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Intelligence



## School-level genetic variation predicts school-level verbal IQ scores: Results from a sample of American middle and high schools<sup>☆</sup>

Kevin M. Beaver<sup>a,\*</sup>, John Paul Wright<sup>b</sup>

<sup>a</sup> Florida State University, College of Criminology and Criminal Justice, 634 W. Call Street, Tallahassee, FL 32306-1127, United States

<sup>b</sup> University of Cincinnati, United States

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### ABSTRACT

Research has consistently revealed that average IQ scores vary significantly across macro-level units, such as states and nations. The reason for this variation in IQ, however, has remained at the center of much controversy. One of the more provocative explanations is that IQ across macro-level units is the result of genetic differences, but empirical studies have yet to examine this possibility directly. The current study partially addresses this gap in the literature by examining whether average IQ scores across thirty-six schools are associated with differences in the allelic distributions of dopaminergic polymorphisms across schools. Analysis of data drawn from subjects (ages 12–19 years) participating in the National Longitudinal Study of Adolescent Health provides support in favor of this perspective, where variation in school-level IQ scores was predicted by school-level genetic variation. This association remained statistically significant even after controlling for the effects of race.

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### 1. Introduction

Substantial variation exists across macro-level units for virtually every measurable characteristic. For example, research has revealed that indicators of wealth, measures of health, and crime rates vary significantly across neighborhoods, states, and nations (Beaver & Wright, 2011; Kanazawa, 2008; McDaniel, 2006; Pesta, McDaniel, & Bertsch, 2010). This same line of research has also documented that variation and inequality tend to be the most pronounced at the level of the nation. Stated

simply, some nations are rich and others are poor; some nations are healthy and others are not; some nations have high rates of crime and others have low rates of crime (Braithwaite, 1989). The question that has plagued researchers, however, is what accounts for such disparities. Most of the explanations that have been advanced to explain nation-level differences have focused on culture, socialization, access to resources, and other socio-environmental factors (e.g., Diamond, 1997; Messner & Rosenfeld, 1994).

Perhaps the most controversial explanation for inequality across nations was advanced in Lynn and Vanhanen's (2002) book, *IQ and a Wealth of Nations*. In this book Lynn and Vanhanen empirically examined the association between the average IQ of the nation and measures of wealth. The result of their analyses revealed a statistically significant association, where nations with higher average IQ scores tended to have more wealth than nations with lower IQ scores. More recently, they expanded their analyses and examined whether nation-level IQ scores were related to other measures of inequalities, such as educational level, life expectancy, and literacy rates (Lynn & Vanhanen, 2006). Their results once again indicated a statistically significant association between IQ and an assortment of measures of inequality.

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\* Corresponding author. Tel.: +1 850 644 9180; fax: +1 850 644 9614.

E-mail address: [kbeaver@fsu.edu](mailto:kbeaver@fsu.edu) (K.M. Beaver).

With evidence mounting in favor of the position that nation-level IQ scores are related to inequality across nations, the next logical question to ask is what accounts for variation in IQ across nations? Lynn and Vanhanen (2002, 2006) (see also Hart, 2007; Rushton, 1997) advanced a very provocative and controversial claim that variation in nation-level IQ scores is produced by genetic variation across nations. Much of the evidence that they cite and discuss in relation to this claim, however, centers on heritability estimates that were generated using data at the individual level. For example, Lynn and Vanhanen (2006) describe the results of twin studies showing that IQ is about .75, meaning that about 75% of the variance in IQ is due to genetic factors. Although the evidence indicating that variation in individual-level IQ scores is due largely to genetic factors is overwhelming, the connection between individual-level IQ scores and nation-level IQ scores is not entirely clear. Heritability estimates are point estimates that are designed to explain variance generated from *individual* scores and thus whether these results can be extrapolated to higher levels of aggregation remains to be determined.

The goal of the current study is to provide a partial test of Lynn and Vanhanen's (2002, 2006) thesis that variation in IQ scores at the nation level is the result of genetic differences. Our analysis focuses on examining whether polymorphisms in dopaminergic genes are related to IQ scores. We employed dopaminergic genes because prior research has provided some theoretical and empirical evidence linking the dopaminergic system, including dopaminergic polymorphisms, to cognitive abilities and IQ (Beaver, DeLisi, Vaughn, & Wright, 2010; Berman & Noble, 1995; Previc, 1999).

