\underbrace{0.376}_{=z^{FST}} - \underbrace{\frac{1}{2} \ln\left(\frac{1 + \overbrace{\left(\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2 + c^2\right)}^{\rho^{FST}(\theta)}}{1 - \left(\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2 + c^2\right)}\right)}_{=\eta^{FST}} \right]^2$$

$$+1,033 \times \left[0.307 - \frac{1}{2} \ln\left(\frac{1 + \left(\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2\right)}{1 - \left(\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2\right)}\right) \right]^2$$

$$+4,088 \times \left[0.188 - \frac{1}{2} \ln\left(\frac{1 + \left(\frac{1}{4}h_A^2 + c^2\right)}{1 - \left(\frac{1}{4}h_A^2 + c^2\right)}\right) \right]^2$$

$$+11,566 \times \left[0.135 - \frac{1}{2} \ln\left(\frac{\frac{1}{4}h_A^2}{\frac{1}{4}h_A^2}\right) \right]^2$$

Toy Code for Example

R Console

```
> #Specify Moments
>
> aval <- c(0.5,0.5,0.25,0.25)
> cval <- c(1,0,1,0)
> dval <- c(0.25,0.25,0,0)
> Moments = cbind(aval, cval, dval)
> N <- c(151789,1033,4088,11566)
>
> BMI  <- sapply(BMI <- c(0.359,0.298,0.186,0.134), function(x) 0.5*log((1+x)/(1-x)))
> BMI
[1] 0.3757375 0.3073232 0.1881906 0.1348108
>
> # Run Analyses
>
> BMInls <- nls(BMI ~ 0.5*log((1+(a*aval+c*cval+d*dval))/(1-(a*aval+c*cval+d*dval))), weights = N, start = list(a = 0.5,c = 0.1,d = 0.1))
> BMI_est <- coef(BMInls)
>
> # Display Estimates
>
> BMI_est
         a          c          d
0.53330652 0.05451202 0.15117743
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```

The solution is: $\hat{\theta} = (\hat{\sigma}_A^2, \hat{\sigma}_D^2, c^2) = (0.533, 0.151, 0.054)$.

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Predicted and Actual Correlations: 4-Kinship Estimator

	Moment	$\rho^k(\hat{\theta})$	$\hat{\rho}^k$	$\triangle\rho$	χ^2_k
Full Sibs Same	$\frac{1}{2}h^2_A + \frac{1}{4}h^2_D + c^2$	0.358			
Full Sibs Apart	$\frac{1}{2}h^2_A + \frac{1}{4}h^2_D$	0.304			
Half Sibs Same	$\frac{1}{4}h^2_A + c^2$	0.187			
Half Sibs Apart	$\frac{1}{4}h^2_A + c^2$	0.133			

For each kinship, can calculate the correlation predicted by the model given $\hat{\theta} = (\hat{\sigma}^2_A, \hat{\sigma}^2_D, c^2) = (0.533, 0.151, 0.054)$ and the moment condition.

In-Sample Comparison: 4-Kinship Estimator

	Moment	$\rho^k(\hat{\theta})$	$\hat{\rho}^k$	$\triangle\rho$	χ^2_k
Full Sibs Same	$\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2 + c^2$	0.358	0.359	-0.001	0.112
Full Sibs Apart	$\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2$	0.304	0.298	0.006	0.049
Half Sibs Same	$\frac{1}{4}h_A^2 + c^2$	0.187	0.186	0.001	0.007
Half Sibs Apart	$\frac{1}{4}h_A^2 + c^2$	0.133	0.134	-0.001	0.007

Predicted and actual correlations are close, so in-sample fit is excellent, as shown by the red-colored column $\triangle\rho$.

