Advances in discordant sibling designs from the NLSY, illustrated with SES and health

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INTRODUCTION

Causal Claims with Experiments

- Randomized experiments are the "gold standard" for inferring causation in psychology.
- Whenever possible, conduct a randomized experiment (Cochran & Chambers, 1965; Dorn, 1953).
- Randomized experiments hit all the characteristics for making causal claims (See, West & Thoemmes, 2010)...
  - under Campbell's approach to causal inference (Campbell, 1957; Shadish, Cook, & Campbell, 2002)
    - focused on eliminating plausible threats to validity; and
  - under the Rubin Causal Model (Holland, 1988; Rubin, 1974, 2005)
    - focused on making verifiable assumptions.

Causal Claims with Experiments

- The design features of randomized experiments are fundamentally appealing:
  - treatment assignment is objective,
  - they are "automatically designed without access to any outcome data,"(Rubin, 2008) and
  - all pre-treatment covariates are "balanced" across conditions.

Causal Claims without Experiments

- However, questions posed by developmental psychology cannot always be answered with experiments.
  - Particularly, within the context of the SES-health gradient.
- If you are interested in
  - outcomes,
  - causes, or
  - exogenous change.

CAUSAL CLAIMS WITHOUT EXPERIMENTS

- However, questions posed by developmental psychology cannot always be answered with experiments.
  - Particularly, within the context of the SES-health gradient.
- If you are interested in
  - outcomes that
    - are rare or unique,
    - are longterm, or
    - require expensive designs;
  - causes, or
  - exogenous change.

Causal Claims without Experiments

- However, questions posed by developmental psychology cannot always be answered with experiments.
  - Particularly, within the context of the SES-health gradient.
- If you are interested in
  - outcomes,
  - causes that
    - cannot be manipulated,
    - should not be manipulated (according to the IRB), or
    - are too expensive to be manipulated; or
  - exogenous change.

Causal Claims without Experiments

- However, questions posed by developmental psychology cannot always be answered with experiments.
  - Particularly, within the context of the SES-health gradient.
- If you are interested in
  - outcomes,
  - causes, or
  - exogenous change
    - (*i.e.*, effects holding all other variables constant),
    - rather than net effects (Todd & Wolpin, 2003).

Causal Claims without Experiments

- Instead, we use quasi-experimental designs,
  - and control for potential confounds as covariates (Campbell, 1957; Rubin, 1974, *etc.*).
- Unlike randomized experiments, most quasi-experimental designs
  - cannot objectively assign treatment,
  - are not "automatically designed without access to any outcome data," and
  - cannot always balance *relevant* pre-treatment covariates across conditions.

Gene and Environmental Confounds

- The typical use of covariates doesn't control for systematically confounded gene and environmental effects (GE).
- In particular, poverty (and individual differences) covary with GE, so much so that
  - the covariate approach is fundamentally biased (Rowe & Rodgers, 1997).
- Yet, the covariate approach is the main method used to test the effect of SES on health.
  - (and many other psychology topics).

Sibling Models

- Classic sibling models rely on rare events (*e.g.*, twins) or advanced methods (*e.g.*, propensity score matching).
- They support causal inference without random assignment, and
  - incorporate GE elements (Lahey & D'Onofrio, 2010; Rutter, 2007).
- We advocate for using sibling (and other kin) models.
- Yet, such models are underused in psychology (Rodgers, Cleveland, van den Oord, & Rowe, 2000).
- Classic models impose barriers, which deter researchers from using sibling-based models.