Due to data limitations we were unable to obtain data that included IQ scores at the nation level and genetic data at the nation level. We were, however, able to locate data that included IQ scores and DNA markers that could be aggregated to the school level. In this way, we were able to test whether variation in IQ scores at the school level was associated with variation in DNA markers that were aggregated to the school level. While using data aggregated to the school level cannot be considered a definitive test of Lynn and Vanhanen's hypotheses at the nation level, the results based on schools can be considered an initial test of their statements for two main reasons. First, schools, like nations, show tremendous variation in terms of health, wealth, crime, and even IQ (Herrnstein & Murray, 1994; Saab & Klinger, 2010; Weissberg, 2010). Second, Lynn and Vanhanen's (2002) arguments linking IQ to various outcomes have been shown to exist at levels of aggregation other than the nation, including the state level and the county level (Beaver & Wright, 2011; Kanazawa, 2008; McDaniel, 2006; Pesta et al., 2010). It is quite likely, then, that Lynn and Vanhanen's explanation may apply to all types of aggregate units of analysis, not just nations. We use this possibility as a springboard to provide the first partial test of Lynn and Vanhanen's provocative thesis that IQ varies across nations because of variation in genetic factors.

## 2. Method

### 2.1. Sample

Data for this study come from waves 1 and 3 of the National Longitudinal Study of Adolescent Health (Add Health). The Add Health is a four-wave study of a nationally representative

sample of American youths who were enrolled in seventh through twelfth grades during the 1994–1995 school year (Udry, 2003). Multi-stage stratified sampling techniques were employed to select 132 middle and high schools included in the study. Students attending these schools were administered a self-report survey during a specified school day. More than 90,000 youths were included in the wave 1 in-school component of the Add Health study. A subsample of youths was then selected to be reinterviewed at their homes to gain more detailed information. Altogether, 20,745 adolescents participated in the wave 1 in-home component of the study (Harris, Florey, Tabor, Bearman, Jones, & Udry, 2003).

One of the distinguishing features of the Add Health data is that at wave 3 a subsample of respondents was genotyped. To be eligible for participation in the DNA subsample, respondents had to have a sibling who was also included in the study. If they were eligible, and if they agreed to participate, then they submitted samples of their buccal cells to be genotyped. Genotyping was conducted in coordination with the Institute of Behavioral Genetics in Boulder, Colorado and Add Health. In total, more than 2500 participants were included in the DNA subsample of the study (Harris, Halpern, Smolen, & Haberstick, 2006).

The final analytic sample consisted of only schools where there were at least 19 students who were included in the sample. In that way, the school-level estimates, which were based on aggregated individual-level scores, were less subject to variability associated with small sample sizes. After removing schools with less than 19 students, we were left with a final analytical sample of 1265 youths nested within 36 schools. Given that our analysis is based on a small sample size ( $N = 36$  schools), the power to detect small-to-moderate effect sizes is compromised. Any statistically significant effects that are detected will thus be moderate-to-large in magnitude.

### 2.2. Measures

#### 2.2.1. School-level IQ scores

At wave 1, Add Health participants completed the Picture Vocabulary Test (PVT). The PVT is an abbreviated version of the full-length Peabody Picture Vocabulary Test (PPVT), a test used to assess verbal abilities and receptive vocabulary. The PVT measure has been used previously as a measure of IQ among researchers analyzing the Add Health data (Rowe, Jacobson, & Van den Oord, 1999). School-level IQ was estimated by aggregating and averaging individual PVT scores at the school level. A similar technique has been used previously to estimate county-level IQ (Beaver & Wright, 2011). The final score represents the average IQ score for respondents attending that school. The average school-level IQ was 99.08 with a standard deviation of 7.54.

#### 2.2.2. School-level dopamine scores

To estimate school-level dopamine scores, we aggregated and averaged (at the school level) genotypic scores for three dopaminergic polymorphisms: one in the dopamine transporter gene (DAT1), one in the dopamine D2 receptor gene (DRD2/ANKK1), and one in the dopamine D4 receptor gene (DRD4). Detailed information about the genotyping of these polymorphisms is available elsewhere (Beaver, Vaughn, Wright, DeLisi, & Howard, 2010; Hopfer, Timberlake, Haberstick,

