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ORIGINAL RESEARCH

A Twin Study of the Genetics of High Cognitive Ability Selected from 11,000 Twin Pairs in Six Studies from Four Countries

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Abstract Although much genetic research has addressed normal variation in intelligence, little is known about the etiology of high cognitive abilities. Using data from 11,000 twin pairs (age range = 6–71 years) from the genetics of high cognitive abilities consortium, we investigated the genetic and environmental etiologies of high general cognitive ability (*g*). Age-appropriate psychometric cognitive tests were administered to the twins and used to create *g* scores standardized within each study. Liability-threshold model fitting was used to estimate genetic and environmental parameters for the top 15% of the distribution of *g*. Genetic influence for high *g* was substantial (0.50, with a 95% confidence interval of 0.41–0.60). Shared environmental influences were moderate (0.28, 0.19–0.37). We conclude that genetic variation contributes substantially to high *g* in Australia, the Netherlands, the United Kingdom and the United States.

Keywords Genetics · High cognitive ability · Twins · Intelligence · Talent

Introduction

A substantial body of genetic research using the classical twin design has demonstrated the important role of genetics as a risk factor in the development of cognitive disabilities (Plomin and Kovas 2005). In contrast, very little is known about the other end of the normal distribution—the genetic and environmental origins of high cognitive abilities—

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despite the societal importance of exceptional talent and the well-documented extraordinary creative potential of this group (Lubinski and Benbow 2006; Lubinski et al. 2006). It cannot be assumed that the etiology of high cognitive ability is the same as cognitive disability or the same as the normal distribution of cognitive ability. For example, an extreme version of epistasis called *emergenesis* has been suggested in which rare combinations of alleles are responsible for exceptional cognitive ability (Lykken 1982, 2006). Such a genetic model would predict high correlations for identical twins and relatively low correlations in first-degree relatives. On the other hand, if exceptional cognitive ability requires an especially favorable environment, we might expect to see greater environmental influence.

In 1869, Francis Galton raised the topic of the etiology of high ability in one of the first books in behavioral genetics, *Hereditary genius: An enquiry into its laws and consequences* (Galton 1869). His conclusion that "there is no escape from the conclusion that nature prevails enormously over nurture" (Galton 1883, p. 241) was not warranted because his research involved family studies which cannot unambiguously disentangle the effects of nature and nurture and he used reputation as an index of ability. In contrast, more than a century later, others have argued that "differences in early experiences, preferences, opportunities, habits, training, and practice are the real determinants of excellence" (Howe et al. 1998). However, these authors note that "relatively little is known about the genetic origins of high-level ability" (Howe et al. 1998, p. 403). In contrast, for general cognitive ability in the normal range the substantial heritability of *g* has been documented in dozens of family, twin and adoption studies (Bouchard and McGue 1981; Deary et al. 2006; Plomin and Spinath 2004).

Although much research on high ability considers athletic and artistic ability, our focus is on general cognitive ability (*g*), often referred to as *intelligence* (Jensen 1998). The normal range of variation in *g* is the target of more genetic research than any other behavioral trait other than self-reported personality (Bouchard and McGue 1981; Deary et al. 2006; Plomin and Spinath 2004), but very little is known about the genetics of high cognitive ability. Three reports of a few dozen twins of high ability in infancy (Petrill et al. 1998), childhood (Plomin and Thompson 1993), and in later life (Saudino et al. 1994), found substantial genetic influence and moderate shared environment (environmental effects that make members of the same family more similar) for high *g*, similar to what was found across the distribution of *g*. The only large twin study of high *g* was selected from a sample of 1,943 young twin pairs (2, 3 and 4 years) assessed by their parents (Ronald et al. 2002). In contrast to the previous two studies with small sample sizes, genetic influence was modest (0.20) and shared environment was substantial (0.70) for high ability as well as for the rest of the distribution, although these results may be due to the method of assessment. Other studies have investigated the etiology of individual differences within high-*g* groups, with mixed results (Thompson et al. 1993); however, such studies ask why high-ability individuals differ from each other in their *g* scores rather than asking why high-ability individuals as a group have so much higher *g* scores than the rest of the population.

In 2007, we formed the genetics of high cognitive abilities (GHCA) consortium with the goal of combining cognitive ability test scores from six twin studies in four countries in order to identify sufficient numbers of twins with high *g* scores to conduct adequately powered analyses of the genetic and environmental etiology of high *g*. Although these studies included different measures of cognitive ability, diverse cognitive tests can be used to create a *g* score that correlates highly with *g* scores derived from other tests (Johnson et al. 2008), which Charles Spearman (1927) referred to as *the indifference of the indicator*. Thus, we created *g* scores standardized within each study and also corrected scores for age within each study because the twins in the six studies varied in average age from 6 to 18 years (age range = 6–71 years). In another paper, we report results for analyses of individual differences in *g* for the combined sample of 11,000 twin pairs (Haworth et al. 2009). Heritability was estimated as 0.56 and shared environment accounted for 0.21 of the variance. Significant heterogeneity was found across the studies, but this heterogeneity is explained by the age differences among the samples. When the 11,000 twin pairs were sorted by three age groups, heritability increased significantly across age: 0.41 in childhood (average age of 9 years), 0.55 in adolescence (12 years), and 0.66 in young adulthood (17 years). Shared environmental influence declined significantly from childhood (0.33) to adolescence (0.18) but no further significant decline emerged in young adulthood (0.16).

In the present paper, we investigated the etiology of high *g*, defined as the top 15% of the distribution, and compared these results from categorical analyses of high *g* using liability-threshold models to the results of our previously reported continuous analyses of the full range of individual differences in *g*. As noted earlier, the dearth of data on the genetics of high *g* permits no strong hypotheses. Nonetheless, we predicted that heritability of high *g* is substantial and similar to heritability for the entire distribution of *g*, because that is generally what is seen at the low end of the *g* distribution (Plomin and Kovas 2005). Although we also explored whether heritability of high *g* increases with age as it does for the entire distribution of *g*,

our study of the top 15% of the distribution is underpowered to detect heritability differences when the sample is divided into subgroups such as age even with the large GHCA sample.

Methods

Samples and measures

Data on general cognitive ability were available from six twin studies from four different countries in the genetics of high cognitive abilities consortium. Three studies came from the United States: from Ohio, Colorado and Minnesota; and one each from Australia, the Netherlands and the United Kingdom. Individuals ranged from 6 to 71 years of age and the samples are organized here in order of the average age of the sample.

