

Additive effects of maternal persistence and general intelligence on infant health: Cause or Confound?

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Introduction

Conscientiousness and intelligence consistently predict later health outcomes (Jokela *et al.*, 2013; Gotfredson & Deary, 2004) across the life course (Roberts *et al.*, 2007). Yet, conscientiousness and intelligence are seldom tested simultaneously (Deary *et al.*, 2010), making it difficult to determine whether both traits determine health or one measure is a proxy for the other. Intelligence and the conscientiousness facet of persistence are confounded under many assessment conditions (Duckworth, 2011). Moreover, persistence is associated with later health (Tores *et al.*, 2001), making the persistence-intelligence confound even more difficult to untangle.

Thus, we examined the joint impact of maternal intelligence and persistence on the birth weight of her first live child, using the National Longitudinal Survey of Youth 1979 (NLSY79). To control for genetic and environmental risk, we employed a discordant kin quasi-experimental design.

Data

The NLSY79 is a nationally representative household probability sample. In 1980, 12,686 adolescents between the ages of 15 and 23 were surveyed on a battery of measures, including the Armed Forces Vocational Aptitude Battery (ASVAB). Subscales of the ASVAB measure intelligence (AFQT) and persistence (Coding Speed). In 1986, the biological children of the female NLSY79 participants were surveyed. Both generations of participants are surveyed on a biannual basis.

	Has Sibling in NLSY?					
	Mean			Sd		
	0	1		0	1	
Mother Age at Birth	23.3	22.6	23.9	5.5	5.2	5.7
Birth Weight	115.0	115.0	114.9	20.6	20.4	20.8
AFQT	65.2	67.9	62.8	22.2	21.7	22.4
Coding Speed	42.2	43.9	40.7	16.8	16.5	16.8

Methods

To control for gene and environmental confounds, we have adapted Kenny's (2001) reciprocal standard dyad model. The adaptation controls for gene and shared environmental influences within a simple regression framework, by taking the difference between the kin:

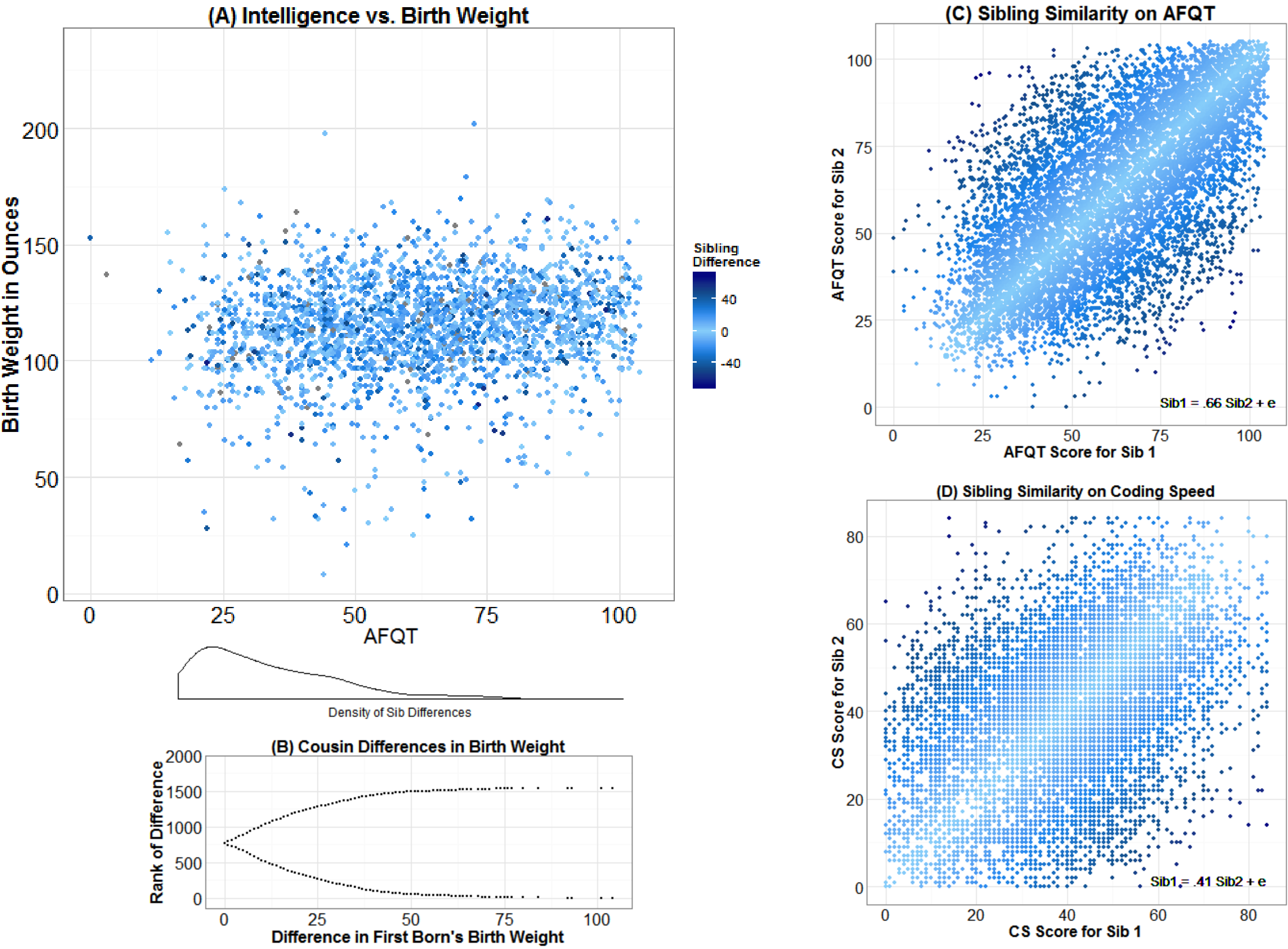
$$\mathbf{Y}_{\text{diff}} = \beta_0 + \beta_1 \bar{\mathbf{Y}} + \beta_2 \bar{\mathbf{X}} + \beta_3 \mathbf{X}_{\text{diff}}$$