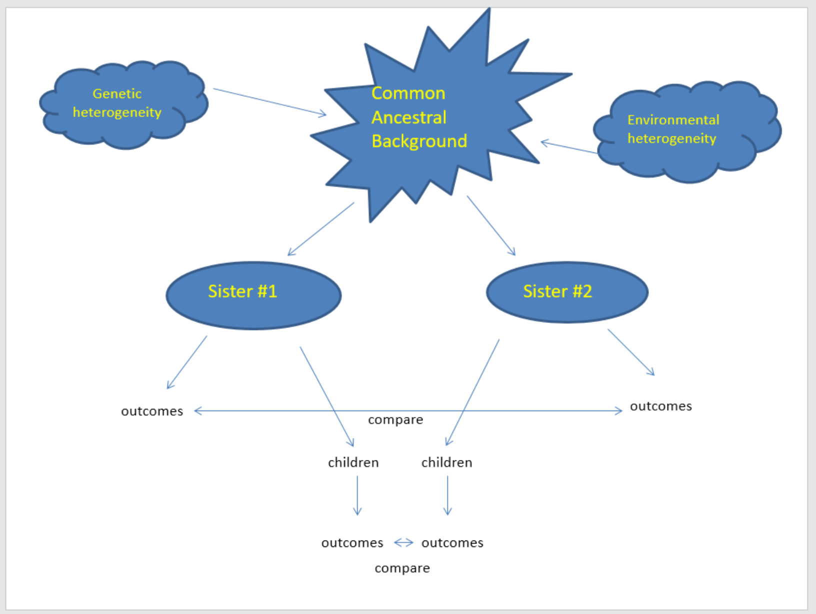
Sibling Models

- To reduce these barriers, we adapted Kenny and colleagues (2006; 2001) reciprocal standard dyad model to facilitate kin comparisons.

$$Y_{i\Delta} = \beta_0 + \beta_1 \bar{Y}_i + \beta_2 \bar{X}_i + \beta_3 X_{i\Delta}$$

- Our model uses differences between kin pairs, which explicitly accounts for within-family variance.
- Further, within-family differences create a powerful control for virtually all background heterogeneity
  - associated with both
    - genetic and
    - environmental differences (Lahey & D'Onofrio, 2010).
  - To illustrate, we examine whether household income influences birth weight.
  - We compare first-born children of sisters from within the family in the context of the following models.

Discordant Kin Model



Discordant Kin Model

- First, we predict the difference in birth weight, $Y_{i\Delta}$, for a given cousin pair (*i.e.*, kin), indexed as i, in the following model:

$$\mathrm{Y_{i\Delta}} = \beta_0 + \beta_1\bar{\mathrm{Y}}_{\mathrm{i}} + \beta_2\bar{\mathrm{X}}_{\mathrm{i}} + \beta_3\mathrm{X_{i\Delta}}$$

where,

$$\mathrm{Y_{i\Delta}} = \mathrm{Y_{i1}} - \mathrm{Y_{i2}};\ \mathrm{X_{i\Delta}} = \mathrm{X_{i1}} - \mathrm{X_{i2}}$$

Note: 1 and 2 identify the individuals within the kin pair, and are defined by

$$\mathrm{Y_{i1}} > \mathrm{Y_{i2}}$$

Discordant Kin Model

- In this model, the relative difference in kin outcomes (Y_Δ; *e.g.*, birth weight) is predicted from the
  - mean level of Y ($\bar{Y}$; *e.g*. mean birth weight),
  - the mean level of X ($\bar{X}$; *e.g.*, household income), and the
  - between-kin household income difference ($X_{i\Delta}$).

$$\mathbf{Y_{i\Delta}} = \beta_0 + \beta_1 \bar{Y}_i + \beta_2 \bar{X}_i + \beta_3 X_{i\Delta}$$

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Discordant Kin Model

- The mean levels, reflecting between-family variance, support causal inference through
  - partial control for genes and shared environment in previous generations.
- Within this model, there is explicit separation of
  - within-family variance (with Y_Δ and X_Δ), and
  - between-family variance (with $\bar{\mathrm{Y}}$ and $\bar{\mathrm{X}}$).

$$\mathrm{Y}_{\mathrm{i}\Delta} = \beta_0 + \beta_1 \bar{\mathrm{Y}}_\mathrm{i} + \beta_2 \bar{\mathrm{X}}_\mathrm{i} + \beta_3 \mathrm{X}_{\mathrm{i}\Delta}$$

- We've used this method in Garrison and Rodgers (*in press*), examining whether intelligence causes age at first intercourse (spoiler: it doesn't)

