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The evolution of human intelligence and the coefficient of additive genetic variance in human brain size

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Abstract

Most theories of human mental evolution assume that selection favored higher intelligence and larger brains, which should have reduced genetic variance in both. However, adult human intelligence remains highly heritable, and is genetically correlated with brain size. This conflict might be resolved by estimating the coefficient of additive genetic variance (CVA) in human brain size, since CVAs are widely used in evolutionary genetics as indexes of recent selection. Here we calculate for the first time that this CVA is about 7.8, based on data from 19 recent MRI studies of adult human brain size *in vivo*: 11 studies on brain size means and standard deviations, and 8 studies on brain size heritabilities. This CVA appears lower than that for any other human organ volume or life-history trait, suggesting that the brain has been under strong stabilizing (average-is-better) selection. This result is hard to reconcile with most current theories of human mental evolution, which emphasize directional (more-is-better) selection for higher intelligence and larger brains. Either these theories are all wrong, or CVAs are not as evolutionarily informative as most evolutionary geneticists believe, or, as we suggest, brain size is not a very good index for understanding the evolutionary genetics of human intelligence.

Keywords: Behavior genetics; Brain size; Coefficients of additive genetic variation; Directional selection; Endophenotypes; Evolutionary genetics; Evolutionary psychology; Heritability; Intelligence; Life history traits; Linear vs. volumetric traits; MRI brain imaging; Organ volumes; Reproductive success; Sexual selection; Sexually antagonistic coevolution; Stabilizing selection

1. Introduction

There has been some tension and mutual misunderstanding recently between intelligence research, which focuses on the factor-analytic structure of individual differences in mental abilities (e.g. Petrill, 1997; Plomin & Spinath, 2004; Stanovich & West, 2000), and evolutionary psychology, which focuses on the adaptive design features of species-typical mental abilities (e.g. Cosmides & Tooby, 2002; Kanazawa, 2004). Can these be reconciled? In the Modern Synthesis of the 1930s, biologists such as Ronald Fisher, Sewall Wright, J. B. S. Haldane, and Ernst Mayr developed evolutionary genetics to reconcile Mendelian genetics (an individual-differences science) with Darwinian evolution (a science of species-typical adaptations) (see Mayr, 1993). In this paper we suggest that some recent advances in evolutionary genetics might also mediate a constructive reconciliation between intelligence research and evolutionary psychology.

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These recent advances in evolutionary genetics have been spurred partly by the revival of Darwinian sexual selection research in recent decades (Andersson, 1994; Cronin, 1991; Kokko, Brooks, Jennions, & Morley, 2003). This research is important not just because mate choice may have shaped human mental evolution (Darwin, 1871; Miller, 2000a), but because it has sparked important new insights into the factors that maintain genetic variance in complex traits (Keller & Miller, in press; Pomiankowski & Møller, 1995; Rowe & Houle, 1996) — such as human brain size and intelligence. These insights have challenged the traditional view that persistent genetic variance in a trait is *prima facie* evidence that it has not been under selection, and has not been evolutionarily important (e.g. Diamond, 1999; Gould, 1991; Kanazawa, 2004; Tooby & Cosmides, 1990, 2005). Throughout most of the 20th century, this view seemed a reasonable corollary of Fisher (1930)'s 'Fundamental Theorem of Natural Selection': Selection should drive advantageous alleles to fixation (100% prevalence), and thereby reduce genetic variance in fitness-related traits. However, this selection-eliminates-variance view never sat comfortably with behavior genetics, which showed that almost all human mental traits remained heritable in modern populations (Turkheimer, 2000) — including traits such as intelligence that seemed most likely to have been under positive selection (Plomin, 1999).

2. What are coefficients of additive genetic variance, and why do they matter?

One of the key insights from recent sexual selection research is that a trait's heritability is often less evolutionarily informative than its 'coefficient of additive genetic variance' (CVA) (Houle, 1992, 1998). A CVA is a mean-standardized index of genetic variance in a trait (Lande, 1977). It is a dimensionless quantity, computed simply as a trait's coefficient of phenotypic variation (CVP) times the square root of its narrow-sense heritability (additive genetic heritability). A CVP in turn is a trait's standard deviation standardized by its mean, times 100 (as a convenient scaling factor). Thus,

$$\text{trait CVA} = (\text{trait SD}) / (\text{trait mean}) * 100 * \sqrt{(\text{trait narrow} - \text{sense heritability})} \quad (1)$$

This is easy to calculate for morphological traits such as height or weight, which can be measured on true ratio scales.