Out-of-Sample Comparison: 4-Kinship Estimator

	Moment	$\rho^k(\hat{\theta})$	$\hat{\rho}^k$	$\triangle\rho$	χ^2_k
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Half Sibs Same	$\frac{1}{4}h_A^2 + c^2$	0.187	0.186	0.001	0.007
Half Sibs Apart	$\frac{1}{4}h_A^2 + c^2$	0.133	0.134	-0.001	0.007
MZ	$h_A^2 + h_D^2 + c^2$	0.738	0.829	-0.091	65.839
DZ	$\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2 + c^2$	0.358	0.402	-0.044	4.190
Adoptees	c^2	0.054	-0.016	0.070	3.156

A more stringent test of the model specification is its out-of-sample fit, which is considerably less impressive.

Key Points

- Adequate in-sample fit does not mean that your estimates are likely to have little bias or that your model is well-specified.
  - If model fit remains "adequate" even after you drop a parameter, that does not mean it is a good idea to drop the parameter!
- Usually a good idea to include as many kinships as possible:
  - Provides more leverage to test and relax, where appropriate, potentially problematic assumptions.
  - Such analyses often yield insights that are relevant for interpreting previously published estimates. They can also suggest new avenues for resarch.
- Consider what happens when we reestimate the ACDE parameters for BMI with all seven kinships included, rather than just the full- and half-sibs.

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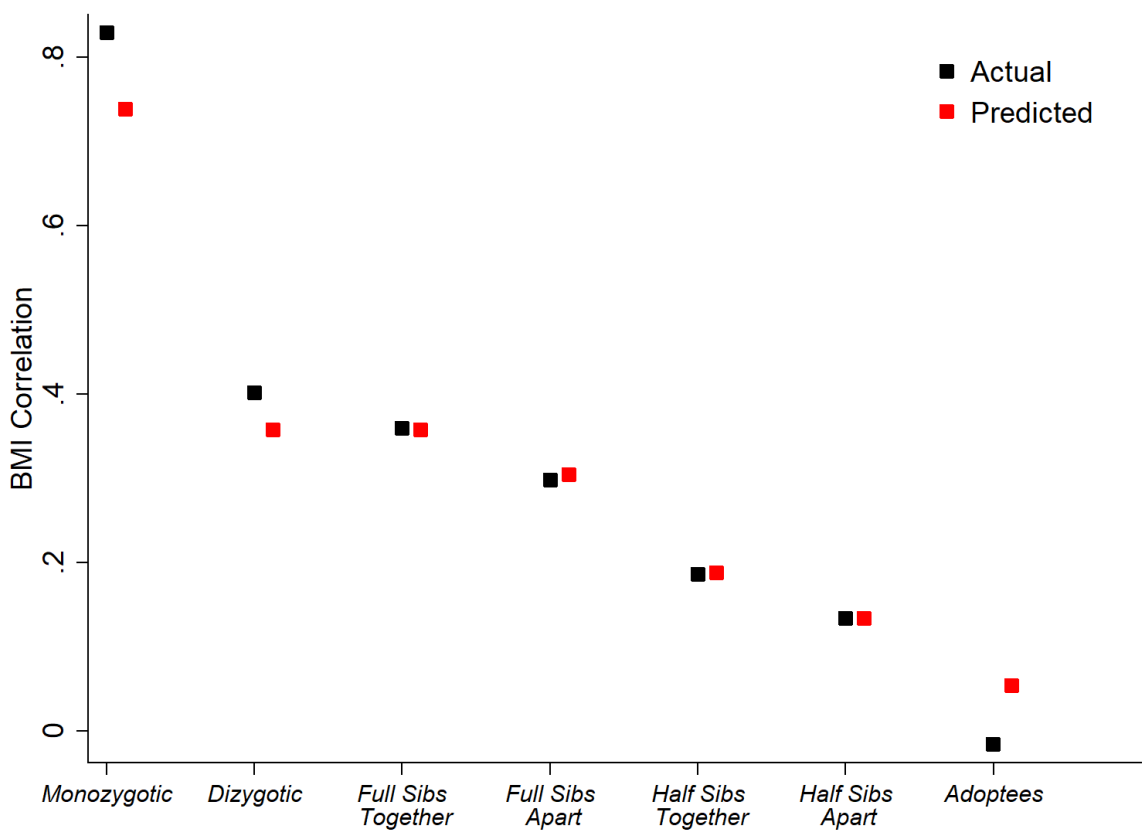
Predicted and Actual Correlations: 7-Kinship Estimator

The estimates are now $\left(\hat{h}_A^2, \hat{h}_D^2, \hat{c}^2\right) = (0.520, 0.274, 0.032)$.