where,

$$\mathbf{Y}_{\text{diff}} = \mathbf{Y}_{1i} - \mathbf{Y}_{2i}; \bar{\mathbf{Y}} = \frac{\mathbf{Y}_{1i} + \mathbf{Y}_{2i}}{2}$$
$$\mathbf{X}_{\text{diff}} = \mathbf{X}_{1i} - \mathbf{X}_{2i}; \bar{\mathbf{X}} = \frac{\mathbf{X}_{1i} + \mathbf{X}_{2i}}{2}$$

The relative difference in outcomes (\mathbf{Y}_{diff}) is predicted from the mean level of the outcome ($\bar{\mathbf{Y}}$), the mean level of the predictor ($\bar{\mathbf{X}}$), and the between-sibling predictor difference (\mathbf{X}_{diff} ; see fig. B). The mean levels support causal inference through at least partial control for genes and shared environment. Therefore, we simultaneously evaluate the individual difference (\mathbf{X}_{diff}) and the joint contribution of genes and shared environment ($\bar{\mathbf{Y}}$ & $\bar{\mathbf{X}}$).

Figures



Mother-Child-Aunt-Nibling Quads

Dependent Variable: Birth Weight of First Born Child						
	Between Family			Within Family		
	(1)	(2)	(3)	(4)	(5)	(6)
Mean Weight					-4.245*** (0.632)	-4.319*** (0.636)
Mean Persistence	0.157*** (0.036)	0.177*** (0.036)		0.158*** (0.036)	-0.762(0.631)	
Mean Intelligence	0.114*** (0.036)		0.208*** (0.036)	0.111*** (0.036)		-0.521(0.635)
lxP				0.043(0.043)		
Diff Persistence					0.547(0.622)	
Diff Intelligence						0.739(0.622)
Observations	753	753	753	753	753	753
R ²	0.044	0.031	0.043	0.045	0.065	0.065
Adjusted R ²	0.041	0.030	0.042	0.041	0.062	0.061
Residual Std. Error	0.978 (df = 750)	0.984 (df = 751)	0.978 (df = 751)	0.978 (df = 749)	17.033 (df = 749)	17.035 (df = 749)
F Statistic	17.265*** (df = 2; 750)	24.280*** (df = 1; 751)	33.989*** (df = 1; 751)	11.831*** (df = 3; 749)	17.466*** (df = 3; 749)	17.402*** (df = 3; 749)
Note:	*p<0.1; **p<0.05; ***p<0.01					

Kin Relationships

Quad Correlation Matrix				
	Self	Kin		
Weight	1			0.537
AFQT	0.174	1		0.668
CS	0.108	0.715	1	0.425

ACE Estimates				
	A ²	C ²	E ²	Pairs
Weight	0	0.154	0.846	629
AFQT	0.301	0.508	0.190	4,002
CS	0.236	0.290	0.473	4,002

Results

A series of kinship dyad regressions used the AFQT and Coding Speed scales in 1980 to predict the first born child's birth weight. The Between Family Models (1-4) ignore genetic and environmental confounds. The Within Family Models (5-6) simultaneously evaluated the influence of the individual difference and the joint contribution of genes and shared environment.

Correlation Structure

Within sibling correlations revealed that health moderately correlated with intelligence and persistence ($r \approx .15$). Persistence was strongly related to AFQT score ($r = .72$), more so than the correlations between siblings, which ranged from .43 to .67.

Models

The Between Family Models (1-4) examined the impacts of persistence (2), intelligence (3), joint influence (1), and their interaction (4) on first born child's birth weight. Each found that respective individual difference was influential. Model 4 revealed that the result was additive because the interaction was not significant.

The Within Family Models (5-6) failed to replicate the Between Family Model results. Once background genetic and environmental influences were controlled for, neither persistence (5) nor intelligence (6) predict birth weight.

Summary

- When genes and environment are ignored, intelligence and persistence predict birth weight. (Models 1-4).
- Once genetic and environmental influences are included (i.e., between-family confounds are controlled), the effects of intelligence and persistence vanish (5, 6).
- Therefore, maternal intelligence and persistence are not direct causal agents affecting changes in birth weight. Rather, they stand in for other more proximate causes.
- Ignoring genetic and environmental influences can result in theoretically compelling, yet spurious results.