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The Developmental Etiology of High IQ

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Abstract

The genetic and environmental trends in IQ development were assessed in 483 same-sex twin pairs in the Colorado Longitudinal Twin Study using maximum-likelihood model-fitting analysis. The twins were assessed periodically from ages 1 to 16. Results show a decreasing influence of shared environment and an increasing influence of heritability across development, with large and increasing age to age stability of genetic influences. Non-shared environment contributes almost exclusively to age to age change. Similar analyses were conducted designating the top 15% of the sample as having high IQ at each age. The developmental etiology of high IQ did not significantly differ from that found for the continuous measure in this relatively novel analysis. These results demonstrate relatively early stability in etiological influences on IQ and have potential implications for gene-finding efforts, suggesting that samples selected for high IQ can be used to find genetic variation that will be applicable to the full range of the IQ distribution, although conclusive demonstration that the same genes are indeed involved was beyond the scope of this study.

Introduction

There is an extensive literature relating to the etiological basis of IQ and its development. However, the question of whether there are differences in etiology at different levels of IQ is less well studied. The current investigation is the first extended longitudinal study of cognitive development in the upper end of the IQ distribution, using an etiologically informative sample (The Colorado Longitudinal Twin Study). We additionally address cognitive development from infancy to late adolescence across the full distribution of ability.

Numerous studies have shown that the genetic and environmental influences on intelligence change from age to age. This phenomenon was first noticed in a small adoption study by Skodak and Skeels in 1949. Since then, research using data from both twins and adoptive children has focused on the resulting pattern of increased heritability and decreased shared environmental effects with age and found it to be robust and widespread (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Boomsma & Molenaar, 1987; Bouchard & McGue, 1981; Cardon, Fulker, DeFries and Plomin, 1992; Cherny & Cardon, 1994; Eaves, Long, & Heath, 1986; Fulker, Cherny, & Cardon, 1993; Humphreys & Davey, 1988; McGue, Bouchard, Iacono & Lykken, 1993).

Petrill *et al.* (2004) used data from siblings who participated in the Colorado Adoption Project (CAP) at ages one through sixteen to examine these factors further. Results showed positive correlations between intelligence scores at all ages except between age one and ages nine and twelve, and a pattern of higher correlations between intelligence scores closer

together in time. The best-fitting model included a common additive genetic factor, no shared environment and only time-specific non-shared environmental factors. Similar results were obtained from looking at combined data from the CAP and the Longitudinal Twin Study (including non-twin siblings) using the same multivariate model by Bishop, Cherny, Corley, Plomin, DeFries and Hewitt (2003) from ages one to ten. Non-shared environment contributed to continuity from age seven through age-to-age transmission and to change (innovation) throughout development. Further, shared environment contributed exclusively to continuity through a common factor and additive genetic factors were accounted for by age-to-age transmission and by innovations up to age nine.

Results of behavioral genetic analyses have been reported from ages two to ten from the Twins Early Development Study (TEDS) -a sample of over 11,000 pairs of MZ and DZ twins recruited in the UK between 1994 and 1996. Spinath, Ronald, Harlaar, Price and Plomin (2003) extracted the first principal component from cognitive tasks given to the participants at ages two, three and four, finding evidence for only modest genetic influence, and a large effect of shared environment. Davis, Arden and Plomin (2008) used the same method at ages seven, nine and ten, demonstrating that continuity was due to genetic and shared environmental factors. Both genetic and shared environmental influences also contributed to differences between ages and methods. Non-shared environment contributed almost entirely to differences. There was a high genetic correlation across age, test composition, and method of administration. Additionally, genetic influences were stronger and shared environmental factors more modest than in earlier childhood.

It is clear from the above discussion that genetic and shared environmental influences can be concluded to contribute mostly to stability, whereas non-shared environment contributes mostly to change across ages. Additionally, genetic contributions to IQ increase across development, while shared environmental influences decrease. One aim of the current study was to extend this research by fitting the simultaneous common factor, simplex and unique factor model in a systematic age-to-age twin sample to provide a more comprehensive analysis of developmental trends from infancy through late adolescence.

The second aim of the current study was to examine whether the factors outlined above apply equally across the distribution of intelligence scores, specifically to the upper levels of ability. Does the available evidence suggest a similar effect for high IQ individuals? Cherny, Cardon, Fulker and DeFries (1992) used an extension of the DeFries-Fulker multiple regression methodology and found no evidence for either linear or quadratic influences of level of cognitive ability on heritability or shared environmental effects in the Longitudinal Twin Study at ages one, two or three. This replicated the results of Horn, Loehlin and Willerman (1982) in the Texas Adoption Project and Vogler and DeFries (1983) in the Hawaii Family Study of Cognition. Sundet, Eilertsen, Tambs & Magnus (1994) found no evidence for differential heritability across the spectrum of ability in a sample of over 3000 Norwegian male young adults.

A more recent study by Ronald, Spinath and Plomin (2002) compared the pattern of etiological influences found for the full ability distribution found for a sample of 1943 preschool twin pairs to those found in a series of DF extremes analyses using increasingly stringent thresholds for proband status from the top 15 to the top 2.5 percent of the sample. Results indicated that genetic influences accounted for 20% of the variance, shared environmental factors accounted for over 70% of the variance, with around 10% attributable to unique environment. This pattern of etiology did not differ according to ability level. This study once again suggests that high cognitive ability is the quantitative extreme of the genetic factors influencing the full ability distribution, in children as well as adults.