	Moment	$\rho^k(\hat{\theta})$	$\hat{\rho}^k$	$\triangle\rho$	χ_k^2
Full Sibs Same	$\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2 + c^2$	0.358	0.359	-0.001	0.451
Full Sibs Apart	$\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2$	0.304	0.298	0.006	1.182
Half Sibs Same	$\frac{1}{4}h_A^2 + c^2$	0.187	0.186	0.001	2.504
Half Sibs Apart	$\frac{1}{4}h_A^2 + c^2$	0.133	0.134	-0.001	0.192
MZ	$h_A^2 + h_D^2 + c^2$	0.738	0.829	-0.091	0.105
DZ	$\frac{1}{2}h_A^2 + \frac{1}{4}h_D^2 + c^2$	0.358	0.402	-0.044	3.777
Adoptees	c^2	0.054	-0.016	0.070	1.482

Actual vs Predicted Correlations: 7-Kinship Estimator



The minimized criterion evaluated at the optimum is 9.66 and a standard χ^2 test rejects the ACDE model specification. The misspecification is similar in magnitude if unweighted estimates $(0.543, 0.250, 0.034)$ are used instead of the WLS estimates $(0.520, 0.274, 0.032)$ to calculate predicted correlations.

Outline

Genetic Covariance for General k

Definition: if two individuals share an allele that can be traced to a copy inherited from a recent common ancestor, the shared allele is said to be identical by descent (IBD). For example:

- A mother-child (or father-child) pair share exactly 1 one allele IBD at each locus.
- Full siblings share zero alleles IBD with probability 0.25, one with probability 0.5 and two with probability 0.25.

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General Formula

If $G(x_i) = A(x_i) + D(x_i)$ and a population is in HWE, the genetic covariance between a pair of relatives of type k is:

$$\begin{aligned} Cov_k(G_i, G_i') &= Cov_k(A_i, A_i') + Cov_k(D_i, D_i') \\ &= \rho_A^k \sigma_A^2 + \rho_D^k \sigma_D^2 \end{aligned}$$

where

$\rho_A^k = 1/2 \times \Pr((1 \text{ allele IBD}) \mid k) + \Pr((2 \text{ alleles IBD}) \mid k)$ and

$\rho_D^k = \Pr((2 \text{ alleles IBD}) \mid k)$.

Rather than derive ρ_A^k and ρ_D^k from first principles, often easier to do IBD calculation and calculate (ρ_A^k, ρ_D^k).

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Example 1: Half Siblings

A half sib (HS) gets exactly one allele from common parent, different alleles from the other parent. The allele from the common parent is IBD with probability 0.5. Therefore:

$$\Pr(\text{IBD} = j \mid \text{HS}) = \begin{cases} 1/2 & \text{if } j = 0 \\ 1/2 & \text{if } j = 1 \\ 0 & \text{if } j = 2. \end{cases}$$

Hence, $\rho_A^{HS} = 0.5 \times \Pr(\text{IBD} = 1 \mid \text{HS}) + \Pr(\text{IBD} = 2 \mid \text{HS}) = 1/4$ and $\rho_D^{HS} = \Pr(\text{IBD} = 2 \mid \text{HS}) = 0$.

Hence, $(\rho_A^{HS}, \rho_D^{HS}) = (1/4, 0)$.

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Hence, $\left(\rho_A^{HS}, \rho_D^{HS}\right) = (1/4, 0)$.