Lessem, Ehringer, Smolen et al., 2005). We used prior research examining the link between dopaminergic genes and cognitive abilities to determine which alleles should be coded as the risk alleles (Beaver, Vaughn et al., 2010). Briefly, respondents were genotyped for a 40 base pair variable number of tandem repeats (VNTR) in the 3' untranslated region of DAT1 (SLC6A3). For this polymorphism, the 10R allele was coded as the risk allele, while the 9R allele was coded as the non-risk allele. Following the lead of prior researchers (Hopfer et al., 2005), alleles other than the 9R and 10R were removed from the analysis. The second dopaminergic polymorphism included in the current study was the DRD2/ANKK1 TaqIA polymorphism. For DRD2/ANKK1, the A1 allele was scored as the risk allele and the A2 allele was scored as the non-risk allele. Last, DRD4 has a 48 base pair VNTR located at 11p15.5 on exon III. Two groups of alleles were created: one that included the 2R, 3R, 4R, 5R, and 6R alleles and one that included the 7R, 8R, 9R, and 10R alleles. The group of alleles that contained alleles of 7R or greater were coded as the risk alleles while the other group of alleles were coded as the non-risk alleles. All of the polymorphisms were coded co-dominantly, where the value indexed the number of risk alleles that each respondent possessed (0 risk alleles, 1 risk allele, or 2 risk alleles).

Following prior research indicating that the combination of genes (as opposed to each gene in isolation) tends to have the strongest and most consistent effects on human phenotypes (Belsky & Beaver, forthcoming; Li et al., 2010), we created an additive dopamine index (Beaver, Vaughn et al., 2010). To create this index, the scores for each of the three polymorphisms were summed at the individual level with values ranging between zero (0) and six. We then aggregated and averaged the individual-level dopamine scores for each school. The average school-level dopamine score was 2.54, with a standard deviation of 0.28.

### 2.2.3. Percentage African American

We included a measure of percentage African American in the analyses as a control variable. To create this measure, we aggregated and averaged scores on an individual-level self-reported race question, where 0 = white and 1 = African American. The resulting value indexed the percentage of African Americans who were attending the school.

### 2.3. Analytical strategy

The analysis for this study was conducted in two main steps. First, our analysis was focused on the interrelationships between IQ and dopaminergic polymorphisms at the individual-level. Specifically, we estimated the means and standard deviations for IQ by each genotype and we also

examined the bivariate correlations between the dopaminergic genes and IQ scores. In addition, we examined whether IQ scores and dopaminergic scores varied across schools by calculating F-tests. The second step in the analysis was to estimate whether school-level dopamine scores predicted school-level IQ scores before and after controlling for percentage African American. To do so, we conducted ordinary least squares (OLS) regression models.

### 3. Results

The analysis begins by first estimating the association between each of the dopaminergic polymorphisms and individual-level IQ scores. Table 1 presents the means, standard deviations, sample sizes, and correlations for each of the genotypes. The results indicate that DAT1 and DRD2 maintain statistically significant and negative associations with IQ scores, while the effect of DRD4 on IQ is non-significant. To further explore the association between dopaminergic polymorphisms and IQ, we employed the additive dopamine scale as a predictor of IQ scores. The results of this analysis indicated a statistically significant and negative association between IQ and dopamine scores, where higher scores on the dopamine index correspond to lower IQ scores ( $r = -.15$ ,  $p < .05$ , two-tailed test).

We continue our analysis of the individual-level data by examining whether IQ scores and dopamine scores vary significantly across the 36 schools. Our aggregate-level analyses hinge on significant variation across schools in both IQ and dopamine scores, otherwise it would be akin to trying to explain a constant with a constant, a variable with a constant, or a constant with a variable. The results of the F-tests revealed that IQ scores varied significantly across schools ( $F = 11.227$ ,  $p < .05$ ) as do dopamine scores ( $F = 2.239$ ,  $p < .05$ ). Fig. 1 reveals additional support that IQ scores and dopamine scores vary significantly across schools. The distributions in this figure reveal the scores for IQ and dopamine, respectively, across schools and clearly indicate a significant amount of dispersion for both variables.

The next set of analyses examines the association between school-level dopamine scores and school-level IQ. Model 1 in Table 2 shows the results of the bivariate analyses revealing a strong and statistically significant negative association between dopamine scores and IQ scores (as measured with a standardized regression coefficient [i.e., Beta]). Given that allelic distributions for certain genes and IQ scores both vary across race/ethnicity, it is possible that the results would be rendered spurious by the confounding effects of race. As a result, in Model 2 we introduce the percentage of African American variable. As can be seen, even after including race in

**Table 1**  
Means, standard deviations, and correlations for IQ by dopaminergic genotypes (N = 1265).

	DAT1			DRD2			DRD4		
	9R/9R	9R/10R	10R/10R	A2/A2	A1/A2	A1/A1	<7R/<7R	<7R/≥7R	≥7R/≥7R
Mean	102.29	99.49	97.42	99.84	97.47	92.97	98.67	97.82	97.07
SD	13.05	14.22	14.56	13.88	14.53	15.79	14.54	14.10	15.21
N	56	428	781	659	502	104	814	397	54
r		-.09*			-.13*			-.03	

\* Significant at the .05-level, two-tailed tests.

