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The nature and nurture of high IQ: An extended sensitive period for intellectual development

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Abstract

IQ predicts many measures of life success, as well as trajectories of brain development. Prolonged cortical thickening observed in individuals with higher IQ might reflect an extended period of synaptogenesis and high environmental sensitivity or plasticity. We tested this hypothesis by examining the timing of changes in the magnitude of genetic and environmental influences on IQ as a function of IQ score. We find that individuals with higher IQ show high environmental influence on IQ into adolescence (resembling younger children), whereas individuals with lower IQ show high heritability of IQ in adolescence (resembling adults), consistent with an extended sensitive period for intellectual development in more intelligent individuals. These patterns hold across a cross-sectional sample of almost 11,000 twin pairs, and a longitudinal sample of twins, biological siblings, and adoptive siblings.

Keywords

Intelligence; Behavior Genetics; Individual Differences; Cognitive Development; Cognitive Ability

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Authorship

A.M.B. developed the study concept, performed analyses and wrote the manuscript under the supervision of J.K.H. Y.M. provided theoretical input to inform interpretation and critical revisions. Testing and longitudinal data collection was directed by D.B., J.C.D., J.K.H., M.M., N.G.M., S.A.P., R.P., S.J.W. & M.J.W. M.C.K. and C.M.A.H. aided in data analysis. All authors provided feedback on the manuscript.

Introduction

Adult IQ is a measure of cognitive ability that is predictive of social and occupational status, educational and job performance, adult health and longevity (Gottfredson, 1997; Neisser *et al*, 1996; Whalley & Deary, 2001). Individuals with IQ scores at the high end of the distribution show distinct timing of postnatal structural changes in cortical regions known to support intelligence, which has been posited to reflect an extended “sensitive period” (Shaw *et al*, 2006). Specifically, change in cortical thickness in frontal and temporal regions is cubic during development, with initial thickening in childhood, followed by thinning in late childhood/adolescence that levels out in young adulthood (see also Shaw *et al*, 2008), matching the patterns of synaptogenesis and pruning observed in postmortem prefrontal tissue (Petanjek *et al*, 2011). Individuals of superior IQ (compared to average and high) show more intense and prolonged cortical thickening, followed by more rapid thinning. This distinct trajectory may reflect prolonged synaptogenesis and an extended sensitive period, during which the brain is particularly responsive to environmental input (Shaw *et al*, 2006).

Further evidence for a link between cortical thickness and IQ comes from the finding that common genes influence change in cortical thickness and IQ in adulthood (Brans *et al*., 2010). In addition, IQ and cortical thickness show similar patterns of change across development in the magnitude of genetic and environmental influences. Specifically, the heritability (magnitude of genetic influence) of IQ and the heritability of cortical thickness in brain regions associated with IQ both increase during childhood and adolescence, while environmental influences decrease in importance (Haworth *et al*., 2010; Bartels, Rietveld, Van Baal & Boomsma, 2002; Brant, Haberstick, Corley, Wadsworth, DeFries & Hewitt., 2009; Lenroot *et al*, 2009).

These results are suggestive of an extended sensitive period for IQ development: cortical thickening, which is associated with IQ, occurs over an extended period for individuals with higher IQ, corresponding to prolonged sensitivity to the environment. These results are only suggestive, however, because developmental changes do not necessarily correspond to changes in sensitivity to the environment. There is no direct evidence for individual differences in the length of a sensitive period for IQ.

We provide an empirical test of the extended sensitive-period hypothesis of high IQ, by examining changes in the magnitude of genetic and environmental influence on individual differences in IQ scores throughout development. As noted above, the magnitude of environmental influences on IQ decreases across development. We test whether these decreases in environmental influence occur later in development for individuals with higher IQ, consistent with a prolonged sensitivity to the environment. We focus on influences of the shared family environment rather than individual-specific environment, because the developmental change in environmental influence on intelligence is mainly driven by a reduction in influence of the shared family environment. Additionally, the shared family environment should arguably be the driving force behind experiential influence on IQ, because shared family environmental influences are highly correlated across different ages such that their effects can accumulate across development, while individual-specific

environmental factors tend to be more age-specific and include measurement error (Brant *et al*, 2009).

