

Heritability of Intelligence

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Intelligence has been studied and measured for about 100 years. During that time researchers initially made casual observations that intelligence was substantially heritable, but they did not have enough data nor the appropriate methodology to quantify heritability. It was not pursued as an important focus of research until the end of the 60s. During the 60s, it was commonly believed (even by appropriately educated scholars) that humans were primarily the product of their environments. This belief was applied to intelligence in a deductive manner, with the starting assumption taken to be true and with all implications accorded equal truth. Since there was no inductive path established, people used their unproved assumptions to design various social programs with the expectation that they would elevate the intelligence of groups known to exhibit low intelligence. Parents were urged to provide stimulating home environments in order to cause their children to become smart.

Thus appeared a classic example of thinking about what should happen, without first bothering to find out what actually happens and then attempting to determine causation. Any researcher can look around him at the typical environment of his erudite colleagues and see that most of them came from high SES backgrounds, grew up in homes filled with books, and were exposed to stimulating educational activities. They can then compare that environment with that of the typical low IQ individual and note that the latter most often grew up in homes without books, had few childhood activities that relate to scholarship, and generally lived in depressed circumstances. When academics did just that, they concluded that intelligence is a matter of attention, encouragement, and a stimulating environment.

One of the first indications that the commonly accepted ideas about environment were wrong was the product of the Civil Rights Act of 1964, which spawned a national survey of US students. In 1966, the findings of this study were published in the Coleman report. The “aptitude” assessment discussed in the report was nothing more than an extraction from various standard IQ tests (Herrnstein & Murray, 1994). Its findings were that the abilities of the large number of students surveyed were not the result of the quality of their schools. This observation remains true today, in spite of continued denial by various political and educational groups. In fact, they have pushed for massive educational spending¹ in some US school districts, only to reconfirm what James Coleman observed in the 60s. At the end of that decade, the now famous Harvard Educational Review article appeared (Jensen, 1969). In his summary, Jensen wrote: “Individual variation in IQ is largely genetic, as shown by heritability analysis of kinship data. Social class differences involve genetic factors, and many lines of evidence suggest it is a reasonable hypothesis that genetic factors may be strongly implicated in the one standard deviation average IQ difference between Whites and Blacks.” For making this accurate observation, Jensen was villanized by various groups in a manner that has become commonplace by those who impose political correctness on scientific research. Subsequently, researchers devised studies to test the heritability of intelligence. Their

findings were consistent and showed very high heritability. As with Jensen, many of these researchers were also damned by politically correct journalists, academics, and politicians.

This article will explore the specific findings that show not only that intelligence is highly heritable, but will quantify the findings. Before proceeding, we must have a definition.

$$\text{Heritability} = (\text{variance in the genotype}) / (\text{variance in the phenotype})$$

Since the above is a ratio of variances, heritability is a variance and is written as h^2 . The variance in the phenotype is the sum of the variances due to the genotype and the environment. That means that heritability increases when the environment contributes less to the variance.

The Biological Basis of Intelligence

Intelligence has been studied mostly by the use of regression analysis as a means of ferreting out the factors that simultaneously appear in the phenotype and which are difficult or impossible to study independently. It has been through such statistical examination that the biological components of intelligence have been identified and quantified. Other statistical methods have also been useful, but none has become as important as factor analysis.² Intelligence researchers (Spearman in particular) observed that all cognitive processes are correlated; this is known as the “positive manifold.” Factor analysis is intended to distill this single common factor. In a hierarchal factor analysis, the data generated by an IQ test are correlated in stages and by groups, such that where there are as many as three³ separate sets of data that relate, a single factor is extracted that shows the commonality between the sets. After the extraction of factors from one level, the resulting factors are treated similarly, resulting in a smaller set of factors that are more general than the prior factors. After a few extractions, the resulting sets have only a single factor that is common to them all and that factor is known as g . The factors from which the final extraction is performed are known as group factors.⁴

In this section, the biological elements of intelligence will be discussed, as evidence of the heritability of intelligence. Heritability estimates are given where such data is known, but even in cases where heritability is not easily located in the literature, it is reasonable to expect that biological elements are largely determined by genetic, as opposed to environmental factors. This is especially true when the biological features appear early in life, before various environmental factors (such as schools) have an opportunity to act.

Nerve Conduction Velocity (NCV)

The importance of NCV is that it presumably affects all of the cognitive processing in the brain, and therefore, is likely a primary component of g . Reed and Jensen (1991) measured NCV in males and found a correlation of +.37 with the Raven IQ (after correction for restriction of range). The portion of the brain that was measured was not part of the brain involved in cognition. They specifically looked at the P100 visual

evoked potential. An important finding was that although both NCV and choice reaction time were both significantly correlated to *g*, they were not significantly correlated with each other; this implies separate mechanisms. A confirmation of the Reed and Jensen finding was performed with both male and female subjects, with similar results (Reed, Vernon, & Johnson, 2004).

NCV correlates positively with the degree of myelination (Reed, Vernon, & Johnson, 2004). Presumably myelin thickness is an actual cause (there are others) of the higher NCV and therefore will co-vary with it.

There are two paths that may explain the function of NCV:

Path 1 – Brain speed and intelligence correlate positively and strongly. Measurements of reaction time (RT) and inspection time (IT) show such strong correlations with *g* that they can substitute for IQ tests as an equally valid measurement. Brand (1996) focused much of his discussion on brain speed as the primary source of variance in intelligence.