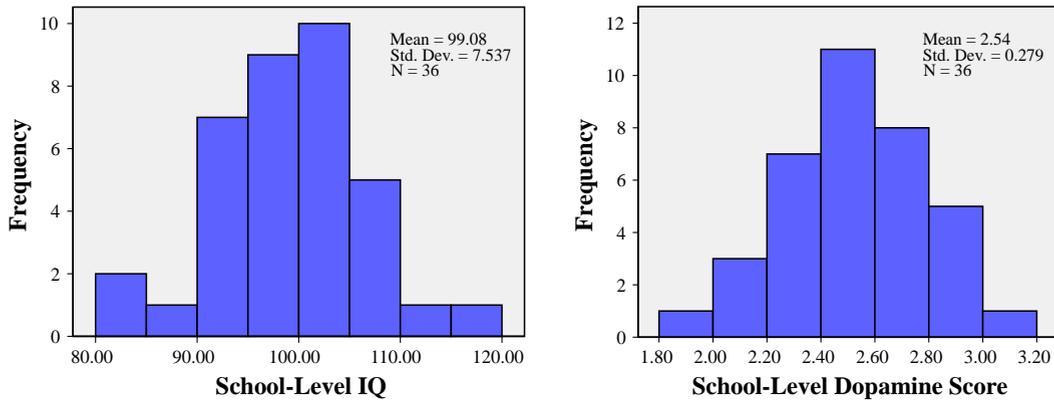


Fig. 1. Distributions of IQ scores and dopamine scores across schools.

the analysis, the partial correlation between school-level dopamine scores and school-level IQ scores remained large and statistically significant.

Last, to examine convergence in the results generated at the individual level with those generated at the school level, we plotted predicted IQ scores across scores on the dopamine scale index. The dopamine scale indexes were z-transformed so that the individual-level analysis could be compared with the school-level analysis. Fig. 2 portrays these plots and shows a high degree of convergence in the slopes and by implication the predicted values, where IQ scores decrease as the total number of risk alleles increases.

4. Discussion

Research has consistently revealed that IQ and other measures of cognitive abilities vary significantly across macro-level units of analysis, such as states, nations, and even schools. Although various explanations have been set forth to explain variation in IQ at the macro-level, the most controversial explanation is that genetic variation across macro-level units explains variation in IQ. To this point, however, empirical research had not directly examined this potential link. The current study partially addressed this gap in the literature by examining whether variation in IQ at the school level was associated with dopaminergic scores aggregated to the school-level. Analysis of data drawn from the Add Health revealed support in favor of this position, where schools that had higher dopamine scores were the same schools that had, on average, lower IQ scores.

Our results also examined the association between dopaminergic polymorphisms and IQ at the individual level.

Consistent with prior research (e.g., Beaver, DeLisi et al., 2010; Berman & Noble, 1995), the associations between dopaminergic genes and individual-level IQ scores were either small and statistically significant or non-significant. Recall, however, that the association between school-level dopamine scores and school-level IQ scores was relatively large in magnitude, which necessarily begs the question of why the effects differed so markedly. While not exhaustive we offer two potential explanations. First, given the small sample size that was employed in the school-level analysis, our statistical power to detect small-to-moderate effect sizes was severely compromised and detecting large effect sizes could be due, in part, to methodological and statistical artifacts. We addressed this possibility by comparing the predicted values of IQ scores at the individual- and school-levels of analysis. The results of these models converged suggesting that the significant effects at the school-level are not solely due to a methodological or statistical artifact.

Second, it is well known that findings detected at one level of analysis cannot be extrapolated to other levels of aggregation (Piantadosi, Byar, & Green, 1988; Samuelson, 1955). This

Table 2

OLS regression models examining the association between school-level dopamine scores and school-level IQ scores.

	Model 1			Model 2		
	b	SE	Beta	b	SE	Beta
School-level dopamine score	-12.57	4.1	-.47*	-9.56	3.8	-.35*
Percentage African American				-10.58	3.6	-.42*

\* Significant at the .05-level, two-tailed tests.