Ohio USA

The Western Reserve Reading Project (Petrill et al. 2007), a longitudinal twin study, provides data for 121 identical (monozygotic, MZ) pairs and 171 same-sex fraternal (dizygotic, DZ) pairs. Recruiting was conducted through school nominations, Ohio State birth records, and media advertisements. Schools were asked to send a packet of information to parents in their school system with twins who have been enrolled for kindergarten but have not finished first grade. Cooperation was secured from 273 schools throughout the state of Ohio. Media advertisements in the Greater Cleveland Metropolitan Area have also been used for the effective recruitment of additional twins. A social worker with long-standing ties to the community was also hired to assist in the recruitment of under-represented groups via face-to-face meetings with churches, community centres, and other service organizations. General cognitive ability was assessed using a short form of the Stanford-Binet Intelligence Scale (Thorndike et al. 1986), including vocabulary, pattern analysis, memory for sentences, memory for digits, and quantitative subtests. These subtests were summed and standardized for age and sex to form a general cognitive ability (g) score. Zygosity was assessed using DNA analysis via a buccal swab procedure. The average age of the sample was 6.07 years (range = 4.33–7.92).

United Kingdom

The Twins Early Development Study (TEDS) is a sample of twins born in the UK between 1994 and 1996 (Oliver and Plomin 2007). The TEDS sample has been shown to be reasonably representative of the general population in terms of parental education, ethnicity and employment status (Kovas et al. 2007). Zygosity was assessed through a parent questionnaire of physical similarity, which has been shown to be over 95% accurate when compared to DNA testing (Price et al. 2000). For cases where zygosity was unclear from this questionnaire, DNA testing was conducted. At age 12 the twins participated in web-based testing (Haworth et al. 2007). The twins were tested on two verbal tests, WISC-III-PI multiple choice information (general knowledge) and vocabulary multiple choice subtests (Wechsler 1992), and two non-verbal reasoning tests, the WISC-III-UK picture completion (Wechsler 1992) and Raven's standard and advanced progressive matrices (Raven et al. 1996, 1998). We created a g score with equal weights for the four tests by summing their standardized scores. Further information about g as measured in TEDS can be found elsewhere (Davis et al. 2009; Haworth et al. 2007). TEDS provides data for 1,518 MZ pairs and 2,500 DZ pairs (1,293 same-sex and 1,207 opposite-sex pairs). The average age of the sample was 11.57 years (range = 10.08–13.74).

Minnesota USA

The Minnesota Center for Twin and Family Research (Iacono et al. 2006) provides data for 1,177 MZ pairs and 679 same-sex DZ pairs. Twins were ascertained from Minnesota state birth records spanning the years 1972 through 1994 and recruited to participate in a broad-ranging longitudinal study of psychological development. At their intake into the study, twins were either age 11 or age 17. Twins with known mental retardation or a developmental disability that would have precluded their completing the intensive in-person MCTFR assessments as well as twins living more than a day's drive from the laboratories in Minneapolis were excluded from participation. Otherwise, the MCTFR sample is broadly representative of twin pairs born in Minnesota for the birth years sampled, with little evidence of participation bias in terms of parental education, socioeconomic status or mental health (Iacono et al. 1999).

The IQs used in the current study were determined from the twins' intake assessment, at which time they completed an abbreviated version of the Wechsler adult intelligence scale-revised (WAIS-R) if they were from the older cohort or the Wechsler intelligence scale for children-revised (WISC-R) if they were from the younger cohort. In both cases, the abbreviated Wechsler assessment consisted of two verbal subtests (information and vocabulary) and two performance subtests (block design and picture arrangement), selected because performance on these four subtests correlates greater than 0.90 with IQ determined by all Wechsler subtests. Performance on the four subtests was prorated and norms for the Wechsler tests used to compute IQs.

Zygosity was initially assessed using the consensus of four indicators: a standard zygosity questionnaire completed by the twins' parents prior to the intake assessment; a diagnosis of zygosity based on trained project staff perception of physical similarity at the time of intake assessment; and an algorithm based on ponderal index, cephalic index, and fingerprint ridge count. If there was any discrepancy among these three methods, zygosity was determined by evaluating 12 blood group antigens from blood samples. In an analysis of 50 twin pairs where the questionnaire, project staff assessment and physical similarity algorithm all agreed, the resulting zygosity determination was always confirmed in the serological analyses. The average age of the sample was 13.00 years (range = 11.00–17.00).

Colorado USA

The data are provided by the Institute for Behavior Genetics (IBG) from 390 twin pairs participating in the Colorado Longitudinal Twin Study (LTS), 696 pairs from the Colorado Twin Study (CTS), and 1,779 pairs from the Colorado Learning Disabilities Research Center (CLDRC). The LTS and CTS are maintained in a single database with no overlap in subjects. The CLDRC subjects were independently ascertained and could include overlapping subjects. For the purposes of this analysis, a search was made for all doubly ascertained families and all known duplicates have been removed from the original LTS and CTS samples; all data for these analyses are for unique individuals with one test per individual. The study samples are 90% white, with approximately equal representation of males (49%) and females (51%).

The LTS sample was collected from 1984 with repeated testing from about 1 year of age through, currently, their early twenties. Ascertainment was through a search of birth records made available by the Colorado Department of Health. A total of 483 pairs have participated at some time in the study, with 412 currently active. IQ testing at approximately age 16 used the WAIS-III (Wechsler 1997). The data from this test was used if available. If not, the next latest test was used: WISC-III (Wechsler 1991) at age 12 or WISC-R at age 7 (Wechsler 1974). Thus age of testing ranged from 6 to 19 years, with a mean age of 15.4 years. Zygosity was determined initially using a modified version of the Nichols and Bilbro (1966) questionnaire. Subsequently these assignments were confirmed using 11 highly polymorphic short tandem repeat markers (the IBG zygosity panel) in 92% of the sample for whom DNA has been collected. Further details of the ascertainment and history of the study are provided elsewhere (Rhea et al. 2006).

The CTS sample was recruited as adolescents through a combination of historical birth records and the use of school records. About 170 of 176 school districts participated at some level. IQ testing used the vocabulary and block design subtests of the age-appropriate WISC-III or WAIS-III. Age of testing ranged from 12 to 25 years, with a mean age of 17.1 years. In almost all cases, zygosity is determined by genotyping the IBG zygosity panel. Further details of the ascertainment and history of the study are provided in Rhea et al. (2006). To estimate full scale IQ scores from the two subtests administered, a regression equation of full scale IQ on the subtests was computed in the LTS sample and applied to the CTS sample.