Path 2 – Jensen (1998) noted that the correlation between RT and *g* is likely to be due to the volatile nature of working memory. A slower brain cannot access and use the representations held in working memory without refreshing them, while a faster brain⁵ is able to use working memory more efficiently, with less refreshing.

One of the most subjectively convincing demonstrations of the relationship between a simple evoked response and brain activity is that of fMRI images over short intervals (ms), assembled into a viewable movie. The author viewed such movies at The Mind Institute in Albuquerque. These show rapid and widely dispersed brain activity following an external (visual) stimulus. It is intuitively obvious that variations in speed would affect the progression of the events that are observed. This imaging demonstrates what is being measured with respect to various RT paradigms and the standard IT measurement.

Chronometric Measurements

RT measurements are performed in the context of elementary cognitive tests (ECTs). A wide variety of such tests⁶ have been devised, ranging from the simple response of pressing a button when a light is turned on, to the selection of a button response based on a more complex (but still very easy) task, such as determining which of 8 lights is illuminated but does not have an adjacent light illuminated. ECTs do not completely co-vary and can be combined when given as a test battery to produce a high quality measurement of *g*. The importance of ECTs in the understanding of heritability is that they act at a very low biological level and their heritability can be measured. Jensen (2006) discusses various measurement results for RT, which are simply summarized by Beaujean (2005) as $h^2 = .40$ for subjectively simpler tasks to $h^2 = .67$ for subjectively more complex tasks.

It should be noted that RT measurements have a limited correlation with *g* because RT consists of both peripheral and central processing components; only the central

processing component loads on g . Since the non- g peripheral component cannot be eliminated, the RT- g correlation is limited. When the g -loaded components are additive (even if only partially so), they can be combined from different ECTs to yield a higher loading than exists with any individual class of ECT. This same condition applies in standard IQ tests. In the case of a composite RT measurement (use of multiple ECTs), heritability is higher than for the individual measurements. This is because the combined measurements reduces the attenuation due to specificity.⁷ It is also partly due to the increased heritability associated with more complex tasks (Jensen 2006).

IT is a measurement of the sensitivity of the individual to a briefly presented stimulus. The IT procedure⁸ consists of the projection of a masked figure;⁹ the removal of the mask to show the figure; and covering of the figure by reapplying the mask. This can be done with both auditory and visual stimuli, but some individuals have problems (unrelated to IQ) in responding to the auditory format, so it is used less often than visual IT. Since there is no motor component in the IT measurement, it has a higher correlation with g than individual RT measurements. A large body of literature has built up around IT measurements; when combined via a meta analysis, they show an IT- g correlation of $-.54$. See Jensen (1998) and, for a more extensive discussion, Brand (1996).

Other measures of speed related parameters have also appeared in the literature. One of these is the latency of troughs as shown in electroencephalography traces. The P300 latency¹⁰ has been of particular interest to some researchers, since that latency has produced strong correlations with IQ in some studies. Processing speed (G_s) is measured by some IQ tests using timed clerical tasks (visual matching, cross out, picture naming, etc.). Although processing speed has a lower g -loading than some other tests, it is still significantly g -loaded.

It is reasonable to accept that the various speed related parameters are biological functions that are determined genetically and all correlate with g in the “faster = higher g ” direction. It is, therefore, not surprising to find that these parameters and g are highly heritable.

Myelination

The importance of myelin in intelligence is well known, but the precise ways in which it operates remains open to investigation. Myelin is an insulating sheath around axons. It contributes directly and significantly to nerve conduction velocity and acts as an insulator. Destruction of myelin (demyelination) is the underlying factor responsible for the symptoms of multiple sclerosis; there is an associated decline in intelligence. Per the discussion concerning nerve conduction velocity, a biological condition that causes faster NCV (Jung and Haier, 2006) is linked to the speed related factors that correlate with intelligence. These presumably act to increase the efficiency of WMC¹¹ and other cognitive functions.

There is a second, and completely different, possible mechanism for myelin to influence intelligence. Case (1995) first speculated that a form of cross-talk may exist between neurons. More myelination means better insulation and less cross-talk; this means fewer retransmissions due to noise. Miller (1994) presented a carefully constructed argument that neural noise may explain various observations, such as the relatively high correlation between the standard deviation of RT and g . He argued that low myelination means more noise and more retransmissions and that this would account for the larger standard deviations of RT (known as RTSD) seen in low g subjects.

The correlation between myopia and high intelligence has been clearly established, but its cause remains open to some speculation. One idea is that myopia is caused by higher than normal myelination in the eye, presumably reflecting high myelination in the brain and possibly accounting for at least some of the correlation between brain size and myelination (Miller, 1994). It is also possible that myelination in the eye is pleiotropic (Rushton and Jensen, 2005).

The myelination explanation for RTSD is not, however, the only workable model. Jensen (2006) discusses oscillation theory as another viable explanation. The oscillation theory argument is based on neural excitatory potential, which oscillates at different rates from one individual to another. The excitatory potential may be thought of as the probability that an external stimulus will set off a train of neural impulses as a response to the stimulus. If the external stimulus happens with the excitatory potential is in a trough, there will be no response until the potential returns to the excitatory threshold. The period in which the brain cannot respond to an external stimulus is necessarily a function to the rate of oscillation. If the oscillatory rate is high, the non-response period will be shorter, and vice versa.