We use a cross-sectional sample of 11,000 twin pairs aged from 4 –71 years, and a smaller longitudinal replication sample of twins, biological siblings and adoptive siblings tested from ages 1 to 16. Previously published investigations using the datasets examined here have tested for differences between high IQ and IQ in the normal range. Although no difference was reported in the etiology of individual differences (Haworth *et al*, 2009; cross-sectional GHCA sample) nor in their trajectories of developmental change (Brant *et al*, 2009; Longitudinal Twin Sample), these investigations discretized IQ rather than examining continuous trends, and did not test whether the relationship between IQ score and heritability/environmentality was specific to adolescence. Here we test this hypothesis explicitly. We predict that environmental influences should remain high for longer in higher IQ individuals, and that genetic influences conversely should remain lower for longer. IQ score should therefore be associated with magnitude of genetic and environmental influence in adolescence (but not in childhood or adulthood, where regardless of IQ, environmental influences should be high or genetic influences should be high, respectively).

Method

Participants and Measures

Participants for the initial cross-sectional analysis were 10,897 monozygotic (MZ; identical) and dizygotic (DZ; fraternal) twin pairs amalgamated from the 6 institutions in four different countries (USA, UK, The Netherlands and Australia) that constitute the Genetics of High Cognitive Ability Consortium (GHCA). Zygosity was determined in almost all cases by analysis of DNA microsatellites, blood group polymorphisms or other genetic variants (for sample-specific detail see supplementary materials). The sample is described in detail elsewhere (Haworth *et al*, 2010) and is summarized in Table 1.

The longitudinal sample included MZ and DZ twins from the Colorado Logitudinal Twin Study (LTS) and adoptive and biological sibling pairs from the Colorado Adoption Project (CAP), two prospective community studies of behavioral development at the Institute for Behavioral Genetics (IBG; University of Colorado at Boulder). A total of 483 same-sex twin pairs have participated in the LTS study, ascertained from local birth records (264 MZ and 219 DZ)*. Twin zygosity status was determined using 12 molecular genetic markers as described elsewhere (Haberstick and Smolen, 2004). In the CAP, families with an adoptive child and matched community families were ascertained in infancy. If siblings were born or adopted into the families they were included in the study. For many families, more than one sibling pair per family was available. The current analysis used the sib pair with complete IQ data at the most ages. The final sample consisted of 185 biological sibling pairs and 184 adoptive sibling pairs. Of these, 64 biological pairs were available only at age 16 and the same was true for 75 adoptive pairs. For more details on the samples see Rhea, Gross, Haberstick and Corley, 2006 (LTS) and DeFries, Plomin and Fulker, 1994 (CAP). The IQ

*This total exceeds that reported in cross-sectional sample, which included only twins that had an IQ score measured at age 7 or above.

tests administered at each of the seven measured ages are outlined in Table 2. The scores were standardized within age and across samples to maintain the slightly higher mean scores in the CAP.

Twin Methodology

Extensions of DeFries-Fulker regression, a special case of linear regression for deriving genetic and environmental components of variance in pairs of related individuals, were employed. DeFries-Fulker regression (for details see Cherny, Cardon, Fulker & DeFries, 1992) predicts the score of one member of a sibling pair from the score of the other, the coefficient of relationship - which takes a value of 1.0 for MZ twins (100% genetic sharing), 0.5 for DZ twins and biological siblings (50% genetically related on average) and 0.0 for adoptive siblings (who are not genetically related) - and the interaction between these two variables. When the data is standardized, as it is here, this regression yields direct estimates of the heritability (h^2) of the measured trait and proportional influence of the family-wide environment (c^2) on differences between individuals in the sampled population. The influence of individual-specific environments (e^2) can be derived by subtraction.