The CLDRC sample participated in either the Colorado Reading Project (DeFries 1985; DeFries et al. 1991) or the Colorado Learning Disabilities Research Center (DeFries et al. 1997). Twin pairs were ascertained through 27 cooperating school districts in the state of Colorado. Twin pairs included those in which at least one member had a school history of reading problems and twin pairs in which neither member had a school history of reading problems. Although this means that the sample is not strictly unselected, the IQ distribution shows no signs of departure from normality, with mean = 105.6, SD = 13.2, skewness = 0.00, kurtosis = 0.11. IQ tests used either the WISC-R or the WAIS-R. The twins were reared in primarily English-speaking, middle-class homes, and were between 8 and 20 years of age at the time of testing, with a mean age of 11 years. The average age of the combined Colorado sample was 13.12 years (range = 6.00–25.00).

Australia

The Twin Cognition Study (Luciano et al. 2003b) provides data for 338 MZ pairs and 513 DZ pairs (265 same-sex and 248 opposite-sex pairs), recruited through primary and secondary schools in the greater Brisbane area (Wright and Martin 2004). Zygosity for dizygotic same-sex twin pairs was established by typing nine independent DNA microsatellite markers (AmpF1STR Profiler Plus Amplification kit; Applied Biosystems, Foster City, CA; polymorphism information content > 0.7), and cross-checked with blood group results (ABO, MNS, and Rh blood typing provided by Australian Red Cross Blood Service, Brisbane) and phenotypic data (hair, skin, eye color). The overall probability of correct zygosity assignment was greater than 99.9% (Nyholt 2006). Parental report indicated no significant head injury, neurological or psychiatric conditions, history of substance abuse/dependence, or taking of medications with significant central nervous system effects. Informed written consent was obtained from the twins and their parent or guardian, and ethical approval was obtained from the Human Research Ethics Committee, Queensland Institute of Medical Research. Twins were tested as close as possible to their 16th birthday on three verbal (information, arithmetic, vocabulary)

and two performance (spatial and object assembly) subtests from the multidimensional aptitude battery (MAB) (Jackson 1998), in addition to other measures of cognitive ability. The MAB is a computerized test, based on the WAIS-R (Wechsler 1981), that generates scores for full-scale IQ based on Canadian normative data. For a full description of the test battery as measured in the Twin Cognition Study see Luciano et al. (2003a). The average age of the sample was 16.00 years (range = 15.00–22.00).

The Netherlands

The Netherlands Twin Register (Boomsma et al. 2006) provides data for 434 MZ pairs and 517 DZ pairs (337 same-sex and 180 opposite-sex pairs). IQ data were available in twins who had taken part in studies on cognition at ages 6, 12 and 18 years (Boomsma et al. 2008) or as adults. At age 6, twins were tested as part of studies on the development of cognition executive function and neuropsychological development (Polderman et al. 2006); IQ was assessed using the Revised Amsterdamse Kinder Intelligentie Test (RAKIT) (Bleichrodt et al. 1984). IQ data at age 12 were collected in twins who took part in developmental studies of cognition and brain development (Bartels et al. 2002); IQ was assessed using the Dutch version of the WISC-R. At age 18 the twins took part in studies of brain development and cognition (Rijsdijk et al. 2002); IQ was assessed using Raven's standard progressive matrices and the Dutch version of the WAIS. The adult twins had also taken part in a study of brain function and IQ (Posthuma et al. 2002), where IQ was assessed using the Dutch WAIS. The large majority of same-sex twins' zygosity was based on typing of DNA or blood group polymorphisms. For the other pairs, zygosity was based on a series of physical similarity questions, answered by the mother of twins repeatedly over time (Rietveld et al. 2000). IQ testing was carried out with standard, age-appropriate IQ tests (see Boomsma et al. 2008; Posthuma et al. 2002). The average age of the sample was 17.99 years (range = 5.67–71.03).

Data preparation and preliminary analyses

All measures were standardized to a mean of zero and a standard deviation of one separately for each sample. ANOVA was used to assess differences in means by sex and zygosity. All measures were residualized for age and sex effects using a regression procedure. Standardized residuals were used because the age and sex of twins is perfectly correlated across pairs, and variation within age at the time of testing and variation within sex could contribute to the correlation between twins, and thus be misrepresented as environmental influences shared by the twins (McGue and Bouchard 1984). Four of the samples (Australia, US Colorado, UK and the Netherlands) included both same-sex pairs as well as opposite-sex DZ twin pairs. We therefore performed preliminary analyses based on sex-limitation models to investigate possible quantitative and qualitative sex differences in etiology. These analyses indicated no significant qualitative differences and therefore we report results here from analyses including opposite-sex as well as same-sex twins. There was a significant quantitative sex difference only in the UK sample, but the difference was small, and the UK sample had the greatest power to detect significant differences. In order to create the largest possible sample to power the analyses of high *g* we combined data from males and females. In a previous paper, we reported twin intraclass correlations for each sample and standard univariate twin model-fitting analyses using raw data (Haworth et al. 2009).

Categorical analyses of high cognitive ability

The focus of this paper is on the high extreme of the distribution of general cognitive ability. For these analyses, we classified high performance as scores above the 85th percentile (in each study). This cut-off provided a balance between extreme scores and power. These categorical data from each sample were used in Mx (Neale et al. 2006) to calculate tetrachoric twin correlations and thresholds, and to perform standard univariate liability-threshold modeling (Falconer 1965; Smith 1974). The standard liability-threshold model uses categorical data—in this case, meeting the criteria for high performance—and concordance rates to assess the relative contributions of genetic and environmental influences to an assumed underlying continuum (Falconer 1965; Smith 1974). Liability-threshold modeling is the categorical equivalent of continuous twin model-fitting analyses, and it allows the estimation of genetic, shared-environmental and non-shared environmental influences on a trait defined categorically.

Data from all six samples were then included in a heterogeneity analysis in Mx to assess whether estimates from the different samples could be equated, and to provide genetic, shared and non-shared environmental estimates from the combined sample.

Because our previous analyses of individual differences for the entire GHCA sample found significant increases in heritability from childhood to adolescence to young adulthood (Haworth et al. 2009), we also tested for heterogeneity for high *g* when the high-*g* twins in the GHCA consortium were sorted not into six studies but into three age groups: childhood (average age 9 years), adolescence (12 years) and young adulthood (17 years).

Results

The means and standard deviations (SD) for *g* in all six samples are shown in Table 1. Results from the ANOVA indicate significant effects of sex on four of the six samples, with males scoring higher than females, although effect sizes of this effect are small. There were significant effects of zygosity in two of the six samples, but no significant interactions between sex and zygosity. All samples were normally distributed for *g*.

From each sample, twins were selected using an 85% cut-off as an index of high *g*. Descriptive statistics of the high *g* probands (total $N = 3{,}300$) and of individuals from the remaining distribution (total $N = 18{,}796$) are shown in Table 2. On average, probands score 1.5 standard deviations above the mean as expected for the top 15% of a normal distribution.