As of today, the magnitudes and mechanisms of these two models are not resolved. It may happen that either or both mechanisms account for some of the variance seen in cognitive processing speed and efficiency. But both models are entirely individual biological features and should be accounted for primarily by genetic factors.

Brain Volume

The correlation between brain volume and intelligence has been known for a long time. As early as the 1800s, scientists¹² were measuring cranial volume¹³ and correctly observed its relationship with intelligence. Brain size is fundamental to the differences in intelligence, not only within humans, but between species. Species comparisons are made on the basis of the encephalization quotient (EQ = brain size to body size ratio). In general, higher EQ means higher intelligence. Lists of EQs can be found in various texts, including Lynn (2006). Larger brains have more neurons and that means greater processing capacity, whether among humans or between species.

Head size (and calculated brain volume) can be used as a proxy for brain volume¹⁴ and correlates with IQ at about $r = 0.20$ (Rushton & Osborne, 1995). The development of methods of measuring brain volume in living subjects via computerized axial tomography

and MRI boosted the correlation to $r = 0.40$.¹⁵ Miller and Penke (2007) tabulated all of the MRI brain volume to intelligence studies up to that date. The mean correlation was 0.431. This is probably the maximum Pearson correlation that can be found, based on total brain volume because much of the brain is devoted to non-cognitive functions; however, using the method of correlated vectors¹⁶, the correlation with g falls between .60 and .70 (Rushton, 1999).

The importance of brain volume advanced further when Haier applied voxel-based morphometry to determine the correlations between the volumes of specific cognitive centers¹⁷ in the brain with IQ (Jung and Haier, 2006). The summed volumes of these areas correlates with g at $r = 0.70$.¹⁸ This is a very high correlation and is most likely the single largest biological component of intelligence. It's importance is particularly strong in the context of heritability, since brain volume heritability is 0.90 (Lynn, 2006) or greater. Atwood (2004) reported a heritability of .94 in 1330 individuals. "That narrow-sense heritability estimate of .94 suggests that the broad-sense heritabilities in other studies¹⁹ capture almost entirely additive genetic variance." (Miller and Penke, 2007). None of the reports of brain volume heritability have shown differences in h^2 between the sexes.

The sex difference in intelligence has been addressed numerous times in the past, but has usually not been pursued because it was explained away. But an inconsistency became apparent when Ankney (1992) and Rushton (1992) published papers showing that males have larger brains, even after correction for body size. Jensen (1998) pointed to a limited study (Witelson et al., 1995) that reported a higher neural density in females. The implication was that females had a similar number of neurons, but that they were more densely packed and that there was no real difference in mean intelligence between the sexes. Lynn (1999) evaluated several explanations for the apparent anomaly and offered the explanation that there is an IQ gap of about 4 points, favoring males and confirming the brain size difference and pointed to the much larger study (Packenberg and Gundersen, 1997) that found that the average male brain contained 22.8 billion neurons and the average female brain 19.3 billion. There was no density difference between the sexes and the neuron difference was as predicted by Rushton. Various papers (especially from Lynn, Irwing, Nyborg, and Rushton) have appeared in the past 4 years, mostly showing a 4-6 point IQ advantage for males.²⁰ Some of these have mentioned that one reason that the difference at the mean was not more apparent was that test designers intentionally structured IQ tests to minimize sex differences in scores.

Brain size differences also account for much of the differences between population groups. The differences between the three major racial groups has been well documented. Rushton (1997) shows brain size at intervals from birth to adulthood for Asians, Whites, and Blacks:

	<u>Brain volume, cm³</u>		
	<u>Birth</u>	<u>7-years</u>	<u>adult</u>
Asian	335	1157	1391
White	332	1154	1378
Black	315	1134	1362

Similar numbers have been widely reported in papers dealing with intelligence differences between population groups. Lynn (2006) reports very large numbers of such data, for many population groups, showing that in virtually all cases the brain size numbers vary with mean IQ. He reported a heritability of brain size of 0.90 (also see Baare, et al., 2001).

Glucose Metabolic Rate

One of the early findings from brain imaging involved the use of PET²¹ scans that show glucose uptake rates in the brain. Glucose, containing a radioactive isotope, is injected into the person being studied. Haier (1993) describes the methodology in detail. He took PET images while subjects were solving problems of a given level of difficulty and found that glucose metabolism correlated with RAPM²² in the range of -.7 to -.8. Thus, more intelligent people used less brain energy than did less intelligent people. Intelligence relates to the efficiency of neural activity. “The method of correlated vectors shows that *g* is specifically related to the total brain’s glucose metabolic rate (GMR) while engaged in a mental activity over a period of time.” Jensen (1998).

Haier et al. (1992) also found that GMR decreased markedly as subjects learned a computer game (Tetris). This is a direct observation of the transfer of a cognitively demanding task from *g* resources to a more automatized function. When subjects were tested by Larson, using a variety of tasks of different difficulty, he found that brighter people were able to increase GMR more than less intelligent people, thereby bringing more mental processing to bear on the task at hand.

These GMR studies were not designed to test heritability, but are important demonstrations of the biological differences in intelligence. Biological differences are most parsimoniously explained by genetics, although environmental constructions can be imagined that would explain the observations, if those observations were not supported by many other observations that all point in the same direction (genetic causes).

Other Biological Factors

A variety of physical traits have been shown to correlate with intelligence to various degrees. In fact, there are so many correlates to intelligence that it is difficult to construct a study of any reasonable behavior or physical condition that does not show some correlation. Most of those are of little interest and many can be traced to aspects of physical appearance (leg length, light eye color, weight, etc.) that influence assortative mating.