Example 2: Full Siblings

Each full sib (FS) gets exactly one allele, selected at random, from each parent. The maternally transmitted allele is shared (IBD) with probability 0.5, as is the paternally transmitted allele. The two events are independent, so:

$$\Pr(\text{IBD} = j \mid \text{FS}) = \begin{cases} 1/4 & \text{if } j = 0 \\ 1/2 & \text{if } j = 1 \\ 1/4 & \text{if } j = 2. \end{cases}$$

Hence, $\rho_A^{FS} = 0.5 \times \Pr(\text{IBD} = 1 \mid \text{FS}) + \Pr(\text{IBD} = 2 \mid \text{FS}) = 1/2$ and $\rho_D^{FS} = \Pr(\text{IBD} = 2 \mid \text{FS}) = 1/4$.

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Hence, $(\rho_A^{FS}, \rho_D^{FS}) = (1/2, 1/4)$.

Genetic Covariances: Random Mating

	$\rho_k(A_i, A_i')$	$\rho_k(D_i, D_i')$	$\rho_k(G_i, G_i')$
MZ Twin	1	1	1
Parent-Child	1	0	$\sigma_A^2/2\sigma_G^2$
Full Sibs	1/2	1/4	$\sigma_A^2/2\sigma_G^2 + \sigma_D^2/4\sigma_G^2$
Half Sibs	1/4	0	$\sigma_A^2/2\sigma_G^2$
First Cousins	1/8	0	$\sigma_A^2/8\sigma_G^2$

Table shows genetic correlations for some common relative types. Formulae derived assuming a randomly mating, non-inbred, population.

Fisher's (1918) Model of Phenotypic Assortment

Fisher derived genetic covariances under assortative mating. Our sketch of his framework follows Crow and Kimura (1970).

- In each generation $t = 0, 1, 2, \ldots$ phenotypes are determined as the sum of three independent components: $Y_t = A_t + D_t + U_t$.
- By independence, $\sigma^2_{t,Y} = \sigma^2_{t,A} + \sigma^2_{t,D} + \sigma^2_{t,U}$.
- Consider a (hypothetical) $t = 0$ "base" population with random mating. Then, at $t = 1$, assortative mating begins.
- In all subsequent generations, there is phenotypic assortative mating such that the phenotypic mate correlation is always r_y.
- For $t = 1, 2, \ldots$ assume $\sigma^2_{t,U} \approx \sigma^2_{0,U} \equiv \sigma^2_U$ and $\sigma^2_{t,D} \approx \sigma^2_{0,D} \equiv \sigma^2_D$.

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Fisher's (1918) Model of Phenotypic Assortment

Fisher derived genetic covariances under assortative mating. Our sketch of his framework follows Crow and Kimura (1970).

- In each generation $t = 0, 1, 2, \ldots$ phenotypes are determined as the sum of three independent components: $Y_t = A_t + D_t + U_t$.
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Fisher's (1918) Model of Phenotypic Assortment (cont'd)

- After one generation of assortative mating[1]:

$$\sigma^2_{1,Y} \approx \sigma^2_{t=1,A}[1 + r_{a,1}/2] + \sigma^2_D + \sigma^2_U$$

  where $r_{a,1}$ is the $t = 1$ correlation between the additive genetic factors of mates induced by r_y.

- Keep iterating...
- Solve for genetic equilibrium by calculating limit of variance parameters as $t \to \infty$.
- Label equilibrium parameters with tildes, e.g. $\lim_{t\to\infty}\left\{\sigma^2_{t,A}\right\} = \tilde{\sigma}^2_A$ and standardize so that $\tilde{\sigma}^2_A + \tilde{\sigma}^2_D + \tilde{\sigma}^2_U = \tilde{\sigma}^2_Y = 1$.

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Often possible to solve for equilibrium parameters as functions of the parameters of the base population. For example, if the number of loci is large, the equilibrium variance is:

$$\tilde{\sigma}_A^2 \approx \frac{\left(\sigma_{0,A}^2/\sigma_{0,Y}^2\right)}{1 - r_y\left(\sigma_{0,A}^2/\sigma_{0,Y}^2\right)}$$

1. Can calculate $\tilde{\sigma}_A^2$ from the phenotypic mate correlation and $\sigma_{0,A}^2/\sigma_{0,Y}^2$, heritability in the randomly mating base population.