Tetrachoric correlations

As shown in Table 3, tetrachoric twin correlations calculated in Mx were higher in MZ then DZ twins, indicating genetic influence. A similar pattern of MZ and DZ correlations was found across the six samples suggesting additive genetic influence, no non-additive genetic influence, and shared environmental influence. Calculating ACE (A = additive genetic; C = shared (common) environment; E = non-shared (unique) environment) estimates from the average MZ (0.79) and DZ (0.58) correlations suggests moderate heritability (0.42) and shared environmental (0.37) influence. Thresholds for twin 1 and twin 2, within zygosity, could be equated in all of the samples. For all six samples it was also possible to equate the MZ and DZ thresholds.

Liability-threshold model-fitting analyses

Univariate liability-threshold model fitting was conducted separately for each sample. Table 4 lists results from these models, which include comparative fit statistics of reduced models (as well as comparisons to the saturated model), and ACE estimates with 95% confidence intervals. The ACE model was the best-fitting model in five of the six samples; in the US Ohio sample the best-fitting model was the CE model. For the full ACE models, estimates of heritability ranged from 0.15 to 0.53, shared environment from 0.20 to 0.65, and non-shared environment from 0.15 to 0.28. Next we conducted heterogeneity analyses across the six samples.

Heterogeneity model-fitting analyses

Data from all six samples were included in a liability-threshold heterogeneity model, where each sample is modeled separately and then estimates are equated across samples to test for heterogeneity. There was no evidence for significant heterogeneity across the six samples. That is, it was possible to equate ACE estimates across the six samples without a significant reduction in fit (difference in chi squared = 20.805, difference in $df = 15$, $P = 0.143$, AIC = −9.195). However, the 95% confidence intervals of estimates for each study indicate that power was modest

Table 1 Means (and standard deviations) for general cognitive ability (*g*) by zygosity and sex at each GHCA site and ANOVA results

GHCA site	Mean age (SD) and range	*g*	Zygosity		Sex		ANOVA		
		All	MZ	DZ	Female	Male	Zygosity	Sex	Zygosity × sex
US Ohio	6.07 (0.68)	0.00 (1.00)	0.03 (1.06)	−0.01 (0.96)	−0.04 (1.03)	0.06 (0.97)	$P = 0.920$	$P = 0.324$	$P = 0.525$
	4.33–7.92	$n = 586$	$n = 244$	$n = 342$	$n = 339$	$n = 247$	$\eta^2 < 0.001$	$\eta^2 = 0.003$	$\eta^2 = 0.001$
United Kingdom	11.57 (0.69)	0.00 (1.00)	−0.06 (0.98)	0.03 (1.01)	−0.07 (0.99)	0.09 (1.00)	$P = 0.002$	$P < 0.001$	$P = 0.126$
	10.08–13.74	$n = 8{,}508$	$n = 3{,}156$	$n = 5{,}352$	$n = 4{,}762$	$n = 3{,}746$	$\eta^2 = 0.002$	$\eta^2 = 0.004$	$\eta^2 = 0.001$
US Minnesota	13.00 (2.83)	0.00 (1.00)	−0.01 (1.00)	0.02 (1.00)	−0.13 (1.00)	0.14 (0.98)	$P = 0.495$	$P < 0.001$	$P = 0.064$
	11.00–17.00	$n = 3{,}740$	$n = 2{,}374$	$n = 1{,}366$	$n = 1{,}948$	$n = 1{,}792$	$\eta^2 < 0.001$	$\eta^2 = 0.011$	$\eta^2 = 0.002$
US Colorado	13.12 (3.86)	0.00 (1.00)	−0.06 (0.99)	0.05 (1.00)	−0.08 (0.99)	0.08 (1.01)	$P = 0.003$	$P < 0.001$	$P = 0.052$
	6.00–25.00	$n = 5{,}728$	$n = 2{,}600$	$n = 3{,}128$	$n = 2{,}931$	$n = 2{,}797$	$\eta^2 = 0.003$	$\eta^2 = 0.006$	$\eta^2 = 0.001$
Australia	16.00 (0.45)	0.00 (1.00)	−0.05 (0.99)	0.03 (1.01)	−0.14 (0.98)	0.15 (1.00)	$P = 0.376$	$P < 0.001$	$P = 0.228$
	15.00–22.00	$n = 1{,}713$	$n = 679$	$n = 1{,}034$	$n = 888$	$n = 825$	$\eta^2 = 0.001$	$\eta^2 = 0.018$	$\eta^2 = 0.002$
The Netherlands	17.99 (14.47)	0.00 (1.00)	−0.02 (1.01)	0.02 (0.99)	−0.04 (1.02)	0.05 (0.98)	$P = 0.494$	$P = 0.056$	$P = 0.811$
	5.67–71.03	$n = 1{,}917$	$n = 874$	$n = 1{,}043$	$n = 1{,}022$	$n = 895$	$\eta^2 < 0.001$	$\eta^2 = 0.004$	$\eta^2 < 0.001$

MZ monozygotic, *DZ* dizygotic, η^2 eta squared (effect size). *n* Indicates number of individuals. ANOVA performed on one randomly selected member of each twin pair

Table 2 Mean (SD) and range of general cognitive ability in top 15% (*high g proband*) and in the rest of the sample

GHCA site	High *g* proband			Rest of sample		
	N	Mean (SD)	Range	*N*	Mean (SD)	Range
US Ohio	90	1.57 (0.42)	1.11–2.98	496	−0.28 (0.79)	−3.00 to 1.11
United Kingdom	1,269	1.41 (0.32)	1.02–3.08	7,190	−0.25 (0.86)	−3.09 to 1.02
US Minnesota	548	1.64 (0.47)	1.05–3.04	3,176	−0.28 (0.77)	−2.81 to 1.03
US Colorado	852	1.59 (0.42)	1.07–3.04	4,856	−0.28 (0.79)	−3.04 to 1.06
Australia	255	1.50 (0.35)	1.10–2.84	1,455	−0.26 (0.83)	−2.62 to 1.09
The Netherlands	286	1.51 (0.34)	1.09–2.78	1,623	−0.27 (0.83)	−3.02 to 1.09

N number of individuals. *N* values differ from Table 1 because outliers have been removed. There were a total of 96 outliers from all the studies: Ohio 0, UK 49, Minnesota 16, Colorado 20, Australia 3, and the Netherlands 8

Table 3 Tetrachoric correlations and thresholds (95% CI) for general cognitive ability at each GHCA site by zygosity