The addition of other variables into the regression equation, which are allowed to interact with the existing predictors, tests whether the magnitude of either h^2 or c^2 is changeable in the population according to the variables of interest. In the current study, we were interested in whether the magnitude of h^2 or c^2 for IQ is moderated by IQ score itself, so we added a quadratic ability term (the predicting siblings' scores squared) and quadratic term $\times$ coefficient of relationship interaction (Cherny *et al*, 1992). The significance of these interaction terms assesses whether there is a linear, continuous relationship between IQ score and c^2 or h^2 respectively.

To directly test the extended-sensitive period hypothesis of high IQ, we were additionally interested in whether any effect of score on h^2 or c^2 was restricted to a certain age range. This was examined by estimating the coefficients for the quadratic score term separately at each measured age. In the cross-sectional GHCA sample, we were able to additionally test for significant differences *between* the magnitude of the ability-dependent terms at each age by adding an age covariate into the regression equation. The sensitive period hypothesis predicts that there is only a relationship between IQ score in adolescence (i.e. the coefficient for the age term should be zero at all other times. For this reason continuous modeling of the effect of age was not possible and it was therefore decided to use discrete age categories. We split the sample into three age groups: childhood (4yrs to 12yrs; n pairs = 6044), adolescence (13–18yr; n pairs = 4304) and adulthood (18yrs +; n pairs = 549) and constructed orthogonal contrast codes based on these criteria: A linear code comparing the childhood and adulthood groups and a quadratic code that compared these groups collectively to the adolescent group. Since our hypothesis predicts the values of h^2 and c^2 to be dependent on IQ score only in adolescence (where higher scoring participants will have a child-like etiology and lower scorers will resemble adults), we expected that the three-way interactions between the quadratic age contrast code, ability and the h^2 and c^2 terms would be significant, while the equivalent terms for the linear age code would not be (as no interactions with ability are expected in either childhood or adulthood). Although the

appropriate boundaries between the age categories were somewhat ambiguous, the broad expected pattern was clear, so we chose childhood, adolescent and adulthood age boundaries as typically defined.

In the longitudinal sample, we added an extra covariate, age gap in days between the siblings in each pair, into the regression (0 for all twin pairs), and all results reported from this sample are from analyses including this as an interacting variable with the c^2 and h^2 terms and the ability-dependent terms. Since maximum sharing of the family environment occurs when siblings are the same age, and the groups in our sample differ systematically not only by genetic relatedness but also by average age gap (adoptive siblings being more disparate than biological siblings and biological siblings more than twins), it is prudent to account for this confounding variable in the analysis, so as not to overestimate the magnitude of the heritability estimates.

For every analysis described, each pair appears twice in the data set, with the score of each member of a pair appearing once as a predictor and once as a dependent variable. This is routine in DeFries-Fulker regression using unselected samples because there is no *a priori* reason to favor a particular twin assignment. This procedure does, however, artificially narrow the standard errors derived from regression analysis (which assumes independence). We addressed this by bootstrapping the regression estimates in the GHCA sample by resampling first at the family level and then at the twin assignment level, and by following the robust standard error correction outlined by Kohler and Rodgers (2001) in the longitudinal follow-up, which accounts for the fact that observations are only independent at the level of the twin pair and not the individual observations. Further explanation and details of all analyses including the regression equations can be found in the supplementary materials.

Results

Cross-sectional analysis of the GHCA sample

Sample characteristics and sample-wide analysis—Table 1 outlines the size and mean age of the 6 subsamples, along with the different tests used to measure IQ. The mean age of the sample is 13.06 years (range :4.33 and 70.03 years). Mean age differs considerably between the subsamples, from 6 in the Western Reserve sample to almost 18 in the Netherlands twin register. There is also a considerable difference in the range between the samples, meaning that some age groups are primarily made up of particular samples. The proportion of pairs for each sub-sample and the total sample falling into each of the three age groups is outlined in Table 1. The IQ tests used differ between samples, reflecting age-appropriate, widely-used and validated tests. For the analyses shown here, after residualization for age and sex, the IQ scores were standardized within each study to maintain the subsample structure.

