Untangling Personality, Intelligence, and the SES-Health Gradient: Evaluating Causal Models with Biometrically Informed Data

The relationship between poverty and health has been widely covered in the popular media; within the last year, the Atlantic, Time Magazine, and the New York Times have all featured the SES-health gradient at length, drawing strongly upon conventional explanations that material disadvantage directly (*e.g*, access to medical care)[1] or indirectly (*e.g*, chronic environmental stress)[2] causes health inequalities. Such explanations account for differences between those who have resources and who do not. However, Gottfredson[3] argued that situational explanations do not account for the finely stratified health differences that exist across the entire range of SES, whereas individual differences in intelligence and personality do. Indeed, Chapman and colleagues[4] found that personality characteristics account for 20% of the SES-health gradient.

Regardless of whether the person or situation is the "elusive fundamental cause"[3] of the SES-health gradient, the debate cannot be settled using experiments. Although randomized studies support inferring causality for most situational explanations, poverty cannot be randomly as-signed. Nor can we randomly assign most person characteristics.[5] Instead, longitudinal quasi-experimental studies are used, and potential confounds are included as covariates.

The typical use of covariates does not provide control for systematically confounded genetic and environmental influences. This approach risks misattributions of causality. Indeed, poverty and individual differences covary with genes and environment, to such a degree that the covariate approach is fundamentally biased (Rowe & Rodgers, 1997).[6] Yet, the covariate approach has been the primary method used to evaluate the cause of the SES-health gradient.

Instead, quasi-experimental designs can be used; such designs support causal inference without random assignment.[7] Sibling-based quasi-experimental models are particularly effective at incorporating genetic and environmental design elements.[8] However, such models are underused in psychology (Rodgers *et al*, 2001),[9] tend to focus on environmental confounds,[10] and do not naturally incorporate varying levels of relatedness.[8] My research proposal aims to address both the methodological problem of evaluating person-driven hypotheses and the substantive problem of explaining the SES-health gradient, using and extending the sibling comparison approach.

Kinship Dyads

Traditional sibling comparison models often rely on rare events (*i.e*, twins) or advanced methodology (*e.g*, propensity score matching, multilevel modeling). As an alternative, my advisor and I have adapted Kenny's reciprocal standard dyad model.[11] Our adaptation controls for gene and shared environmental influences within a simple regression framework, by taking the difference between the two siblings (Rodgers, **Garrison** *et al*, 2014):[12]

$$Y_{diff} = \beta_0 + \beta_1 Y_{mean} + \beta_2 X_{mean} + \beta_3 X_{diff}$$

where $$Y_{diff} = Y_{1i} - Y_{2i};\ Y_{mean} = \frac{Y_{1i} + Y_{2i}}{2};\ X_{diff} = X_{1i} - X_{2i};\ X_{mean} = \frac{X_{1i} + X_{2i}}{2}$$

In this model, the relative difference in outcomes (Y_{diff}) is predicted from the mean level of the outcome (Y_{mean}), the mean level of the predictor (X_{mean}), and the between-sibling predictor difference (X_{diff}). The mean levels support causal inference through at least partial control for genes and shared environment. Therefore, we simultaneously evaluate the individual difference (X_{diff}) and the joint contribution of genes and shared environment (Y_{mean} & X_{mean}). Preliminary applications have given estimates consistent with the literature (Garrison *et al*, 2015).[13]

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Proposed Studies

Application I will apply the kinship dyad model to two nationally representative household samples: the National Longitudinal Survey of Youth 1979 (NLSY79) and the NLSY97. The two household sampling techniques have resulted in 15,589 families with 19,374 sibling pairs, which our research team has identified and validated. Both surveys include measures of conscientiousness and intelligence, the most consistent predictors of health and SES. The NLSY97 also includes self-reported Big Five personality indicators.

I will directly test the impacts of SES, personality, and intelligence on health, estimating how much of the SES-health gradient is caused by each (including covariance among the predictors). Then, I will evaluate specific causal mechanisms, such as access to health care, neighborhood quality, and education level. Identifying specific mechanisms will facilitate translating findings into interventions. To ensure that these findings are externally valid, I will compare results across samples to test whether the gradient has changed across cohorts.

Extension After validating the model using real-world data, I will extend the model in three ways: **1** extend the model to include additional highly correlated predictors, **2** evaluate the model's robustness under measurement error; **3** incorporate multiple generations.

First, although the Big Five are orthogonal by design, intelligence is correlated with multiple facets. For my 1st year project, I evaluated the separate impact of intelligence and conscientiousness by partialing out the common variance, resulting in uncorrelated and uncontaminated measures of each.[13] Generalizing this approach will enable the model to test more complicated relationships between predictors. **2** To evaluate the model's robustness, I will conduct a series of Monte Carlo simulations, using NSF's high-performance computing service, XSEDE under various levels of measurement error for both the outcome and predictor variables. Moreover, I will evaluate the model's external validity in relation to previous research that has provided parameters estimates for the relationship between individual differences, health, and SES. If the kinship dyad model correctly estimates the parameters, this further supports that the model has performed correctly under real-world conditions. **3** The model can be further extended by examining the children of the siblings, in the same way that the children of twins design works – by exploiting the common genetic traits and dissimilar environmental effects within standard biometrical models. This would allow the model to distinguish between genes and shared environmental causes. Moreover, the effectiveness of this extension can be tested using the multigenerational structure of the NLSY79.

Merit & Impact

This research has the potential to help untilt the SES-health gradient. Directly, by applying this model to test the causes of the SES-health gradient, I will determine whether the person or situation is driving the gradient. Identifying specific mechanisms will facilitate translating these findings into actionable interventions. Indirectly, my research will support accessible and parsimonious solution to make causal inferences about person-driven hypotheses. Other researchers will be able to employ this model in their own work on the SES-health gradient.

To enhance the model's accessibility to other researchers, I will develop R, SAS, and SPSS syntax, and make it available on my website with detailed tutorials. Moreover, as this approach can be used on many datasets, I will identify and link to compatible files from various academic databases, such as Harvard's Dataverse and University of Michigan's ICPSR.

Refs 1 Adler *et al* (1994) *Ameri Psych*. **2** Baum *et al* (1999) *NY Acad Sci*. **3** Gottfredson (2004) *JPSP*. **4** Chapman *et al* (2009) *Ameri J of Epi*. **5** West (2009) *Cur Dir Psych Sci*. **6** Rowe & Rodgers(1997) *Dev Rev*. **7** Shadish *et al* (2002) Wadsworth. **8** Rutter (2007) *Persp Psych Sci*. **9** Rodgers *et al* (2000) *Ameri Psych*. **10** Lahey *et al* (2010) *Cur Dir Psych Sci*. **11** Kenny *et al* (2001) *Psych Bul*. **12** Rodgers *et al* (2014 Jun) *BGA*. **13** Garrison *et al* (2015 Feb) *SPSP*.