GHCA site	MZ	DZ	Threshold
US Ohio	0.80 ((0.59–0.92)	0.72 (0.47–0.88)	MZ = DZ = 1.03 (0.88–1.18)
United Kingdom	0.72 (0.65–0.78)	0.46 (0.39–0.53)	MZ = DZ = 1.04 (1.02–1.07)
US Minnesota	0.77 (0.69–0.82)	0.52 (0.39–0.64)	MZ = DZ = 1.04 (0.99–1.10)
US Colorado	0.85 (0.80–0.89)	0.59 (0.51–0.66)	MZ = DZ = 1.03 (0.99–1.08)
Australia	0.81 (0.67–0.89)	0.57 (0.42–0.69)	MZ = DZ = 1.04 (0.95–1.12)
The Netherlands	0.79 (0.67–0.87)	0.62 (0.47–0.74)	MZ = DZ = 1.04 (0.96–1.12)

MZ monozygotic, *DZ* dizygotic same and opposite-sex twins, *CI* confidence interval. Probandwise concordances were also calculated and yield a similar pattern of results as the tetrachoric correlations except that the concordances for both MZ and DZ twins are about 75% the magnitude of the tetrachoric correlations; we report the tetrachoric correlations because they convey more information and are used in the model-fitting analyses

for detecting significant differences between studies. Estimates and 95% confidence intervals from the equated model were $A = 0.50$ (0.41–0.60); $C = 0.28$ (0.19–0.37); and $E = 0.22$ (0.19–0.25). Although the lowest heritability in Table 4 is for the US Ohio study with the youngest twins, there is otherwise no trend for increasing heritability for the studies with older twins. Nonetheless, we re-sorted the high-*g* twins into the same three age groups as in our previous publication on the entire GHCA sample: childhood (average age of 9 years; range = 4–10), adolescence (12 years; range = 11–13), and young adulthood (17 years; range = 14–34) (Haworth et al. 2009). For these age analyses it was not possible to include a fourth group of individuals above the age of 34 years because there were too few individuals to provide adequate power in the twin analyses. We also performed analyses for the young adult group with a restricted age range of only 14–26 years, which produced similar results; we therefore present the larger age range (14–34 years) here for the young adulthood sample. There was no evidence for significant heterogeneity across the three age groups. That is, it was possible to equate *A*, *C*, and *E* estimates across the three age groups without a significant reduction in fit. Again, however, the 95% confidence intervals of estimates for each age group indicate that power was limited to detect significant differences between the age groups (Table 5). A more sophisticated analysis of the age effect is possible by including age as a continuous moderator in the model. Such an analysis on this sample failed to optimize, so we do not present results from this model here.

Discussion

In this first adequately powered analysis of the genetic and environmental etiology of high general cognitive ability (*g*), defined as the top 15% of the distribution, we find evidence for substantial heritability (0.50 with 95% confidence intervals of 0.41–0.60) and moderate shared environmental influence (0.28, 0.19–0.37). How do these results for high *g* compare to results for the normal distribution of *g*? We have previously reported that results for the normal distribution of *g* were similar for the entire GHCA sample of 11,000 twin pairs: heritability was estimated as 0.55 (0.51–0.59) and shared environment was 0.21 (0.17–0.25) (Haworth et al. 2009). The overlapping confidence intervals suggest that the etiology of high *g* is not significantly different from the origins of individual differences in *g* throughout the normal distribution. However, the large confidence intervals for high *g* suggest caution in concluding that there are no differences in the etiology of high *g* and the normal distribution of *g*. Moreover, similar heritabilities do not necessarily imply

Table 4 Univariate liability-threshold model fitting for each GHCA site (85% cut-off): model fit and parameter estimates (95% confidence intervals in parentheses)

GHCA site	Model	$\Delta\chi^2$	Δdf	P	AIC	A	C	E
US Ohio	ACE	4.739	3	0.192	−1.261	0.15 (0.00–0.73)	0.65 (0.12–0.87)	0.20 (0.08–0.38)
	CE	0.316	1	0.574	−1.684		0.76 (0.61–0.87)	0.24 (0.13–0.39)
	AE	5.412	1	0.020	3.412	0.84 (0.67–0.93)		0.16 (0.07–0.33)
United Kingdom	**ACE**	4.302	3	0.231	−1.698	0.52 (0.33–0.70)	0.20 (0.05–0.35)	0.28 (0.24–0.35)
	CE	26.483	1	<0.001	24.483		0.56 (0.51–0.61)	0.44 (0.39–0.49)
	AE	6.679	1	0.010	4.676	0.75 (0.70–0.80)		0.25 (0.20–0.30)
US Minnesota	**ACE**	1.287	3	0.732	−4.713	0.48 (0.21–0.78)	0.28 (0.01–0.53)	0.23 (0.18–0.31)
	CE	12.530	1	<0.001	10.530		0.68 (0.62–0.74)	0.32 (0.26–0.38)
	AE	4.057	1	0.044	2.057	0.78 (0.71–0.83)		0.22 (0.17–0.29)
US Colorado	**ACE**	6.592	3	0.086	0.592	0.53 (0.35–0.71)	0.32 (0.16–0.48)	0.15 (0.11–0.20)
	CE	33.808	1	<0.001	31.808		0.72 (0.67–0.76)	0.28 (0.24–0.33)
	AE	14.029	1	<0.001	12.029	0.87 (0.83–0.91)		0.13 (0.09–0.17)
Australia	**ACE**	2.348	3	0.503	−3.652	0.48 (0.12–0.83)	0.33 (0.01–0.60)	0.19 (0.11–0.33)
	CE	6.620	1	0.010	4.620		0.66 (0.56–0.75)	0.34 (0.25–0.44)
	AE	4.196	1	0.041	2.196	0.84 (0.73–0.91)		0.16 (0.09–0.27)
The Netherlands	**ACE**	2.021	3	0.568	−3.979	0.34 (0.01–0.68)	0.45 (0.14–0.71)	0.21 (0.13–0.33)
	CE	4.089	1	0.043	2.089		0.71 (0.62–0.78)	0.29 (0.22–0.38)
	AE	7.612	1	0.006	5.612	0.82 (0.73–0.89)		0.18 (0.11–0.27)

Two fit indices are reported: Δchi-squared (χ^2) and Akaike's information criterion, (AIC; Akaike 1987). CE and AE models are nested within the ACE model; the ACE model is nested in the fully saturated model. The best fitting model (in boldface) was chosen on the basis of a change in χ^2 not representing a significant worsening of fit (for a change of df of 1, the statistically significant change in χ^2 is 3.84). Δdf change in degrees of freedom, A additive genetic influence, C shared environmental influence, E non-shared environmental influence

Table 5 Univariate liability-threshold model fitting for each age category (85% cut-off): model fit and parameter estimates (95% confidence intervals in parentheses)