The finding that pH is correlated with *g* is apparently related to the influence of hydrogen ions on the excitability of adjacent nerves. The reported correlations (Rae, et al., 1996) are at and above 0.50 and are, therefore, significant. Not much research has been done on this subject,²³ but it is another significant biological vector that again shows low level biological factors as the root cause of the variance in intelligence and the probable components of *g*.

Numerous correlations of biological variables suggest that intelligence varies with robustness or varies negatively with physiological faults. Vital capacity, handgrip strength, general health,²⁴ and longevity all correlate positively with *g* (Jensen, 1998; Whaley and Deary, 2001; Rushton, 2004; Gottfredson and Deary, 2004). Referring back to the discussion on brain size, it is worth noting that Rushton (2004) found a correlation between brain size and longevity of .70 for his study group of 234 mammalian species. The human differences in longevity persist, in spite of much greater access to health care, nutrition, etc.

Bates (2007) and Prokosch, et al. (2005) have demonstrated that fluctuating asymmetry (FA)²⁵ correlates negatively with intelligence, supporting the notion of a general fitness factor that underlies intelligence. Prokosch, et al. (2005) also found that the FA correlation is stronger for tests that are more *g* loaded. The FA finding has been extrapolated by several other researchers as an indication of correlation between physical beauty and intelligence (along with selective mating).

Evidence of the Heritability of Intelligence

The following discussions deal with observations beyond the biological traits that have been discussed so far.

Regression to the Mean

For any population group, IQ can be statistically predicted as falling half way between the mean IQ for that population group and the mean for the two parents. This regression point becomes the mean for a Gaussian distribution of the children's IQs. Thus, parents with IQs below the mean for their population group will have an increased probability of producing children who are above the mean IQ of the parents and vice versa. This effect causes children to regress (statistically, on a group basis) differently for parents of matched IQs, when those parents are from population groups that differ at the mean. For

example, Black parents of IQ 110 will have children with IQs distributed around a mean of 97.5 and white parents with the same 110 IQ will have children (assuming a mean of 100 for Whites) with a mean of 105. This phenomenon has been documented and accounts for the lower IQs of Black children when compared to White children with equally intelligent parents (Rushton and Jensen, 2005). One way this can be demonstrated is by comparing Black and White children of equal IQ to their siblings, thereby eliminating environmental variables. Jensen (1973) did such a comparison, where pairs of Black and White children were matched for equal IQs:

<u>mean</u>	<u>White sibling mean</u>	<u>Black sibling</u>
Blacks and Whites paired with IQs of 120	110	100
Blacks and Whites paired with IQs of 70	85	78

MZA Studies

Abbreviations used in twin studies:

MZ	monozygotic
DZ	dizygotic
A	reared apart
T	reared together

One of the most cited means of determining heritability is to compare the IQs of identical twins separated at birth. MZAs have essentially identical genes and do not have a shared environment.²⁶ The intelligence correlation between MZAs is a direct estimate of the heritability of intelligence. This correlation is directly converted to h^2 and is not squared first.²⁷ That is, $r_{MZA} = h^2$.

There have been various studies of twins that included MZAs:

Newman, Freeman and Holzinger, 1937
 Burt, 1955, 1958, 1966
 Shields 1962
 Juel-Nielson, 1965
 Minnesota twin study (Bouchard et al. ongoing)

Of these, the Minnesota twin study (Bouchard, 1990, 1993) is the largest and has been valuable in the study of not only intelligence, but many other human traits. The studies by Cyril Burt were the subject of some scandal and suspicion after Burt's death. Burt found a heritability of 0.771 for 53 pairs of identical twins, reared apart. Some claims were made to the effect that Burt had tampered with the data; this caused researchers to stop citing his work. Kamin claimed that Burt's finding was too high and differed too much from a small study Burt reported many years earlier. Today, Burt's heritability number has to be regarded as either dead on or slightly too low. After a considerable amount of examination by various parties, Burt has been exonerated, thanks largely to Nick Mackintosh (1995) and J. P. Rushton (2002).

The remarkable thing about MZA studies is that they have been consistent, showing heritabilities no lower than .70. Lynn (2006) gives the weighted average of the five studies as 0.75. When corrected for attenuation, this becomes $h^2 = 0.83$. Lynn also mentioned a study of identical twins in Russia. The h^2 measured was 0.87, after correction for unreliability.

Inbreeding Depression

One of the strongest demonstrations that IQ is heritable comes from the study of the intelligence of children from close relatives. The mechanism that works in this special case is entirely genetic and is observed in all sexually reproducing organisms. Eckert (1994) defines inbreeding as *the reduction in fitness of offspring derived from mating between relatives (inbreeding) compared to offspring resulting from mating among unrelated individuals (outcrossing)*. [This reference is entirely about plants and is used here to demonstrate the general applicability of inbreeding depression effects in very different organisms. The mechanism of inbreeding depression is discussed in Jensen (1998) and a wide range of other easily located texts.]

The degree to which intelligence is depressed by inbreeding is a function of the closeness of the relation. Parent-child and brother-sister cases are relatively rare, but have been observed enough to establish that there is a high probability of depressed intelligence. Marriage between first cousins, however, is more common and is practiced enough within some societies that statistically significant measurements have been reported. First cousin matings typically show $.05d^{28}$ to $.10d$ trait effect magnitudes, but for unknown reasons the average trait size is $.18d$ for reaction times (Jensen, 1998). At least 14 studies of the effects of inbreeding depression on intelligence have been published, all showing depression of measures of intelligence and scholastic achievement.