For the sample as a whole, the proportional heritability (h^2) was .55 (95% CI .49–.61), influence of the family environment (c^2) .22 (95% CI .18–.26) and of the individual-specific environment (e^2) .23 (95% CI .16–.39). This finding closely matches the results found in the same sample using different methodology (structural equation modeling; Haworth *et al*,

2010). Examining the influence of IQ score on these parameters, there was a significant effect on c^2 ($\beta = .036$, $p = .026$), such that the influence of c^2 increased as IQ score increased. There was a slight trend for a decrease in h^2 as IQ score increased ($\beta = -.027$, bootstrapped $p = .187$). “Etiology” in the following section collectively refers to the estimates for c^2 and h^2 . As anticipated (for reasons outlined in the introduction), there were no detectable influences of IQ score on the magnitude of e^2 . For this reason we do not report results for this predictor beyond the sample-wide value.

Age as a moderating variable—Separate analysis of the subsamples indicated variability in the strength of the relationship between IQ score and the causal influences on IQ, suggesting a moderation of this relationship by age. We therefore performed the regression analysis with age as an interacting variable, as described in the methods to test the age-dependence of the interaction between score and both heritability and family environmental effects described above. r . As expected there was no moderation by the linear age contrast on the score-etiology relationship (on separate analysis of the age groups, the score-etiology relationship in both childhood (ages 4–12) and adulthood (age 18+) was not significantly different from zero). However, the quadratic contrast code, comparing the adolescent (age 12–18) group to the childhood and adulthood groups collectively, showed that the adolescent group had a larger association between IQ and both higher environmental influence and lower genetic influence, consistent with the extended sensitive period hypothesis. Specifically, both the increase in c^2 and the decrease in h^2 as IQ score increased were significantly greater in adolescence ($\beta = .05$, $p = .04$ and $\beta = -.06$, $p = .04$, respectively). In adolescence, IQ score predicts the pattern of genetic influence ($\beta = -.14$, $p < .001$) and environmental influence ($\beta = .12$, $p < .001$), in a manner consistent with lower IQ individuals transitioning earlier to an adult-like pattern of these influences*.

Analyses removing scores below the 5th and above the 95th percentile ruled out undue influence of extreme scores on the results. We also assessed whether any of these results differed according to the sex by repeating the analysis with non sex-residualized data and adding sex as an interacting variable. Males have a slightly higher mean IQ in this sample ($\beta_{\text{sex}} = .061$, $p < .001$) as would be predicted given the age range of our sample (Lynn & Kanazawa, 2011). However, no significant interactions by sex were found.

Transitions in causal influences—Figure 1 displays estimates for heritability and the influence of the shared family environment in the 4–12, 12–18 and 18+ year old participants separately estimated for the top and bottom half of the ability distribution (median split) at each age, to visualizing the relationship between age, IQ score and the etiological influences on IQ[†]. It can be seen that the estimates of both c^2 and h^2 change with age, with the magnitude of shared environmental influence decreasing and genetic influence increasing between childhood and adulthood, consistent with previous results in this sample and others (see e.g. Haworth *et al*, 2010). The magnitude of these effects is largely equal across ability for the two groups, representing a consistent beginning and end point in developmental change irrespective of ability level. However, the timing of this transition is different for the

[†]For these analyses, a sibling pair was only double entered if both siblings met the score criteria.

two ability groups. For the lower ability group the period of maximum change occurs between childhood and adolescence, indexed by the steeper slope of the hashed lines between these two time points. There is largely no change between adolescence and adulthood, as reflected by the relatively flat hashed lines between these points. For the higher ability group, however, a reciprocal relationship exists. In this group there is largely no change between childhood and adolescence (the solid lines between these points are again nearly flat), with the change in causal influence occurring between adolescence and adulthood.