A trait's CVA reflects the amount of genetic variance that currently exists in the trait — not relative to the environmental variance that affects the trait (as in a heritability estimate), but relative to the trait's current average value. Thus, unlike heritabilities, CVAs are robust to environmental variation effects across time, space, and samples. Different populations may have different CVAs because they have different relative amounts of genetic variance in a trait, but they will not show different CVAs just because they have different amounts of environmental variance. (Theoretically, such environmental variance differences should affect both trait SD and trait heritability such that their effects cancel out.)

Although behavior genetics traditionally focuses on heritabilities, CVAs can be more evolutionarily informative in two key respects. Animal and plant breeders have understood for decades that CVAs reflect 'evolvability' – artificial selection's ability to drive further increases in a domesticated species' productivity with regard to a trait (Lynch & Walsh, 1997). More recently, evolutionary theorists have realized that high CVAs are typical of fitness-related traits, especially those under directional (more-is-better) selection, and those that depend on very many genes that are vulnerable to harmful mutations (Houle, 1992, 1998; Pomiankowski & Møller, 1995; Rowe & Houle, 1996). CVAs can be high in traits with low heritabilities if there is high residual error variance (or phenotypic plasticity) in trait development, as is expected for most fitness-related traits such as mating success, fertility, and longevity (Houle, 1992).

To calculate a trait's CVA, we need to know three things: its mean (on a true ratio scale), its standard deviation (on the same scale), and its (narrow-sense, additive genetic) heritability. Although there is overwhelming evidence for human intelligence being highly heritable (McClearn et al., 1997; Plomin & Spinath, 2004), intelligence cannot yet be measured on a true ratio scale with a lower boundary of zero (Jensen, 1998). As all intelligence researchers know, the distribution of adult human IQ has a mean of 100 and a standard deviation of 15 only by historical convention (Plomin, 1999), so a 150-IQ person is not twice as bright as an IQ-75 person in any straightforward sense. Thus, the CVA for human intelligence cannot be estimated directly. However, we can estimate the CVA for any intelligence-related (*g*-loaded) trait that can be measured on a true ratio scale.

Brain size in cubic centimeters is one such ratio-scale trait known to be moderately correlated with intelligence. A 1200-cc brain (typical of humans) really is three times the volume of a 400-cc brain (typical of chimpanzees), and seems to support higher intelligence. In this paper, we

estimate for the first time the CVA of human brain size, by combining data from all 28 published studies we could find that used *in vivo* magnetic resonance imaging (MRI) to estimate brain size means, standard deviations, heritabilities, and/or correlations with intelligence. We also compare the brain's CVA to the CVAs of other human organ volumes and life-history traits.

Our goal was to see if the CVA for human brain size is consistent with current models of human mental evolution. All such models posit directional selection over recent evolutionary time for higher intelligence and larger brain size in humans (e.g. Dunbar, 2003; Flinn, Geary, & Ward, 2005; Gottfredson, in press; Kanazawa, 2004; Miller, 2000a; Robson & Kaplan, 2003; Richerson & Boyd, 2004; Rushton, 2004; Suddendorf & Whiten, 2001). All of these models are based on cost/benefit reasoning derived from behavioral ecology (Alcock, 2005), and all seek to identify fitness payoffs for larger brains that would out-weigh their energetic costs and obstetric risks. All suppose that brain development depends on very many genes, and thus should be vulnerable to many possible harmful mutations. Thus, if evolutionary genetics is right that high CVAs suggest a history of recent, directional selection on highly polygenic, mutation-vulnerable traits, then the human brain should show a rather high CVA — perhaps much higher than the CVAs of other organ volumes.

3. Does brain size correlate with intelligence?

To find relevant studies for all analyses reported in this paper, we performed searches in SciSearch, MedLine, and PsycInfo, covering the years 1950 through 2005, using keywords such as "brain size", "intelligence", "heritability", "genetic variation", and other related terms and synonyms. We read the online abstracts, located and read the relevant-looking papers, and recorded their relevant data if their samples and methods fit our selection criteria. We also followed their citations forwards and backwards to other relevant papers.