Lower intelligence, due to inbreeding depression has been documented for top SES groups in Muslim populations in the Middle East and India. This finding demonstrates that the lowered IQs were not due to low SES, but rather to inbreeding (Jensen, 1998).

Rushton (1989) studied a group of 1,894 Japanese cousin marriages (data collected and initially analyzed by Schull & Neel, 1965). Using the 11 subtests of the WISC²⁹, he determined that the inbreeding depression of intelligence among those Japanese, correlated at $r = .48$ with the Black-White IQ gap, as measured by the same 11 subtests. Jensen (1998) used the WISC-R standardization sample to demonstrate a high g loading associated with inbreeding depression and found that as the magnitude of g loading increases, the more test scores are depressed from inbreeding.

Brain Imaging

Thompson et al. (2001) reported a heritability of 80% or more based on brain imaging³⁰ of MZ and DZ twins. They found that frontal gray matter volume correlated significantly with IQ (.37–.45). Although the study was small (10 MZ and 10 DZ pairs), it was replicated by Posthuma, D., et al. (2002). This direct observation is a very powerful tool for the demonstration of the role of heredity in brain formation and the determination of intelligence.

Population Group Differences

For any population group (breeding group, race, etc.) there will be a mean IQ that is associated with that group. The most studied of group differences are associated with people living in the United States, Europe, and to a lesser extent, other industrialized locations, such as Japan. However, there is enough data for most groups to identify the probable means for each. These are discussed in detail by Lynn (2006).

Some of the observations that demonstrate the high heritability of intelligence can be performed either within-group or between-group. Regression to the mean has already been discussed and is an example of a phenomenon that can be seen within any group, but which becomes particularly informative when groups are compared, since members of any group will regress to the mean of their group.

Children – Between group differences are the subject of some bitter disagreements that are based, not on science, but on politics and political correctness. Although virtually every population group has a unique mean, the difference between the Black and White means in the US has been particularly subjected to close study and sometimes to gutter-level name calling by those who do not like the findings. The Black mean falls at about 1*d* below the White mean³¹ and about 1.36*d* below the mean for US Whites of European ancestry (Jensen, 1998). The argument that this difference is due to various environmental factors has been refuted by multiple means, but one of the important ones is that the difference is present in 2 ½ year-old children (Rushton & Jensen, 2005). That eliminates all later factors (such as schooling) as causes. Lynn (2006) reported that the low mean (IQ 67) for sub-Saharan Africans is found in preschool children and showed similar results for preschool children from various low mean IQ countries.

Clines and admixture – Clines are hybrids between two pure breeding groups. When cline populations form, the mean for the cline group (assuming a 50/50 admixture) is midway between the means of the two “pure” populations. Most racial mixtures follow a simple proportion between the means of the groups in question.³² In the US, Blacks have a mean IQ of about 85. Lynn (2006) found that there is no difference in the environmental components of Blacks and Whites in the US and that Black IQs gain about 0.2 IQ points per percent of European admixture. Zero European admixture (pure Black genetic makeup) would mean an IQ of 80. The mean European admixture in US blacks is about

25%. This relationship was confirmed by comparing the IQs of low admixture Blacks in Georgia and South Carolina. If the primary factor determining IQ was not genetic, the admixture observations would not be found.

Evolutionary paths – It is beyond the scope of this discussion to outline the complex paths that are projected by people who have specialized in the study of human evolution. Most, however, point to a migration from Africa that ultimately led to population groups being established at points throughout the world. Among the findings of traits between these groups is that they correlate with latitude. Skin color, brain volume, morphology, and intelligence each correlate, although there is no reason to connect cause and effect between all of these traits; rather, a common cause is more likely with some of them. For example, skin color correlates strongly with intelligence, but has no direct causative path. The cause of the variance between population groups in skin color is presumably related to latitude, which is presumably responsible for at least a significant amount of the total variances in cranial capacity and intelligence. All of these traits are ultimately genetic and are the consequence of evolutionary pressures.

Correlations

Absolute distance from the equator and cranial capacity (Beals, Smith, & Dodd, 1984):

$$r = +.62 \text{ (} p < 10 \text{)}$$

$$\text{cranial capacity} = 2.5 \text{ cm}^3 \times \text{degrees latitude} + 1257.3 \text{ cm}^3$$

Templer and Arikawa (2006):

skin color to winter high temperature	$r = .85$	$(p < 0.001)$
IQ to skin color ³³	$r = -.92$	$(p < 0.001)$

Various combinations of these variables can be found in the cited papers. The underlying hypothesis is that cold creates a host of survival challenges and has eliminated groups that were unable to meet the challenges (planning, difficult hunting, robust heating and shelter, etc.). This same idea has been used by Lynn to explain the somewhat higher intelligence of Mongoloids, who were particularly stressed by the Ice Age (see Lynn, 2006 and his various earlier papers on the subject).

