Intelligence and Age at First Intercourse: Cause or Confound?

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Abstract

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Anecdotal evidence from the popular media, such as MTV's reality television franchise, *16 and Pregnant*, suggests that teenage promiscuity is on the rise. Academic evidence confirms such anecdotes; age at first intercourse (AFI) is indeed declining and has so for some time (Bozon, 2003; Finer, 2007). Early age at first intercourse is associated with a plethora of negative downstream consequences, including lower education attainment (Harden, 2012; Spriggs & Halpern, 2008; Wellings et al., 2001), failure to meet education and career goals (Halpern, Joyner, Udry, & Suchindran, 2000), increased risk of teenage pregnancy (Leitenberg & Saltzman, 2000; Wellings et al., 2001), and increased rates of sexually transmitted infections (Kaestle, Halpern, Miller, & Ford, 2005). Moreover, beyond the obvious benefit of avoiding the aforementioned negative outcomes, delaying AFI is associated with greater relationship satisfaction, perception of increased attractiveness, and higher household income (Harden, 2012). Because the aforementioned consequences are severe and long-reaching, psychology has begun to explore potential causal mechanisms of early AFI. Indeed, the field has found a consistent correlate in the literature – intelligence.

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in fact, it looks like shows such as this one reduce rates of teen pregnancy...(Kearney & Levine, 2014)

Higher levels of intelligence are positively associated with delaying first intercourse (Woodward, Fergusson, & Horwood, 2001; Paul, Fitzjohn, Herbison, & Dickson, 2000; Halpern et al., 2000; Mott, 1983). Specifically, it seems that intelligent individuals delay intercourse to "safeguard" their futures (Kirby, 2002b; Manlove, 1998; Raffaelli & Crockett, 2003). They perceive the risks associated with early intercourse, (e.g., pregnancy, STIs) to have career–shattering outcomes (Halpern et al., 2000; Harden & Mendle, 2011). Although this correlate holds promise – much of the field has treated this finding as causal and non-spurious. Yet, there is a fundamental confound in the existing literature that makes it impossible to infer causality.

Practically, all of the AFI-intelligence literature has used between family analyses. In all such analyses, gene and environmental influences, such as education and maternal intelligence are hopelessly confounded (See Harden, 2014). By ignoring such confounds, results are uninterpretable and risk misattributions of causality (Rowe & Rodgers, 1997;

Rutter, 2007). Indeed, both intelligence and AFI are highly heritable and have sizeable shared environmental variances (Harden & Mendle, 2011; Harden, 2014; Plomin & Spinath, 2004). Thus, we need to critically evaluate whether intelligence is a cause of AFI or merely a theoretically attractive confound.

Cause or Confound?

There are numerous theories on the motivations behind adolescents initiation of first intercourse (See Rodgers, 1996 or (Buhi & Goodson, 2007) for a review), and even more specific antecedents (Buhi & Goodson, 2007; Kirby, 2002a; B. C. Miller et al., 1997; Santelli & Beilenson, 1992). Many of these theories either emphasize biology/genetics, where typical adolescent development through puberty (and various hormone changes) drives the interest in sexual behavior (W. B. Miller et al., 1999; Udry, 1979), or social/environmental processes, such as Social Learning (DiBlasio & Benda, 1990; Hogben & Byrne, 1998), where social norms alter the likelihood of early sexual behavior; or Social Control theory (Hirschi, 2002), where societal convention reduces the likelihood that individuals will act on their naturally deviant behavior. Under these environmentally centric theories the underlying biology is either ignored or actively resisted (in the case of Social Control theory, while under many of the genetic centric theories, the environmental components are ignored.

Recently, there have been numerous articles advocating integrative models (See Harden, Mendle, Hill, Turkheimer, & Emery, 2008 and Harden, 2014). The integrative Biopsychosocial Model acknowledges both genetic and environmental contributions (Petersen, 1987; Rodgers, Rowe, & Buster, 1999). Indeed, biology, psychology, and society jointly influence adolescents' decisions to engage in sexual intercourse (Meschke, Zweig, Barber, & Eccles, 2000; Zimmer-Gembeck & Helfand, 2008). Even though this paper focuses on a single predictor – intelligence, we are doing so within the broader context.

Intelligence as the Cause

We've previously mentioned that the short-term risks of early AFI are overwhelming negative, whereas the rewards for delay are equally positive. These consequences extend into adulthood – early AFI is associated with adult delinquency (Harden et al., 2008), anti-social behavior, and substance abuse (Boislard & Poulin, 2011), while those who delayed had higher household incomes in adulthood(Harden, 2012). It is intuitively appealing to believe that intelligent individuals are more likely to observe this high risk, low reward tradeoff, and act upon such observations by delaying intercourse. Accordingly, intelligent individuals perceive the consequences of early AFI to have career–shattering outcomes (Halpern et al., 2000; Harden & Mendle, 2011).

Indeed, the literature is consistent with this theory. Those with higher educational goals delay their first intercourse (Boislard & Poulin, 2011; Schvaneveldt, Miller, Berry, & Lee, 2001), while those who had previously reported higher goals, but engaged in early sexual intercourse reduced their goals (Schvaneveldt et al., 2001). Beyond academic goals, those with a greater affinity for risk and those who perceive benefits from teen-pregnancy are more likely to engage in risky sexual activities (Raffaelli & Crockett, 2003). A greater understanding of the risks associated with sexual intercourse, such as HIV transmission, is also associated with delayed AFI (C. Mathews et al., 2009).

Smarter adolescents are more likely to report delayed intercourse (Woodward et al., 2001; Paul et al., 2000; Halpern et al., 2000; Mott, 1983). Beyond intercourse, smarter individuals appear to postpone all sexual/romantic activity Halpern et al. (2000). Such blanket delays may be a proactive attempt to avoid first intercourse precursors.

Intelligence as a Confound

However, there is an equally valid family of explanations in which intelligence is not the driver of the AFI-intelligence relationship. Instead, various confounds including family level selection effects, or third variables at the individual level could be causing the relationship. Indeed many such findings that link intelligence with various outcomes

are the product of misattributing between family confounds to individual level predictors. The relationship between birth order and intelligence is a classic example of this misattribution. INSERT THREE SENTENCE SUMMARY(See Rodgers, Cleveland, van den Oord, & Rowe, 2000).

Family level influences such as SES and maternal intelligence could drive the relationship because these variables are associated with the onset of first intercourse (Lammers, Ireland, Resnick, & Blum, 2000) and correlated with intelligence (Devlin, Daniels, & Roeder, 1997; Murray, 1998; Neisser et al., 1996; Strenze, 2007). Indeed many of the negative consequences of early AFI, such as reduced education attainment vanish when analyses are adjusted for intelligence and various family level variables (such as parental education) (Spriggs & Halpern, 2008). Accordingly it stands to reason that the relationship between intelligence and AFI could be a product of the same phenomena.

The link between intelligence and AFI could also be the product of individual level confounds that are highly correlated with both intelligence and AFI.

