Intelligence 41 (2013) 428–438

Contents lists available at SciVerse ScienceDirect

Intelligence

The genetic and environmental architecture to the stability of IQ: Results from two independent samples of kinship pairs

Kevin M. Beaver [a,b,*], Joseph A. Schwartz [a], Eric J. Connolly [a], Joseph L. Nedelec [c], Mohammed Said Al-Ghamdi [b], Ahmed Nezar Kobeisy [b]

[a] College of Criminology and Criminal Justice, Florida State University, Tallahassee, FL 32306-1127, USA
[b] Center for Social and Humanities Research, King Abdulaziz University, Jeddah, Saudi Arabia
[c] School of Criminal Justice, University of Cincinnati, Cincinnati, OH 45221-0389, USA

ARTICLE INFO

Article history:
Received 14 May 2013
Received in revised form 11 June 2013
Accepted 13 June 2013
Available online xxxx

Keywords:
Add Health
Siblings
Stability

ABSTRACT

Existing research has revealed that IQ remains relatively stable over the life course, though questions remain about how stable IQ is and whether the stability of IQ varies across different developmental periods of the life course. Despite this stability, there are also questions surrounding the factors that might explain the stability of IQ. Against this backdrop, we conduct bivariate genetic models to estimate genetic, shared environmental, and nonshared environmental influences on the stability of IQ. To do so, we analyze kinship pairs drawn from two separate longitudinal samples: The National Collaborative Perinatal Project (CPP) and the National Longitudinal Study of Adolescent Health (Add Health). Across both samples, IQ was found to be relatively stable. Moreover, the genetic analyses revealed that between 66% and 83% of the stability in IQ was due to genetic factors and between 43% and 69% of the change in IQ was due to genetic factors. The remainder of the stability and change in IQ was the result of a combination of shared and nonshared environmental influences. Importantly, some substantive race differences emerged in respect to genetic and environmental influences on the stability of IQ. We conclude with a discussion of the limitations of the study and avenues for future research.

1. Introduction

The stability of phenotypes across time is an indelible characteristic of human nature. Over long swaths of the life course, it is commonplace to observe extremely high levels of relative stability in virtually every trait and behavior that has been measured. Personality traits, for instance, such as emotionality and self-regulation, have been found to be highly stable from early childhood up through early adulthood (Moffitt, 1990; Tremblay et al., 2004). Antisocial behaviors, moreover, have been found to be highly stable, with the best predictor of future criminal behavior being a history of aberrant and aggressive behaviors (Campbell, Shaw, & Gilliom, 2000; Farrington, 1991). Even different psychopathologies, including depression (Zuroff, Blatt, Sanislow, Bondi, & Pilkonis, 1999), schizophrenia (Heaton et al., 2001), and bulimia (Joiner, Heatherton, & Keel, 1997), have consistently been shown to be a stable feature in the lives of certain people. With these findings in mind, it is probably not surprising that cognitive abilities, including intelligence (IQ), have also been found to be relatively stable over different sections of the life course (Raguet, Campbell, Berry, Schmitt, & Smith, 1996; Deary, Whalley, Lemmon, Crawford, & Starr, 2000).

Although IQ has been found to be stable, there still remains a degree of uncertainty over the precise mechanisms that are involved in producing this stability. Certainly different theoretical arguments have been made and there has been a good deal of research examining the factors that might give rise to

* Corresponding author at: College of Criminology and Criminal Justice, Florida State University, 634 W. Call Street, Tallahassee, FL 32306-1122, USA. Tel.: +1 850 644 9180; fax: +1 850 644 9614.
E-mail address: kbeaver@fsu.edu (K.M. Beaver).

0160-2896/$ – see front matter © 2013 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.intell.2013.06.012

stability and change in IQ (Carlson & Corcoran, 2001; Ellis & Bonin, 2003; van den Oord and Rowe, 1999). Based on the existing literature, however, it appears as though the most consistent finding is that stability in IQ is the result of both genetic and environmental factors (Jensen, 1981; Plomin, DeFries, Knopik, & Neiderhiser, 2013). The extent to which genetic and environmental factors affect IQ stability remains undetermined, in part, because genetic and environmental influences appear to be contingent on an array of factors. For instance, genetic and environmental effects on variance in IQ scores have been shown to vary across different sections of the life course (Plomin, Fulker, Corley, & DeFries, 1997; Deary, Johnson, & Houlihan, 2009) and across different geographical regions (Lynn & Vanhanen, 2006). Against this backdrop, the current study is designed to add to the existing IQ literature in three key ways. First, we examine the stability of IQ from the age of 4 years old to the age of 7 years old in a sample of sibling pairs drawn from a large and widely analyzed sample of Americans. Second, we estimate the stability of IQ from adolescence into young adulthood in a different sample of sibling pairs drawn from a prospective and nationally representative sample of American youth. Third, we estimate the genetic and environmental contributors to the stability of IQ in both samples.

2. The stability of IQ

A body of empirical research has examined the stability of IQ. Although the studies analyze very different samples, employ different statistical techniques to assess stability, estimate stability at different developmental time periods and with different lag times, the findings tend to converge on the same general conclusion—that IQ is relatively stable (Deary et al., 2000; Deary, Strand, Smith, & Fernandes, 2007; Larson, Hartmann, & Nyborg, 2008). Given the heterogeneity in studies, identifying a precise stability estimate that would apply to all studies is not possible. Nonetheless, there are three main conclusions that can be drawn from the existing literature on the stability of IQ. First, and most importantly, IQ tends to be relatively stable across different developmental time periods, with most stability estimates ranging between $r = .50$ and $r = .80$. The precise stability estimate fluctuates across studies based on study-specific factors, such as sample characteristics and the measure of IQ, but across almost all studies there are at least moderate levels of stability in IQ. Second, although stability estimates remain relatively high, they become attenuated as the time between observations increases. This general pattern is not unique to the stability of IQ, but rather has been found in respect to most other phenotypes, including antisocial behaviors (Moffitt, 1993). Third, the stability of IQ tends to increase as childhood progresses, and by adulthood, the stability of IQ reaches its pinnacle. It is not uncommon, for example, to have studies report that the stability of IQ in childhood is around $r = .40–.50$, but in late adulthood these stability estimates are usually around $r = .80$ (Larson et al., 2008).