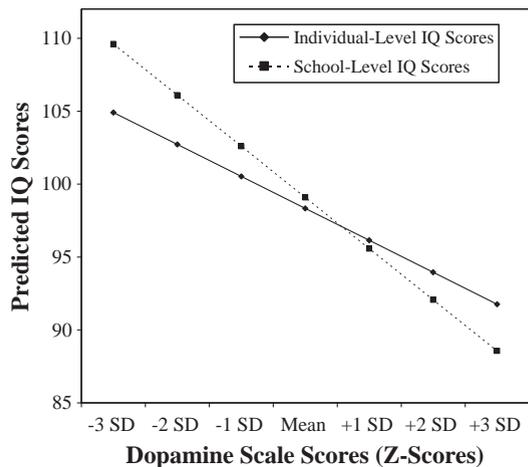


Fig. 2. Predicted IQ scores for individuals and schools across scores on the dopamine scale.

phenomenon is particularly salient in the social sciences where research often spans multiple units of analysis, but the effects can differ considerably among units of analysis (Kramer, 1983). Criminological research, for instance, consistently reveals a strong and robust association between poverty and crime rates among macrosocial units (e.g., states or neighborhoods), while the association between poverty and criminal involvement at the individual-level is weak and oftentimes non-significant. It is quite possible that this pattern also applies to genetic research, where the usual small effects of single genes detected at the individual level become much larger at higher levels of aggregation. Future research will need to explore this possibility in much greater detail.

To our knowledge, this is the first study to aggregate DNA markers to a unit of analysis higher than the individual. Moreover, this is the first study to our knowledge that has revealed that variation in aggregate IQ scores is associated with variation in aggregate DNA markers. These results are in line with Lynn and Vanhanen's (2002, 2006) (see also Hart, 2007; Rushton, 1997) thesis that the average IQ of nations is the result of genetic differences across those nations. Of course, the current study used schools, not nations, as the unit of analysis, meaning that the results reported here may not generalize to other levels of aggregation, including the nation level. There is good reason to believe, however, that the association between DNA and IQ would be even stronger at the nation level in comparison with the school level. There is much more variation in both genetic markers and IQ scores cross-nationally than there is across schools. Schools in the current study were all drawn from the same country (i.e., the United States) creating more genetic homogeneity among schools than there is among nations. Given that nations can vary quite drastically in terms of the allelic distributions of certain genes (Cavalli-Sforza, Menozzi, & Piazza, 1994), it stands to reason that this increased genetic variation would be able to explain more of the variance in IQ scores. Future research is needed to address this issue more fully and examine whether the link between DNA markers and IQ scores would be detected at other levels of aggregation.

The results of the current study provide some of the first evidence indicating that IQ scores across macro-level units are the result of genetic factors. As with all research, though, the current study is host to at least three limitations that need to be rectified in follow-up studies. First, only three dopaminergic genes were used to create the dopamine scale. Although the dopaminergic system has previously been linked to IQ (Beaver, DeLisi et al., 2010; Berman & Noble, 1995; Previc, 1999), future research would benefit by examining a broader range of genes from the dopaminergic system and other systems of genes that may be linked to IQ. Second, the data that were available only allowed for IQ and DNA to be aggregated to the school level. It would be interesting to examine what types of associations are visible at other levels of aggregation, including the neighborhood level, the state level, and the nation level. Third, the measure of IQ was based on scores garnered from the PVT, a test designed to assess verbal skills. Whether the results would be observed using different measures of IQ is an empirical question awaiting future research. Until these limitations are addressed, the results of the current study should be interpreted with caution. If future researchers are able to replicate these findings, then the results would begin to provide additional support that

cross-national inequalities may be produced, in part, by genetic variation.