Table 1 reviews all relevant studies we could find that (1) used *in vivo* MRI to estimate total brain volume (excluding cerebro-spinal fluid and parenchyma), (2) used a reliable, valid measure of general intelligence, and (3) included at least 15 healthy normal adults over the age of 18, recruited from a general community sample with minimal IQ range restriction. All samples were from homogenous ethnic groups, mostly of white European descent, plus one sample from Chile and one from Turkey. These 15 studies show an n-weighted mean correlation of +.416 (and an unweighted mean correlation of +.431) between intelligence and whole brain size, in a total sample of 935 normal, healthy adults.

Table 1
Correlations (*r*) between general intelligence and whole brain size in normal human adults, from 15 MRI studies

r	IQ measure	Sample	Source
.35	WAIS	40 US students	Willerman (1991)
.38	WAIS	67 US adults	Andreasen (1993)
.43	CFIT	29 US adults	Raz (1993)
.40	MAB	39 Canadian women	Wickett (1994)
.69	NART	34 UK adults	Harvey (1994)
.48	WAIS-R	40 UK adults	Egan (1994, 1995)
.25	WAIS-R	90 US adults	Flashman (1997)
.38	WAIS-R	62 US adults	Paradiso (1997)
.40	CFIT	103 Turkish students	Tan (1999)
.41	WAIS-R, CVLT	80 US adults	Gur (1999)
.51	MAB, ZVT	68 Canadian brothers	Wickett (2000)
.42	WAIS-R	96 US adults	Pennington (2000)
.45	PC1	72 US women	Schoenemann (2000)
.44	WAIS-R	96 Chilean students	Ivanovic (2004)
.48	RAPM	19 US men	Thoma (2005)

Note. To save space, sources are cited by first author only, without any et al.'s
.416 *n*-weighted mean *r* from 15 studies (total $n=935$).
.431 unweighted mean *r*.

A recent meta-analysis found a similar average correlation of +.33 between MRI-measured brain size and intelligence (McDaniel, 2005). Higher correlations (around +.6) were found in the first study of postmortem fresh brain volume in relation to prospectively measured intelligence (Witelson, Beresh, & Kigar, in press). These correlations seem likely to hold within families, and not just between families. Although Schoenemann et al. (2000) found a zero within-family correlation between intelligence and brain size in 36 young adult twin pairs, Wickett, Vernon, and Lee (1997) found a within-family correlation of +.25 in 34 adult male siblings. Recent work (Pennington et al., 2000; Posthuma et al., 2002, 2003) also shows a substantial positive genetic correlation between intelligence and brain size, confirming within-family effects.

Thus, brain size seems a reasonable ratio-scale marker, or 'endophenotype' (Boomsma, Anokhin, & De Geus, 1997), for studying the evolutionary genetics of human intelligence. Further, comparative biologists have found brain size to be an accurate marker of cross-species intelligence differences (Reader & Laland, 2002), and paleontologists have routinely argued that larger hominid brain sizes reveal increased cognitive abilities over evolutionary time (e.g. Falk et al., 2005).

4. The CVP for adult human brain size

As mentioned above, the coefficient of phenotypic variation (CVP) is calculated as:

$$\text{trait CVP} = [(\text{trait SD})/(\text{trait mean})]*100 \quad (2)$$

Table 2 reviews all 11 studies we could find that report means (in cubic centimeters) and standard deviations in each sex separately for whole brain sizes, measured by *in vivo* MRI brain imaging, in at least 15 normal, healthy adults drawn from reasonably representative community samples. Most measures are from control groups of normal individuals in MRI studies of psychopathology. From these reported means and SDs, we calculated CVPs for adult human brain sizes. The *n*-weighted mean brain size CVP across studies is 8.06 for 1614 total males, and 8.50 for 1103 total females. (The unweighted CVPs are very similar, at 8.02 for males and 8.20 for females.) Averaged across both sexes, brain size showed an *n*-weighted mean of 1261.07 cc and SD of 103.76 cc (n=2717). This yields an *n*-weighted mean CVP across both sexes of 8.228.