Longitudinal sample: Table 2 presents descriptive statistics for the longitudinal sample. The estimates of h^2 and c^2 for each of the seven testing ages (with age gap modeled) are presented in Table 3. The pattern of increasing genetic influence and decreasing influence of the shared environment corroborates that seen previously, rising from .42 at age one to .85 at age 16. The influence of the shared environment shows the opposite effect, reducing in importance from a high of .39 to a low of .01. Additionally, we confirmed the influence of IQ score on the estimates of these parameters in adolescence in the same direction as in the cross-sectional analysis (last two columns of Table 3). At age 16, the estimate of c^2 increased as ability increased and h^2 decreased in importance, with no significant influence of ability at the earlier ages. We were, however, unable to test the sample in adulthood to confirm the transience of this effect.

Discussion

We have presented evidence from two separate sets of data that supports the existence of a sensitive period in IQ development that is extended in individuals of higher IQ. Using a large-cross-sectional dataset of twins, we found a shift in causal influences on IQ between childhood and adulthood, away from environmental and towards genetic influences. Moreover, we found that the period of child-like levels of environmental influence was prolonged in higher IQ individuals, while lower IQ individuals shifted earlier to an adult-like pattern, demonstrating that higher IQ is associated with a prolonged sensitive period. This result was replicated in a longitudinal sample of twin, biological and adoptive siblings. These results were found for the influence of the family-wide environment and not the individual specific environment (including measurement error), consistent with predictions from prior longitudinal behavior genetic research showing age related changes in the relative magnitude of the former but not the latter component of variance.

Alternative explanations of these results can be ruled out (see supplementary materials for details of supporting analyses). First, assortative mating (the tendency for parents to resemble each other in cognitive ability) could artifactually increase the influence of the family-wide environment, and so could contribute to our results if assortative mating were higher in the parents of higher IQ individuals. However, we find that higher IQ parents actually show less assortative mating; the difference between parental IQ scores is positively correlated with mean parental IQ score. Thus, assortative mating could only contribute to an underestimation of the strength of the results reported here. Second, if different traits were being measured at different IQ levels, and these traits differed in their extent of genetic and environmental influences, this could give a false impression of a single trait that varied by

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IQ in the extent of genetic and environmental influences. However, principal component analyses showed that the same trait was measured across IQ levels. Finally, genotype-environment interactions could contribute to our results, if the environmental variables were correlated with IQ, and estimates of environmental influence were greater for higher levels of the environmental variable. We tested for gene-environment interactions with parental education and parental IQ in the LTS twins' age 16 scores. However, no interaction was present for parental education, and heritability of IQ was higher at higher levels of parental IQ, which would cause *underestimation* of the interaction between the individual's own score and their environmental sensitivity. Moreover, all of these alternative explanations would face an additional challenge in explaining why the link between IQ and genetic and environmental influence *changes* across development.

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Our findings raise the question of why a prolonged sensitive period in IQ development might be associated with higher IQ. One possibility is that protracted development is beneficial for development of higher and uniquely human cognitive functions, such as those measured by IQ tests (Rougier et al., 2005). This pattern may be supported via genetic polymorphisms in higher IQ individuals which limit the rate of developmental cellular changes. Similar arguments have been made for prolonged immaturity being beneficial for other aspects of cognitive development (Bjorkland, 1997; Newport, 1990; Thompson-Schill, 2009). *However*, individuals with an eventual high IQ show this tendency from early in development (Deary, Whalley, Lemmon, Crawford & Starr, 2000), challenging the idea that prolonged immaturity alone leads to higher IQ. An alternative possibility is that having a higher IQ prolongs sensitivity to the environment. For example, heightened levels of attention and arousal, as one may find in individuals of higher IQ, may allow plasticity to occur later into development (Knudsen, 2004). Relatedly, individuals of higher IQ may be more open to experience, more likely to try things and change in response to experience, whereas lower IQ individuals are less motivated as they do not get as much positive feedback from learning experiences. However, this explanation is not without its own issues. The increase in genetic influence over development comes from both an increase in importance of existing genetic influences and addition of new genetic influences (Brant *et al*, 2009). If the extension of the sensitive period is a feedback process from increased cognitive ability, it is unclear how this feedback process would lead to a delay in the introduction of new genetic influences.