Other studies *have* found a differential heritability estimate depending on level of cognitive ability, but with conflicting results. Specifically, Detterman, Thompson and Plomin (1990) found higher heritability and less influence of common environment at lower ability levels in a small sample of twins, as did Bailey and Revelle (1991). However, Jensen (1987) found higher heritability at higher ability levels in a much larger sample by examining the mean weighted correlations between intrapair sums and absolute differences in MZ versus DZ twins. Thompson, Detterman & Plomin (1993), in an extension to their previous investigations found no differences in heritability across ability level in another small sample from the Western Twin Project, although they note a trend towards higher heritability in the upper ranges.

Conflicting results are evident in this field, showing the need for a large-scale investigation of this question. Additionally, two descriptive studies examining intelligence test scores from 'gifted' children with IQs of 120 and above (Wilkinson, 1993) and 130 plus (Sweetland, Reina and Tatti (2006) show patterns that are not typical of the general population. Large verbal-performance discrepancies are seen in IQ scores, as well as more idiosyncratic subtest score scatter. The majority of children tested in these studies were third graders, which suggests an early difference in the characteristics of high IQ. The different characteristics of the phenotype in the upper range suggest at least the possibility of different etiological factors.

Regardless of similarity or differences in etiological influences on high ability at a specific age compared to those of the rest of the distribution, it is possible that the developmental model of stability and change of causative factors in development may vary depending on ability level. This is a theory that has some intuitive merit. For example, the existence of child prodigies suggests that, in some cases at least, high ability children reach their potential earlier, with less influence of structured educational resources, than other children. This could suggest an earlier and stronger influence of stable genetic factors in childhood. On the other hand, some historically renowned geniuses did not stand out early in life, suggesting a quite different pattern of influence. It is possible that for such children, specific genetic influences come into play later in development, which may be related to *g* or to other characteristics, such as creativity or motivation. In this case, one may expect genetic influences on stability of high IQ to be the same as in the general population, with a strong possibility of age-specific genetic or environmental factors.

One other study, to the authors' knowledge, has examined longitudinal trends in the development of high IQ. Petrill et al (1998) used data from the Longitudinal Twin Study sample in infancy-14, 20, 24 and 36 months. Using a cut-off of the ninetieth percentile and conducting DeFries-Fulker extremes analysis, it was found that the heritability of high IQ at each age was not different from that of the unselected sample. However, the sample used was small and the age-to-age analyses, indicating significant shared genetic influence only between the 24 and 36 month time points, were not compared to those of the unselected sample. Additionally, the reliability of the measurement of early childhood IQ is lower than that at later time points, meaning that a systematic effect is less likely to be found in infancy than in later childhood/adolescence.

One issue that arises from previous studies using truncated samples is the criterion used for selection. Previous research presented here use various approaches, but the criterion chosen for our analyses was to select as being of high IQ those twins in our community sample that scored above the 85th percentile in IQ. This criterion was applied on an age-by-age basis, so different individuals can fulfill the criterion at each age. Using this value as a threshold enabled a good balance between selection on the trait of interest and keeping the sample large enough to maintain statistical power. It is also in accordance with the selection criteria

used in the meta-analysis presented in this special journal issue, allowing the reader greater scope to compare results across studies.

Methods and Materials

Sample and Procedure

Twins participating in the Longitudinal Twin Study (LTS; Rhea et al, 2006) served as our sample for the current analyses. The LTS is an ongoing, prospective study of behavioral development conducted at the Institute for Behavioral Genetics (IBG; University of Colorado, Boulder). A total of 483 families participated and included 966 individual twins (240 male-male twin pairs and 243 female-female twin pairs). Those twins who were of the same sex and lived within 300 kilometers of the University were recruited by IBG between 1985 and 1991. At the time of enrollment into LTS, the average age of mothers and fathers was 29.65 and 31.65 years of age, respectively. Over 95% of these parents had completed high school, 50% of whom subsequently completed two or more years of college (Rhea et al, 2006). Self-reported ethnicity of the families participating in LTS was primarily Caucasian (>95%), with the remaining 5% of the sample consisting of African-American, Hispanic-American, and/or Native American. Twin zygosity status was determined using observer ratings and, subsequently, 12 molecular genetic markers as described elsewhere (Haberstick et al, 2004). The analysis presented here uses IQ information collected from the twins at seven time points from age about 1 year to age 16 years.

We obtained written consent from parents prior to their children's participation in the LTS, and/or parental or individual assent/consent (as appropriate) at each testing session. The Human Research Committee of the University of Colorado at Boulder approved the study protocols.

Measures

IQ scores at ages 1 and 2 were calculated using the mental development component of the Bayley Scales of Infant Development (BSID; Bayley, 1969). The Stanford-Binet Intelligence Scale (Terman & Merrill, 1973) was administered at ages 3 and 4. These tests were administered in the twins' homes by separate examiners. Intelligence at age 7 was measured in person using the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974), at age 12 also by the WISC-III (Wechsler, 1991) and at age 16 using the Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997).