Table 2
Coefficients of phenotypic variation in whole brain size in normal human adults, from 11 MRI studies

Mean (cc)±SD	CVP (%)	Sample	Source
Males (10 studies)			
1421.31±99.1	6.97	89 US Utah men	Blatter (1995)
1243±110	8.85	69 German men	Peters (1998)
1269±103	8.12	418 older US men	DeCarli (1999)
1438.0±85.3	5.93	25 Scottish men	Warwick (1999)
1352.2±104.9	7.76	40 US men	Gur (1999)
1286.4±133	8.95	79 US San Diego men	Courchesne (2000)
1323.66±97.7	7.38	140 Dutch men	Baaré (2001)
1113.1±92.5	8.31	27 US Boston men	Goldstein (2001)
1273.6±115.0	9.03	23 US Iowa men	Allen (2002)
1343.43±107.4	8.00	704 US Framingham men	Atwood (2004)
n-weighted total means for men			
1316.5±106.1	8.06	1614 total men	
Unweighted total means for men			
1306.4±104.8	8.02	1614 total men	
Females (10 studies)			
1240.0±103.8	8.37	105 US Utah women	Blatter (1995)
1130±112	9.91	48 German women	Peters (1998)
1251.9±67.7	5.41	13 Scottish women	Warwick (1999)
1154.4±85.1	7.37	40 US women	Gur (1999)
1196±77	6.44	72 US women	Schoenemann (2000)
1137.8±109	9.58	37 US San Diego women	Courchesne (2000)
1181.6±108.5	9.18	118 Dutch women	Baaré (2001)
1021.8±89.5	8.76	21 US Boston women	Goldstein (2001)
1131.1±99.5	8.80	23 US Iowa women	Allen (2002)
1181.3±100.8	8.53	626 US Framingham women	Atwood (2004)
n-weighted total means for women			
1180.0±100.3	8.50	1103 total women	
Unweighted total means for women			
1162.6±95.3	8.20	1103 total women	

Note. To save space, sources are cited by first author only, without any et al.'s

Table 3
Estimated heritabilities (h^2) of normal adult human brain size from 8 studies

h^2	Sex	Mean age	Relations	Total *n*	Source
.94	Mixed	27	10 MZ, 9 DZ pairs	28	Bartley (1997)
.91	Males	72	74 MZ, 71 DZ pairs	290	Carmelli (1998)
.97	Mixed	18	25 MZ, 23 DZ pairs	96	Pennington (2000)
.90	Mixed	31	54 MZ, 58 DZ, 34 sib pairs	258	Baaré (2001)
.81	Males	70	45 MZ, 40 DZ pairs	170	Pfefferbaum (2001)
.64	Males	71	72 MZ, 67 DZ pairs	278	Geshwind (2002)
.92	Mixed	35	11 MZ, 11 DZ pairs	44	Hulshoff Pol (2004)
.94	Mixed	61	608 sib pairs, 312 cousin pairs, etc.	1330	Atwood (2004)

Note. To save space, sources are cited by first author only, without any et al.'s
.891=*n*-weighted mean heritability from 8 studies (total n=2,494).
.879=unweighted mean heritability.

5. The heritability of adult human brain size

Table 3 reviews all 8 relevant studies we could find that (1) used *in vivo* MRI to estimate total brain volume, (2) included at least 15 healthy, normal adult pairs of kin (i.e. 30 individuals, most often twins), recruited from a general community sample with minimal brain size range restriction, and (3) used a genetically informative design that reported exact (mostly broad-sense) heritability estimates. These 8 studies show an *n*-weighted mean broad-sense heritability of .891 for whole brain size in a total sample of 2494 normal, healthy adults (the unweighted mean heritability is very similar, at .879). The range of reported brain size heritabilities is .64 to .97, with no apparent sex difference in heritability. The largest study (Atwood et al., 2004) directly estimated a narrow-sense (additive) heritability of .94 in 1330 individuals. (An impressively high estimate given the demographic uniformity of the sample: stroke-free,

dementia-free, mostly college-educated residents of an affluent town near Boston; mean age 61; 75% white non-Hispanic). That narrow-sense heritability estimate of .94 suggests that the broad-sense heritabilities in other studies capture almost entirely additive genetic variance.

This means that the human brain size heritability of .891 is one of the highest heritabilities found for any human trait. It is also substantially higher than the brain size heritability estimates available for other primates, such as the .60 estimated for rhesus macaques (Cheverud et al., 1990) and the .41 estimated for baboons (Mahaney, Williams-Blangero, Blangero, & Leland, 1993). Since the square root of this human brain size heritability (.891) is .944, the CVA for human brain size is nearly identical to its CVP.