Chronometric measurements – As previously discussed, chronometric measurements correlate with *g* so well that they can be used to measure *g* with about the same accuracy as by using IQ tests. The relations between speed and intelligence apply to all population groups and are a sound way to verify that IQ measurements from standard tests are correct for distinctly different groups. Lynn (2006) gives the following IQ and RT data for Africans and Europeans:

<u>Test</u>	<u>African Illiterates</u>	<u>African Students</u>	<u>Europeans</u>
IQ	68	82	105
RT-S	420 ms	400 ms	350 ms
RT-C	1950 ms	1650 ms	1220 ms
EEG	534 ms	526 ms	506 ms

Notes: S = simple RT; C = choice RT; EEG = electroencephalography evoked potential latency

Lynn also showed RT data for Japanese and Chinese children (age 9). Compared to European children, the Japanese were .50*d* faster and the Chinese were .96*d* faster.

Test *g* loading – The *g* loading of a test is an indication of the degree to which the test is measuring the general factor (the source of almost all of the test validity over the range of +/- 2*d* or so). Every indication is that *g* is a reflection of biological factors (as previously discussed) and that it is very highly heritable. When different population groups are compared, the usual finding is that the differences between their means increases as the test *g* loading increases. This indicates that the differences between population groups are biologically based and not due to environmental factors. Similarly, the between-group differences in intelligence are most pronounced on the more heritable components of the tests, leading to the same conclusion. (Rushton & Jensen, 2005).

Environmental Factors

Although the heritability of IQ is very high, the genotype and phenotype are not equal. This means that part of the variance is caused by environmental factors. Environmental factors consist of those that are from the shared environment (family, school, job, social, etc.) and those that are nonshared (mostly exposures to chemicals, diseases, etc.). Ergo, the phenotype consists of genetic, shared, and nonshared components.

Shared Environment

In early childhood, the shared environment accounts for 30%³⁴ of the variance in intelligence. Thus, adopted children show an IQ correlation with their adopted siblings that is due to the shared family environment. This led various researchers³⁵ to devise studies to prove that the IQs of minority groups would increase when the children were adopted in infancy and reared by the higher scoring groups.³⁶ Those studies quickly detected the shared environmental factor and reported IQ boosts for the adopted children. The researchers wrote glowing reports of their success in demonstrating the strong influence of the family environment. Fortunately, the researchers followed appropriate scientific protocols and did follow-up testing. Scarr and Weinberg (and others, but the Minnesota Transracial Adoption Study is the most cited) discovered that the IQ gains they had found in the adopted children at age 7 had vanished by age 17 (Weinberg, et al. 1992). Plomin (1986) reported virtually identical findings from an independent adoption study. It is now well established that the effects of the shared environment evaporate by about age 12.³⁷ The family (and other social) environment has only a brief effect on the variance in intelligence, leaving adopted siblings with no correlation after childhood. This means that all environmental factors in adults are from the nonshared environment and that the only correlation between related adults is genetic (Jensen, 1998).

The Minnesota Transracial Adoption Study considered various combinations of Black and White adoptions. The adopted children matured to IQ levels that would be predicted

from their biological parents. Black children adopted by White parents matured to be less intelligent than their adopted parents. But there were three independent studies of Asian children³⁸ adopted by White parents in the United States and Belgium. These adopted children developed IQs (by school age) that exceeded that of the mean for White children. The gap was on the order of ten IQ points, even though many of the adopted children were suffering from malnutrition before they were adopted (Jensen, 1998). Lynn (2006) lists six studies of children from China, South Korea, and Taiwan, who were adopted by families in Europe and the U.S. In all cases the children had higher IQs than those of the environment in which they were reared.

Scarr and Weinberg (1978) concluded (this was an all-White adoption study) that within the range of humane environments, variations in family SES and child rearing have little or no effect on IQ measured in adolescence. “By adulthood, all of the IQ correlation between biologically related persons is genetic. In other words, to the extent that there is a correlation between the IQs of genetically related postpubertal family members, the correlation is entirely due to genetic factors; the environmental contribution to the familial correlations is nil.” (Jensen, 1998, P. 178)

Another way to demonstrate the vanishing nature of the shared environment is to look at non-twin siblings. When reared together, their IQ correlation is 0.49 in adulthood. When reared apart, their IQs correlate at 0.24 as children, but this rises to 0.49 in adulthood. Unrelated children, reared together (adopted) correlate at 0.25 in childhood and 0.01 in adulthood. (Rushton and Jensen, 2005)

SES³⁹ is frequently mentioned as an explanation for various intelligence related findings. But, it is not SES (taken as an environmental variable) that drives intelligence differences, it is the opposite. As adults, siblings (reared in the same environment and at the same SES) statistically reach SES levels that are predicted by their *g*. This is, in part, due to the highly predictive relationship between IQ and vocation. When individuals are sorted by IQ, they will usually show a range of SES that reflects their intelligence, but if their genetic variability is accounted for, there is no remaining SES to IQ correlation. The finding that poor people have lower IQs than rich people does not stem from the environmental effects of high SES, but rather is a reflection of high intelligence producing higher SES and low intelligence producing low SES (Jensen, 1998).

Nonshared Environment

The variance associated with the nonshared environment also declines in childhood, to about age 12, then becomes relatively stable through age 20 and then declines slightly. From age 16 onward, the magnitude of the nonshared environment is about 18%. The components of the nonshared environment are largely chemical and biological exposures, such as the ingestion of toxins and the contraction of diseases. Of these, the intrauterine environment is one of the most important. Alcohol, tobacco, prescription and illicit drugs all create a prenatal environment that has the potential to degrade intelligence.