National Longitudinal Survey of Youth 79

- Generation 1
  - The NLSY79 is a nationally representative household probability sample.
  - On December 31, 1978, ≈13,00 adolescents (ages 14-21) were sampled from ≈ 9,000 households.
- Generation 2
  - In 1986, all biological children of the female NLSY79 participants were surveyed (≈ 12,000).
- We have identified and validated the kinship pairs within these two datasets (Rodgers et al., 2016).
- We provide a supporting R package NlsyLinks (Beasley et al., 2015).
  - ≈ 4,000 Sibling Pairs
  - ≈ 450 Mother-Child-Aunt-Nibling Tetrads

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Illustration Overview

- Illustrations
  - Maternal SES on Birthweight of First Born Child
  - SES at Age 40 on Physical Health (Age 40)
  - Intelligence in Adolescence on Physical Health (Age 40)
- Measures
  - SES
  - Health
  - Cognitive Ability

Illustration Overview

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  - Maternal SES on Birthweight of First Born Child
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- Measures
  - SES measure is
    - based on Myrianthopoulos and French (1968) and used more recently by Turkheimer, Haley, Waldron, D'Onofrio, and Gottesman (2003).
    - aggregate score based on the mean of their total net family income, education, and occupation quantile scores.
  - Health
  - Cognitive Ability

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  - SES
  - Health measures are
    - Birth Weight of Gen2 Participants
    - SF-12 Physical Component Summary Score (PCS) of Gen1 Participants
  - Cognitive Ability

Illustration Overview

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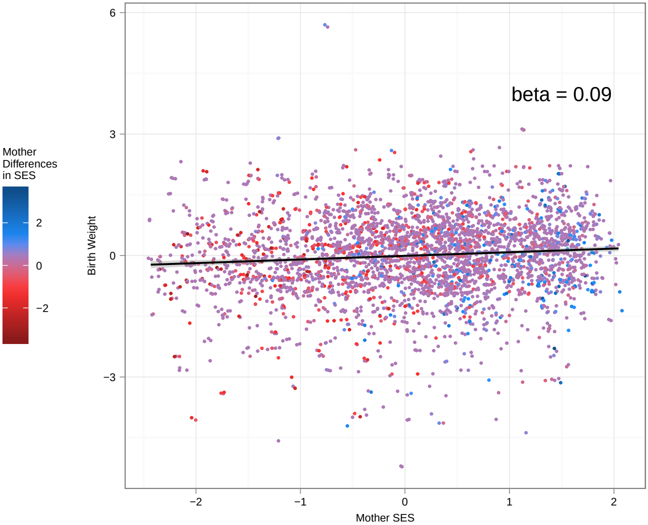
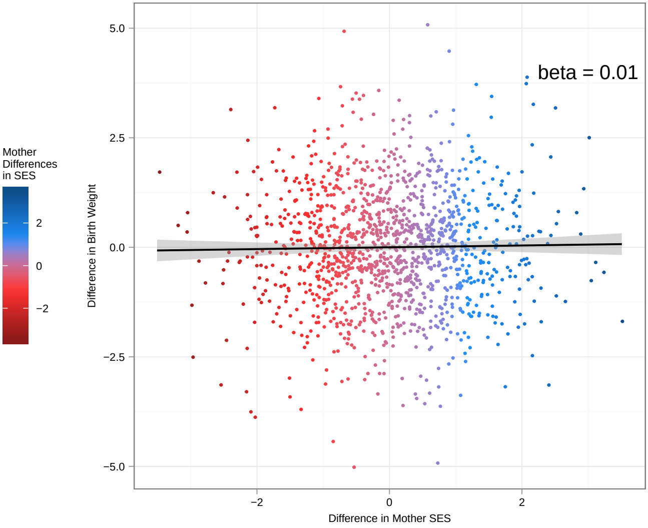
  - Intelligence in Adolescence on Physical Health (Age 40)
- Measures
  - SES
  - Health
  - Cognitive Ability measure is
    - Armed Service Qualification Test (Ages 14-21)