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The most prominent theory of developmental increases in heritability of IQ posits that individuals gain more scope throughout development to shape their own environments, based on their genetic propensities (active gene-environment correlation), which causes an increase in genetic influence over time (Plomin, DeFries & Loehlin, 1977; Haworth *et al*, 2010). Our results challenge this explanation as they show a later increase in heritability for individuals of higher IQ. To explain these results in the context of active gene-environment correlations, one would need to posit, counter-intuitively, that higher IQ individuals seek out environments concordant with their genetic propensities later in development than lower IQ individuals.

The reason for developmental increases in heritability of IQ thus remains unclear - other possibilities include amplification of existing genetic influence by increasing population

variance in cognitive ability and the simultaneous limiting of environmental influences/ introduction of new genetic influences by synaptic pruning processes and myelination at the end of the sensitive period (Plomin, 1986; Plomin, DeFries & Loehlin, 1977; Tau & Peterson, 2010). While resolving that debate is beyond the scope of the current work, our key contribution is in showing for the first time that the timing of the decline in the magnitude of environmental influence depends upon IQ, consistent with the extended sensitive period hypothesis. Further research investigating the developmental influence of specific genes and environments and aided by a better molecular-level understanding of the mechanisms underlying typical brain development will help resolve this question.

Our results suggest that, like cortical thickness, other brain-related measures (such as functional connectivity, synaptic density, and characteristics of neurotransmitter systems) will show differing relationships to IQ across development, and that the timing of this change will be dependent on IQ score. This indicates an important new direction in the search for biological and cognitive markers of IQ, and in the study of the genetic variation and developmental processes underlying individual differences in cognitive ability.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Ability-related differences in the magnitude of genetic and environmental influence were observed specifically during adolescence. Notably, the lower-ability subjects underwent more age-related change before this point, as indicated by the sloped dotted lines (left side). In contrast, the higher-ability subjects underwent more age-related change after this point, as indicated by the sloped solid lines (right side). c_squ = proportion of variance accounted for by the family-wide environment, h_squ = proportion of variance accounted for by genetic influences. *Note: High/Low IQ refers to subjects scoring above/below the median score at each age. Estimates at each age do not sum to 1 as e^2 is not plotted.*

TABLE 1

Genetics of High Cognitive Ability Consortium Sample Characteristics

Sample	Number of pairs	Mean Age (Range)	IQ measure
Ohio, USA: The Western Reserve Reading Project	292 (121MZ, 171DZ)	6.07 (4.33–7.92) 100% child	Stanford Binet Intelligence Scale (short form). Summed and standardized for age and sex.
United Kingdom: Twins Early Development Study (TEDS)	4061 (1529MZ, 2532DZ)	11.57 (10.08–12.84) 100% child	Two WISC-III verbal subtests (information and vocabulary), WISC-III picture completion, Ravens Standard and Advanced Progressive Matrices. Standardized and summed.
Minnesota, USA: The Minnesota Center for Twin and Family Research (MCTFR)	1870 (1187MZ, 683DZ)	13 (11–17) 51% child 49% adolescent	Abbreviated WISC-R or WAIS-R as age-appropriate.
Colorado, USA: Longitudinal Twin Study (LTS), Colorado Twin Study (CTS), Colorado Learning Disabilities Research Center (CLDRC)	2863 (LTS=390, CTS=696, CLDRC=1777; 1299MZ, 1564DZ).	13.12 (6–25) 47% child 45% adolescent 8% adult	WISC-R, WISC-III, WAIS-III or WAIS-R (block design & vocab. only in CTS).
Australia: The Twin Cognition Study	853 (338MZ, 515DZ)	16.00 (15–22) ~100% adolescent <1% adult	3 verbal and 2 performance subtests from the Multidimensional Aptitude Battery.
Netherlands: The Netherlands Twin Register	958 (437MZ, 521DZ)	17.99 (5.67–71.03) 54% child 19% adolescent 27% adult	Standard age-appropriate IQ tests (see Boomsma <i>et al.</i> , 2008 for further details)
Total Sample	10897 (4911MZ, 5986DZ)	13.06 (4.33–71.03) 55.5% child 39.5% adolescent 5% adulthood	<i>g</i> scores standardized within each study after residualization for age and sex

note: WISC-III = Wechsler Intelligence Scales for children -Third Edition; WISC-R = Wechsler Intelligence Scales for children - Revised; WAIS-R = Wechsler Adult Intelligence Scale - Revised; WAIS-III = Wechsler Adult Intelligence Scale - Third Edition.