	Model	$\Delta\chi^2$	Δdf	P	AIC	A	C	E
Childhood	**ACE**	0.798	3	0.850	−5.202	0.54 (0.34–0.74)	0.28 (0.10–0.45)	0.18 (0.13–0.25)
	CE	27.110	1	<0.001	25.110		0.67 (0.62–0.72)	0.33 (0.28–0.38)
	AE	9.033	1	0.003	7.033	0.84 (0.79–0.88)		0.16 (0.12–0.21)
Adolescence	ACE	2.608	2*	0.272	−1.392	0.60 (0.42–0.77)	0.13 (0.00–0.27)	0.27 (0.22–0.33)
	CE	43.656	1	<0.001	41.656		0.57 (0.52–0.61)	0.43 (0.39–0.48)
	AE	2.896	1	0.089	0.896	0.74 (0.69–0.79)		0.26 (0.21–0.30)
Young adulthood	**ACE**	1.732	3	0.630	−4.268	0.47 (0.28–0.66)	0.32 (0.15–0.48)	0.21 (0.16–0.27)
	CE	22.907	1	<0.001	20.907		0.68 (0.62–0.72)	0.32 (0.28–0.38)
	AE	12.306	1	<0.001	10.306	0.81 (0.76–0.84)		0.19 (0.15–0.24)

Two fit indices are reported: Δchi-squared (χ^2) and Akaike's information criterion, (AIC; Akaike 1987). CE and AE models are nested within the ACE model; the ACE model is nested in the fully saturated model. The best fitting model (in boldface) was chosen on the basis of a change in χ^2 not representing a significant worsening of fit (for a change of df of 1, the statistically significant change in χ^2 is 3.84). Δdf change in degrees of freedom, A additive genetic influence, C shared environmental influence, E non-shared environmental influence

* In the adolescence age group, MZ and DZ thresholds could not be equated. In all other samples the MZ and DZ thresholds could be equated

Using the heterogeneity model there was no evidence for significant heterogeneity across the three age groups

For whole model: difference in chi squared = 12.728, difference in $df = 6$, $P = 0.048$, AIC = 0.728

For A: difference in chi squared = 0.974, difference in $df = 2$, $P = 0.614$, AIC = −3.026

For C: difference in chi squared = 3.236, difference in $df = 2$, $P = 0.198$, AIC = −0.764

For E: difference in chi squared = 5.377, difference in $df = 2$, $P = 0.068$, AIC = 1.377

that the same genes affect high *g* and the normal distribution of *g*. Proof of this critical issue will come when genes are found associated with *g*: The test will be the extent to which genes associated with high *g* are also associated with *g* throughout the normal distribution and vice versa.

In the Introduction, we mentioned a hypothesis called *emergenesis* which suggests that exceptional cognitive ability may be due to epistasis, especially rare combinations of alleles (Lykken 1982, 2006). The hallmark of a highly epistatic trait is high MZ correlations and low DZ correlations, lower than half the MZ correlation even though their coefficient of genetic relationship is 0.50. This pattern of twin correlations is expected for a highly epistatic trait because MZ twins share all non-additive interactions whereas epistasis scarcely contributes to similarity for DZ twins and other first-degree relatives (Plomin et al. 2008). As can be seen in Table 3, the MZ and DZ tetrachoric correlations are not at all consistent with non-additive genetic influence because the DZ correlations exceed half the MZ correlation. However, we cannot rule out the effect of epistatic genetic effects, particularly as the twin design is not ideal for assessing these effects. Molecular genetic studies on very high *g* individuals will provide a better test of the role of epistasis in high cognitive ability.

Three limitations of the present study should be mentioned. The first limitation is a consequence of combining six studies in the GHCA consortium. The major strength of the present study is its large sample which provides the power needed to investigate the genetic and environmental etiology of high *g*. However, the six twin studies in the GHCA consortium used different measures of *g*. As mentioned in the Introduction, the creation of *g* scores within each study can be defended because of the high correlation between *g* scores from different test batteries (Johnson et al. 2008). A related potential limitation lies in combining data from four countries. However, the use of different measures and different samples can also be viewed as strengths because the results were nonetheless similar across the studies, which adds to the robustness of our conclusion that genetic variation contributes substantially to high *g* (Lykken 1968).

A second limitation of our study is that we used a cut-off of the top 15% of the distribution as our index of high *g*. Although individuals in the top 15% of the distribution of *g* are by definition high *g*, it is an open question whether similar results would be obtained for truly exceptional individuals such as individuals in the studies of mathematically precocious youth which represent the top 0.01% of the distribution (Lubinski and Benbow 2006). We chose the 15% cut-off for two reasons. First, it mirrors the cut-off often used at the low end of the distribution in studies of cognitive disability. Second, for the twin studies in the GHCA, the 15% cut-off represents a reasonable balance between severity of selection and sample size, which is a crucial consideration in relation to power.

The third limitation is not specific to our study but general to the use of the twin method to estimate shared environmental influence in the cognitive domain. It seems likely that estimates of shared environment for *g* in twin studies are greater than estimates from family and adoption sibling designs, especially after childhood. It is reasonable to assume that because twins are the same age and grow up in the same family at the same time, they share their experiences to a greater extent than other siblings. One study reported that for cognitive abilities, but not for behavior problems, estimates of shared environment were more than twice as large for twins as compared to non-twin siblings (Koeppen-Schomerus et al. 2003). Future research on the genetics of high *g* could estimate the extent of a special twin shared environmental effect by including non-twin siblings. For now, it would be prudent to assume that our estimate of 0.28 for shared environment for high *g*, although accurate for twins, may be an overestimate of shared environmental influence for non-twin populations.

Finding substantial genetic influence of high *g* suggests several directions for future research. The goal of the GHCA consortium is to conduct a genome-wide association study of high *g* in order to identify specific genes that contribute to its heritability. Twins of course are not necessary for molecular genetic analyses and there is a need for a much larger sample of much higher *g* individuals than in the GHCA consortium in order to meet the daunting demands for power in genome-wide association scans for associations of small effect size, especially when individuals are genotyped for as many as a million DNA markers thus creating a huge multiple-testing problem. For these reasons, in collaboration with Martha Putallaz at Duke University, the consortium has launched a study of participants in the talent identification program (TIP; Putallaz et al. 2005).

Since 1980, TIP has screened 1.8 million gifted youth in talent searches. This screening was followed by above-level testing in order to select some of the brightest children in the United States. The goal of GHCA is to obtain DNA from as many TIP participants as possible. Although many genome-wide association studies are underway (Kruglyak 2008) nearly all of these focus on diseases, disorders and the low end of distributions. High *g* provides an interesting angle for gene-finding studies because exceptionally high *g* presumably requires an individual to have many ability-enhancing alleles and few ability-detracting alleles. GHCA intends to increase power to detect associations by conducting a genome-wide association study of a large sample of extremely high *g* individuals.