Maternal SES vs. Birth Weight



Δ SES vs. Δ Birth Weight

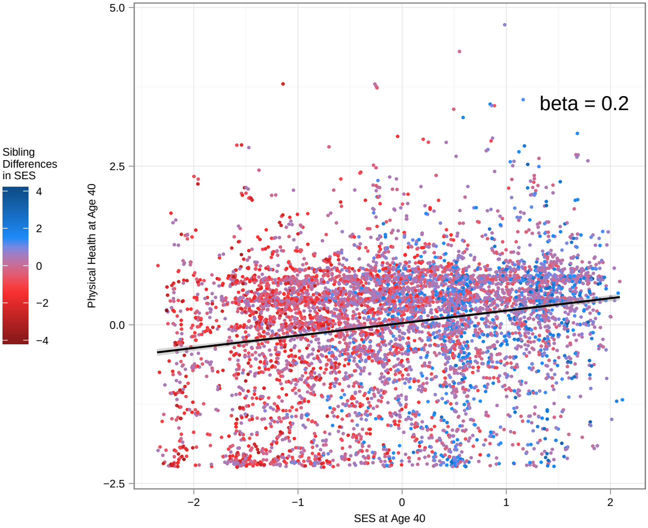


```
## Call:
## lm(formula = DIF_W ~ M_W + M_SES + DIF_SES)

## Coefficients:
##              Estimate(SE)   t value  P(>|t|)
## (Intercept)   1.00  (0.03)  31.92    < 0.001
## M_W          -0.32  (0.04)  -7.67    < 0.001
## M_SES        -0.02  (0.04)  -0.03      0.98
## DIF_SES       0.01  (0.03)  -0.32      0.75

## Multiple R-squared:  0.081,  Adjusted R-
   squared:  0.077
## F-statistic: 20 on 3 and 683 DF
```

SES vs. Physical Health



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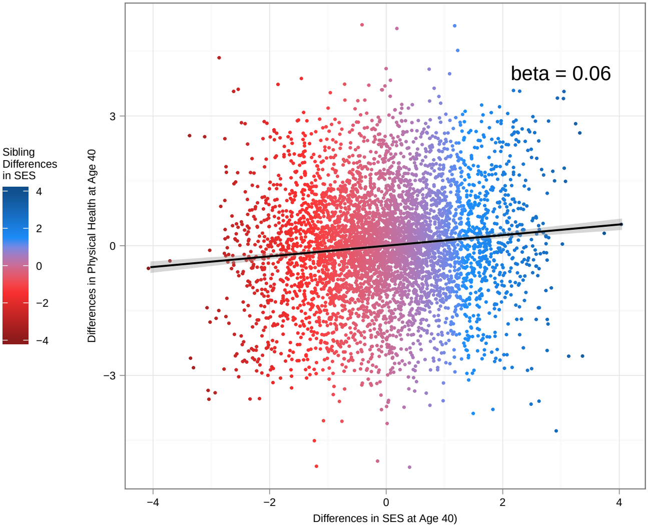
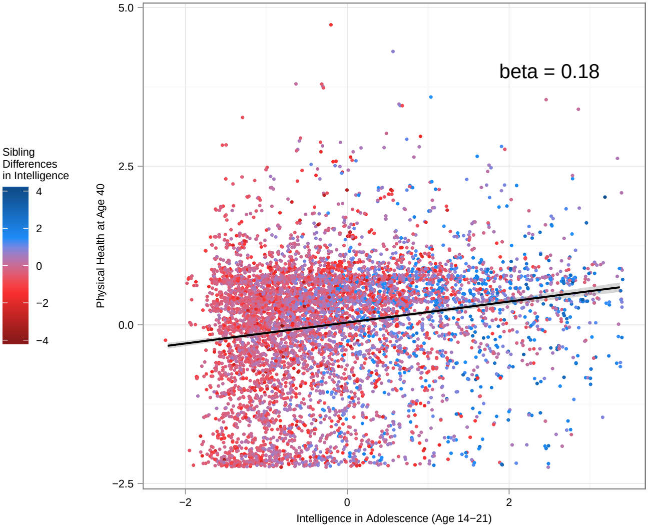
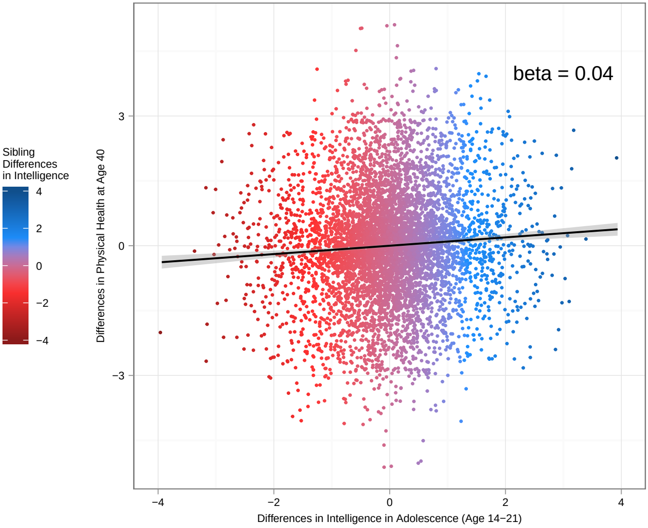


```
## Call:
## lm(formula = DIF_H ~ M_H + M_S + DIF_S)

## Coefficients:
##                 Estimate (SE)  t value P(>|t|)
## (Intercept)      0.97 (0.02)   60.86   <0.001
## M_H             -0.33 (0.02)  -14.22   <0.001
## M_S             -0.01 (0.02)   -0.30    0.765
## DIF_S            0.06 (0.01)    3.21    0.001

## Multiple R-squared:  0.084,  Adjusted R-
   squared:  0.083
## F-statistic: 78.9 on 3 and 2575 DF
```