- family level factors (ses, mother education, use mother intelligence as a transition)
  - Disadvantaged Backgrounds are associated with both lower levels of intelligence and earlier AFI.
    - poverty
    - larger family
    - lower SES
    - being on welfare
  - Parental Characteristics
    - parental education
    - marital status
    - parental intelligence
- alternative individual level factors (genes, education, presplit) school performance, delinquency

substantive confounds

statistical confounds

, use to transition to explaining between and within family findings. IF a result is truly causal, then findings from between family analyses will be replicated by within family results. IF said replication fails, then the characteristic cannot be a direct cause of the effect. That isn't to say intelligence isn't a distal cause – any many of the AFI-intelligence theories suggest that risk perception is an essential element . However, being intelligent in and of itself does not cause changes in AFI

Between vs. Within Family Analyses

Therefore, in order to determine whether the link between intelligence and AFI is causal, we need to find a way to untangle the between family influences from the within family (i.e. causal) influences. One of the fundamental challenges in psychological research is evaluating causal hypotheses that are either situation driven or person driven. Randomized experiments are particularly effective at determining causality for situational hypotheses because random assignment controls for genetic and environmental confounds across repeated trials. However, some situation driven explanations, such as maternal influence, cannot be ethically evaluated via randomized experiment. Moreover, evaluating person-driven hypotheses are comparably problematic, as random assignment of person characteristics (e.g., personality) isn't feasible (see West, 2009). Traditional alternatives to evaluating such hypotheses tend to focus upon between-family longitudinal studies, where potential confounds are included as covariates.

However, the between-family covariate approach cannot control for systematically confounded genetic characteristics and environmental influences. The approach can result in misattributions of causality, as the misattributed cause might sufficiently covary with the true cause. Indeed, poverty and individual differences covary with genes and environment, to such a degree that between family analyses are fundamentally biased (Rowe & Rodgers, 1997). Yet, the covariate approach has been the primary

method used to evaluate the causal mechanism behind the AFI-intelligence relationship.

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Instead, quasi-experimental designs can be used; such designs support causal inference without random assignment. Sibling-based quasi-experimental models are particularly effective at incorporating genetic and environmental design elements. However, such models are underused in psychology (Rodgers *et al*, 2001), tend to focus on environmental confounds and do not naturally incorporate varying levels of relatedness.

Traditional sibling comparison models often rely on rare events (i.e., twins) or advanced methodology (e.g., propensity score matching, multilevel modeling). As an alternative, we have adapted Kenny's reciprocal standard dyad model(Kenny, Mohr, & Levesque, 2001; Kenny, Kashy, & Cook, 2006). Our adaptation controls for gene and shared environmental influences within a simple regression framework, by taking the difference between the two siblings

Prior Within Family Analyses

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Current Study

- We wanted to know whether the Harden & Mendle (2011) results would hold using a different design and data set.
- We examined the relationship between AFI and Intelligence using siblings and their children from a multigenerational nationally representative sample.
- Specifically, we addressed the following questions:
  - Does intelligence (mother or child) predict child AFI, after controlling for gene and environmental confounds?
  - Is this relationship consistent between– and within–families?

To summarize, the current study examines the relationship between intelligence and age at first intercourse, using siblings and their children from a multigenerational

nationally representative sample. This examination extends the intelligence literature in several key ways. We

1. tested whether the relationship between intelligence and age at first intercourse was consistent using between and within family analyses;
2. evaluated the alternative explanation that maternal intelligence influences child AFI; and
3. whether these effects would replicate using earlier measures of intelligence.

We made the following predictions, based upon recent meta-analyses XX and findings from Harden and Mendle:

1.
2.
3.

Methods

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Design

We adapted Kenny and colleagues (2001; 2006) reciprocal standard dyad model.

$$\mathrm{Y_{i\Delta}} = \beta_0 + \beta_1 \bar{\mathrm{Y}}_\mathrm{i} + \beta_2 \bar{\mathrm{X}}_\mathrm{i} + \beta_3 \mathrm{X_{i\Delta}}$$

where

$$Y_{i1} = \max(Y_{ij});\ Y_{i2} = \min(Y_{ij});\ Y_{i\Delta} = Y_{i1} - Y_{i2};\ X_{i\Delta} = X_{i1} - X_{i2}$$

In this model, the relative difference in outcomes ($\mathrm{Y_\Delta}$) is predicted from the mean level of the outcome ($\mathrm{Y_{mean}}$), the mean level of the predictor ($\mathrm{X_{mean}}$), and the between-sibling predictor difference ($\mathrm{X_\Delta}$). The mean levels support causal inference through at least partial control for genes and shared environment. Therefore, we simultaneously evaluate the individual difference ($\mathrm{X_\Delta}$) and the joint contribution of

genes and shared environment (Y_{mean} & X_{mean}). Preliminary applications have given estimates consistent with the literature (Garrison, Hadd, & Rodgers, 2015; Rodgers, Garrison, & Hadd, 2014).

Sample

The National Longitudinal Survey of Youth 1979 (NLSY79) is a nationally representative household probability sample, jointly sponsored by the US Department of Labor and US Department of Defense. In 1980, 12,686 adolescents were surveyed from 8,770 households on a battery of measures. The initial survey consisted of three subsamples:

- a cross–sectional probability sample of 6,111 non-institutionalized adolescents residing in the United States on December 31^{st} of 1978,
- an over–sampled civilian subsample of 5,295 racial minorities and disadvantaged whites, and
- a representative sample of 1,280 youths serving in the US Military on September 30^{th}, 1978.

In the civilian samples, subjects' birthdates ranged from January 1, 1957 to December 31, 1964, and were between the ages of 14 and 21 on 31^{st} of 1978, whereas military subject's birthdates ranged from January 1, 1957 to December 31, 1961, and were likewise between 17 and 21 years old. Participants were surveyed annually until 1994, and then surveyed biannually to the present day. Two waves of planned attrition occurred. After the 1984 interview, all but 201 randomly selected members of the military sample were dropped. After the 1990 interview, all 1,643 disadvantaged whites from the oversample were dropped. More information, such as the data and documentation can be found on the Bureau of Labor Statistics (BLS) website: `http://www.bls.gov/nls/nlsy79.htm`.

In 1986, the biological children of the female NLSY79 participants were surveyed, resulting in the NLSY79 Children and Young Adults (NLSY79–CYA) survey. These 11,512 participants are also surveyed on a biannual basis. Accordingly, participants in

the NLSY79 will be periodically referred to as the Generation 1 (Gen1) sample, whereas the NLSY79–CYA will be referred to as the Generation 2 (Gen2) sample.

Tetrads

Mother-Child-Aunt-Nibling (MCAN) tetrads were created using the NLSY Kinship Links (Rodgers et al., 2015) and supporting R package (Beasley et al., 2015). The oldest two female kin (Mother, Aunt) were selected from each household; additional female Gen1 kin were excluded. Three tetrad designs were employed, in which the genders of Gen2 were the defining feature:

- Mother-Daughter-Aunt-Niece (MDAN) included the oldest female child from each of the sisters,
- Mother-Son-Aunt-Nephew (MSAN) included the oldest male child from each of the sisters, and
- Mother-Child-Aunt-Nibling (MCAN) included the first born child from each of the sisters.