The available evidence thus suggests that IQ is stable, that stability estimates are highest when the time lag is relatively short, and that IQ becomes increasingly stable from childhood to adulthood. Given that IQ is stable over time, a wave of research has examined the various factors that might be able to explain the stability of IQ. Some of the most illuminating research on this topic comes from studies which estimate the genetic and environmental influences on the stability of IQ by analyzing samples of kinship pairs. This approach represents a variant of the widely used methodology that decomposes variance in a single phenotype. Typically, twin pairs are analyzed with this methodology. In doing so, the similarity of monozygotic (MZ) twin pairs is compared to the similarity of dizyogtic (DZ) twin pairs. As long as the assumptions of the twin-based methodology are fulfilled, the only reason that MZ twins (from the same twin pair) should be more similar to DZ twins (from the same twin pair) is because MZ twins share twice as much genetic material as DZ twins. The greater the similarity of MZ twins versus the similarity of DZ twins, the stronger the genetic effect. In the parlance of behavioral genetic research, the proportion of variance accounted for by genetic factors is referred to as heritability.

The proportion of variance that is not accounted for by heritability must be accounted for by environmental influences (and error). The twin-based methodology, however, distinguishes between two types of environmental influences: shared environmental influences and nonshared environmental influences. Shared environmental influences capture the effects of environments that are the same between twins and that make them similar to each other. Nonshared environmental influences, in contrast, capture the effects of environments that are unique to each twin and/or that make twins dissimilar from each other. It is also important to note that the effects of error are pooled into the nonshared environmental component. Together, heritability, the shared environment, and the nonshared environment account for 100% of the variance in any phenotype that is being studied.

The methodology described above is frequently referred to as a univariate model because it examines the variance components (i.e., heritability, shared environmental, and nonshared environmental) in a single variable. An extension of the univariate model is the bivariate model which can be used to examine the genetic, shared environmental, and nonshared environmental influences on the covariance between two variables. This type of methodology has direct application to examining the stability of IQ as it is typically assessed by measuring IQ at two different periods of time. The stability coefficient therefore represents the degree of covariance between IQ scores at one point in time and IQ scores at a different point in time and, as a result, is a bivariate model. With bivariate decomposition models, the focus is on the covariance between the two variables (i.e., the stability). The covariance is decomposed into the proportion that is the result of genetic influences (i.e., heritability), the proportion that is the result of shared environmental influences, and the proportion that is the result of nonshared environmental influences. Once again, these three components will account for 100% of the covariance between the two variables.

A line of research has employed bivariate decomposition models to examine the genetic and environmental influences on the stability of IQ. Three main findings can be garnered from this body of research. First, and quite generally, the stability of IQ has been found to be strongly affected by genetic influences, with estimates hovering around 60% (Boomsma & van Baal, 1998). The genetic origins to the stability of IQ

have been detected in childhood (LaBuda, DeFries, Plomin, & Fulker, 1986; Boomsma & van Baal, 1998; Cherny et al., 2001; Trzaskowski, Yang, Visscher, & Plomin, 2013), adolescence (Bishop et al., 2003; Hoekstra, Bartels, & Boomsma, 2007), and adulthood (Bouchard, 1998; Hoekstra et al., 2007), with some evidence suggesting that the genetic influences on stability increase with age (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Petrill et al., 2004; Bergen, Gardner, & Kendler, 2007; Haworth et al., 2010). For example, Plomin, Pedersen, Lichtenstein, and McClearn (1994) analyzed data from the Swedish Adoption/Twin Study of Aging and reported that genetic factors accounted for 90% of the stability in IQ during adulthood, a heritability estimate that tends to be much larger than those that are generated with samples of children, adolescents, or young adults. Second, environmental influences have also been found to account for some of the stability in IQ (Bishop et al., 2003; Davis, Arden, & Plomin, 2008). Earlier in life, shared environmental influences appear to be slightly more influential than nonshared environmental influences (Plomin et al., 1993), though later in life genetic influences tend to emerge as the most salient (Plomin et al., 1994). Third, changes in IQ over time also have been found to be affected by a combination of genetic, shared environmental, and nonshared environmental influences (Bishop et al., 2003; Davis et al., 2008; for a review see Thompson, 1993). The precise estimates, however, vary and appear to be a function of the developmental time period being studied, the exact measure of IQ, and other sample-specific characteristics.

3. The current study

Given the heterogeneity in effect sizes owing to differences in studies, the current study examined the genetic and environmental architecture to the stability in IQ in two distinct samples. The first sample consisted of children who were born in the 1950s and assessed on IQ at age 4 and again at age 7. The second sample consisted of youth who were in middle and high school in the 1990s and who were assessed on vocabulary IQ during adolescence and again in early adulthood. By using two samples we will not only be able to compare the results of the current study with those previously published, but we will also be able to compare the findings generated in this study across a period of development beginning in early childhood and ending in early adulthood.

4. Methods

4.1. Data

4.1.1. National Collaborative Perinatal Project (CPP)

The CPP was used to examine the stability and change in IQ during early childhood. The CPP is a prospective birth cohort study which initiated in 1959 and continued subject enrollment until 1965. Pregnant women were recruited for inclusion in the study from 12 university affiliated hospitals across the United States, typically during their first clinical visit. Various medical professionals collected extensive data during each mother's pregnancy, during delivery, and after the birth of the child. Additional information was collected regarding each mother's lifestyle including socioeconomic information, exposure to potential carcinogens or chemicals, and substance use (Hardy, 2003). Overall, the CPP sample included approximately 60,000 pregnancies and over 58,000 live births (Hardy, 2003).

Children included in the sample were subjected to a wide variety of tests taking place across multiple ages. More specifically, children were subjected to various tests carried out by trained medical and psychological professionals tapping multiple aspects of development including general health, neurological functioning, physical and mental development, and behavioral patterns. Children included in the CPP underwent extensive examinations at five time periods: 4 months, 8 months, 1 year, 4 years, and 7 years. The results of multiple studies indicate that sample attrition was relatively minimal, with retention rates ranging between 75 and 88% across each time period (Hardy, 2003; Klebanoff, 2008). The resulting data cover a critically important developmental period—early childhood—and provide information on a wide variety of subject areas. Importantly, several aspects of the 4-year and 7-year examinations were aimed at directly assessing the cognitive and neurological development of the children included in the sample (Nichols & Chen, 1981).

Nested within the overall sample of children in the CPP is a large subsample of twin births. In total, there were 615 pairs of twins born during the CPP study period. Zygosity was determined via blood typing and both microscopic and gross examination of the placenta. Of the 615 twin births, 187 were monozygotic (MZ) twin pairs, 112 were same-sex dizygotic (DZ) twin pairs, and 204 were different-sex DZ twin pairs (316 total DZ twin pairs). In addition, there were 117 twin pairs whose zygosity was not determined (Myrianthopoulos, 1970). In the current study, all ambiguous twin pairs were coded half way between MZ and DZ twin status ($r = .75$).[1]

In addition to twin births, the CPP also contains over 6,000 women who were registered for more than one pregnancy over the study period. Consequently, various types of non-twin kinship pairs (e.g., full siblings, half siblings) can be identified within the full sample by assessing whether siblings born to the same mother shared the same father. Siblings who share the same father were coded as full siblings and siblings who have different fathers (but the same mother) were coded as half siblings. Importantly, these coding procedures have been used in prior research (Nichols & Chen, 1981). For all households with more than one sibling pair, a single pair was randomly selected, but all twin pairs were retained with certainty. In this way, for all households with a twin pair and additional sibling pairs, only the twin pair was retained in the final analytical sample. In all, the current study includes MZ twin pairs, ambiguous twin pairs, DZ twin pairs, full-sibling pairs, and half-sibling pairs.