Intelligence vs. Physical Health



Δ Intelligence vs. Δ Physical Health



```
## Call:
## lm(formula = DIF_H ~ M_H + M_I + DIF_I)

## Coefficients:
##                 Estimate(SE)   t value   P(>|t|)
## (Intercept)      0.98 (0.02)     65.97    <0.001
## M_H             -0.35 (0.02)    -16.61    <0.001
## M_I              0.02 (0.02)      1.59     0.112
## DIF_I            0.04 (0.02)      2.28     0.023

## Multiple R-squared:  0.084,  Adjusted R-
   squared:  0.084
## F-statistic: 97.3 on 3 and 3164 DF
```

Recall: 3 appealing design features of experiments

- treatment assignment is objective,
- "automatically designed without access to any outcome data," and
- all pre-treatment covariates are "balanced" across conditions.

Recall: 3 appealing design features of experiments

- treatment assignment is objective,
  - Within a kin pair, the kin are assigned based on whichever has more of outcome.

$$Y_{i1} > Y_{i2}$$

  - Theoretically, you could assign the kin based on whichever has more of treatment.

$$X_{i1} > X_{i2}$$

  - But, modeling multiple predictors (*e.g.*, Garrison & Rodgers, in press), complicates assignment.
- "automatically designed without access to any outcome data," and
- all pre-treatment covariates are "balanced" across conditions.

Recall: 3 appealing design features of experiments

- treatment assignment is objective,
- "automatically designed without access to any outcome data," and
  - The design is automatic... but it requires the outcome data.
- all pre-treatment covariates are "balanced" across conditions.

Recall: 3 appealing design features of experiments

- treatment assignment is objective,
- "automatically designed without access to any outcome data," and
- all pre-treatment covariates are "balanced" across conditions.
  - Within a sibling pair, all background covariates are balanced at birth.
    - Independent assortment of genes occurs during meiosis (Mendel, 1901).

Caveats

- After birth,
  - genetic influences are still balanced, as are
  - shared-environmental influences.
- As our siblings age, they accumulate non-shared experiences.
  - These non-shared environmental influences are not balanced.
  - Accordingly, spurious significant results can arise from differences in non-shared environmental influences.

Conclusion

- The discordant sibling model is a parsimonious,
  - but powerful quasi-experimental design.
- It provides a framework for casting doubt on causal claims.
- Within the NLSY79 alone, there are $>$ 12,000 variables, which include psychological constructs
- `nlsinfo.org`
- Our lab has grants from NIH (and NSF), which provide user support for work in this area,
  - including help applying these models to NLSY data.

Acknowledgments

NSF

VANDERBILT

SMEP

Society of Multivariate Experimental Psychology

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