Analyses

Means, standard deviations, and ANOVA results as a function of zygosity and gender and their interaction were calculated using SPSS (Version 16.2, SPSS, 2005) using one randomly selected member of each twin pair. Estimates of the skewness and kurtosis were calculated to determine whether IQ scores at each age were normally distributed. Prior to model-fitting analyses, the IQ scores were standardized within age and across zygosity and sex. Four individuals (2 pairs of twins) were excluded from the analysis and the subsequent genetic modeling due to unknown zygosity.

Genetic modeling

Univariate genetic modeling

Genetic contributions included the summed influences of many genes acting additively (A) and non-additively (referred to as 'dominance', D) in their effects. Environmental effects included those sources that are shared by both siblings of a pair and serve to make them more similar (C) and non-shared unique environmental effects (E) that include influences of

child-specific experiences with their environment and measurement error. As MZ twins share 100% of their genes, while DZ twins share on average 50% of their genes identical by descent, quantitative genetic analyses of twin data assumes that: (1) MZ twins correlate perfectly for all A contributions to attention problems while DZ twins correlate 0.50, (2) an MZ twin correlation of 1.0 and a DZ twin correlation of 0.25 for D effects, (3) and that siblings experience the effects of shared environments equally regardless of zygosity status and are therefore correlated 1.0.

The magnitude of latent genetic and environmental influences on observed variation can be inferred from the extent MZ and DZ twin pairs correlate. If only additive genetic effects were involved in making a pair of twins similar, it is expected that the DZ twin correlation would be one-half that of the MZ twin correlation. Shared environmental influences are implicated when the correlation between DZ twins is more than one-half that between MZ twins. Conversely, when the correlation between DZ twins is less than one-half that between MZ twins, non-additive genetic effects are implicated. With MZ and DZ twin pairs reared in the same home, estimates of non-additive genetic and shared environmental contributions to observed variation are confounded and so only one, but not both, of these can be estimated in a given model (Jinks and Fulker, 1970).

Variation in IQ is assumed to be a function of two or more latent variables: A, E and either D or C. This baseline model is refined by equating the contribution of one or more latent variables to zero and testing the difference in model fit. In same-sex twin pairs, gender differences in the magnitude of genetic and environmental effects are tested by estimating the fit of a model that allows the latent influences for boys and girls to differ and comparing its fit with one that constrained them to be equal. However, as variances for male and female twins at each age were not significantly different, and the covariances were also found not to differ significantly across gender, these factors were not considered in the genetic analyses.

Univariate analyses were conducted the IQ data at each age. This analysis was additionally conducted on factor scores derived from a latent IQ factor constructed from the last three ages, using the program MPlus (Muthén & Muthén, 1998) to test the generalization of our results to individuals that scored consistently high on IQ. It was decided to use this measure, rather than an aggregate over the full range of ages, as the pattern of etiology changes substantially across early development, whereas the influences on IQ from age seven onwards are relatively stable.

Developmental genetic modeling

We adopted the repeated-measures design shown in Figure 1 to estimate the extent that genetic and environmental influences contributed to the correlation of IQ across multiple years (Eaves et al, 1986). This approach partitions the genetic and environmental variances at each age and the covariances across all ages into influences that can be common to all ages, age-specific, and transmitted forward into later ages. Common genetic effects are denoted as a_c , age-specific genetic effects as a_s , and age-to-age or transmitted genetic effects as j . Non-shared environmental influences are denoted as e_c , e_s , and k , respectively, with similar parameters for the shared environmental influences (not shown in the Figure.) Stability is conceptualized as resulting from two different processes. The first postulates that a common latent factor influences IQ at all ages through paths a_c and e_c . The second postulates that genetic and environmental influences on earlier ages persist or are transmitted forward to subsequent ages through paths j and k . Because of their similarity to well known psychometric models, we refer to the model for the first process as the common factor model and the second as the simplex model (Boomsma and Molenaar, 1987; Joreskog, 1970). From our developmental model we obtained the genetic and environmental correlations that indexed the extent earlier influences overlapped with later ones.

Genetic models were fit to standardized age-scaled scores, using the raw maximum-likelihood estimation option in Mx. The significance of model parameters was evaluated by a comparison of twice the log-likelihood ($-2LL$) for models with or without the parameters, with the difference distributed as a chi-square and the degrees of freedom being equal to the difference between the number of parameters estimated. A non-significant difference chi-square between the two models indicates that the parameters dropped from the more parsimonious model were not significantly different from zero. Classes of models were compared on the basis of the Akaike Information Criterion (AIC; Akaike, 1987), calculated by subtracting twice the difference in the degrees of freedom ($2\Delta df$) from the difference chi-square ($\Delta\chi^2$) between any particular model and the fullest, i.e. least parsimonious model, considered. The AIC indexes the extent that a given model offers the most parsimonious, but adequate, explanation of the data.