2. Positive (negative) assortment increases (depress) the genetic variance, $\tilde{\sigma}_A^2$.
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Correlations Between Relatives under Assortative Mating

	Random ($r_y = 0$)	Assortative ($r_y \neq 0$)
	$\rho_k(Y_i, Y_i')$	$\rho_k(\tilde{Y}_i, \tilde{Y}_i')$
MZ Twin	$\left(\sigma^2_{0,A} + \sigma^2_{0,D}\right)/\sigma^2_{0,Y}$	$(\tilde{\sigma}^2_A + \tilde{\sigma}^2_D)$
Parent-Child	$\sigma^2_{0,A}/2\sigma^2_{0,Y}$	$\tilde{\sigma}^2_A(1+r_y)/2$
Full Sib	$\sigma^2_{0,A}/2\sigma^2_{0,Y} + \sigma^2_{0,D}/4\sigma^2_{0,Y}$	$\tilde{\sigma}^2_A(1+r_y)/2 + \tilde{\sigma}^2_D/4$
Half Sib	$\sigma^2_{0,A}/2\sigma^2_{0,Y}$	$\tilde{\sigma}^2_A(1+\tilde{\sigma}^2_A r_y)(1+r_y)/4$
First Cousin	$\sigma^2_{0,A}/8\sigma^2_{0,Y}$	$\tilde{\sigma}^2_A(1+\tilde{\sigma}^2_A r_y)^3/4 + \tilde{\sigma}^2_D r_y^2 \tilde{\sigma}^4_A/16$

r_y is the spousal correlation. Parameters *with* tildes are standardized variances components in a population at equilibrium under assortative mating (e.g. $\tilde{\sigma}^2_A = V\left(\tilde{A}\right)/V\left(\tilde{Y}\right)$), whereas those *without* hats are random-mating variances.

▸ Return to Main Presentation

Some Problems

With one exception, the model rules out correlation between the environments of relatives. This feature

- seems at odds both with common sense and *Stylized Fact 2.*
- limits the scope for credible applications of the model!
- the only exception allowed is for...spouses?!

Goldberger on (1978, p.10) on what this means:

> *"my wife's childhood environment was similar to mine, but not to her brother's, nor indeed to our children's. This makes the model not only implausible but empirically inadequate..."*

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Outline

Sibling Interactions: Motivation

- Sibs close in age spend considerable time together and may be expected to influence each other's developmental trajectories.
- Here, we illustrate how one might go about exploring implications of such sibling interactions more formally.
- Our treatment follows Carey (1986).

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Sibling Interactions: Setup

- Let Y_t and Y'_t stand for the phenotypes of two siblings measured in period $t = -1, 0, 1, 2, ...$
- Define $\triangle Y_t = Y_t - Y_{t-1}$ and $\triangle Y'_t = Y'_t - Y'_{t-1}$.
- At $t = -1$, everyone's phenotype is zero ($Y_{-1} = Y'_{-1} = 0$).
- The starting point of the dynamic feedback process is a pair of antecedent phenotypes, denoted by Y_0 or Y'_0. These
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Feedback Cycle

Feedback cycle is given by:

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- My Y_t is a weighted average of my phenotype yesterday (Y_{t-1}) and the lagged growth in my sib's phenotype.
- a is an interaction parameter satisfying $|a| < 1$.
  - imitation process if $a > 0$ and a contrast process if $a < 0$.

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Equilibrium

A pattern quickly emerges

$$
\begin{aligned}
Y_1\left(Y_0, Y_0'\right) &= Y_0 + aY_0' \\
Y_2\left(Y_0, Y_0'\right) &= Y_0 + aY_0' + a^2 Y_0 \\
Y_3\left(Y_0, Y_0'\right) &= Y_0 + aY_0' + a^2 Y_0 + a^3 Y_0' \\
Y_4\left(Y_0, Y_0'\right) &= Y_0 + aY_0' + a^2 Y_0 + a^3 Y_0' + a^4 Y_0
\end{aligned}
$$

(with a symmetric set of conditions for the primed sib).