6. The CVA for adult human brain size

Multiplying the brain size CVP estimate (8.228, from Section 4) by the square root (.944) of the heritability estimate (.891, from Section 5), we estimate that the coefficient of additive genetic variance in normal adult human brain size is 7.767, or about 7.8 (see Formula (1)).

7. What does the brain's CVA mean?

At first glance, the human brain's CVA of 7.8 seems higher than might be expected for a trait under strong stabilizing (average-is-better) selection, which would favor strict canalization and mutation-resistance during development (driving the CVA down towards 0). For example, Pomiankowski and Møller (1995) surveyed 30 sexual ornaments known to be under directional selection in 24 species (e.g. calling time in the field cricket, badge size in the great tit), and, from their data, we calculated that these ornaments showed a median CVA of 9.9. By contrast, their median CVA for seven sexual traits under stabilizing selection (e.g. pheromone blend in the bollworm moth, number of tibial cilia in the fruit fly) was 2.8, and their median CVA for non-sexual traits (e.g. pupal mass in the flour beetle, wing length in the barn swallow) was 3.6 (Their reported mean CVAs of 20.0, 3.6, and 4.8 for these trait types were inflated by high positive skews in each case of 1.75, 2.04, and 1.13). Thus, the human brain's CVA (7.8) seems closer to the median CVA for sexual ornaments under directional selection (9.9) than to the median CVA for sexual ornaments under stabilizing selection (2.8) or to the median CVA for non-sexual traits (3.6). This seems like good news for the sexual selection theory of human mental evolution (Miller, 2000a), and is concordant with other theories that posit recent directional selection for larger brains and higher intelligence.

However, it has been known since 1935 that, for an organ of a given shape, the relative magnitudes of coefficients of variation of linear, surface, and volume measures should be about 1:2:3 (Lande, 1977). That is, organ volumes should generally show higher CVAs than organ areas or organ diameters. The sexual ornament CVAs reported in Pomiankowski and Møller (1995) were mostly for ornaments measured on a linear scale (e.g. eye-span in the stalk-eyed fly, chest-badge diameter in the great tit, tail length in the barn swallow), and their widely-cited paper did not mention the dimensionality problem with CVAs. Thus, the human brain's CVA of 7.8 may be high for a linear trait but low for a volumetric trait.

To clarify this issue, we reviewed recent, high-quality, large-sample studies from which CVPs, CVAs, and/or CVGs can be calculated for other human traits. (CVAs reflect additive genetic variance, as revealed by narrow-sense heritabilities, whereas CVGs – coefficients of genetic variance – reflect all types of genetic variance (additive, dominance, epistatic), as revealed by broad-sense heritabilities.) Tables 4.1, 4.2 and 4.3 lists these, arranged by dimensionality (linear or volumetric) and trait type—morphological (body organ size) or life-history (achieved survival and reproductive success).

For linear morphological traits (Table 4.1), CVAs and CVGs are sometimes below 5.0 — such as 3.6 for human height in 2 Scandinavian samples, or 4.9 for the axial length of the eyeball. Other CVGs for linear measures are higher though – such as 8.3 for the heart's aortic diameter, or 8.86 and 6.64 respectively for male and female heights in India – probably reflecting strong within-caste assortative mating (Arya et al., 2002). Even for the human eye – the premier example of a complex morphological trait under strong stabilizing selection (for efficient vision) (Darwin, 1859) – the CVAs for other linear measures are often much higher (e.g. 6.9 for central corneal thickness, 9.1 for dilated pupil diameter, 10.1 for anterior chamber depth). This is especially notable because (1) the eye is actually an extended part of the diencephalon, (2) the eye, like the rest of the brain, reaches near-adult size by middle childhood, (3) the eye, like the rest of the brain, is encased in bone, and (4) these CVAs are not much reduced when these eye dimensions are controlled for overall body size.

For volumetric morphological traits (Table 4.2), CVAs and CVGs tend to be much higher, as expected from their higher dimensionality (Lande, 1977). The CVA of human brain volume (7.8) is much lower than the CVAs for total body weight (ranging from 16.1 to 45.4),