Threshold models

The model-fitting analyses for the top 15 percent of the distribution of IQ scores proceeded in largely the same fashion as those for the full distribution. A threshold of 1.036, the z-score corresponding to the 85th percentile of a normal distribution was set, and the data file on which this analysis was made was ordinal, with '1's representing a twin whose score passed this threshold, and a '0' for a twin that did not. This criterion was applied to each age separately, so an individual could reach the threshold at one age, but not at others. The aggregate univariate analysis differed in this respect, as the individuals that were above the eighty-fifth percentile on average across the last three ages were labeled high IQ. Differences between the best-fitting models for the full IQ range and the high IQ range was tested for by comparing the fit of a freely estimated threshold model to one in which the parameters were fixed to those derived from the continuous analysis. For the developmental model we compared the best-fitting model from the threshold analysis in which the parameters were freely estimated to the same model in which the parameter values were fixed to those from the continuous analysis. This analysis assumes a similar structure of etiological influences on the development of high IQ to the rest of the distribution, while testing whether the relative importance of etiological factors is different for highly intelligent individuals.

Results

Table 1 shows the mean IQs and the standard deviation around them for each age in the full sample. The mean age, standard deviation and range is also shown. The ANOVA results using data from one randomly picked twin from each pair demonstrates no significant effect of zygosity on IQ score at any age. There are sex differences in the mean IQ observed at four ages, but the effect sizes are very small, and there are no *a priori* reasons to expect such an effect. No interactions of zygosity by sex effects are observed. Skewness and kurtosis at each age are within acceptable limits to assume a normal distribution of IQ scores at each age.

Cross-twin, cross-age correlations

As can be seen in Table 2, the cross-twin correlations at all ages are higher for monozygotic than for dizygotic twins, considering the entire distribution, indicating some genetic influence at each age. The cross-age twin correlations are larger for closer ages than for those that are further away in time, suggesting at least some influence of age-to-age transmission effects. The approximate heritability estimates from simply doubling the difference between the MZ and DZ correlations range from a low of .38 at age 1 to a high of .74 at age 12.

Univariate continuous model-fitting analyses

Table 3 presents the continuous univariate analyses for the full sample distribution of IQ scores. The fit of the ACE models was compared to a saturated phenotypic model. As can be seen, the amount of variance in the sample attributable to additive genetic variance (A) increases from .3-.4 at earlier ages to just above .7 at later ages. The pattern is reversed for shared environmental (C) variance, reducing from its highest value of .46 at age 2 to just above .1 at ages 12 and 16. Non-shared environmental variance (E) does decrease somewhat over age, but not to the same extent. It should be noted that these E estimates include measurement error. As different tests were used at different ages, it is possible that the reliabilities varied, which would change the proportion of error. Examining the chi-square difference tests ($\Delta\chi^2$) and the Akaike Information Criteria (AIC) for the sub-models at each age, it appears that the full ACE model is the best-fitting at all ages, as the AIC estimate never reaches below -1, even when the $\Delta\chi^2$ indicates that the drop in fit is non-significant. It could be argued, however, that an AE model fits the best at ages 12 and 16 and the aggregate measure of the last three ages.

Developmental continuous model-fitting analyses

Table 4a presents the model fit statistics for the developmental model shown in Figure 1 when applied to the full sample. As can be seen, a model that includes C and E common factors, an A age-to-age transmission factor, and A, C and E innovations at each age, gives very little reduction in fit, while reducing complexity substantially, when compared to the full model. The parameter estimates yielded by the full model are presented in Figure 2, and the best more parsimonious model by AIC, with parameter estimates, is presented in Figure 3.

Extensive age-to-age additive genetic transmission effects can be seen at all ages. In fact, transmission is so high from ages 7–12 and 12–16, that the simplex model is indistinguishable from a common factor. However, we included just a simplex model rather than a common factor at later ages for parsimony. There are also moderate to high levels of specific genetic influences at each age, even the highest ages, suggesting that the etiology of individual differences in cognitive development isn't completely stable even at 16 years of age. Loadings on the common environmental factor are steady and moderately high at all ages, and innovation shared environmental effects are low, suggesting that the same aspects of the family environment are important at all ages. One exception to this is the higher C innovation at age 1. The common non-shared environmental factor in the model contributes only modestly to developmental stability and primarily after age 7. This is consistent with real individual environmental influences on IQ for school age children that could include biological factors, such as poor health, or psychological or educational influences. We should remember that circumstances that have large effects for individual children, but that occur relatively infrequently among the population, will contribute only modestly to overall variance. Age-specific E effects are at a consistent level over time. One conclusion that can be drawn from this model and from the univariate analyses is that parameter estimates from ages 7 onwards are remarkably stable. This suggests that the importance of different kinds of causal influences on intelligence does not change much once children have attained school age.

Table 4b presents the total variance components for additive genetic, shared environment and non-shared environmental factors. These largely reflect the patterns observed in the univariate analyses of increasing genetic and decreasing environmental effects across development. It is reassuring that these estimates replicate those of the univariate analyses, and this lends credence to the results of the developmental model-fitting. Table 4b also presents age-to-age genetic and non-shared environment correlations. The shared

environmental correlation approaches 1 at each age after age one because of the common factor and the low level of age-specific innovations after this time point. The genetic correlations demonstrate the increasing similarity of genetic influences over time that was seen in the larger transmission effects for ages 7–16. The correlations are, however, high across age groups, signifying that the same genetic effects are evident from infancy to late adolescence, but that these influences increase in importance across development.