Equilibrium

Let k_1 be the greatest integer less than or equal to $(t+2)/2$ and k_2 the greatest integer less than or equal to $(t+1)/2$. General pattern:

$$Y_t = \underbrace{\left(1+a^2+a^4\ldots\right)}_{=k_1 \text{ terms}} Y_0 + \underbrace{\left(1+a^2+a^4\ldots\right)}_{=k_2 \text{ terms}} aY_0'$$

$$= Y_0 \underbrace{\sum_{s=0}^{k_1} a^{2s}}_{=S_{k_1}} + aY_0' \underbrace{\sum_{s=0}^{k_2}}_{=S_{k_2}}$$

$$= S_{k_1}\left(Y_0\right) + S_{k_2}\left(aY_0'\right)$$

Since $|a|<1$, $\lim_{t\to\infty}\{S_{k_1}\} = \lim_{t\to\infty}\{S_{k_2}\} = \frac{1}{1-a^2}$, equilibrium values are:

$$\left(\tilde{Y}, \tilde{Y}'\right) = \left(\frac{Y_0 + aY_0'}{1-a^2} = \frac{Y_0' + aY_0}{1-a^2}\right).$$

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Equilibrium (cont'd)

Therefore:

$$V(\tilde{Y}) = \left[\frac{1}{1-a^2}\right]^2 \left[(1+a^2) \times V_0 + 2a \times cov(Y_0, Y_0')\right]$$

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Key Prediction: imitation/contrast effects result in different phenotypic variance across sib types:

- e.g. if $cov_{MZ}(Y_0, Y_0') = h^2 + c^2 > cov_{DZ}(Y_0, Y_0') = \frac{1}{2}h^2 + c^2 > cov_{ADO}(Y_0, Y_0') = c^2$, can identify a, at least in principle.
- in practice, of course, phenotypic variances could differ for reasons other than $a \neq 0$.
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Possible Extensions/Generalizations

Carey (1986) discusses how the model can be generalized to accomodate several extensions that enhance its realism.

- Sibships size ≥ 2.
- Nonsymmetric sibling effects. For example:
  - older siblings might impact younger siblings more.
  - stronger effects in same-sex dyads than mixed-sex ones.
- Time-varying imitation/contrast effects.
- Time-varying values of A, C and E.

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Key Readings for Extra Material: BG Models

Estimating BG Models

- Goldberger AS. (2005). "Structural Equation Models in Human Behavior Genetics." Chapter 2 in Identification and Inference for Econometric Models, DW Andrews and JS Stock. Cambridge, eds: Cambridge University Press, 11–26.
- Goldberger AS. (1978). "Models and Methods in the IQ Debate: Part 1. Revised**". *Social Systems Research Institute Working Paper*, University of Wisconsin-Madison.
- Boker *et al.* (2011). "OpenMx: An Open Source Extended Structural Equation Modeling Framework ," *Psychometrika* 76(2), 306-317.

Key Readings for Extra Material: Resemblance and Relatedness

Resemblance and Relatedness

- Lynch, M and B Walsh (1998). "Resemblance Between Relatives". Chapter 7 in *Genetics and Analysis of Quantitative Traits. Sunderland, MA: Sinauer Associates*, Inc.
- Crow JF, and Kimura M. (1970). "Correlation between Relatives and Assortative Mating," Chapter 4 in *An Introduction to Population Genetics Theory*, Holt, Rinehart and Winston.
- Nagylaki TJ. (1982). "Assortative Mating for a Quantitative Character". *Journal of Mathematical Biology* 16(57), 57-74.

Key Readings for Extra Material: Interactions

Sibling Interactions

- Carey G (1986). "Sibling Imitation and Contrast Effects". *Behavior Genetics*, 16(3), 319-341.
- Eaves LJ. (1976). "A Model for Sibling Effects in Man." *Heredity* 36, 205-214.