## References

- Beaver, K. M., DeLisi, M., Vaughn, M. G., & Wright, J. P. (2010). Association between the A1 allele of the DRD2 gene and reduced verbal abilities in adolescence and early adulthood. *Journal of Neural Transmission*, *117*, 827–830.
- Beaver, K. M., Vaughn, M. G., Wright, J. P., DeLisi, M., & Howard, M. O. (2010). Three dopaminergic polymorphisms are associated with academic achievement in middle and high school. *Intelligence*, *38*, 596–604.
- Beaver, K. M., & Wright, J. P. (2011). The association between county-level IQ and county-level crime rates. *Intelligence*, *39*, 22–26.
- Belsky, J., & Beaver, K. M. (forthcoming). Cumulative-genetic plasticity, parenting, and adolescent self-regulation. *Journal of Child Psychology and Psychiatry*.
- Berman, S. M., & Noble, E. P. (1995). Reduced visuospatial performance in children with the D2 dopamine receptor A1 allele. *Behavior Genetics*, *25*, 45–58.
- Braithwaite, J. (1989). *Crime, shame, and reintegration*. New York NY: Cambridge University Press.
- Cavalli-Sforza, L. L., Menozzi, P., & Piazza, A. (1994). *History and geography of human genes*. Princeton NJ: Princeton University Press.
- Diamond, J. (1997). *Guns, germs, and steel: The fates of human societies*. New York, NY: W. W. Norton.
- Harris, K. M., Florey, F., Tabor, J., Bearman, P. S., Jones, J., & Udry, J. R. (2003). The national longitudinal study of adolescent health: Research design [www document], <http://www.cpc.unc.edu/projects/addhealth/design> URL.
- Harris, K. M., Halpern, C. T., Smolen, A., & Haberstick, B. C. (2006). The national longitudinal study of adolescent health (Add Health) twin data. *Twin Research and Human Genetics*, *9*, 988–997.
- Hart, M. H. (2007). *Understanding human history: An analysis including the effects of geography and differential evolution*. Augusta, GA: Washington Summit.
- Herrnstein, R. J., & Murray, C. (1994). *The bell curve: Intelligence and class structure in American life*. New York, NY: Free Press.
- Hopfer, C. J., Timberlake, D., Haberstick, B. C., Lessem, J. M., Ehringer, M. A., Smolen, A., et al. (2005). Genetic influences on quantity of alcohol consumed by adolescents and young adults. *Drug and Alcohol Dependence*, *78*, 187–193.
- Kanazawa, S. (2008). IQ and the health of states. *Biodemography and Social Biology*, *54*, 200–213.
- Kramer, G. H. (1983). The ecological fallacy revisited: Aggregate- versus individual-level findings on economics and elections, and sociotropic voting. *The American Political Science Review*, *77*, 92–111.
- Li, S., Zhao, J. H., Luan, J., Ekelund, U., Luben, R. N., Khaw, K. -T., et al. (2010). Physical activity attenuates the genetic predisposition to obesity in 20,000 men and women from EPIC-Norfolk prospective population study. *PLoS Medicine*, *7*, 1–9.
- Lynn, R., & Vanhanen, T. (2002). *IQ and the wealth of nations*. Westport: Praeger.
- Lynn, R., & Vanhanen, T. (2006). *IQ & global inequality*. Augusta, GA: Washington Summit Publishers.
- McDaniel, M. A. (2006). Estimating state IQ: Measurement challenges and preliminary correlates. *Intelligence*, *34*, 607–619.
- Messner, S. F., & Rosenfeld, R. (1994). *Crime and the American dream*. Belmont, CA: Wadsworth.
- Pesta, B. J., McDaniel, M. A., & Bertsch, S. (2010). Toward an index of well-being for the fifty U.S. states. *Intelligence*, *38*, 160–168.
- Piantadosi, S., Byar, D. P., & Green, S. B. (1988). The ecological fallacy. *American Journal of Epidemiology*, *127*, 893–904.
- Previc, F. H. (1999). Dopamine and the origins of human intelligence. *Brain and Cognition*, *41*, 299–350.
- Rowe, D. C., Jacobson, K. C., & Van den Oord, E. J. C. G. (1999). Genetic and environmental influences on vocabulary IQ: Parental education level as moderator. *Child Development*, *70*, 1151–1162.
- Rushton, J. P. (1997). *Race, evolution, and behavior: A life history perspective*. New Brunswick, NJ: Transaction.
- Saab, H., & Klinger, D. (2010). School differences in adolescent health and wellbeing: Findings from the Canadian health behaviour and school-aged children study. *Social Science & Medicine*, *70*, 850–858.
- Samuelson, P. A. (1955). *Economics: An introductory analysis*. New York, NY: McGraw-Hill.
- Udry, J. R. (2003). *The national longitudinal study of adolescent health (Add Health), waves I and II, 1994–1996; wave III, 2001–2002 [machine-readable data file and documentation]*. Chapel Hill, NC: Carolina Population Center, University of North Carolina.
- Weissberg, R. (2010). *Bad students, not bad schools*. New Brunswick, NJ: Transaction.