All outcomes were standardized by gender prior to tetrad creation. Table 3 on page 21 reports descriptive statistics for all variables used throughout this paper by whether the respondent has a sibling in the sample.

Age at First Intercourse

Over the life-time of the NLSY-CYA survey, participants were surveyed about their AFI. The exact phrasing of the question varied by administration. Between 1988 and 2000, subjects were asked for age, year, and month of first intercourse. After 2000, subjects were only asked their age. The reason for this change is unknown. However, the first author suspects that the change had to do with the fact that the modal response for month was consistently: “Don’t Know”. Indeed, only 1147 subjects reported a viable month of first intercourse. Regardless, we calculated AFI as follows, using SAS (citation). First, we transformed year of first intercourse into age. If subjects reported both age and year within the same survey, we averaged the age scores. Across

all survey years, we identified the minimum AFI and maximum AFI for each subject. Then we took the average of those two scores. Given that the expected AFI of a subject ≠ the reported AFI, we added 1 to the Maximum AFI. Therefore, if the subject only reported one instance of AFI, their AFI would now reflect their expected AFI. For example, a subject who reports AFI at 16 could be 16 years and 1 day old OR 16 years and 364 days old. Thus the expected value for 16 is in fact 16.5. We calculated AFI in this manner because we wished to include the maximum amount of information without ignoring the expected value problem with self-reported age. Using this method, the average AFI was 16.01 years (sd = 2.30; n = 6288).

Taking the average of all AFIs (without addressing expected value), results in 15.49 (sd equal 2.30; n equal 6288). Adding in expected value of .5 changes this value to 15.99. Pretty much indistinguishable.)

After transforming all AFI scores, we recoded all impossible AFIs as missing. We considered a score to be impossible if the reported AFI that exceeded participant's age at time of survey ($\overline{\text{AFI}}$ = 15.99, sd = 2.30, n = 6235). Next, we excluded all AFIs below age 12 (16.14, sd = 2.10, n = 6087). Finally we excluded subjects who reported AFI prior to menstruation (16.16, sd = 2.09, n = 6047). We excluded those below age 12 because those responses likely are the result of misunderstanding or non-consensual sexual activity, while we excluded those with premenstrual AFI because of we were only interested in post-pubescent sexual activity. AFI varied by gender and race. Most notably, women reported AFIs that were 6 months later than men, and black men reported the lowest AFI (15 yrs). See Table 3 on page 21 for a complete breakdown.

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Measures

Generation 1

The Armed Services Vocational Aptitude Battery (ASVAB; Form 8A; Palmer, Hartke, Ree, Welsh, & Valentine, Jr., 1988) was administered to Gen1 participants during the summer and fall of 1980 (U.S. Department of Defense, 1982), and was used to establish national norms for the Department of Defense (Waters, Laurence, Camara, & Green, 1987). The Armed Forces Qualification Test (AFQT) is derived from the ASVAB, and used as a measure of general trainability (Maier & Sims, 1986). It is a

composite of four subscales: Arithmetic Reasoning (AR; 30 items), Math Knowledge (MK; 25 items), Paragraph Comprehension (PC; 15 items), and Word Knowledge (WK; 35 items). Arithmetic Reasoning targets the ability to solve word problems. Math Knowledge also tests quantitative ability, by assessing knowledge of high school level mathematics, with special emphasis on algebra, fractions, and geometry. The remaining subscales focus on verbal ability, and are sometimes referred to as the Verbal Composite (VE). Specifically, Word Knowledge tests the subjects' knowledge of the meaning of words within a given context, whereas Paragraph Comprehension targets a subject's ability to understand the meanings of paragraphs. Other administrations of the pencil and paper ASVAB reveal that all the AFQT subscales have high internal consistency ($\alpha_{AR} = .91$; $\alpha_{WK} = .92$; $\alpha_{PC} = .81$; $\alpha_{MK} = .87$; Kass, Mitchell, Grafton, & Wing, 1982)

Methods of calculating the AFQT have varied throughout the ASVAB's administrative lifetime (Mayberry & Hiatt, 1992). For pencil and paper administrations, standard scores were created for each of the subscale scores ($\bar{x} = 50$, sd = 10), and then combined into a standard score. Then, the AFQT standard score is derived from the following formula:

$$\text{AFQT} = \text{AR} + \text{MK} + 2\text{VE}, \tag{1}$$

$$\text{where VE} = \text{PC} + \text{WK}. \tag{2}$$

This score is then converted into a percentile, which determines an applicant's basic qualification for enlistment. All applicants must earn a score at or above the 10^{th}percentile(Defense Manpower Data Center, 2012). Each branch has its own minimum score, ranging from 31 to 36 (U.S. Department of the Army, 2013; U.S. Coast Guard, 2004), and each branch uses different linear combinations of these subtests to determine an applicant's eligibility for specialty positions. Additionally, multiple researchers have used the AFQT standard score as a proxy for general intelligence (g) (Herrnstein & Murray, 1994; Der, Batty, & Deary, 2009). Indeed, the military has found that the AFQT correlated 0.8 with the Wechsler Adult Intelligence Scale (WAIS; McGrevy, Knouse, & Thompson, 1974). Moreover, the AFQT consistently predicts outcomes traditionally associated with intelligence (See Welsh, Kucinkas, & Curran,

1990), including grades (Wilbourn, Valentine, Jr., & Ree, 1984; J. J. Mathews, 1977).

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Generation 2

Administration of ability measures has varied considerably across the lifecourse of the NLSY-CYA survey (See Table 2.12 from Center for Human Resources Research, 2006 for a summary). However, the vast majority of subjects have completed the following test batteries:

- Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970):
  - Math Subtest (84 items),
  - Reading Recognition Subtest (84 items),
  - Reading Comprehension Subtest (84 items),
- The Peabody Picture Vocabulary Test-Revised (PPVT-R; Form L; Dunn & Dunn, 1981; 175 items), and
- Wechsler Intelligence Scales for Children–Revised (WISC-R; Wechsler, 1974) Digit Span Subscale (28 items).

Although individual item level data was available for all of the aforementioned tests, conducting a unidimensional 2-PL is not a viable means of estimating general ability because of the nature of test construction and administration. The PIATs and PPVT-R were administered to subjects in an adaptive manner. The starting items on the PIAT Math and PVVT-R were determined by age, whereas the starting items for the remaining PIAT subtests were determined based on PIAT Math performance. Moreover, administration of a given test were terminated when a subject reached a "ceiling." For example, testing was terminated for the PIAT Math if a subject incorrectly answered 5 of the most recent 7 questions (See Baker, Keck, Mott, & Quinlan, 1993 for a thorough overview of NLSY-CYA test administration protocols). In essence, this administration procedure results in a tremendous amount of non-randomly missing data.