4.1.2. National Longitudinal Study of Adolescent Health (Add Health)

The Add Health was used to examine the stability and change in IQ during adolescence and young adulthood. The

[1] The analyses were repeated using two additional coding schemes for the ambiguous twin pairs. First, all ambiguous twins pairs were omitted from the final analytic sample. Second, all ambiguous twin pairs were coded as DZ twins ($r = .50$). In both sets of supplemental analyses, the overall pattern of results was not substantively different from those reported in the current study.

Add Health is a prospective nationally representative sample of American youths consisting of multiple waves of data collection. The first wave of data collection was carried out between 1994 and 1995, and included a nationally representative sample of middle and high schools in the United States. Over 90,000 youths participated in the in-school portion of the wave 1 interviews. In an effort to obtain more detailed information, a subsample of youths who participated in the in-school portion of the study were also asked to participate in the in-home portion. More than 20,000 youth—between the ages of 12 and 21—and over 17,000 of their primary caregivers (most often the respondent's biological mother) participated in the in-home portion of the study (Harris et al., 2003; Udry, 2003). Respondents were asked questions pertaining to a wide variety of topics including family relationships, school interactions, and substance use.

The second wave of data collection was carried out during 1995 and 1996, approximately 1–2 years after the completion of wave 1. Due to the short period of time that had passed since the completion of wave 1 of the project, many of the same survey items used during wave 1 interviews were retained in the questionnaires administered during wave 2 interviews. In total, 14,738 youths who participated in the in-home portion of the wave 1 interviews also participated in wave 2 of the study. Wave 3 of the study was carried out between 2001 and 2002, approximately 5 years after the completion of wave 2 interviews and included more than 15,000 youths. Due to the age range of the respondents at wave 3 (between 18 and 26 years old), the administered questionnaires were modified to take into account more age appropriate topics such as employment history, parenting practices, and contact with the criminal justice system (Harris et al., 2003; Udry, 2003).

A unique aspect of the Add Health is that a subsample of sibling pairs from the same household are nested within the full sample (Harris, Halpern, Smolen, & Haberstick, 2006). Sibling pairs were identified during wave 1 interviews by asking respondents whether they had a co-twin or sibling that lived with them. Cousins, genetically unrelated siblings, half-siblings, and co-twins were included in the sample with certainty. In addition, a random sample of full-siblings between the ages of 11 and 20 was also included in the study. In total, over 3,000 sibling pairs were included in the study. The final analytic sample used in the current study was limited to MZ twins, DZ twins, full-sibling pairs, and half-sibling pairs.

5. Measures

5.1. IQ scores

CPP. Mental and motor performance of the children included in the CPP was assessed at age 4 with a wide variety of assessments, including an abbreviated version of the third edition of the Stanford–Binet IQ scale, form L–M (Nichols & Chen, 1981). The abbreviated version of the scale included four of the six items at each age level included in the full scale. A more in-depth discussion of the construction, reliability, and validity of the revised Stanford–Binet scale can be found elsewhere (Broman, Nichols, & Kennedy, 1975). Children included in the CPP sample were reassessed at age 7, at which time they underwent an extensive psychological examination. During the 7 year examination, an abbreviated version of the Wechsler Intelligence Scale for Children (WISC) was administered. Scores on the WISC were used to generate verbal IQ (VIQ), performance IQ (PIQ) and full-scale IQ (FSIQ) scores (Nichols & Chen, 1981). For both IQ measures, all individuals with scores lower than 50 were considered outliers and omitted from the final analytic sample.[2] Importantly, the WISC has been used in previous studies analyzing the CPP (Jusko, Klebanoff, Brock, & Longnecker, 2012; Rushton, 1997; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003).

Add Health. IQ was assessed in the Add Health using vocabulary IQ scores (Rowe, Jacobson, & Van den Oord, 1999). Vocabulary IQ was measured using the Picture Vocabulary Test (PVT), which is an abbreviated version of the full Peabody Picture Vocabulary Test Revised (PPVT-R). The PVT was administered during wave 1 and wave 3 interviews and was standardized by age. Once again, individuals scoring less than 50 on either measure of vocabulary IQ were omitted from the final analytic sample. Importantly, previous studies analyzing the Add Health data have used the PVT as a measure of vocabulary IQ (Rowe et al., 1999; Schwartz & Beaver, 2013; van den Oord & Rowe, 1999).

5.2. Covariates

CPP. Two demographic controls were included in the statistical models involving the CPP data. First, race was measured dichotomously where 0 = White and 1 = African American. Second, gender was also coded dichotomously where 0 = female and 1 = male.

Add Health. Three demographic control variables were included in the analyses involving the Add Health data. First, race was measured as a dichotomous variable where 0 = White and 1 = African American. Second, age was measured as a continuous variable measured in years. Third, gender was measured dichotomously where 0 = female and 1 = male.

5.3. Analysis plan

The analysis for the current study was carried out in four interconnected steps. First, descriptive statistics of all study variables were produced for both the CPP and the Add Health. Second, cross-sibling correlations were estimated for each of the IQ measures across all levels of genetic relatedness separately for each sample. Third, a series of univariate ACE models were fit to all four IQ measures. This behavior genetic modeling strategy allows for the decomposition of variance within a given measure into three separate latent constructs: additive genetic (symbolized as A); shared environmental (symbolized as C); and nonshared environmental (symbolized as E) influences. Variance decomposition is performed by estimating the cross-sibling covariance explained by each construct on a single phenotype. Importantly, the covariance estimates for each latent construct are constrained to reflect the extent to which siblings covary across each construct. Additive genetic influences (A) were constrained based on the level of genetic relatedness between each sibling pair with the correlation between MZ twins constrained to 1.00, the correlation between

[2] Including the outliers in the final analytic sample did not result in a substantive change in the findings of any of the estimated models.

ambiguous twin pairs constrained to .75, the correlation between DZ twins and full siblings constrained to .50, and the correlation between half siblings constrained to .25. Shared environmental influences (C) are constrained to correlate at 1.00 for all sibling pairs since such influences equally impact both siblings. Finally, nonshared environmental influences (E) capture effects that result in differences between siblings from the same household (in addition to measurement error), and were allowed to vary freely. Fig. 1 presents a graphical representation of a univariate ACE model in which the variance within a single IQ measure is decomposed into A, C, and E components.