Low birth weight, in the range of 2,000 grams, begins to show a depression of IQ that becomes considerably more severe when the weight is at 1,500 grams or less (Storfer, 1990). Data from the National Longitudinal Study of Youth show that the mother's IQ is the only significant factor in accounting for low birth weight babies. Factors such as the mother's age and SES were insignificant. Herrnstein and Murray (1994) show that the frequency of low birth weight births is highest among the group they label as "dull" and is high for "very dull." As the mother's intelligence increases, the frequency drops for "normal" and "bright" mothers, then jumps to almost the frequency of "very dull" for mothers in the "very bright" category. The authors explain that this unexpected finding may be an anomaly associated with the small sample size in the "very bright" range.

Nutrition, especially as it applies to certain items, such as iodine,⁴⁰ iron, and folate (Arija, 2006), is a source of IQ depression. It is not found in enough instances in the industrialized world to be a significant factor, but in nations where extreme poverty is common, intelligence is depressed. Lynn (2006) estimated that most of the IQ depression below a mean of 80 in sub-Saharan Africa (mean IQ = 67) is due to nutrition.

Environmental factors do not boost IQ, they depress it. At one time, it was believed that breast feeding was a positive environmental factor, but it was ultimately shown that brighter mothers were more likely to breast feed their children than duller mothers. One standard deviation advantage in maternal IQ more than doubled the odds of breast feeding (Der, 2006). The effect was genetic, and not caused by the milk.

Measuring Heritability

Fortunately, there are several diverse means of quantifying heritability. These different approaches yield estimates that fall within a narrow range. Heritability increases throughout childhood and early adulthood, due to the evaporation of the shared environmental component and a reduction in the nonshared component.

Identical Twins Reared Apart (MZA)

This method of determining h^2 has already been discussed in detail. The method is completely straightforward, since it takes the correlation between genetically identical individuals, with no common environmental factors (other than prenatal) and takes the correlation as a direct measure of heritability. As previously noted, Lynn's average $h^2 = 0.75$. Corrected for attenuation, this becomes $h^2 = 0.83$.

Falconer's Formula

This method (Falconer and MacKay, 1996) estimates h^2 by doubling the difference between the correlations of same-sex MZT ($r = .88$) and DZT ($r = .51$) twins. Correcting these numbers for test reliability (as before, assumed to be 0.90), the numbers become .98 and .56, respectively. The difference is 0.42, so the computed h^2 is 0.84 (Lynn, 2006). Lynn also reported two studies of heritability in India, both using Falconer's Formula.

One study yielded h^2 of 0.81 and the other 0.90. After correction for reliability, these become 0.90 and 1.00, respectively.

1.0 Minus the Environmental Component

Adopted, unrelated children reared together have the same shared environment. A correlation of their IQs is an estimate of the adopted family environmental effect. Lynn (2006) uses is 0.04,⁴¹ which would yield a h^2 of 0.96, but the environmental number is somewhat too small, since it does not account for the nonshared environment. It is also questionable in light of various sources that show correlations closer to zero. For example Jensen (1998) states: “The IQs of unrelated persons who were reared together correlate +.25 in childhood and –.01 in adulthood.” If the tiny negative correlation is taken to be zero, this method should consist of subtracting an estimate of the nonshared environment from 1.0. Posthuma (2002) claimed the nonshared environmental component is 15%, although this may be a bit of circular reasoning, since that number was computed from Dutch twin studies (by subtracting h^2 from 1.0).

Path Analysis

This technique was invented in the 1920s by Sewall Wright. The method incorporates multiple linear regression to apportion the contributions of each of the multiple causal variables to the variance in the single outcome. The assumed links between the causal variables can be tested and rejected if they do not fit the assumed causation. This is not a test of causation, but provides a means of determining magnitude and of establishing the existence or nonexistence of the assumed causality link. The method is general and has been used to study diseases, occupations, etc. See Jensen (1980), Li (1975) and Loehlin, J. (1991).

Relatively few path analysis studies have been reported in the literature. One that used this method was based on the Texas Adoption Project (300 adoptees). The analysis used the IQs of mother, father, their natural children, and their adopted children (after about 17 years of adoption). The h^2 derived from this study was 0.78 (Horn, et al., 1978).

Summary and Final Comments

Heritability in early childhood is at its lowest point—about 40-45%. At that time, the shared environment contributes significantly to observed intelligence, but this environmental component declines to zero or near zero sometime in the age range of 12 to 20. Heritability continues to increase from somewhere around 70% in early adulthood to over 80%.⁴²

Genes have the first and last say in determining intelligence. First, they set the biological factors that determine intelligence. Phenotypic intelligence is a small (statistically) reduction in the intelligence that would emerge, absent environmental factors. For whatever reason, the environmental factors ultimately decline to yield adult intelligence that is heritable at the rates shown under “Measuring Heritability.”

Researchers have attempted to locate the intelligence genes and have made some progress. The researcher to watch is Robert Plomin, who is by far the most active in this narrow field. Plomin has concluded that there are a hundred or more genes that each account for only a small amount of the total variance. This makes his job especially difficult, in the sense of looking for a needle in a haystack. Plomin (2006) contains a good discussion of the available genetic research tools and the nature of the task of trying to identify the small-effect intelligence genes.