TABLE 2

Demographic and descriptive information for the LTS/CAP samples

Age	n pairs LTS	n pairs CAP	mean age (sd)	Test administered	mean score (sd)
1 yr	342 (245MZ, 197DZ)	291 (150Bio., 141Ad.)	1.12 (.09)	BSMD	106.86 (13.83)
2 yrs	398 (215MZ, 183DZ)	270 (139Bio., 131Ad.)	2.03 (.05)	BSMD	108.00 (17.86)
3 yrs	381 (204MZ, 177DZ)	254 (130Bio, 124Ad.)	3.03 (.06)	S. Binet Intell. Scale	104.61 (16.93)
4 yrs	378 (203MZ, 175DZ)	260 (134Bio., 126Ad.)	4.01 (.03)	S. Binet Intell. Scale	105.73 (13.94)
7 yrs	410 (222MZ, 188DZ)	262 (134Bio., 128Ad.)	7.41 (.37)	WISC-III; WISC-R	108.66 (13.43)
12 yrs	377 (195MZ, 182DZ)	267 (137Bio., 130Ad.)	12.45 (.38)	WISC-III; WISC-R	106.02 (12.95)
16 yrs	399 (213MZ, 186DZ)	352 (178Bio., 174Ad.)	16.6 (1.02)	WAIS-III; WAIS-R	103.92 (11.60)
Full	483 (264MZ, 219DZ)	384 (193Bio., 191Ad.)			

note: MZ = monozygotic twin pairs; DZ = dizygotic twin pairs, Bio. = Biological sibships, Ad. = adoptive sibships (no genetic relationship); BSMD = Bayley Scales of Mental Development, S. Binet = Stanford Binet Intelligence Scale, WISC-III = Wechsler Intelligence Scale for Children - Third Edition; WISC-R = Wechsler Intelligence Scale for Children - Revised; WAIS-III = Wechsler Adult Intelligence Scale - Third Edition; WAIS-R = Wechsler Adult Intelligence Scale - Revised

TABLE 3

Heritability and shared environmental effects in the LTS/CAP combined sample when age gap between sibling pairs is modeled as an interacting variable, with 95% confidence intervals. Rightmost two columns report the moderating effect of ability score on these estimates.

Age group	h^2 (95% c.i.s)	c^2 (95% c.i.s)	ability* h^2 (95% c.i.s)	ability* c^2 (95% c.i.s)
1 (n = 635 pairs)	0.42 (.12,.72)*	0.17 (-.07,.41)	-0.03 (-.13,.08)	0.00 (-.07,.06)
2 (n = 583 pairs)	0.42 (.23,.62)*	0.39 (.21,.57)*	-0.01 (-.12,.09)	-0.03 (-.12, .07)
3 (n = 556 pairs)	0.33 (-.02,.67)	0.35 (.08,.62)*	-0.14 (-.36,.08)	0.05 (-.08, .18)
4 (n = 561 pairs)	0.55 (.30,.79)*	0.21 (0.00,.43)	-0.07 (-.17,.03)	0.01 (-.06,.08)
7 (n = 601 pairs)	0.54 (.33,.75)*	0.28 (.09,.47)*	-0.03 (-.10,.44)	-0.01 (-.07,.04)
12 (n = 571 pairs)	0.63 (.43,.82)*	0.20 (.02,.38)*	-0.01 (-.29,.21)	0.04 (-.15,.23)
16 (n =730 pairs)	0.85 (.67,1.03)*	0.01 (-.16,.19)	-0.08 (-.16, -.001)*	0.07 (.003, .14)*

* = significant at $P < .05$ when s.e.s are corrected for non-independence due to double entry