It is our hope that finding substantial heritability for high *g* does not re-ignite controversies in relation to expert training

(e.g., Howe et al. 1998). Heritability and expert training address different issues: 'what is' vs. 'what could be.' Heritability describes the extent to which individual differences in *g* can be attributed to genetic differences between individuals given the genetic and environmental differences that exist in a particular population at a particular time. In contrast, training experiments are not concerned about describing the origins of individual differences; their focus is on the potential for change. That is, heritability of *g* could be 100% but a training regime or other environmental interventions could improve performance on tests that assess *g*. Conversely, showing that environmental interventions can improve performance says nothing about the genetic and environmental origins of individual differences. However, beyond this nature vs. nurture level of debate, there are interesting and largely unexplored issues at the interface between training and heritability. For example, are there genotype–environment interactions, differential sensitivity to the quantity or quality of training as a function of genotype? Or genotype–environment correlations, differential exposure to training as a function of genotype, in which children seek, modify and create environments correlated with their genetic propensities? One interesting example of this interface is a study of performance on a motor task which showed that heritability was substantial before, during and after training (Fox et al. 1996). Further analyses of gene–environment interaction and correlation are also needed. As one of many possible examples, these results for high *g* may be moderated by socioeconomic class as has been suggested for the full range of *g* (Turkheimer et al. 2003).

Finally, we hope that our study, the many interesting and unanswered questions about high cognitive ability, and the importance of studying the high end of the distribution of ability as well as the low end will stimulate much-needed research on the genetics of high cognitive ability.

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References

Akaike H (1987) Factor analysis and AIC. Psychometrika 52:317–332

Bartels M, Rietveld MJ, van Baal GC, Boomsma DI (2002) Genetic and environmental influences on the development of intelligence. Behav Genet 32:237–249. doi:10.1023/A:1019772628912

Bleichrodt N, Drenth PJD, Zaal JN, Resing WCM (1984) Revisie Amsterdams kinder intelligentie test. Swets and Zeitlinger B.V, Lisse

Boomsma DI, De Geus EJC, Vink JM, Stubbe JH, Distel MA, Hottenga JJ et al (2006) Netherlands twin register: from twins to twin families. Twin Res Hum Genet 9:849–857. doi:10.1375/twin.9.6.849

Boomsma DI, van Beijsterveld TCEM, Beem AL, Hoekstra RA, Polderman TJC, Bartels M (2008) Intelligence and birth order in boys and girls. Intelligence 36:630–634. doi:10.1016/j.intell.2008.01.005

Bouchard TJ Jr, McGue M (1981) Familial studies of intelligence: a review. Science 212:1055–1059. doi:10.1126/science.7195071

Davis OSP, Haworth CMA, Plomin R (2009) Generalist genes for cognition: etiology of learning abilities and disabilities in early adolescence. Cogn Neuropsychiatr (in press)

Deary IJ, Spinath FM, Bates TC (2006) Genetics of intelligence. Eur J Hum Genet 14:690–700. doi:10.1038/sj.ejhg.5201588

DeFries JC (1985) Colorado reading project. In: Gray DB, Kavanagh JF (eds) Biobehavioral measures of dyslexia. York Press, Parkton, pp 107–122

DeFries JC, Olson RK, Pennington RF, Smith SD (1991) Colorado reading project: an update. In: Duane DD, Gray DB (eds) The reading brain: the biological basis of dyslexia, York Press, Parkton, pp 53–87

DeFries JC, Filipek PA, Fulker DW, Olson RK, Pennington BF, Smith SD et al (1997) Colorado learning disabilities research center. Learn Disabil Q 8:7–19

Falconer DS (1965) The inheritance of liability to certain diseases estimated from the incidence among relatives. Ann Hum Genet 29:51–76. doi:10.1111/j.1469-1809.1965.tb00500.x

Fox PW, Hershberger SL, Bouchard TJ Jr (1996) Genetic and environmental contributions to the acquisition of a motor skill. Nature 384:356–357. doi:10.1038/384356a0

Galton F (1869) Hereditary genius: an enquiry into its laws and consequences. Macmillan, London

Galton F (1883) Inquiries into human faculty and its development. Macmillan, London

Haworth CMA, Harlaar N, Kovas Y, Davis OSP, Oliver BR, Hayiou-Thomas ME et al (2007) Internet cognitive testing of large samples needed in genetic research. Twin Res Hum Genet 10:554–563. doi:10.1375/twin.10.4.554

Haworth CMA, Wright MJ, Luciano M, Martin NG, de Geus EJC, van Beijsterveldt CEM et al (2009) The heritability of general cognitive ability increases linearly from childhood to young adulthood. Mol Psychiatr (in press)

Howe MJA, Davidson JW, Sloboda JA (1998) Innate talents: reality or myth? Behav Brain Sci 21:399–442. doi:10.1017/S0140525X9800123X

Iacono WG, Carlson SR, Taylor J, Elkins IJ, McGue M (1999) Behavioral disinhibition and the development of substance-use disorders: findings from the Minnesota twin family study. Dev Psychopathol 11:869–900. doi:10.1017/S0954579499002369

Iacono WG, McGue M, Krueger RF (2006) Minnesota Center for twin and family research. Twin Res Hum Genet 9:978–984. doi:10.1375/twin.9.6.978