The fourth step in the analysis involved estimating the extent to which additive genetic, shared environmental, and nonshared environmental factors influenced the stability of IQ from one time period to the next. A bivariate Cholesky decomposition model was calculated to estimate the proportion of covariance between each of the IQ measures within each sample explained by additive genetic, shared environmental, and nonshared environmental influences (Neale & Cardon, 1992). Fig. 2 presents a graphical representation of a bivariate Cholesky decomposition model in which the covariance between IQ measures taken at two time periods is decomposed into A, C, and E components. The path estimates of a11, a21, c11, c21, e11, e21 were used to estimate the influence of A, C, and E on the stability of IQ over time within each sample separately. Corresponding path estimates were multiplied and then summed to estimate the total correlation between two IQ estimates:

$$r = (a11 * a21) + (c11 * c21) + (e11 * e21) \quad (1)$$

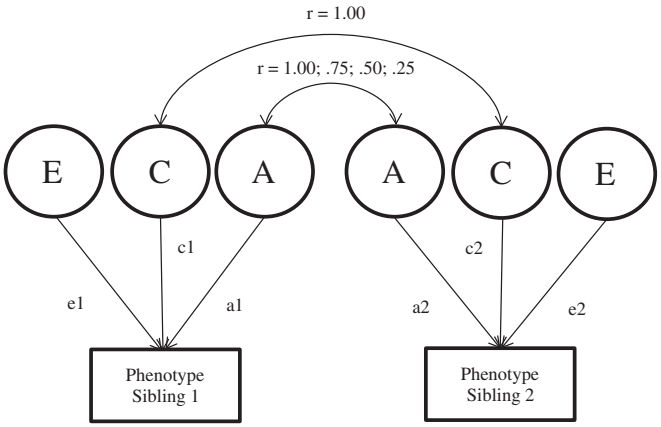
In order to estimate the proportion of the total correlation explained by genetic factors, the product of (a11 * a21) was divided by the value of the total correlation. Similarly, the proportion of the total correlation explained by the shared environment was estimated by dividing the product of (c11 * c21) by the total correlation. Finally, the proportion of the total correlation explained by nonshared environmental factors was estimated by dividing the product of (e11 * 21) by the total correlation. A more detailed description of the analytic procedure can be found elsewhere (Larsson, Larsson, & Lichtenstein, 2004; Neale & Cardon, 1992).

The fifth and final step in the analytic strategy involved estimating the extent to which genetic and environmental factors contribute to changes in IQ from one observation to the next within each sample. The path coefficients of a22, c22, and e22, were used to estimate the influence of A, C, and E on changes in IQ from one observation to the next within each sample separately. In order to estimate overall change in IQ from one observation to the next each path coefficient was squared and then all three were summed ($a22^2 + c22^2 + e22^2$). Each squared coefficient was then divided by the overall change score to estimate the proportion of overall change that was explained by that component. For example, the proportion of overall change explained by genetic influences would be estimated by dividing $a22^2$ by the overall change score.

Importantly, the effects of all demographic control variables were residualized out of each examined outcome measure prior to estimating all univariate ACE and bivariate Cholesky models. This procedure was performed by regressing each outcome measure on all demographic controls, effectively removing any variance that could be attributed to demographic differences between respondents included in either sample. In addition, all models were calculated using a maximum likelihood estimator with robust standard errors (MLR), using the structural equation modeling software M*plus* version 7.1 (Muthén & Muthén, 2012).

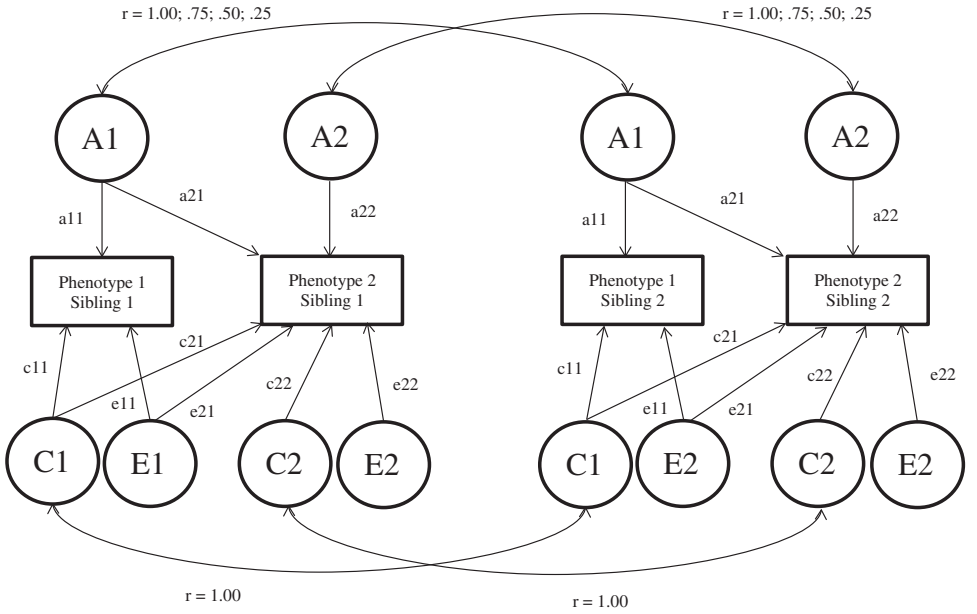
6. Results

The analysis began by producing the descriptive statistics for all study variables within each sample. Means, frequencies, standard deviations, maximum/minimum values, and other descriptive statistics for all variables included in the analysis are presented in Table 1. Within the CPP sample, the average age 4 IQ was 96.73 (SD = 16.45), and the average age 7 IQ was 95.05 (SD = 14.78), with a stability coefficient of .67 ($p < .001$). Additionally, 51.38% of the final analytical



Note: A = genetic influences, C = shared environmental influences, E = nonshared environmental influences.

Fig. 1. Univariate ACE model for all IQ measures.



Note: A1, C1, and E1 represent genetic, shared environmental, and nonshared environmental effects common to IQ at time 1 and time 2; A2, C2, and E2 represent genetic shared environmental, and nonshared environmental effects unique to IQ at time 2.

Fig. 2. Bivariate Cholesky decomposition model for the stability and change in IQ.

sample within the CPP was male and the remaining 46.28% of the sample was female. Approximately 48% of the CPP sample was African American and 52% was White. The mean wave 1 vocabulary IQ score within the Add Health data was 98.19 (SD = 14.19) and the mean wave 3 vocabulary IQ score was 100.41 (SD = 13.04), with a stability coefficient of .68 ($p < .001$). There were slightly more males (50.29%) than females (49.71%) within the Add Health sample. Finally, 74.04% of the Add Health sample was White and 25.98% were African American.

Table 1
Descriptive statistics for CPP and Add Health study variables.