Although the administration created non-randomly missing data, the standard scores of the PVVT-R, PIATs, and WISC-R Digit Span themselves are valid and very

reliable assessments of cognitive ability (Mott & Baker, 1995). Accordingly, we elected to use the standard scores of all the Gen2 ability measures already mentioned. However, subjects were surveyed on a biannual basis. Thus we could not use cognitive tests at a given age. Instead, we aggregated scores across a 4 year window, and targeted ages 9 and 10. We targeted 9.5 because all cognitive tests were administered within the 8–11 age window, we wanted to maximize the number of subjects with viable ability scores, and we wanted to ensure temporal precedence with respect to AFI. In the case of missing subtests, we allowed age 11 scores to replace age 9 scores, and age 8 scores to replace age 10 scores. By employing a 4 year window, all subjects had an equal chance of replacing the primary test administration. Our replacement strategy ensured that the average age of testing matched the average of our targeted ages.

In order to confirm the validity of this method, we replicated all age 9.5 ability analyses using ages 10.5 and 11.5. These replications can be found in the Appendices A and B, respectively. Appendix A begins on page 31 and appendix B begins on page 39.

Measurement. A unidimensional confirmatory factor analytic model was run in Mplus 7.3 (Muthén & Muthén, 2014), and used a robust maximum likelihood estimator (MLR). There were 8,254 useable observations in 3,742 clusters. A single factor solution fit the model decently (RMSEA = .101, p(RMESA < .05) = 0; CFI = .973; TLI = .946, SRMR = .027). Table 4 on page 22 contains a full summary of the model fit statistics, and Table 5 on page 23 contains the factor loadings.

Results

We examined the relationship between AFI and intelligence using two designs: between and within families. The between family analyses report the relationships between the average AFI and various measures of ability. The within family analyses attempt to replicate the between family findings by testing whether differences in AFI can be explained by differences in various measures of ability. If there is a causal relationship between intelligence and AFI then differences in AFI will be significantly associated with differences in ability. If the relationship is the result of between family

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confounds, such as shared environmental influences, then differences in AFI will not be significantly associated with differences in ability, and accordingly, AFI cannot be caused by intelligence.

Between Family Anlayses

First, we examined the between family results. We tested whether the family average of Gen2 AFI could be predicted by the family averages of Gen1 ability and of Gen2 ability. We evaluated the influences both independently and simultaneously. All ability scores have been standardized by generation ($\overline{g} = 0$, sd $= 1$), prior to averaging by household. AFI scores have been standardized by gender ($\overline{\text{AFI}} = 0$, sd $= 1$), prior to averaging by household.

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Mean Gen1 Intelligence → Mean Gen2 AFI. Gen1 sister averages of standardized AFQT scores were used to predict Gen2 averages of gender standardized AFI. Table 6 on page 24 displays the results by Gen2 linking. The Mixed model reports the averages of the first borns of each sister (n = 342), the Daughters model reports the averages of the first born girls (n = 264), and the Sons model reports the averages of the first born sons (n = 282). All three models reveal similar results. A one unit increase in the average standardized intelligence of the children's mothers predicted $\approx .013$ increase in average Gen2 AFI. The adjusted R^2 varied slightly by Gen2 linking (Mixed = .087, Daughters = .097, Sons = .103).

Mean Gen2 Intelligence → Mean Gen2 AFI. Gen2 cousin averages of standardized ability scores were used to predict Gen2 averages of gender standardized AFI. Table 7 on page 25 displays the results by Gen2 linking. The Mixed model reports the averages of the first borns of each sister (n = 344), the Daughters model reports the averages of the first born girls (n = 267), and the Sons model reports the averages of the first born sons (n = 283). All three models reveal similar results. A one unit increase in the average standardized intelligence of the children predicted $\approx .075$ increase in average Gen2 AFI. The adjusted R^2 varied slightly by Gen2 linking (Mixed = .014, Daughters = .016, Sons = .009).

Mean Joint Intelligence → Mean Gen2 AFI. Results from the Gen1 sister averages of standardized AFQT scores and Gen2 cousin averages of standardized ability scores predictions of Gen2 averages of gender standardized AFI are displayed in Table 8 on page 26. Again, three models based on Gen2 linking are displayed: Mixed (n = 337), Daughters(n = 260), and the Sons(n = 278). All three models reveal similar results. Gen1 intelligence was significantly associated with Gen2 AFI ($p < .01$), while Gen2 intelligence was not significantly associated with Gen2 AFI. A one unit increase in the average standardized intelligence of the children's mothers predicted $\approx .014$ increase in average Gen2 AFI. The adjusted R^2 varied slightly by Gen2 linking (Mixed = .086, Daughters = .097, Sons = .100), but each were practically identical to the Mean Gen1 Intelligence models.

Within Family Analyses

We attempted to replicate the between family analyses reported in the previous subsection, using within family analyses. Using the discordant sibling model, we predicted the differences in Gen2 AFI as a function of differences in intelligence, controlling for means of the outcomes and predictors. We ran three series of models, where we examined the individual and then joint influence of Gen1 intelligence and Gen2 intelligence. Moreover, within each series we included three Gen2 linking variants, just as we did in the between family analyses: Mixed model reports the differences of the first borns of each sister, the Daughters model reports the differences of the first born girls„ and the Sons model reports the differences of the first born sons.

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Dif Gen1 Intelligence → Dif Gen2 AFI. Gen1 sister differences in standardized AFQT scores were used to predict Gen2 differences of gender standardized AFI, controlling for Gen1 sister averages of standardized AFQT scores and Gen2 averages of gender standardized AFI. Table 9 on page 27 displays the results by Gen2 linking. The Mixed model reports the averages and differences of the first borns of each sister (n = 336), the Daughters model reports the averages and differences of the first born girls (n = 258), and the Sons model reports the averages and differences of the

first born sons (n = 278). All three models reveal similar results. Gen2 averages of gender standardized AFI were significant predictors of Gen2 differences in gender standardized AFI ($p < .01$), across all three linking methods. A one unit increase in the average gender standardized AFI predicted ≈ 0.34 increase in average Gen2 AFI difference, controlling for all over variables in the model.

In the Sons model, the Gen1 sister average of standardized AFQT scores was a significant predictor of differences in Gen2 AFI ($p < .01$). A one unit increase in the average standardized intelligence of the children's mothers predicted $\approx .0083$ decrease in the AFI difference between siblings. All other variables were not significant, including all kin difference variables. The adjusted R^2 varied slightly by Gen2 linking (Mixed = .066, Daughters = .072, Sons = .106).

Dif Gen2 Intelligence → Dif Gen2 AFI. Gen2 cousin differences in standardized ability scores were used to predict Gen2 differences of gender standardized AFI, controlling for Gen2 cousin averages of standardized ability scores and gender standardized AFI. Table **??** on page ?? displays the results by Gen2 linking. The Mixed model reports the averages and differences of the first borns of each sister (n = 291), the Daughters model reports the averages and differences of the first born girls (n = 223), and the Sons model reports the averages and differences of the first born sons (n = 238). All three models reveal similar results. Gen2 averages of gender standardized AFI were significant predictors of Gen2 differences in gender standardized AFI ($p < .01$), across all three linking methods. A one unit increase in the average gender standardized AFI predicted ≈ 0.38 increase in average Gen2 AFI difference, controlling for all over variables in the model.