Table 4.1
CVPs, CVAs and CVGs for linear morphological traits

Trait (measurement unit, geographical origin of sample)							
N	Sex	Mean±S.D.	CVP	h^2	CVA	CVG	Source
Height (cm, Finland)							
2532	M	176±6.25	3.6	.85	–	3.27	Silventoinen (2003)
3084	F	163±5.58	3.4	.83	–	3.12	Silventoinen (2003)
Height (cm, Denmark)							
598	M	179.7±6.8	3.8	.69	3.14	–	Schousboe (2004)
650	F	166.6±6.2	3.7	.81	3.35	–	Schousboe (2004)
Height (cm, India)							
983	M	143.36±21.2	14.8	.358	–	8.86	Arya (2002)
926	F	140.20±15.6	11.1	.358	–	6.64	Arya (2002)
Head length (cm, from top of nose (nasion) to back of head (inion), India)							
987	M	18.02±0.95	5.3	.413	–	3.41	Arya (2002)
930	F	17.63±0.84	4.8	.413	–	3.08	Arya (2002)
Head breadth (cm, greatest breadth of skull, India)							
985	M	13.83±0.68	4.9	.447	–	3.28	Arya (2002)
925	F	13.68±0.66	4.8	.447	–	3.21	Arya (2002)
Nose breadth (cm, greatest width between lateral borders of nostrils, India)							
983	M	3.37±0.42	12.5	.498	–	8.82	Arya (2002)
927	F	3.13±0.30	9.6	.498	–	6.77	Arya (2002)
Facial height (cm, from chin point (gnathion) to top of nose (nasion), India)							
983	M	9.88±1.02	10.3	.414	–	6.63	Arya (2002)
927	F	9.79±0.87	8.9	.414	–	5.73	Arya (2002)
Eye: axial length (mm, from outer cornea to macula, right eye, ultrasound, Sardinia)							
776	Mix	23.57±1.15	4.9	.46	–	3.31	Biino (2005)
Eye: central corneal thickness (microns, by ultrasound, Britain and Australia)							
512	Mix	544.5±37.3	6.9	.95	6.68	–	Toh (2005)
Eye: dilated pupil diameter (mm, by retroillumination, Britain)							
962	F	7.80±.71	9.1	.79	8.09	–	Hammond (2000)
Eye: anterior chamber depth (mm, from outer cornea to iris, right eye, ultrasound, Sardinia)							
741	Mix	3.45±0.35	10.1	.46	–	6.88	Biino (2005)
Heart: aortic root diameter (cm, echocardiography, Native American)							
2610	Mix	3.45±.4	11.6	.51	–	8.28	Bella (2002)
Penis: length (cm, erect, Germany, young adults)							
111	M	14.48±1.99	13.7	–	–	–	Schneider (2001)
Penis: length (cm, stretched, Greece)							
52	M	12.18±1.7	14.0	–	–	–	Spyropoulos (2002)
Penis: length (cm, stretched, Jordan)							
271	M	13.5±2.3	17.0	–	–	–	Awwad (2005)

Note. To save space, sources are cited by first author only, without any et al.'s

For each study, h^2=narrow-sense (additive) heritability if CVA is calculated; h^2=broad-sense heritability (including additive, dominance, and epistatic effects) if CVG is calculated.

From each study, only samples of healthy, normal participants were included.

All samples are from mature adults (roughly aged 20–70), except where otherwise noted.

lung volume (10.2), knee cartilage volume (20.5), kneecap bone volume (23.9), thyroid gland volume (24.7), and heart left ventricle volume (31.7). Presumably most of these are under strong stabilizing selection for physiological efficiency and appropriate fit within the body. Notably, the brain's CVP (8.2) is also much lower than the CVPs for volumetric traits that are probably under directional sexual selection (Miller, 2000a), such as penis volume (37.0) and breast volume (61.5). If human female brains showed the same proportional phenotypic variation as human female breasts, then the distribution of modern female brain sizes would be 1162 (mean)±686 (SD) cubic centimeters — such that 15.9% of women would have brains larger than 1848 cc (5 SDs larger than the male average given the current male distribution), and 15.9% would have brains smaller than 476 cc (smaller than the average gorilla's).

The dimensionality effect is obvious when one compares CVAs, CVGs, and CVPs derived from linear measures (Table 4.1) to those derived from volumetric measures (Table 4.2) on the same traits. Within the same population of 1909 adults from India (Arya et al., 2002), the CVAs for male weight (25.44) and female weight (21.29) are about three times higher than those for male height (8.86) and female height (6.64). Likewise, the CVA for the heart's left ventricle volume (31.7) is about three times higher than for the heart's aortic diameter (11.6), which empties the left ventricle directly through the aortic valve. Similarly the CVP for penis volume (37.0) is much higher than for penis length (14.0) in the same Greek sample (Spyropoulos et al., 2002).