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- ¹ “Though over the past 35 years we have poured billions of dollars into inner-city schools, and though we have fiddled with practically everything you could think to fiddle with, we have done almost nothing to raise the trajectory of ghetto children.” Traub, James (January 16, 2000). What No School Can Do, New York Times Magazine.
- ² The method that is briefly described is known as a hierarchical factor analysis and was invented by Charles Spearman, who used it to discover *g*. Various factor analysis procedures have been defined and used for similar purposes. When applied to intelligence test items, all methods should yield a single common factor. When applied to other sets of data, such as personality test items, a single factor does not necessarily emerge.
- ³ For a discussion of the three variables rule, see P. 141 of Jensen (2006).
- ⁴ A number of recent papers have presented both hierarchical and bi-factor factor analyses. The bi-factor (or nested) model extracts *g* from the correlation matrix as the first principal factor. Group factors are then extracted from the variance remaining in the matrix. (Jensen 2006)
- ⁵ The “faster brain” can be thought of as displaying a higher speed of neural activity, which equates to faster NCV.
- ⁶ ECTs are described in some detail in Jensen (1998) and in more detail in Jensen (2006).
- ⁷ Specificity = $s = \sqrt{1 - g^2 - e^2}$; where *g* is psychometric *g* and *e* is random error.
- ⁸ The procedures for IT and RT measurements are explained in detail in Jensen (1998) and in more detail in Jensen (2006).
- ⁹ In the case of visual inspection time measurements, the figure used is two vertical lines, joined by a horizontal line at the top. The figure resembles the Greek letter pi. When the figure is projected (using a tachistoscope), one of the two vertical legs is shorter than the other. The testee is asked to identify which side is the longer one (he presses a button).
- ¹⁰ The time from stimulus to the third recorded trough.
- ¹¹ WMC = working memory capacity
- ¹² One of the early brain volume researchers was Sir Francis Galton.
- ¹³ The standard method for measuring cranial volume is to fill the skull with lead shot or seed, which is then transferred to a container to measure the volume.
- ¹⁴ Various methods have been used to calculate head size. A simple circumference measurement is one option, but most measurements are based on measurements of circumference, length, breadth, and height of the head. The correlation between head size and brain volume falls in the range of 0.50 to 0.60 in adults (Jensen, 1998).
- ¹⁵ When corrected for IQ test measurement reliability (0.90), the value rises slightly to $r = 0.44$ (Lynn, 2006).
- ¹⁶ The method of correlated vectors was developed by Arthur Jensen and is one of his great contributions to intelligence research methodology. It is explained in Jensen (1998).
- ¹⁷ The regions associated with cognition are distributed across the entire brain. Jung and Haier (2006) list 14 Brodmann Areas (BAs) associated with various aspects of cognition. In the same paper, there is a figure delineating 32 BAs from a survey of various imaging studies.
- ¹⁸ From a direct question the author asked of Haier, during a conversation. (December 2005)
- ¹⁹ Most of the cited studies found heritabilities at or above 0.90.
- ²⁰ The male IQ advantage does not appear prior to about age 16. This has been attributed to the faster maturation of females.
- ²¹ Positron emission tomography. PET scans provide images of the specific locations and amounts of neural metabolism that occurred during an immediately preceding period of mental activity.
- ²² Raven Advanced Progressive Matrices
- ²³ Anderson, et al. (1998) attempted to verify the Rae findings and failed. The subjects being examined by the two researchers were different in many aspects. This difference probably accounts for the different findings.
- ²⁴ The correlation between health and *g* when socioeconomic status is partialled out is $+0.326$ (Jensen, 1998).
- ²⁵ FA is an index of left to right body symmetry. Larger FA means less symmetry. Measurements are made in places where soft tissue is minimal and includes finger length and the length of bones at the ankles, and wrists.
- ²⁶ Bouchard (1983) demonstrated that criticisms that MZA studies do not represent truly different environments is not valid.
- ²⁷ For a detailed explanation of why the regression coefficient is not squared, see pages 134 and 135 of Jensen (2006).
- ²⁸ *d* is used to indicate units of standard deviations.
- ²⁹ Naturally, the Japanese language version of the WISC was used.
- ³⁰ Anyone who is interested in seeing the brain images can easily locate them on the Internet; just search on the report information given in the References.
- ³¹ The mean in question includes Whites of European, American Indian, Mexican, etc. ancestry. See Jensen (1998) page 17.
- ³² There are few instances of departure from simple, proportionate outcomes from hybrids. Nagoshi & Johnson (1986) reported higher than expected IQs from the children of Asian and European parents in Hawaii. The difference was about 4 IQ points and may be attributable to heterosis.
- ³³ The referenced paper was based on average skin colors of large breeding groups, not on individual variation.
- ³⁴ Some sources may give a slightly higher number.
- ³⁵ There is a large amount of material in the literature that deals with various adoption and intervention programs. These may be easily identified by consulting detailed textbooks on the subject of intelligence. Jensen (1998) is the most cited

reference in the field of psychometrics and has many references to such studies.

³⁶ The studies being referenced involved Black children adopted by White families.

³⁷ 12 is the most frequently referenced age, but some studies have put the age at which the shared environmental factor disappears as high as 20.

³⁸ From Cambodia, Korea, Thailand, and Vietnam.

³⁹ Socioeconomic Status

⁴⁰ A meta-analysis of 18 studies by Bleichrodt and Born led them to conclude that the effect of severe iodine deficiency could reduce intelligence by 13.5 IQ points.

⁴¹ See page 27 of the referenced text for Lynn's discussion for how this number was selected.

⁴² As a point of comparison, the heritability of height in present day industrialized nations is about 90%.