Univariate High IQ model-fitting analyses

The threshold was set at 1.036 - the z-score corresponding to the 85th percentile of the normal distribution. The full-model results were variable, demonstrating the large drop in power from dichotomizing the data at this relatively high threshold and the resulting difficulty in estimating all parameters. Comparison of the freely estimated to the fixed-parameter models did not demonstrate a decrement of fit for any of the models from fixing the A C and E parameters to those from the continuous models. Chi-square values were small and non-significant, ranging from 0.245 to 1.973 for 3 degrees of freedom. Due to the power reduction, the results from these analyses are arguably inconclusive, but are suggestive of the presence of similar proportions of genetic and environmental influences at the top 15th percentile of the distribution and those found in the full range.

Developmental high IQ model-fitting analyses

Fitting the full model with all parameters freely estimated gave a $-2LL$ of 3785.70 for $df=5571$. The extra seven degrees of freedom for this model compared to the same model estimated for the continuous data (Model 1 in Table 4) correspond to the constraining of the phenotypic variance to one at each age. The fit of the best continuous developmental model (Model 10 in Table 4: A transmission, C and E common factor and ACE innovations) to the high IQ, in which the parameters were freely estimated, yielded a -2 log-likelihood ($-2LL$) value of 3798.90 with 5590 degrees of freedom (df). The difference chi-square was 23.35, $p = .222$ and $AIC = -14.65$. Fixing all the parameter values to those estimated for the continuous data gives a $-2LL$ value of 3818.21 with 5631 df . The comparison of these fit measures gives a $\Delta\chi^2(41)=19.31$, $p=0.998$, and $AIC = -87.49$. This means that, assuming the same structure of etiological influences for high IQ and normally distributed IQ, there is no significant difference between the parameters estimated for the continuous distribution and those estimated dichotomizing the distribution for high IQ..

Discussion

Our model-fitting analyses demonstrate developmental trends across the full spectrum of ability that largely accord with those found previously (Bishop *et al*, 2003; Davis *et al*, 2008; Petrill *et al*, 2004; Spinath *et al*, 2003). Specifically, the common shared environment factor was found to contribute to age-to-age continuity, although its influence on variance in intelligence was very small from age twelve onwards. Genetic factors were also found to largely contribute to continuity, but the higher correlations for assessments closer together in time mean that the simplex transmission model fits the additive genetic factors best. Non-shared environmental factors contribute mainly to changes across development, although there is some age-to-age correlation of non-shared environmental effects, particularly at later time points. The great similarity of the pattern of contributing factors from between ages twelve and sixteen, and even from age seven, suggests that the etiology of individual differences in intelligence development is highly stable by early adolescence. Genetic influences increase in importance throughout development, and shared environmental influences decrease in importance.

The analysis additionally suggests that there is not only no difference between the proportion of variance attributable to genetic and environmental influence at each age for high ability individuals and the rest of the distribution, but also that the pattern of transmission, common and age specific factors from age-to-age is e similar for individuals scoring above the 85th percentile at each age and that of the full normal distribution. This result is consistent with the hypothesis that the etiological influences contributing to the development of high intelligence are the same as those contributing to individual differences in the full range, but a conclusive demonstration of this were not possible in this study.

Validity of Results

The continuous developmental results relating to the full distribution can be fully accepted as the power with this sample is sufficiently high and the confidence intervals are correspondingly narrow. However, power decreases with the use of threshold models, up to 10 times with a 10 percent threshold (Neale, Eaves & Kendler, 1994). This power increases with the use of multiple measures over time (Schmitz, Cherny & Fulker, 1998), but remains markedly reduced in comparison to the continuous analysis. It remains possible, therefore, that some different correlational patterns for high IQ were missed in the threshold models.

The sample used in this study gives it strength as it is a longitudinal sample with a range of assessment periods fully representative of the course of intelligence development. The analysis is also novel in the field as it allows modeling of developmental trends in high IQ and direct comparison of the fit of this model to that found for the full distribution of ability.

As Petrill *et al* (1998) used the early data from the LTS, it is reassuring that a similar result was found using a different methodology and a less stringent threshold (85th as opposed to 90th percentile) for high IQ selection. This holds for both the univariate and the cross-age analyses. As mentioned earlier, Petrill *et al* (1998) is the only previous longitudinal and etiologically informative study of high IQ. Others have presented cross-sectional analyses, and the current study supports those that find no difference in the relative importance of genetic, shared environmental and unique environmental influences (Cheney, *et al*, 1992; Horn *et al*, 1982; Ronald *et al*, 2002; Sundet *et al*, 1994; Vogler & DeFries, 1983). It also largely supports the specific values found, in an age-specific fashion, with the exception of the considerably higher shared environment effects found for preschool cognitive ability by Ronald *et al* (2002). These results come from a variety of twin and family studies, indicating that the finding is representative of the population, at least in the United States.

Detterman *et al* (1990) and Bailey and Revelle (1991) found increased heritability in the lower range of the ability distribution. The current investigation did not address this issue. One study that found higher heritability for higher ability levels was that of Jensen (1987). The method used in that study, of correlating absolute difference scores with total twin scores on IQ tests, and comparing this correlation in MZ and DZ twins is quite different to that used here and could potentially pick up more subtle ability-related differences- especially if these differences are gradual and cumulative and IQ gets higher. However, one would still expect that that this cumulative effect would still be seen when a comparison was made between an upper cut-off and the general population.