	Mean/Frequency	SD/%	Min–Max
CPP			
Age 4 IQ	96.73	16.45	50–160
Age 7 IQ	95.05	14.78	50–153
Gender			
Male	4259	51.38%	–
Female	4030	46.28%	–
Race			
White	3781	52.08%	–
African American	3479	47.92%	–
Sibling pairs			
MZ twin pairs	187	2.15%	–
Ambiguous twin pairs	117	1.34%	
DZ twin pairs	305	3.50%	–
Full sibling pairs	7194	82.61%	–
Half sibling pairs	905	10.39%	–
Add Health			
Wave 1 Vocabulary IQ	98.19	14.19	51–141
Wave 3 Vocabulary IQ	100.41	13.04	54–123
Age	16.07	1.73	12–21
Gender			
Male	2099	50.29%	–
Female	2075	49.71%	–
Race			
White	2656	74.02%	–
African American	932	25.98%	–
Sibling pairs			
MZ twin pairs	278	13.32%	–
DZ twin pairs	437	20.94%	–
Full sibling pairs	1023	49.02%	–
Half sibling pairs	349	16.72%	–

Note: MZ = monozygotic, DZ = dizygotic.

Table 2 presents the cross-sibling correlation coefficients for each of the examined IQ measures across various levels of genetic relatedness. The results for both the CPP and Add Health samples revealed that the magnitude of the correlation coefficients increased as the level of genetic relatedness increased. For example, within the CPP sample, MZ twins more closely resemble one another across both measures of IQ than ambiguous twins, DZ twins and full sibling pairs, and half-sibling pairs. The same pattern was present within the Add Health data. These results provide preliminary evidence suggesting that genetic factors play a significant role in the development of IQ.

For all univariate ACE models, model fit statistics were used to assess the best fitting and most parsimonious model. Specifically, the comparative fit index (CFI), Tucker–Lewis index (TLI), and root mean square error of approximation (RMSEA) were used to determine goodness of fit. Difference in coefficients Wald's χ^2 tests were used to assess whether constraining any of the components in the baseline ACE model resulted in a more parsimonious model. More specifically, if the constraint of any of the components of the baseline ACE model resulted in a nonsignificant change in χ^2, this model would be considered the most parsimonious and best-fitting model.

Table 3 presents the results of a series of univariate ACE models that decompose the variance of the age 4 and age 7 IQ scores within the CPP sample. As a reminder, the effects of

Table 2
Cross-sibling correlations for CPP and Add Health IQ measures.

CPP	Age 4 IQ	Age 7 IQ
MZ	.78**	.81**
Ambiguous twins	.75**	.62**
DZ twins/full siblings	.57**	.55**
Half siblings	.36**	.34**
Add Health	**Wave 1 Vocabulary IQ**	**Wave 3 Vocabulary IQ**
MZ Twins	.79**	.71**
DZ Twins/Full Siblings	.54**	.55**
Half Siblings	.41**	.43**

** $p < .01$.

race and gender were residualized out of both IQ measures prior to the estimation of the ACE models. The best fitting model for the age 4 IQ measure was the full ACE model with genetic influences explaining 45% of the variance, shared environmental influences explaining 27% of the variance, and nonshared environmental influences explaining the remaining 28% of the variance. For the 7 year IQ measure, the full ACE model fit the data well and constraining any of the components of the baseline model resulted in a significant change in χ^2, indicating that the baseline model was the best fitting and most parsimonious model. The results indicated that 55% of the variance in the 7 year IQ measure was explained by additive genetic influences, 19% was explained by shared environmental influences, and the remaining 26% of the variance was explained by nonshared environmental influences.

Table 4 presents the results of a series of univariate ACE models examining genetic and environmental influences on wave 1 and wave 3 vocabulary IQ measures within the Add Health. Importantly, the effects of age, race, and gender were residualized out of both vocabulary IQ measures prior to the calculation of the ACE models. For the wave 1 vocabulary IQ measure, the full ACE model was the best-fitting model. The results indicated that 47% of the variance in vocabulary IQ at wave 1 was explained by genetic influences, 23% was explained by shared environmental influences, and the remaining 30% was explained by nonshared environmental influences. For the wave 3 vocabulary IQ measure, the full ACE model was also the best fitting model. The results indicated that 49% of the variance in the wave 3 vocabulary IQ measure was explained by genetic influences, 20% of the variance was explained by the shared environment, and the remaining 31% was explained by the nonshared environment.

Table 5 provides the proportion of stability and change in IQ that can be explained by A, C, and E within each sample separately. Unstandardized path coefficients from a bivariate Cholesky decomposition model were utilized to examine genetic and environmental contributions to the stability and change between age 4 and age 7 IQ within the CPP. The results revealed that genetic influences explain 54% of the stability in IQ from age 4 to age 7, while the shared environment explained 41% of the stability and the nonshared environment explained the remaining 5%. Conversely, 60% of the change in IQ between the ages of 4 and 7 was explained by genetic factors, 15% was explained by the shared environment, and the remaining 25% was explained by nonshared environmental influences. A second bivariate Cholesky decomposition model was estimated to examine the stability and change between the wave 1 and wave 3 vocabulary IQ measures in the Add Health. The results indicated that 75% of the stability in vocabulary IQ was explained by genetic influences, 24% was explained by shared environmental influences, and the remaining 1% of the stability was explained by nonshared environmental influences. Alternatively, 61% of the change in vocabulary IQ was explained by genetic influences, 9% was explained by the shared environment, and the remaining 30% was explained by the nonshared environment.

Table 3
Univariate biometric models for age 4 IQ and age 7 IQ: CPP.

	A	C	E	χ^2	$\Delta\chi^2$	CFI	TLI	RMSEA
4 year IQ								
ACE	**.45****	**.27****	**.28****	**173.83****		**.97**	**.98**	**.04**
	(**.33–.57**)	(**.21–.34**)	(**.22–.34**)					
AE	.84**	.00	.16**	261.33**	46.60**	.95	.96	.06
	(.82–.87)	(.00–.00)	(.13–.18)					
CE	.00	.49	.51	176.42**	38.58**	.96	.97	.05
	(.00–.00)	(.47–.51)	(.49–.53)					
E	.00	.00	1.00	1891.37	2090.48**	.56	.67	.16**
	(.00–.00)	(.00–.00)	(1.00–1.00)					
7 year full-scale IQ								
ACE	**.55****	**.19****	**.26****	**132.22****		**.98**	**.99**	**.04**
	(**.42–.67**)	(**.12–.26**)	(**.20–.33**)					
AE	.83**	.00	.17**	178.202**	21.43**	.97	.98	.04
	(.81–.86)	(.00–.00)	(.14–.20)					
CE	.00	.46**	.54**	175.67**	51.63**	.97	.98	.04
	(.00–.00)	(.44–.49)	(.52–.56)					
E	.00	.00	1.00	1744.47**	1756.19**	.64	.73	.15**
	(.00–.00)	(.00–.00)	(1.00–1.00)					

Note: All models were estimated using a maximum likelihood estimator with robust standard errors (MLR).
Race and sex were residualized prior to estimating each model.
Best fitting model bolded.
** $p < .01$.