In the Sons model, the Gen2 cousin average of standardized ability scores was a significant predictor of differences in Gen2 AFI ($p < .05$). A one unit increase in the average standardized intelligence of the children predicted $\approx .107$ decrease in the AFI difference between siblings. All other variables were not significant, including all kin difference variables. The adjusted R^2 varied slightly by Gen2 linking (Mixed = .103, Daughters = .121, Sons = .132).

Dif Joint Intelligence → Dif Gen2 AFI. Gen1 sister differences in standardized AFQT scores and Gen2 cousin differences in standardized ability scores were used to predict Gen2 differences of gender standardized AFI, controlling for Gen1 sister averages of standardized AFQT scores, Gen2 cousin averages of standardized ability scores, and Gen2 cousin averages of gender standardized AFI. Table 11 on page 29 displays the results by Gen2 linking. The Mixed model reports the averages and differences of the first borns of each sister (n = 285), the Daughters model reports the averages and differences of the first born girls (n = 217), and the Sons model reports the averages and differences of the first born sons (n = 235). All three models reveal similar results. Gen2 averages of gender standardized AFI were significant predictors of Gen2 differences in gender standardized AFI ($p < .01$), across all three linking methods. A one unit increase in the average gender standardized AFI predicted ≈ 0.38 increase in average Gen2 AFI difference, controlling for all over variables in the model.

All other variables were not significant, including all kin difference variables. The adjusted R^2 varied slightly by Gen2 linking (Mixed = .090, Daughters = .105, Sons = .131).

Discussion

Tables

Descriptive Statistics

Table 1

Gen1 Summary Statistics by Sibling Status.

		Has Sibling in NLSY?				
		Mean_3			Sd_2	
		0	1		0	1
Mother Age at Birth	23.31	22.595	23.949	5.49	5.16	5.69
Standardized AFQT	0.00	0.108	− 0.088	1.09	1.07	1.10
AFI	17.84	17.796	17.870	2.33	2.27	2.39

Table 2

Gen2 Summary Statistics by Sibling Status.

		Has Sibling in NLSY?				
		Mean_3			Sd_2	
		0	1		0	1
Year Born	1985.73	1986.160	1985.679	26.99	6.62	28.39
Standardized Ability Score	0.00	0.275	− 0.023	1.00	0.89	1.01
AFI	16.16	16.052	16.173	2.10	2.09	2.10

Table 3

Gen2 AFI by Gender, Race, and GenderxRace.

			AFI	
			$Mean_3$	Sd_2
			16.16	2.10
		MALE	15.88	2.15
		FEMALE	16.47	1.99
		HISPANIC	16.22	2.14
		BLACK	15.66	2.01
		NON-BLACK, NON-HISPANIC	16.57	2.05
MALE	RACE	HISPANIC	15.92	2.16
		BLACK	15.04	1.96
		NON-BLACK, NON-HISPANIC	16.54	2.06
FEMALE		HISPANIC	16.60	2.05
		BLACK	16.26	1.88
		NON-BLACK, NON-HISPANIC	16.61	2.05

Table 4

Gen2 Measurement Model.

	g at Age 9.5
Estimator	MLR
Observations	8254
Parameters	15
ChiSqM_Value	423
ChiSqM_DF	5
ChiSqM_PValue	0
ChiSqBaseline_PValue	0
LL	-142896
UnrestrictedLL	-142653
LLCorrectionFactor	1.44
UnrestrictedLLCorrectionFactor	1.37
CFI	0.973
TLI	0.946
AIC	285821
BIC	285927
aBIC	285879
RMSEA_Estimate	0.101
RMSEA_90CI_LB	0.093
RMSEA_90CI_UB	0.109
RMSEA_pLT05	0
SRMR	0.027
AICC	285821

Table 5

Gen2 Factor Loadings.

	Test	Estimate	S.E.	Est./.S.E.	P.Value
1	DIGIT	0.530	0.010	52.400	0
2	MATH	0.720	0.007	98.100	0
3	RECOG	0.871	0.005	184.000	0
4	COMP	0.855	0.006	154.000	0
5	PPVT	0.695	0.010	68.800	0

Between Family Analyses

Table 6

Mean Gen1 Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean Gen2 AFI		
	Mixed	Daughers	Sons
Gen1 Intel	0.012^{***}	0.013^{***}	0.014^{***}
	(0.008, 0.017)	(0.009, 0.018)	(0.009, 0.019)
Constant	-0.754^{***}	-0.801^{***}	-0.856^{***}
	(−1.010, −0.500)	(−1.090, −0.513)	(−1.140, −0.569)
Observations	342	264	282
R^2	0.090	0.101	0.106
Adjusted R^2	0.087	0.097	0.103
Residual Std. Error	0.700 (df = 340)	0.703 (df = 262)	0.691 (df = 280)
F Statistic	33.500^{***} (df = 1; 340)	29.400^{***} (df = 1; 262)	33.200^{***} (df = 1; 280)

Note: *p<0.1; **p<0.05; ***p<0.01

Table 7

Mean Gen2 Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean Gen2 AFI		
	Mixed	Daughers	Sons
Gen2 Intel	0.077**	0.086**	0.066*
	(0.014, 0.140)	(0.013, 0.159)	(−0.004, 0.135)
Constant	−0.021	−0.020	−0.039
	(−0.100, 0.057)	(−0.109, 0.070)	(−0.125, 0.047)
Observations	344	267	283
R^2	0.016	0.020	0.012
Adjusted R^2	0.014	0.016	0.009
Residual Std. Error	0.739 (df = 342)	0.744 (df = 265)	0.732 (df = 281)
F Statistic	5.700** (df = 1; 342)	5.320** (df = 1; 265)	3.430* (df = 1; 281)

Note: *p<0.1; **p<0.05; ***p<0.01

Table 8

Mean Joint Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean Gen2 AFI		
	Mixed	Daughers	Sons
Gen1 Intel	0.013***	0.014***	0.015***
	(0.008, 0.017)	(0.008, 0.019)	(0.009, 0.020)
Gen2 Intel	−0.002	−0.0002	−0.015
	(−0.070, 0.066)	(−0.077, 0.077)	(−0.089, 0.060)
Constant	−0.766***	−0.814***	−0.892***
	(−1.050, −0.482)	(−1.130, −0.497)	(−1.220, −0.568)
Observations	337	260	278
R^2	0.091	0.104	0.107
Adjusted R^2	0.086	0.097	0.100
Residual Std. Error	0.704 (df = 334)	0.706 (df = 257)	0.695 (df = 275)
F Statistic	16.700*** (df = 2; 334)	14.900*** (df = 2; 257)	16.500*** (df = 2; 275)