The descriptive pattern of a greater verbal-performance discrepancy in ‘gifted’ children observed by Wilkinson (1993) and Sweetland *et al* (2006) was not addressed in our analyses. However, if correct, it does not appear to result in differential causal factors in either kind or proportion. It could be explained by less reliable measurement at the higher level of ability, or environmental, rather than heritable factors could account for the differences between performance and verbal IQ. This would accord with Cattell’s (1971) idea of Fluid and Crystallized intelligence. Under this hypothesis, verbal subtests in the

Wechsler intelligence tests tap the crystallized aspect and performance subtests tap the fluid aspect of intelligence. It is hypothesized that crystallized intelligence is more amenable to the influences of the environment, and so the same aspects of the environment could account for individual differences along this dimension. This could also be the aspect of intelligence that is acted upon by gene-environment correlation, allowing a greater performance discrepancy between these factors at higher ability levels. This hypothesis is supported by the fact that the average verbal intelligence score is somewhat higher than the average performance intelligence score in the Wilkinson (1993) and Sweetland *et al* (2006) samples.

Implications

The results of this study have implications for gene-finding efforts in the area of intelligence. As it appears that the magnitude and possibly nature, of genetic influences on intelligence are the same for higher and average ability levels, linkage and association studies do not have to concentrate on a specific ability level when looking for genes associated with intelligence. It is likely that higher IQs result from the cumulative impact of many genes each individually increasing IQ by some small amount. If so, molecular genetics findings from high IQ individuals will be applicable to the rest of the population. As using extreme samples gives increased power in the search for specific genetic influences, this is an important finding and is indicative of the utility of high IQ individuals in gene finding efforts. Of course, the advantage of such an approach must be counter-balanced by the difficulty in ascertaining extreme samples and the resulting lack of power mentioned above.

There are also implications of the results of this study that relate to the environment. Our results suggest that it is unlikely that there are dramatic environmental influences that only highly intelligent children experience, and more likely that development of high cognitive ability comes from quantitatively better engagement with or access to, available educational materials. It is also clear that the relative impact of shared environmental influences is reduced after the age of seven, although several factors could contribute to this pattern.

Limitations and Conclusions

The selection criteria used in the current study allowed a different subset of individuals to be included in the high IQ subsample at each age. This approach has limitations relating to sample uniformity across ages. Just fifteen percent of the sample who were above threshold at one year of age was above threshold at age sixteen, and a mere three participants met criteria at all ages. The pattern is more stable for remaining above threshold from age seven to age sixteen; half of the participants above threshold at age seven maintained that status at age sixteen. This pattern reflects the strong similarity of influences on developmental from age seven onwards. There is some consistency from even the earliest ages, however. Those of greater than average intelligence at age one are 1.74 times more likely to meet threshold criteria at age sixteen.

The 85th percentile was chosen to maintain power to detect possible differential causal influences at the higher level of ability. However, it could be argued that this is an insufficiently high selection criterion for high IQ, masking possible ability-related etiological effects. However, for a high cut-off to be viable, a much larger sample would be required to retain adequate power. Also, as mentioned earlier, our results accord with those using samples using more stringent selection, as well as those using fully continuous methods. It would, however, be interesting to see if our results replicate in a larger sample with more extreme selection..

Overall, these analyses demonstrate a dynamic pattern of genetic and environmental influences across development that confirms and extends that of previous studies.

Additionally, our results are equally applicable to individuals with high IQ and those in the normal range. This conclusion has implications for gene-finding and for the search for specific environmental influences on the development of intelligence.

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Figure 1.

Developmental model. Ac, Common genetic influences; As, Age-specific genetic influences; J, Age-to-age genetic influences; E, environmental influences; Ec, Common environmental influences; Es, Age-specific environmental influences; K, Age-to-age environmental influences; IQ, IQ score at each age.

Figure 2.

Standardized parameter estimates for baseline developmental model of IQ scores at seven ages during childhood and adolescence. A, additive genetic effects; A_C , common additive genetic effects; C, shared environmental effects; C_C , common shared environmental effects; E, non-shared or individual specific environmental effects; E_C , common non-shared environmental effects. Parameter estimates whose 95% confidence intervals did not include zero are **bolded**.

Figure 3.

Best-fitting developmental model of IQ scores at seven ages during childhood and adolescence. A, additive genetic effects; C, shared environmental effects; C_C, common shared environmental effects; E, non-shared or individual specific environmental effects; E_C, common non-shared environmental effects. Parameter estimates whose 95% confidence intervals did not include zero are **bolded**.