Table 4
Univariate biometric models for Wave 1 and Wave 3 Vocabulary IQ: Add Health.

	A	C	E	χ^2	$\Delta\chi^2$	CFI	TLI	RMSEA
Wave 1 IQ								
ACE	**.47****	**.23****	**.30****	**47.26**		**1.00**	**1.00**	**.01**
	(**.35–.60**)	(**.15–.31**)	(**.24–.36**)					
AE	.75**	.00	.25**	77.15**	21.63**	.96	.97	.04
	(.70–.79)	(.00–.00)	(.21–.30)					
CE	.00	.48**	.52**	91.04**	38.63**	.95	.96	.04
	(.00–.00)	(.45–.52)	(.48–.55)					
E	.00	.00	1.00	579.63**	690.52**	.40	.49	.14**
	(.00–.00)	(.00–.00)	(1.00–1.00)					
Wave 3 IQ								
ACE	**.49****	**.20****	**.31****	**36.81**		**1.00**	**1.01**	**.00**
	(**.33–.65**)	(**.10–.30**)	(**.23–.39**)					
AE	.72**	.00	.28**	53.17	10.56**	.99	.99	.02
	(.66–.78)	(.00–.00)	(.22–.34)					
CE	.00	.46**	.54**	77.74**	25.24**	.95	.96	.04
	(.00–.00)	(.42–.50)	(.50–.58)					
E	.00	.00	1.00	342.23**	365.85**	.56	.63	.11*
	(.00–.00)	(.00–.00)	(1.00–1.00)					

Note: All models were estimated using a maximum likelihood estimator with robust standard errors (MLR).
Race and sex were residualized prior to estimating each model.
Best fitting model bolded.
* $p < .05$.
** $p < .01$.

6.1. Supplemental analyses

Given that there are important racial differences in IQ (Jensen, 1998), we also examined whether the bivariate Cholesky models revealed a similar pattern of results for Whites and African Americans by reestimating the models within race.[3] Table 6 presents the results of these supplemental analyses. As can be seen, for the White CPP sample, genetic factors explained 36% of the stability in IQ from age 4 to 7, while the shared environment explained 60% of the stability and the nonshared environment explained the remaining 4%. Moreover, 50% of the change in IQ was explained by genetic influences, 26% was explained by the shared environment, and the remaining 24% of the change was explained by nonshared environmental influences. For the White Add Health sample, 79% of the stability in vocabulary IQ was explained by genetic factors and the remaining 21% of the stability was explained by the shared environment whereas 73% of the change in vocabulary IQ was explained by genetic influences, 7% was explained by the shared environment, and the remaining 20% was explained by nonshared environmental influences.

The second set of columns in Table 6 presents the results for African-American subsamples within the CPP and the Add Health. The results of these models revealed that 73% of the stability in IQ in the CPP sample was explained by genetic factors, 22% was explained by shared environmental factors, and the remaining 6% of the stability was explained by the nonshared environment. In addition, 66% of the change in IQ was explained by genetic factors, 8% was explained by the shared environment, and the remaining 25% of the change was explained by nonshared environmental influences. Similarly, 88% of the stability in vocabulary IQ in the Add Health sample was explained by genetic factors and the remaining 12% of the stability was explained by shared environmental influences. Finally, 48% of the change in vocabulary IQ was explained by genetic influences, 6% was explained by the shared environment, and the remaining 47% was explained by nonshared environmental influences.

[3] The univariate ACE models were also reestimated separately for Whites and African Americans within the CPP and the Add Health. The overall pattern of results from these models was similar to results presented in Tables 3 and 4, with most of the estimates having overlapping confidence intervals. For this reason, the results of the race-specific univariate models are not presented.

7. Discussion

Studies examining the effects of IQ on later-life outcomes have found significant associations, with IQ scores in childhood and adolescence predicting behaviors, educational achievement, occupational choices, and salary well into adulthood (Jensen, 1998; Deary, 2012). This should not be too surprising given that IQ scores have been found to be relatively stable over long periods of time. Even so, there remains much to be learned about the stability of IQ within different periods of human

Table 5
Bivariate biometric models for stability and change in IQ for CPP and Add Health samples.

	A	C	E
Age 4 to Age 7 IQ (CPP)			
Stability	.54	.41	.05
Change	.60	.15	.25
Wave 1 to Wave 3 IQ (Add Health)			
Stability	.75	.24	.01
Change	.61	.09	.30

Notes: All models were estimated using a maximum likelihood estimator with robust standard errors (MLR).
Race and sex (and age for models analyzing data from the Add Health) were residualized prior to estimating each model.

Table 6
Bivariate biometric models for stability and change in IQ for White and African-American subsamples within the CPP and Add Health.

	White subsample			African American subsample		
	A	C	E	A	C	E
Age 4 to Age 7 IQ (CPP)						
Stability	.36	.60	.04	.73	.22	.06
Change	.50	.26	.24	.66	.08	.25
Wave 1 to Wave 3 IQ (Add Health)						
Stability	.79	.21	.00	.88	.12	.00
Change	.73	.07	.20	.48	.06	.47

Notes: All models were estimated using a maximum likelihood estimator with robust standard errors (MLR).
Sex (and age for models analyzing data from the Add Health) was residualized prior to estimating each model.

development and, moreover, there remains much to be learned about the various mechanisms that account for the stability of IQ over different developmental time periods. The current study was designed to shed some light on these issues by examining the stability of IQ in two independent samples of kinship pairs. The results generated from the analyses revealed three main findings.

First, and in line with previous research (Deary et al., 2000; Raguet et al., 1996), IQ scores were found to be relatively stable in both samples. More specifically, the CPP data were used to assess the stability of IQ over a three-year time period during childhood. The results revealed a relatively high stability coefficient of $r = .67$. Very similar results were generated with the Add Health data wherein the stability of vocabulary IQ over a seven-year time period from adolescence to adulthood was $r = .68$. Taken together these findings suggest that IQ is relatively stable and this stability was detected in childhood and in adolescence/young adulthood and did not appear to decrease as the time lag between waves increased.