Note: *p<0.1; **p<0.05; ***p<0.01

Within Family Analyses

Table 9

Dif Gen1 Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.303***	0.328***	0.376***
	(0.187, 0.419)	(0.192, 0.465)	(0.251, 0.501)
INTMOMmean	−0.003	−0.002	−0.008***
	(−0.007, 0.002)	(−0.008, 0.004)	(−0.013, −0.003)
INTMOMdiff	0.001	0.001	0.0003
	(−0.003, 0.006)	(−0.005, 0.006)	(−0.005, 0.005)
Constant	1.180***	1.180***	1.500***
	(0.893, 1.470)	(0.838, 1.520)	(1.180, 1.830)
Observations	336	258	278
R^2	0.074	0.083	0.116
Adjusted R^2	0.066	0.072	0.106
Residual Std. Error	0.757 (df = 332)	0.780 (df = 254)	0.734 (df = 274)
F Statistic	8.830*** (df = 3; 332)	7.640*** (df = 3; 254)	12.000*** (df = 3; 274)

Note: *p<0.1; **p<0.05; ***p<0.01

Table 10

Dif Gen2 Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.357***	0.395***	0.398***
	(0.239, 0.475)	(0.259, 0.531)	(0.269, 0.527)
Int_Mean	−0.064	−0.069	−0.107**
	(−0.142, 0.014)	(−0.160, 0.021)	(−0.193, −0.021)
Intdiff	0.029	0.035	0.009
	(−0.018, 0.075)	(−0.019, 0.089)	(−0.041, 0.059)
Constant	1.050***	1.070***	1.070***
	(0.962, 1.140)	(0.970, 1.180)	(0.970, 1.160)
Observations	291	223	238
R^2	0.112	0.133	0.143
Adjusted R^2	0.103	0.121	0.132
Residual Std. Error	0.769 (df = 287)	0.787 (df = 219)	0.753 (df = 234)
F Statistic	12.100*** (df = 3; 287)	11.200*** (df = 3; 219)	13.000*** (df = 3; 234)

Note: *p<0.1; **p<0.05; ***p<0.01

Table 11

Dif Joint Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.345***	0.379***	0.428***
	(0.221, 0.469)	(0.234, 0.524)	(0.293, 0.562)
Int_Mean	−0.064	−0.071	−0.068
	(−0.153, 0.024)	(−0.174, 0.032)	(−0.164, 0.028)
INTMOMmean	0.001	0.001	−0.005
	(−0.005, 0.007)	(−0.006, 0.008)	(−0.012, 0.001)
Intdiff	0.025	0.034	0.007
	(−0.023, 0.073)	(−0.022, 0.090)	(−0.044, 0.058)
INTMOMdiff	0.001	−0.001	0.001
	(−0.004, 0.006)	(−0.007, 0.005)	(−0.005, 0.006)
Constant	0.999***	1.010***	1.370***
	(0.642, 1.360)	(0.591, 1.430)	(0.975, 1.770)
Observations	285	217	235
R^2	0.106	0.125	0.150
Adjusted R^2	0.090	0.105	0.131
Residual Std. Error	0.774 (df = 279)	0.795 (df = 211)	0.754 (df = 229)
F Statistic	6.610*** (df = 5; 279)	6.040*** (df = 5; 211)	8.080*** (df = 5; 229)

Note: *p<0.1; **p<0.05; ***p<0.01

Figures

Figure 1. Comparable Between Family and Within Family Effects

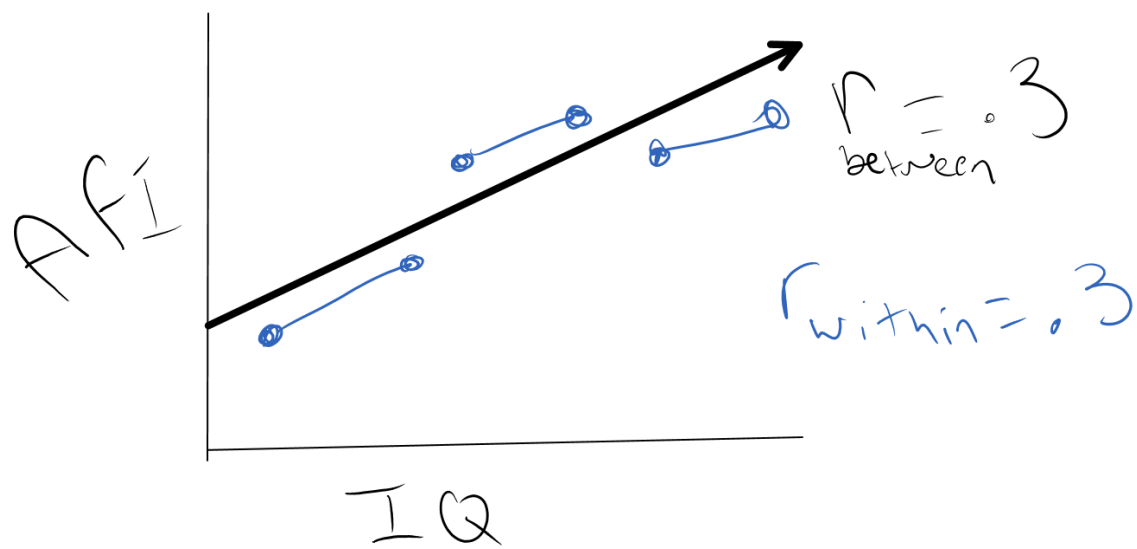
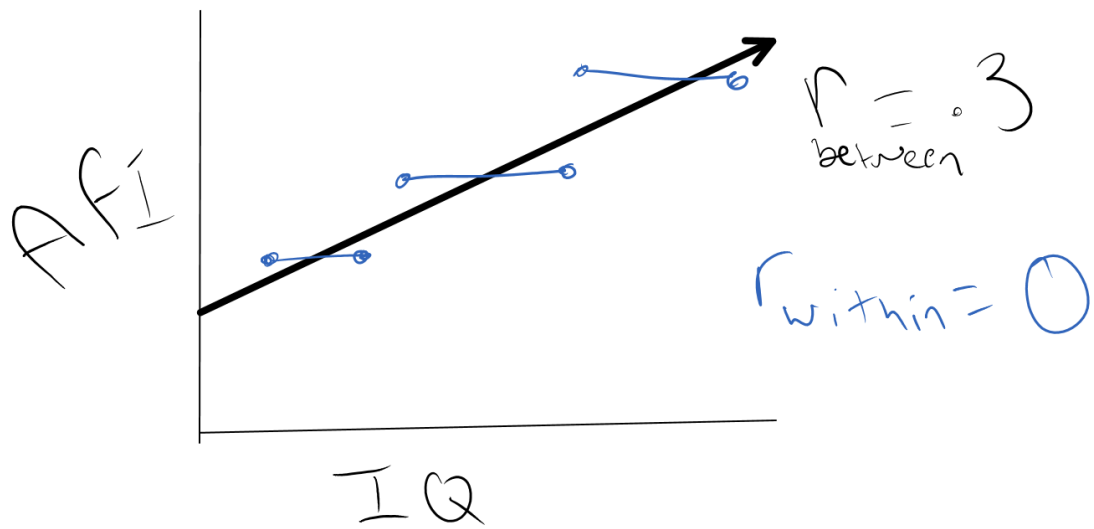


Figure 2. Unequal Between Family and Within Family Effects



Appendix A

Age 10.5 Replication

Table A1

Gen2 Measurement Model.

	g at Age 10.5
Estimator	MLR
Observations	8143
Parameters	15
ChiSqM_Value	404
ChiSqM_DF	5
ChiSqM_PValue	0
ChiSqBaseline_PValue	0
LL	-143535
UnrestrictedLL	-143304
LLCorrectionFactor	1.48
UnrestrictedLLCorrectionFactor	1.4
CFI	0.974
TLI	0.947
AIC	287100
BIC	287205
aBIC	287158
RMSEA_Estimate	0.099
RMSEA_90CI_LB	0.091
RMSEA_90CI_UB	0.107
RMSEA_pLT05	0
SRMR	0.024
AICC	287100

Table A2

Gen2 Factor Loadings.

