

Impact of adolescent conscientiousness and intelligence on health at middle age:  
A sibling comparison approach

S. Mason Garrison, Alexandria R. Hadd, & Joseph Lee Rodgers  
Vanderbilt University



VANDERBILT  
PEABODY COLLEGE

Introduction

Conscientiousness and intelligence consistently predict later health outcomes (Jokela *et al.*, 2013; Gotfredson & Deary, 2004) across the life course (Roberts *et al.*, 2007). However, 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.

Using the National Longitudinal Survey of Youth (NLSY) 1979, we examined the joint impact of intelligence and persistence assessed at adolescence on self-reported health at age 40. To control for genetic and environmental risk, we developed a sibling-based quasi-experimental method.

Data

The National Longitudinal Survey of Youth 1979 (NLSY79) is a nationally representative household probability sample. In 1980, 12,686 adolescents between the ages of 14 and 22 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). Participants are surveyed biannually by the Bureau of Labor Statistics (BLS).

	Has Sibling in NLSY?					
	Mean			Sd		
	0	1		0	1	
Age	54.4	54.9	54.0	2.3	2.3	2.2
Health	2.4	2.4	2.3	1.0	1.0	1.0
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 (2014) 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 two siblings:

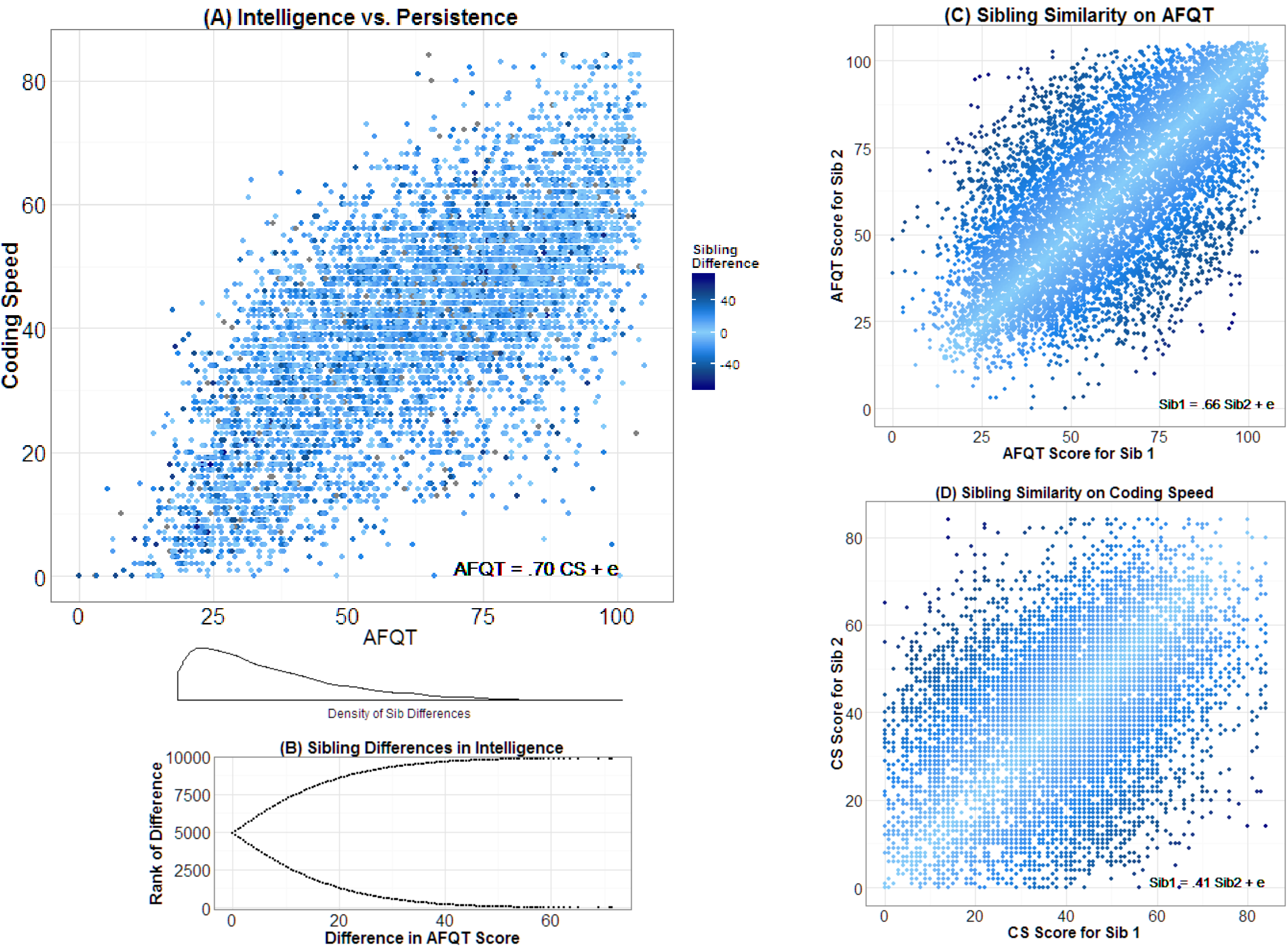
$$\mathbf{Y}_{diff} = \beta_0 + \beta_1 \bar{\mathbf{Y}} + \beta_2 \bar{\mathbf{X}} + \beta_3 \mathbf{X}_{diff}$$