Jackson DN (1998) Multidimensional aptitude battery II: manual. Sigma Assessment Systems, Port Huron
Jensen AR (1998) The g factor: the science of mental ability. Praeger, Wesport
Johnson W, Nijenhuis J, Bouchard TJ (2008) Still just 1 g: consistent results from five test batteries. Intelligence 36:81–95. doi:10.1016/j.intell.2007.06.001
Koeppen-Schomerus G, Spinath FM, Plomin R (2003) Twins and non-twin siblings: different estimates of shared environmental influence in early childhood. Twin Res 6:97–105. doi:10.1375/136905203321536227
Kovas Y, Haworth CMA, Dale PS, Plomin R (2007) The genetic and environmental origins of learning abilities and disabilities in the early school years. Monogr Soc Res Child Dev 72:1–144
Kruglyak L (2008) The road to genome-wide association studies. Nat Rev Genet 9:314–318. doi:10.1038/nrg2316
Lubinski D, Benbow CP (2006) Study of mathematically precocious youth after 35 years: uncovering antecedents for the development of math-science expertise. Perspect Psychol Sci 1:316–345
Lubinski D, Benbow CP, Webb RM, Bleske-Rechek A (2006) Tracking exceptional human capital over two decades. Psychol Sci 17:194–199. doi:10.1111/j.1467-9280.2006.01685.x
Luciano M, Wright MJ, Geffen GM, Geffen LB, Smith GA, Evans DM et al (2003a) A genetic two-factor model of the covariation among a subset of multidimensional aptitude battery and Wechsler adult intelligence scale-revised subtests. Intelligence 31:589–605. doi:10.1016/S0160-2896(03)00057-6
Luciano M, Wright MJ, Smith GA, Geffen GM, Geffen LB, Martin NG (2003b) Genetic covariance between processing speed and IQ. In: Plomin R, DeFries JC, Craig IW, McGuffin P (eds) Behavioral genetics in the postgenomic era. American Psychological Association, Washington, DC, pp 163–181
Lykken DT (1968) Statistical significance in psychological research. Psychol Bull 70:151–159. doi:10.1037/h0026141
Lykken DT (1982) Research with twins: the concept of emergenesis. Psychophysiology 19:361–373. doi:10.1111/j.1469-8986.1982.tb02489.x
Lykken DT (2006) The mechanism of emergenesis. Genes Brain Behav 5:306–310. doi:10.1111/j.1601-183X.2006.00233.x
McGue M, Bouchard TJ Jr (1984) Adjustment of twin data for the effects of age and sex. Behav Genet 14:325–343. doi:10.1007/BF01080045
Neale MC, Boker SM, Xie G, Maes H (2006) Mx: statistical modeling, 7th edn. Department of Psychiatry, Richmond
Nichols RC, Bilbro WC (1966) The diagnosis of twin zygosity. Acta Genet 16:265–275. doi:10.1159/000151973
Nyholt DR (2006) On the probability of dizygotic twins being concordant for two alleles at multiple polymorphic loci. Twin Res Hum Genet 9:194–197. doi:10.1375/twin.9.2.194
Oliver BR, Plomin R (2007) Twins early development study (TEDS): a multivariate, longitudinal genetic investigation of language, cognition and behavior problems from childhood through adolescence. Twin Res Hum Genet 10:96–105. doi:10.1375/twin.10.1.96
Petrill SA, Saudino KJ, Cherny SC, Emde RN, Fulker DW, Hewitt JK et al (1998) Exploring the genetic and environmental etiology of high general cognitive ability in 14 to 36 month-old twins. Child Dev 69:68–74
Petrill SA, Deater-Deckard K, Thompson LA, Schatschneider C, DeThorne LS, Vandenbergh DJ (2007) Longitudinal genetic analysis of early reading: the Western Reserve reading project. Read Writ 20:127–146. doi:10.1007/s11145-006-9021-2
Plomin R, Kovas Y (2005) Generalist genes and learning disabilities. Psychol Bull 131:592–617. doi:10.1037/0033-2909.131.4.592
Plomin R, Spinath FM (2004) Intelligence: genetics, genes, and genomics. J Pers Soc Psychol 86:112–129. doi:10.1037/0022-3514.86.1.112
Plomin R, Thompson LA (1993) Genetics and high cognitive ability. In: Bock GR, Ackrill K (eds) The origins and development of high ability. Wiley (CIBA Foundation Symposium 178), Chichester, pp 62–84
Plomin R, DeFries JC, McClearn GE, McGuffin P (2008) Behavioral genetics, 5th edn. Worth, New York
Polderman TJC, Gosso MF, Posthuma D, van Beijsterveldt TC, Heutink P, Verhulst FC et al (2006) A longitudinal twin study on IQ, executive functioning, and attention problems during childhood and early adolescence. Acta Neurol Belg 106:191
Posthuma D, Mulder E, Boomsma DI, De Geus EJC (2002) Genetic analysis of IQ, processing speed and stimulus-response incongruency effects. Biol Psychol 61:157–182. doi:10.1016/S0301-0511(02)00057-1
Price TS, Freeman B, Craig IW, Petrill SA, Ebersole L, Plomin R (2000) Infant zygosity can be assigned by parental report questionnaire data. Twin Res 3:129–133. doi:10.1375/136905200320565391
Putallaz M, Baldwin J, Selph H (2005) The Duke University talent identification program. High Abil Stud 16:41–54. doi:10.1080/13598130500115221
Raven JC, Court JH, Raven J (1996) Manual for Raven's progressive matrices and vocabulary scales. Oxford University Press, Oxford
Raven JC, Court JH, Raven J (1998) Manual for Raven's progressive matrices. H.K. Lewis, London
Rhea SA, Gross AA, Haberstick BC, Corley RP (2006) Colorado twin registry. Twin Res Hum Genet 9:941–949. doi:10.1375/twin.9.6.941
Rietveld MJH, van der Valk JC, Bongers IL, Stroet TM, Slagboom PE, Boomsma DI (2000) Zygosity diagnosis in young twins by parental report. Twin Res Hum Genet 3:134–141. doi:10.1375/twin.3.3.134
Rijsdijk FV, Vernon PA, Boomsma DI (2002) Application of hierarchical genetic models to Raven and WAIS subtests: a Dutch twin study. Behav Genet 32:199–210. doi:10.1023/A:1016021128949
Ronald A, Spinath F, Plomin R (2002) The aetiology of high cognitive ability in early childhood. High Abil Stud 13:103–114. doi:10.1080/1359813022000048761
Saudino KJ, Plomin R, Pedersen NL, McClearn GE (1994) The etiology of high and low cognitive ability during the second half of the life span. Intelligence 19:353–371. doi:10.1016/0160-2896(94)90007-8
Smith C (1974) Concordance in twins: methods and interpretation. Am J Hum Genet 26:454–466
Spearman C (1927) The abilities of man: their nature and measurement. Macmillan, New York
Thompson LA, Detterman DK, Plomin R (1993) Differences in heritability across groups differing in ability, revisited. Behav Genet 23:331–336. doi:10.1007/BF01067433
Thorndike RL, Hagen EP, Sattler JM (1986) Guide for administering and scoring the fourth edition: Stanford-Binet intelligence scale. Riverside, Chicago
Turkheimer E, Haley A, Waldron M, D'Onofrio B, Gottesman II (2003) Socioeconomic status modifies heritability of IQ in young children. Psychol Sci 14:623–628. doi:10.1046/j.0956-7976.2003.psci_1475.x
Wechsler D (1974) Manual for the Wechsler intelligence scale for children-revised. (Revised ed.). Psychological Corporation, New York
Wechsler D (1981) Examiner's manual: Wechsler adult intelligence scale: revised. The Psychological Corporation, New York

Wechsler D (1991) WISC-III. The Psychological Corporation, San Antonio

Wechsler D (1992) Wechsler intelligence scale for children—third edition UK (WISC-IIIUK) manual. The Psychological Corporation, London

Wechsler D (1997) Wechsler adult intelligence scale-III. The Psychological Corporation, New York

Wright MJ, Martin NG (2004) Brisbane adolescent twin study: outline of study methods and research projects. Aust J Psychol 56:65–78. doi:10.1080/00049530410001734865