	Test	Estimate	S.E.	Est./.S.E.	P Value
1	DIGIT	0.547	0.010	54.400	0
2	MATH	0.731	0.007	97.900	0
3	RECOG	0.852	0.005	168.000	0
4	COMP	0.848	0.006	149.000	0
5	PPVT	0.743	0.009	81.400	0

Between Family Analyses

Table A3

Mean Gen1 Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean AFI		
	Mixed	Daughers	Sons
Gen1 Intel	0.012***	0.013***	0.014***
	(0.008, 0.017)	(0.009, 0.018)	(0.009, 0.019)
Constant	−0.754***	−0.801***	−0.856***
	(−1.010, −0.500)	(−1.090, −0.513)	(−1.140, −0.569)
Observations	342	264	282
R^2	0.090	0.101	0.106
Adjusted R^2	0.087	0.097	0.103
Residual Std. Error	0.700 (df = 340)	0.703 (df = 262)	0.691 (df = 280)
F Statistic	33.500*** (df = 1; 340)	29.400*** (df = 1; 262)	33.200*** (df = 1; 280)

Note: *p<0.1; **p<0.05; ***p<0.01

Table A4

Mean Gen2 Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean AFI		
	Mixed	Daughers	Sons
Gen2 Intel	0.098***	0.105***	0.088***
	(0.039, 0.157)	(0.036, 0.173)	(0.022, 0.153)
Constant	−0.015	−0.015	−0.031
	(−0.093, 0.062)	(−0.104, 0.074)	(−0.117, 0.054)
Observations	345	267	283
R^2	0.030	0.033	0.024
Adjusted R^2	0.027	0.029	0.021
Residual Std. Error	0.734 (df = 343)	0.739 (df = 265)	0.728 (df = 281)
F Statistic	10.500*** (df = 1; 343)	8.990*** (df = 1; 265)	6.910*** (df = 1; 281)

Note: *p<0.1; **p<0.05; ***p<0.01

Table A5

Mean Joint Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean AFI		
	Mixed	Daughers	Sons
INTMOMmean	0.012***	0.013***	0.014***
	(0.007, 0.017)	(0.007, 0.018)	(0.008, 0.019)
Int_Mean	0.021	0.018	0.008
	(−0.046, 0.088)	(−0.058, 0.094)	(−0.066, 0.082)
Constant	−0.714***	−0.771***	−0.844***
	(−1.010, −0.418)	(−1.100, −0.440)	(−1.180, −0.506)
Observations	338	260	278
R^2	0.092	0.103	0.107
Adjusted R^2	0.086	0.096	0.101
Residual Std. Error	0.703 (df = 335)	0.707 (df = 257)	0.695 (df = 275)
F Statistic	16.900*** (df = 2; 335)	14.700*** (df = 2; 257)	16.500*** (df = 2; 275)

Note: *p<0.1; **p<0.05; ***p<0.01

Within Family Analyses

Table A6

Dif Gen1 Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.303***	0.328***	0.376***
	(0.187, 0.419)	(0.192, 0.465)	(0.251, 0.501)
INTMOMmean	−0.003	−0.002	−0.008***
	(−0.007, 0.002)	(−0.008, 0.004)	(−0.013, −0.003)
INTMOMdiff	0.001	0.001	0.0003
	(−0.003, 0.006)	(−0.005, 0.006)	(−0.005, 0.005)
Constant	1.180***	1.180***	1.500***
	(0.893, 1.470)	(0.838, 1.520)	(1.180, 1.830)
Observations	336	258	278
R^2	0.074	0.083	0.116
Adjusted R^2	0.066	0.072	0.106
Residual Std. Error	0.757 (df = 332)	0.780 (df = 254)	0.734 (df = 274)
F Statistic	8.830*** (df = 3; 332)	7.640*** (df = 3; 254)	12.000*** (df = 3; 274)

Note: *p<0.1; **p<0.05; ***p<0.01

Table A7

Dif Gen2 Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.326***	0.366***	0.357***
	(0.208, 0.444)	(0.228, 0.504)	(0.227, 0.486)
Int_Mean	−0.079**	−0.088**	−0.111***
	(−0.151, −0.008)	(−0.173, −0.003)	(−0.190, −0.032)
Intdiff	0.024	0.023	0.002
	(−0.020, 0.068)	(−0.029, 0.074)	(−0.044, 0.048)
Constant	1.040***	1.060***	1.050***
	(0.951, 1.130)	(0.959, 1.170)	(0.957, 1.150)
Observations	287	219	234
R^2	0.100	0.118	0.125
Adjusted R^2	0.091	0.105	0.114
Residual Std. Error	0.756 (df = 283)	0.778 (df = 215)	0.738 (df = 230)
F Statistic	10.500*** (df = 3; 283)	9.570*** (df = 3; 215)	11.000*** (df = 3; 230)

Note: *p<0.1; **p<0.05; ***p<0.01

Table A8

Dif Joint Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.315^{***}	0.347^{***}	0.388^{***}
	(0.191, 0.438)	(0.202, 0.493)	(0.255, 0.521)
Int_Mean	-0.077^{*}	-0.088^{*}	-0.059
	(−0.161, 0.007)	(−0.187, 0.011)	(−0.150, 0.032)
INTMOMmean	0.0002	0.001	-0.007^{**}
	(−0.006, 0.006)	(−0.007, 0.008)	(−0.013, −0.0001)
Intdiff	0.018	0.021	0.00003
	(−0.028, 0.063)	(−0.033, 0.074)	(−0.047, 0.047)
INTMOMdiff	0.002	−0.001	0.002
	(−0.004, 0.007)	(−0.008, 0.005)	(−0.004, 0.007)
Constant	1.020^{***}	1.020^{***}	1.440^{***}
	(0.656, 1.380)	(0.597, 1.440)	(1.040, 1.840)
Observations	282	214	232
R^2	0.094	0.109	0.138
Adjusted R^2	0.077	0.087	0.119
Residual Std. Error	0.759 (df = 276)	0.783 (df = 208)	0.735 (df = 226)
F Statistic	5.710^{***} (df = 5; 276)	5.080^{***} (df = 5; 208)	7.260^{***} (df = 5; 226)

Note: *p<0.1; **p<0.05; ***p<0.01

Appendix B

Age 11.5 Replication

Table B1

Gen2 Measurement Model.

	g at Age 11.5
Estimator	MLR
Observations	7970
Parameters	15
ChiSqM_Value	273
ChiSqM_DF	5
ChiSqM_PValue	0
ChiSqBaseline_PValue	0
LL	-140683
UnrestrictedLL	-140524
LLCorrectionFactor	1.47
UnrestrictedLLCorrectionFactor	1.39
CFI	0.982
TLI	0.963
AIC	281396
BIC	281501
aBIC	281453
RMSEA_Estimate	0.082
RMSEA_90CI_LB	0.074
RMSEA_90CI_UB	0.09
RMSEA_pLT05	0
SRMR	0.02
AICC	281396

Table B2

Gen2 Factor Loadings.