Table 1

Means (standard deviations) for age and IQ at each age, ANOVA results for IQ by age and zygosity and skewness and kurtosis results for one randomly selected member of each twin pair

Mean Age in years (SD) and Range	N		Male (N)	Female (N)	Mean IQ (SD)	Skewness (SE)	Kurtosis (SE)	ANOVA	
	MZ	DZ						Zygosity	Sex
One Year									
1.17 (0.07)	494	393	445	438	104.56 (13.80)	-0.31 (0.08)	0.92 (0.16)	p= 0.5 $\eta^2= 0.002$	p= 0.04 $\eta^2< 0.001$
1.00-1.33									
Two Years									
2.04 (0.06)	433	366	403	392	107.44 (18.82)	0.07 (0.09)	-0.38 (0.17)	p= 0.4 $\eta^2= 0.003$	p< 0.01 $\eta^2= 0.097$
1.92-2.25									
Three Years									
3.04 (0.07)	410	352	370	390	103.22 (17.50)	-0.06 (0.09)	0.08 (0.18)	p= 0.3 $\eta^2= 0.005$	p< 0.01 $\eta^2= 0.076$
2.83-3.5									
Four Years									
4.01 (0.04)	405	351	387	367	103.77 (14.12)	0.12 (0.09)	0.55 (0.18)	p= 0.5 $\eta^2= 0.002$	p<0.01 $\eta^2= 0.028$
3.92-4.08									
Seven Years									
7.39 (0.36)	444	376	412	408	106.33 (13.40)	-0.24 (0.09)	0.57 (0.17)	p= 0.16 $\eta^2= 0.008$	p= 0.76 $\eta^2< 0.001$
6.67-8.42									
Twelve Years									
12.43 (0.37)	390	364	370	382	103.21 (12.86)	-0.01 (0.09)	-0.39 (0.18)	P= 0.78 $\eta^2< 0.001$	p= 0.74 $\eta^2< 0.001$
11.33-14.00									
Sixteen Years									
16.55 (0.76)	427	373	384	414	102.20 (11.47)	0.21 (0.09)	0.22 (0.17)	p= 0.38 $\eta^2= 0.003$	p= 0.59 $\eta^2= 0.001$
16.00-20.00									

Table 2

Within-pair and Cross-age correlations for Monozygotic and Dizygotic twins. [†]

Twin 1	Twin 2							
	Age 1	Age 2	Age 3	Age 4	Age 7	Age 12	Age 16	
MZ								
Age 1	.58	.39	.22	.24	.29	.18	.17	
Age 2		.82	.66	.59	.52	.38	.38	
Age 3			.70	.69	.48	.51	.34	
Age 4				.76	.60	.53	.47	
Age 7					.81	.71	.69	
Age 12						.85	.80	
Age 16							.84	
DZ								
Age 1	.39	.27	.15	.12	.01	-.02	.01	
Age 2		.63	.44	.48	.41	.31	.25	
Age 3			.51	.45	.41	.34	.31	
Age 4				.50	.39	.33	.30	
Age 7					.59	.50	.45	
Age 12						.48	.40	
Age 16							.51	

[†] All correlations are significant at p < 0.05.

Table 3

Results of Univariate Model Fitting of IQ Scores at Seven Ages.

Model	Additive Genetic Variance	Shared Environmental Variance	Non-Shared Environmental Variance	$\Delta\chi^2$	Δdf	P	AIC
AGE 1							
ACE †	0.40 (0.14 to 0.64)	0.18 (0.00 to 0.41)	0.42 (0.35 to 0.50)	3.86	3	0.28	-2.14
AE	0.59 (0.52 to 0.66)	...	0.41 (0.34 to 0.48)	2.11	1	.146	0.11
CE	...	0.49 (0.42 to 0.56)	0.51 (0.44 to 0.58)	9.22	1	.002	7.22
AGE 2							
ACE †	0.36 (0.21 to 0.54)	0.46 (0.28 to 0.60)	0.19 (0.15 to 0.23)	4.89	3	0.18	-1.11
AE	0.82 (0.78 to 0.85)	...	0.18 (0.15 to 0.22)	18.7	1	.000	16.7
CE	...	0.73 (0.69 to 0.78)	0.27 (0.22 to 0.31)	23.1	1	.000	21.1
AGE 3							
ACE †	0.30 (0.08 to 0.54)	0.39 (0.16 to 0.57)	0.32 (0.26 to 0.39)	3.73	3	0.29	-2.27
AE	0.70 (0.63 to 0.75)	...	0.30 (0.25 to 0.37)	9.69	1	.002	7.69
CE	...	0.62 (0.55 to 0.68)	0.38 (0.32 to 0.45)	7.34	1	.007	5.35
AGE 4							
ACE †	0.51 (0.30 to 0.75)	0.26 (0.03 to 0.45)	0.23 (0.19 to 0.29)	1.03	3	0.79	-4.97
AE	0.77 (0.72 to 0.81)	...	0.23 (0.19 to 0.28)	4.20	1	0.03	2.70
CE	...	0.65 (0.59 to 0.70)	0.35 (0.30 to 0.41)	25.9	1	.000	23.9
AGE 7							
ACE †	0.48 (0.32 to 0.68)	0.34 (0.15 to 0.49)	0.18 (0.15 to 0.22)	0.84	3	0.84	-5.16
AE	0.82 (0.78 to 0.85)	...	0.18 (0.15 to 0.22)	10.6	1	.001	8.56
CE	...	0.71 (0.65 to 0.75)	0.29 (0.25 to 0.35)	37.7	1	.000	35.7
AGE 12							
ACE	0.73 (0.53 to 0.87)	0.12 (0.00 to 0.31)	0.15 (0.12 to 0.19)	3.56	3	0.31	-2.44
AE	0.85 (0.81 to 0.88)	...	0.15 (0.12 to 0.19)	1.09	1	0.30	-0.91
CE	...	0.67 (0.61 to 0.72)	0.33 (0.28 to 0.39)	68.8	1	.000	66.8
AGE 16							