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

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

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 a 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



Sibling Dyads

	Difference in Health					
	□(1)⊙	(2)⊙	(3)	(4)	(5)	(6)
<b>Individual</b>						
Persistence	-0.099* (0.024)	-0.059* (0.014)	-0.023(0.015)			-0.044* (0.013)
Intelligence	-0.095* (0.024)	-0.037* (0.014)	-0.039* (0.015)		-0.052* (0.013)	
Common Variance				-0.053* (0.013)		
<b>Family</b>						
Persistence	-0.053* (0.017)	-0.043* (0.013)	-0.057* (0.020)			-0.041* (0.013)
Intelligence	-0.027(0.017)	0.014(0.013)	0.022(0.020)		-0.022(0.014)	
Common Variance				-0.034* (0.013)		
Health	0.155* (0.014)	0.155* (0.014)	0.155* (0.014)	0.154* (0.014)	0.157* (0.014)	0.154* (0.013)
Observations	4,268	4,268	4,268	4,268	4,268	4,268
Adjusted R <sup>2</sup>	0.042	0.042	0.042	0.041	0.040	0.041
F Statistic	38.665* (df = 5; 4262)	38.665* (df = 5; 4262)	38.665* (df = 5; 4262)	62.551* (df = 3; 4264)	60.923* (df = 3; 4264)	61.693* (df = 3; 4264)
Notes:	⊙ Persistence was partialled out of Intelligence □ Intelligence was partialled out of Persistence.			*p<0.05 Standardized β(SE)		

Dyad Correlation Matrix

	Self		Sibling	
Health	1			0.543
AFQT	0.281	1		0.668
CS	0.233	0.715	1	0.425
Overlap	0.274	0.903	0.946	1
				0.580

Results

A series of kinship dyad regressions used the AFQT and Coding Speed scales from the ASVAB at Wave 1 to predict self-reported health at age 40. The models 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, persistence, and their overlap ( $r \approx .25$ ). Persistence was strongly related to AFQT score ( $r = .72$ ), more so than the correlations between siblings, which ranged from .43 to .67.

Models

Models 6, 5, and 4 examined the incremental impacts of persistence, intelligence, and their common variance – respectively – on health. Each found that respective individual difference was influential.

Model 3 examined the incremental impacts of both persistence and intelligence on health, without controlling for their covariance. Intelligence was significantly predicted health, while persistence was not.

Model 2 examined the incremental impacts of both persistence and intelligence, on health, after partialing persistence from intelligence. Both significantly predicted health. Model 1 fully extracted the common variance by partialing persistence from intelligence and intelligence from persistence. Both still significantly predicted health with comparably sized effects.

Summary

- ▶ Intelligence and persistence individually predict health, even after controlling for genes and shared environment (Models 5, 6).
- ▶ Their shared covariance was an equally effective predictor, beyond genes and shared environment (4).
- ▶ When modeled jointly, partialled intelligence and persistence comparably accounted for health (1, 2).
- ▶ When the covariance between intelligence and persistence was not modeled, intelligence masked the effect of persistence (3).