Table 4.2
CVPs, CVAs and CVGs for volumetric morphological traits

Trait (measurement unit, geographical origin of sample)							
N	Sex	Mean±S.D.	CVP	h^2	CVA	CVG	Source
Weight (kg, Australia, young adults)							
674	M	76.7±12.34	16.1	.837	14.72	–	Harrap (2000)
736	F	61.7±10.58	17.2	.837	15.69	–	Harrap (2000)
Weight (kg, India)							
982	M	37.82±17.16	45.4	.314	–	25.44	Arya (2002)
926	F	37.04±14.08	38.0	.314	–	21.29	Arya (2002)
Lung: volume (liters, forced vital capacity, Australia)							
468	M	4.73±0.74	15.6	.406	–	9.94	Palmer (2001)
468	F	3.36±0.55	16.4	.406	–	10.45	Palmer (2001)
Heart: left ventricular mass (grams, France, teenagers)							
150	M	112.0±29.9	26.7	.34	–	15.57	Garner (2000)
176	F	98.9±20.1	20.3	.34	–	11.85	Garner (2000)
Heart: left ventricular volume (cubic mm, Germany)							
332	Mix	170.5±55	32.3	.68	26.6	–	Busjahn (2000)
Knee: cartilage total volume (milliliters, by MRI, Australia)							
136	F	17.35±3.55	20.5	.73	–	17.5	Hunter (2003)
Knee: patella bone volume (milliliters, by MRI, Tasmania)							
128	Mix	13.8±3.3	23.9	.70	–	20.0	Zhai (2004)
Thyroid gland: volume (milliliters, Denmark)							
281	F	14.56±5.09	35.0	.50	–	24.7	Hansen (2004)
Penis: volume (cubic cm, Greece)							
52	M	46.5±17.2	37.0	–	–	–	Spyropoulos (2002)
Breast: volume (milliliters, China, young adults)							
250	F	325.4±200.2	61.5	–	–	–	Qiao (1997)

For life-history traits (Table 4.3), CVAs are generally higher than for morphological traits. The highly fitness-related trait of reproductive success (number of live births) has a CVP mean that varies from 31 to 87 across various samples, and CVAs consistently higher than 30 (these CVs are inflated by high skew, especially in males). High CVPs and CVAs are also shown by other complex, fitness-related traits such as male hunting ability in indigenous small-scale societies (CVP=65.6, from Hill & Hurtado, 1996), and 'developmental stability' (CVA=14, from Gangestad & Thornhill, 2003) — the theoretical construct that underlies body symmetry, and that has provoked so much fruitful work in sexual selection research (e.g. Møller & Swaddle, 1998), evolutionary psychology (e.g. Gangestad, Bennett, & Thornhill, 2001), and Darwinian psychiatry (e.g. Yeo, Gangestad, Edgar, & Thoma, 1999).

Table 4.3
CVPs, CVAs and CVGs for life-history traits

Trait (measurement unit, geographical origin of sample)							
N	Sex	Mean±S.D.	CVP	h^2	CVA	CVG	Source
Longevity (years, pre-1900 Finland)							
1388	M	56.80±18.86	33.2	.167	–	13.5	Pettay (2005)
1226	F	61.31±20.10	32.8	.175	–	13.7	Pettay (2005)
Hunting ability (mean kg meat acquired per hour, Ache tribal people from Paraguay)							
42	M	.538±.353	65.6	–	–	–	Walker (2002)
Developmental stability (from body symmetry measures, mixed-sex USA sample)							
1,735	–	–	25	.30	14	–	Gangestad (2003)
Reproductive success (# children surviving to age 10, Dogon tribal people from Mali)							
55	F	8.1±2.56	31.6	–	–	–	Strassman and Gillespie (2002)
Reproductive success (# live births, Sami tribal people from Scandinavia)							
236	M	5.88±2.38	40.5	–	–	–	Helle (2002)
327	F	5.70±2.42	42.5	–	–	–	Helle (2002)
Reproductive success (# live births, Ache tribal people from Paraguay)							
41	M	6.12±3.75	61.3	–	–	–	Hill (1996)
42	F	7.67±2.34	30.5	–	–	–	Hill (1996)
Reproductive success (# live births, Kipsigi tribal people from East Africa)							
82	M	12.78±11.07	86.6	–	–	–	Borgerhoff (2000)
64	F	5.81±2.83	48.7	–	–	–	Borgerhoff (2000)
Reproductive success (# live births, Denmark)							
1678	M	1.49±1.10	73.8	.28	39	–	Rodgers (2001)
1540	F	1.61±1.06	65.8	.29	35	–	Rodgers (2001)
Reproductive success (# live births, Denmark)							
334	M	2.49±1.40	56.2	.39	–	35	Kohler and Christensen (2000)
914	F	2.27±1.82	80.2	.11	–	27	Kohler and Christensen (2000)
Reproductive success (# live births, Australia)							
2710	F	–	–	–	–	39	Kirk (2001)