Model	Additive Genetic Variance	Shared Environmental Variance	Non-Shared Environmental Variance	$\Delta\chi^2$	Δdf	P	AIC
ACE	0.71 (0.52 to 0.87)	0.14 (0.00 to 0.32)	0.15 (0.13 to 0.19)	4.20	3	0.24	-1.80
AE	0.85 (0.81 to 0.87)	...	0.15 (0.13 to 0.19)	1.63	1	0.20	-0.36
CE	...	0.67 (0.62 to 0.72)	0.33 (0.28 to 0.38)	71.1	1	.000	69.1
AGGREGATE 7,12 & 16							
ACE	0.63(0.43 to 0.84)	0.18 (0.00 to 0.37)	0.18 (0.15 to 0.22)	4.35	3	0.36	-3.65
AE	0.82 (0.78 to 0.85)	...	0.18 (0.15 to 0.22)	2.46	1	0.12	0.45
CE	...	0.68 (0.62 to 0.72)	0.32 (0.28 to 0.37)	47.5	1	.000	45.5

Table 4a

Model fit statistics for developmental models of IQ. [†]

Model	Model specification			Model fit statistics					
	Common Factor	Age-to-Age Transmission	Age-Specific	-2LL	Df	$\Delta\chi^2$	Δdf	p	AIC
1. [‡]	A,C,E	A,C,E	A,C,E	11758.30	5564	18.90	24	.76	-29.10
2.	A,E	A,C,E	A,C,E	11761.59	5571	3.29	7	.86	-10.71
3.	C,E	A,C,E	A,C,E	11763.41	5571	5.11	7	.65	-8.89
4.	A,C	A,C,E	A,C,E	11787.01	5571	28.72	7	.00	14.7
5.	--	A,C,E	A,C,E	11809.24	5585	50.94	21	.00	8.94
6.	A,C,E	A,E	A,C,E	11763.33	5570	5.03	6	.54	-6.97
7.	A,C,E	C,E	A,C,E	11783.01	5570	24.71	6	.00	12.7
8.	A,C,E	A,C	A,C,E	11763.33	5570	5.03	6	.54	-6.97
9	A,C,E,	--	A,C,E	11837.11	5582	78.81	18	.00	42.8
10.	C,E	A	A,C,E	11772.31	5583	14.01	19	.78	-23.9
11.	C	A	A,C,E	11758.30	5591	83.10	27	.00	29.1
12.	E	A	A,C,E	11811.66	5590	53.37	26	.00	1.37

Bolded indicates the best-fitting developmental model.

[†] Genetic analyses were performed using z-scored continuous IQ scores.

[‡] Baseline developmental model vs. Saturated Model (Genetic Cholesky)

Abbreviations: A, additive genetic effects; C, shared environmental effects; E, non-shared environmental effects; AIC, Akaike Information Criteria.

Variance Component Estimates and Correlations for the Best-Fitting Developmental Model of IQ at seven ages during childhood and adolescence.

Table 4b

Age	Variance Components [†]			Correlations [‡]											
	Additive Genetic Variance	Environmental Variance	Non-Shared Environmental Variance	1	2	3	4	7	12	16					
1	0.37 (0.12 to 0.61)	0.20 (0.01 to 0.42)	0.43 (0.35 to 0.51)	1.0	.73	.45	.39	.23	.21	.20					
2	0.35 (0.21 to 0.52)	0.46 (0.29 to 0.60)	0.19 (0.15 to 0.23)	.48	1.0	.62	.54	.32	.29	.27					
3	0.31 (0.16 to 0.47)	0.39 (0.24 to 0.54)	0.30 (0.25 to 0.36)	.49	.97	1.0	.88	.52	.47	.44					
4	0.43 (0.27 to 0.57)	0.33 (0.20 to 0.48)	0.24 (0.19 to 0.29)	.49	.97	.99	1.0	.59	.54	.51					
7	0.52 (0.37 to 0.66)	0.31 (0.17 to 0.45)	0.17 (0.14 to 0.20)	.47	.93	.96	.96	1.0	.91	.86					
12	0.64 (0.53 to 0.75)	0.22 (0.10 to 0.33)	0.14 (0.12 to 0.18)	.46	.91	.94	.94	.91	1.0	.94					
16	0.66 (0.53 to 0.76)	0.19 (0.09 to 0.32)	0.15 (0.12 to 0.19)	.48	.95	.98	.98	.94	.93	1.0					

* Parameter estimates are standardized.
[†] 95% Confidence intervals in parentheses.

[‡] Genetic correlations are above the leading diagonal, shared environmental correlations ranged between 0.01 and .10 for early ages with later ages, they are not shown. Increases in the non-shared environmental correlations were observed between ages 12 and 16, however, and ranged between 0.20 and .034.