	Test	Estimate	S.E.	Est./.S.E.	P.Value
1	DIGIT	0.551	0.010	53.500	0
2	MATH	0.744	0.007	102.000	0
3	RECOG	0.837	0.005	153.000	0
4	COMP	0.843	0.006	148.000	0
5	PPVT	0.751	0.009	83.500	0

Between Family Analyses

Table B3

Mean Gen1 Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean AFI		
	Mixed	Daughers	Sons
Gen1 Intel	0.012***	0.013***	0.014***
	(0.008, 0.017)	(0.009, 0.018)	(0.009, 0.019)
Constant	−0.754***	−0.801***	−0.856***
	(−1.010, −0.500)	(−1.090, −0.513)	(−1.140, −0.569)
Observations	342	264	282
R^2	0.090	0.101	0.106
Adjusted R^2	0.087	0.097	0.103
Residual Std. Error	0.700 (df = 340)	0.703 (df = 262)	0.691 (df = 280)
F Statistic	33.500*** (df = 1; 340)	29.400*** (df = 1; 262)	33.200*** (df = 1; 280)

Note: *p<0.1; **p<0.05; ***p<0.01

Table B4

Mean Gen2 Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean AFI		
	Mixed	Daughers	Sons
Gen2 Intel	0.104^{***}	0.117^{***}	0.091^{***}
	(0.044, 0.163)	(0.047, 0.187)	(0.027, 0.156)
Constant	−0.016	−0.007	−0.035
	(−0.094, 0.062)	(−0.097, 0.083)	(−0.121, 0.050)
Observations	339	262	278
R^2	0.034	0.040	0.027
Adjusted R^2	0.031	0.036	0.023
Residual Std. Error	0.729 (df = 337)	0.738 (df = 260)	0.721 (df = 276)
F Statistic	11.700^{***} (df = 1; 337)	10.700^{***} (df = 1; 260)	7.650^{***} (df = 1; 276)

Note: *p<0.1; **p<0.05; ***p<0.01

Table B5

Mean Joint Intelligence → Mean Gen2 AFI

	Dependent variable:		
	Mean AFI		
	Mixed	Daughers	Sons
INTMOMmean	0.012***	0.013***	0.014***
	(0.007, 0.017)	(0.007, 0.018)	(0.008, 0.020)
Int_Mean	0.010	0.017	−0.006
	(−0.060, 0.079)	(−0.064, 0.099)	(−0.081, 0.070)
Constant	−0.722***	−0.751***	−0.861***
	(−1.030, −0.416)	(−1.100, −0.401)	(−1.210, −0.511)
Observations	333	255	274
R^2	0.087	0.098	0.103
Adjusted R^2	0.082	0.091	0.096
Residual Std. Error	0.705 (df = 330)	0.709 (df = 252)	0.696 (df = 271)
F Statistic	15.800*** (df = 2; 330)	13.800*** (df = 2; 252)	15.600*** (df = 2; 271)

Note: *p<0.1; **p<0.05; ***p<0.01

Within Family Analyses

Table B6

Dif Gen1 Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.303***	0.328***	0.376***
	(0.187, 0.419)	(0.192, 0.465)	(0.251, 0.501)
INTMOMmean	−0.003	−0.002	−0.008***
	(−0.007, 0.002)	(−0.008, 0.004)	(−0.013, −0.003)
INTMOMdiff	0.001	0.001	0.0003
	(−0.003, 0.006)	(−0.005, 0.006)	(−0.005, 0.005)
Constant	1.180***	1.180***	1.500***
	(0.893, 1.470)	(0.838, 1.520)	(1.180, 1.830)
Observations	336	258	278
R^2	0.074	0.083	0.116
Adjusted R^2	0.066	0.072	0.106
Residual Std. Error	0.757 (df = 332)	0.780 (df = 254)	0.734 (df = 274)
F Statistic	8.830*** (df = 3; 332)	7.640*** (df = 3; 254)	12.000*** (df = 3; 274)

Note: *p<0.1; **p<0.05; ***p<0.01

Table B7

Dif Gen2 Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.344***	0.369***	0.382***
	(0.225, 0.463)	(0.232, 0.506)	(0.251, 0.512)
Int_Mean	−0.097***	−0.120***	−0.130***
	(−0.168, −0.026)	(−0.205, −0.035)	(−0.207, −0.052)
Intdiff	0.037	0.034	0.015
	(−0.007, 0.081)	(−0.016, 0.084)	(−0.032, 0.062)
Constant	1.050***	1.070***	1.080***
	(0.962, 1.140)	(0.963, 1.170)	(0.979, 1.170)
Observations	286	223	230
R^2	0.117	0.131	0.149
Adjusted R^2	0.108	0.119	0.138
Residual Std. Error	0.751 (df = 282)	0.773 (df = 219)	0.734 (df = 226)
F Statistic	12.500*** (df = 3; 282)	11.000*** (df = 3; 219)	13.200*** (df = 3; 226)

Note: *p<0.1; **p<0.05; ***p<0.01

Table B8

Dif Joint Intelligence → Dif Gen2 AFI

	Dependent variable:		
	Gen2 AFI Differences		
	Mixed	Daughers	Sons
AFIKIDmean	0.328^{***}	0.339^{***}	0.417^{***}
	(0.203, 0.452)	(0.194, 0.483)	(0.282, 0.552)
Int_Mean	-0.098^{**}	-0.131^{**}	-0.071
	(−0.184, −0.013)	(−0.234, −0.029)	(−0.163, 0.021)
INTMOMmean	0.001	0.002	-0.007^{**}
	(−0.005, 0.007)	(−0.005, 0.010)	(−0.014, −0.0001)
Intdiff	0.034	0.037	0.012
	(−0.011, 0.080)	(−0.016, 0.089)	(−0.036, 0.059)
INTMOMdiff	0.002	−0.002	0.001
	(−0.004, 0.007)	(−0.008, 0.005)	(−0.004, 0.007)
Constant	0.984^{***}	0.925^{***}	1.470^{***}
	(0.615, 1.350)	(0.489, 1.360)	(1.060, 1.890)
Observations	278	215	226
R^2	0.112	0.125	0.162
Adjusted R^2	0.096	0.104	0.143
Residual Std. Error	0.751 (df = 272)	0.775 (df = 209)	0.727 (df = 220)
F Statistic	6.860^{***} (df = 5; 272)	5.970^{***} (df = 5; 209)	8.500^{***} (df = 5; 220)

Note: *p<0.1; **p<0.05; ***p<0.01

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