The second main finding to emerge from the analyses was that a combination of genetic and environmental factors accounted for the wave-to-wave stability in IQ scores, with the majority of stability being attributable to genetic factors. For the CPP, 54% of the stability was due to genetic factors, 41% was the result of shared environmental factors, and 5% was accounted for by nonshared environmental influences. Similarly, for the Add Health, 75% of the stability was due to genetic factors, 24% was due to shared environmental influences, and 1% was the result of nonshared environmental factors. Comparing across the two samples, the genetic influence on stability appears to increase from childhood to adolescence/adulthood, while the shared environmental influence decreases. These findings comport with those reported previously showing that genetic influences on IQ increase across the life course whereas shared environmental influences tend to decrease (Plomin et al., 1993, 1994). Importantly, in the race-specific models, genetic factors were more influential for the stability of IQ for African-Americans when compared to Whites. Given the lack of existing research on this topic, replication studies are needed to determine whether similar estimates would be garnered in other samples.

Third, statistical models were also estimated revealing that genetic, shared environmental, and nonshared environmental influences accounted for changes in IQ scores. Once again, genetic factors accounted for most of the change. In the CPP data, genetic influences accounted for 60% of the change, shared environmental factors accounted for 15% of the change, and nonshared environmental influences accounted for 25% of the change. In the Add Health, the genetic effect accounted for 61% of the change, while the shared environmental and nonshared environmental factors accounted for 9% and 30% of the change, respectively.

Given how central IQ is to life outcomes (Deary, 2012), it is not surprising that there is a great deal of interest in determining the factors that affect the unfolding of IQ across the life course (Jensen, 1998). There have been efforts to increase IQ in childhood and adolescence, as well as a great deal of interest in trying to bring relatively low IQ scores up to the normal range of variation. However, for the most part these efforts have been relatively unsuccessful (Jensen, 1969, 1998; Rushton & Jensen, 2005). Perhaps part of the reason that improving IQ scores is so difficult is because they are largely influenced by genetic factors and genetic factors also underlie the stability of IQ. Of course, genetic influences do not represent a fixed, immutable effect, but rather fluctuate based on exposure to different environments (Turkheimer et al., 2003). As a result, future efforts designed to improve IQ scores and perhaps alter IQ trajectories should begin to examine the various ways that genetic influences can be manipulated through concerted intervention efforts.

Although this study analyzed two separate samples to estimate the stability of IQ and the genetic and environmental underpinnings to such stability, there are a number of limitations that need to be addressed in future studies. First, although the stability of IQ was estimated in childhood and in adolescence/adulthood, we were unable to examine the stability of IQ from childhood to adulthood. It would be interesting to examine the stability of IQ over the entire life course and to estimate the genetic and environmental influences on IQ during this time period. Second, the CPP data are somewhat outdated as they were collected in the 1950s. Whether the findings generated from this sample would generalize to more contemporary samples remains to be determined, though it is important to point out that the estimates are in line with those reported in more recent samples (e.g., Boomsma & van Baal, 1998). Third, the Add Health data only included measures of vocabulary IQ at waves 1 and 3 and so we were only able to assess the stability of IQ in the Add Health data from vocabulary IQ scores. Whether this same pattern of findings would have been detected with other measures of IQ awaits future research.

Acknowledgments

This research uses data from Add Health, a program project designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris, and funded by a grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies. Special acknowledgment is due to Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 W. Franklin Street, Chapel Hill, NC 27516-2524

(addhealth@unc.edu). No direct support was received from grant P01-HD31921 for this analysis.

This research uses data from the Modernization and Enhancement of the Collaborative Perinatal Project conducted by J.P. Lawlor, E. Gladen, D. Dhavale, D. Tamagoglu, J.B. Hardy, A.K. Duggan, W.W. Eaton, and E.F. Torrey. "Modernization and Enhancement of the Collaborative Perinatal Project (1959–74)", The Johns Hopkins University 2005. Data can be found at ftp://sph-ftp.jhsph.edu/cpp. The project was funded by the Stanley Medical Research Institute, Bethesda MD and NIMH grant 070333. No direct support was received from these sources for this analysis.

References

Bartels, M., Rietveld, M. J. H., Van Baal, G. C. M., & Boomsma, D. I. (2002). Heritability of educational achievement in 12-year-olds and the overlap with cognitive ability. *Twin Research and Human Genetics*, *5*, 544–553.

Bergen, S. E., Gardner, C. O., & Kendler, K. S. (2007). Age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood: A meta-analysis. *Twin Research and Human Genetics*, *10*, 423–433.

Bishop, E. G., Cherny, S. S., Corley, R., Plomin, R., DeFries, J. C., & Hewitt, J. K. (2003). Development genetic analysis of general cognitive ability from 1 to 12 years in a sample of adoptees, biological siblings, and twins. *Intelligence*, *31*, 31–49.

Boomsma, D. I., & van Baal, G. C. M. (1998). Genetic influences on childhood IQ in 5- and 7-year-old Dutch twins. *Developmental Neuropsychology*, *14*, 115–126.

Bouchard, T. (1998). Genetic and environmental influences on adult intelligence and special mental abilities. *Human Biology*, *70*, 257–279.

Broman, S. H., Nichols, P. L., & Kennedy, W. A. (1975). *Preschool IQ: Prenatal and early developmental correlates*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Campbell, S. B., Shaw, D. S., & Gilliom, M. (2000). Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Development and Psychopathology*, *12*, 467–488.

Carlson, M. J., & Corcoran, M. E. (2001). Family structure and children's behavioral and cognitive outcomes. *Journal of Marriage and the Family*, *63*, 779–792.

Cherny, S. S., Fulker, D. W., Emde, R. N., Plomin, R., Corley, R. P., & DeFries, J. C. (2001). Continuity and change in general cognitive ability from 14 to 36 months. In R. N. Emde, & J. K. Hewitt (Eds.), *Infancy to early childhood: Genetic and environmental influences on developmental change* (pp. 206–220). New York, NY: Oxford University Press.

Davis, O. S. P., Arden, R., & Plomin, R. (2008). *g* in middle childhood: Moderate genetic and shared environmental influence using diverse measures of general cognitive ability at 7, 9 and 10 years in a large population sample of twins. *Intelligence*, *36*, 68–80.

Deary, I. J. (2012). Intelligence. *Annual Review of Psychology*, *63*, 453–482.

Deary, I. J., Johnson, W., & Houlihan, L. M. (2009). Genetic foundations of human intelligence. *Human Genetics*, *126*, 215–232.

Deary, I. J., Strand, S., Smith, P., & Fernandes, C. (2007). Intelligence and educational achievement. *Intelligence*, *35*, 13–21.

Deary, I. J., Whalley, L. J., Lemmon, H., Crawford, J. R., & Starr, J. M. (2000). The stability of individual differences in mental ability from childhood to old age: Follow-up of the 1932 Scottish Mental Survey. *Intelligence*, *28*, 49–55.

Ellis, L., & Bonin, S. L. (2003). Genetic and occupation-related preferences. Evidence from adoptive and non-adoptive families. *Personality and Individual Differences*, *35*, 929–937.

Farrington, D. P. (1991). Childhood aggression and adult violence: Early precursors and later-life outcomes. In D. J. Pepler, & K. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 5–29). Hillsdale, NJ: Erlbaum.

Hardy, J. B. (2003). The Collaborative Perinatal Project: Lessons and legacy. *Annals of Epidemiology*, *13*, 303.

Harris, K. M., Florey, T., Tabor, J., Bearman, P. S., Jones, J., & Udry, J. R. (2003). The national longitudinal study of adolescent health: Research design. http://www.cpc.unc.edu/projects/addhealth/design [Available from].

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.

Haworth, C. M. A., Wright, M. J., Luciano, M., Martin, N. G., de Geus, E. J. C., van Beijsterveldt, C. E. M., et al. (2010). The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Molecular Psychiatry*, *15*, 1112–1120.

Heaton, R. K., Gladsjo, J. A., Palmer, B. W., Kuck, J., Marcotte, T. D., & Jeste, D. V. (2001). Stability and course of neuropsychological deficits in schizophrenia. *Archives of General Psychiatry*, *58*, 24–32.

Hoekstra, R. A., Bartels, M., & Boomsma, D. I. (2007). Longitudinal genetic study of verbal and nonverbal IQ from early childhood to young adulthood. *Learning and Individual Differences*, *17*, 97–114.

Jensen, A. R. (1969). How much can we boost IQ and scholastic achievement? *Harvard Educational Review*, *39*, 1–123.

Jensen, A. R. (1981). Raising the IQ: The Ramey and Haskins study. *Intelligence*, *5*, 29–40.

Jensen, A. R. (1998). *The g factor*. Westport, CT: Praeger.

Joiner, T. E., Heatherton, T. F., & Keel, P. K. (1997). Ten-year stability and predictive validity of five bulimia-related indicators. *The American Journal of Psychiatry*, *154*, 1133–1138.

Jusko, T. A., Klebanoff, M. A., Brock, J. W., & Longnecker, M. P. (2012). In-utero exposure to dichlorodiphenyltrichloroethane and cognitive development among infants and school-aged children. *Epidemiology*, *23*, 689–698.

Klebanoff, M. A. (2008). The Collaborative Perinatal Project: A 50-year retrospective. *Paediatric and Perinatal Epidemiology*, *23*, 2–8.

LaBuda, M. C., DeFries, J. C., Plomin, R., & Fulker, D. W. (1986). Longitudinal stability of cognitive ability from infancy to early childhood: Genetic and environmental etiologies. *Child Development*, *57*, 1142–1150.

Larson, L., Hartmann, P., & Nyborg, H. (2008). The stability of general intelligence from early adulthood to middle-age. *Intelligence*, *36*, 29–34.

Larsson, J. O., Larsson, H., & Lichtenstein, P. (2004). Genetic and environmental contributions to stability and change of ADHD symptoms between 8 and 13 years of age: A longitudinal twin study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*, 1267–1275.

Lynn, R., & Vanhanen, T. (2006). *IQ and global inequality*. Washington: Summit Publishers.

Moffitt, T. E. (1990). Juvenile delinquency and attention deficit disorder: Boys' developmental trajectories from age 3 to age 15. *Child Development*, *61*, 893–910.

Moffitt, T. E. (1993). Adolescence-limited and life-course persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, *100*, 674–701.

Muthén, L. K., & Muthén, B. O. (2012). *Mplus user's guide* (7th ed.) Los Angeles, CA: Muthén and Muthén.

Myrianthopoulos, N. C. (1970). An epidemiological survey of twins in a large, prospectively studied population. *American Journal of Human Genetics*, *22*, 611–629.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, Netherlands: Kluwer.

Nichols, P. L., & Chen, T. (1981). *Minimal brain dysfunction: A prospective study*. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Petrill, S. A., Lipton, P. A., Hewitt, J. K., Plomin, R., Cherny, S. S., Corley, R., et al. (2004). Genetic and environmental contributions to general cognitive ability through the first 16 years of life. *Developmental Psychology*, *40*, 805–812.

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2013). *Behavioral genetics* (6th ed.)New York: Worth.

Plomin, R., Fulker, D. W., Corley, R., & DeFries, J. C. (1997). Nature, nurture, and cognitive development from 1 to 16 years: A parent–offspring adoption study. *Psychological Science*, *8*, 442–447.

Plomin, R., Kagan, J., Emde, R. N., Reznick, J. S., Braungart, J. M., Robinson, J., et al. (1993). Genetic change and continuity from fourteen to twenty months: The MacArthur Longitudinal Twin Study. *Child Development*, *64*, 1354–1376.

Plomin, R., Pedersen, N. L., Lichtenstein, P., & McClearn, G. E. (1994). Variability and stability in cognitive abilities are largely genetic later in life. *Behavior Genetics*, *24*, 207–215.

Raguet, M. L., Campbell, D. A., Berry, D. T. R., Schmitt, F. A., & Smith, G. T. (1996). Stability of intelligence and intellectual predictors in older persons. *Psychological Assessment*, *8*, 154–160.

Rowe, D. C., Jacobson, K. C., & Van den Oord, E. J. (1999). Genetic and environmental influences on vocabulary IQ: Parental education level as moderator. *Child Development*, *70*, 1151–1162.

Rushton, J. P. (1997). Cranial size and IQ in Asian Americans from birth to age seven. *Intelligence*, *25*, 7–20.

Rushton, J. P., & Jensen, A. R. (2005). Thirty years of research on race differences in cognitive ability. *Psychology, Public Policy, and Law*, *11*, 235–294.

Schwartz, J. A., & Beaver, K. M. (2013). Examining the effects of dopamine genes on verbal IQ within and between families. *Journal of Neural Transmission*, *120*, 477–486.

Thompson, L. A. (1993). Genetic contributions to intellectual development in infancy and childhood. In P. A. Vernon (Ed.), *Biological approaches to the study of human intelligence* (pp. 95–138). Norwood, NJ: Ablex.

Tremblay, T. E., Nagin, D. S., Seguin, J. R., Zoccolillo, M., Zelazo, P. D., Boivin, et al. (2004). Physical aggression during early childhood: Trajectories and predictors. *Pediatrics*, 114.

Trzaskowski, M., Yang, J., Visscher, P. M., & Plomin, R. (2013). DNA evidence for strong genetic stability and increasing heritability of intelligence from age 7 to 12. *Molecular Psychiatry*, 1–5.

Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, *14*, 623–628.

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 at Chapel Hill.

van den Oord, E. J. C. G., & Rowe, D. C. (1999). A cousin study of associations between family demographic characteristics and childrens' intellectual ability. *Intelligence*, *27*, 251–266.

Zuroff, D. C., Blatt, S. J., Sanislow, C. A., III, Bondi, C. M., & Pilkonis, P. A. (1999). Vulnerability to depression: Reexamining the state dependence and relative stability. *Journal of Abnormal Psychology*, *108*, 76–89.