Note. To save space, sources are cited by first author only, without any et al.'s

These comparisons from Tables 4.1, 4.2 and 4.3 suggest that the CVA for human brain size is surprisingly low — lower than the CVA for the volume of any other human organ for which we could find good data. Apparently, brain and skull sizes are much more tightly constrained by evolution than the sizes of other organs. This view is supported by the data from Arya et al. (2002) that linear skull dimensions show much lower CVGs than linear facial dimensions do (Table 4.1). For example, head breadth shows a CVG of 3.28 (males) and 3.21 (females), whereas nose breadth shows CVGs of 8.82 (males) and 6.77 (females). Likewise, head length (reflecting skull length) shows CVGs of 3.41 (males) and 3.08 (females), whereas facial height (from chin to top of nose) shows CVGs of 6.63 (males) and 5.73 (females).

8. Discussion

Brain size is not the same as intelligence, but it is one of the few ratio-scale endophenotypes of intelligence that have been measured well enough for its CVA to be calculated. Based on 19 *in vivo* MRI studies of brain size means, standard deviations, and heritabilities, the CVA for adult human brain size is about 7.8.

By traditional standards in sexual selection theory (Pomiankowski & Møller, 1995), the human brain's CVA looks fairly high, comparable to that for linear-scale sexual ornaments under directional selection. However, if the dimensionality problem (Lande, 1977) is confronted ditrectly, and the brain's CVA (7.8) is compared to the CVAs of other human organs (which range from 15 to 30), we have a problem: brain size seems to be under stronger stabilizing selection than any other organ in the human body.

Comparing the brain to the eye is especially instructive, because both are early-maturing, bone-encased, complex organs. If the CVAs for linear eye measurements (ranging from 4.9 to 10.1) scaled up as expected by a factor of 3 to yield volumetric CVAs, these would range from 15 to 30. Thus, brain volume shows a CVA at the lower end of CVAs for eye structure volumes. Here we reach a quandary. Ever since Darwin (1859), the human eye has been the premier example of a complex morphological adaptation under stabilizing selection for all of its components to work together efficiently. And ever since Darwin (1871), the human brain has been the premier example of a complex morphological adaptation under directional selection to support higher intelligence, which presumably yielded survival and reproductive benefits. Finally, ever since the revival of sexual selection research and the CVA revolution in evolutionary genetics (Houle, 1992), traits under stabilizing selection are expected to show lower CVAs than traits under directional selection. It seems difficult to reconcile these views.

This raises serious problems for most current models of human mental evolution that view the human brain as a good proxy for human intelligence. Most models posit directional selection in recent evolutionary history (the last 2 million years) for higher intelligence, whether they emphasize survival payoffs (Flinn et al., 2005; Geary, 2005; Gottfredson, in press; Kanazawa, 2004; Robson & Kaplan, 2003; Rushton, 2004), social payoffs (Cosmides & Tooby, 2002; Dunbar, 2003; Suddendorf & Whiten, 2001), culture-learning payoffs (Henrich & Gil-White, 2001; Richerson & Boyd, 2004), group-level payoffs (Boehm, 1996; Wilson, Timmel, & Miller, 2004), or sexual payoffs (Darwin, 1871; Miller, 2000a; Prokosch, Yeo, & Miller, 2005). Only a few of these models explicitly acknowledge the importance, heritability, and cross-domain pervasiveness of the *g* factor and its relationship to brain size (e.g. Gottfredson, in press; Miller, 2000a,b,c; Rushton, 2004), but most of these models take the tripling of hominid brain size in the last 2 million years as evidence that such directional selection has been acting on the human brain. If such models are correct, the human brain should show a much higher CVA than we have found.

There are several possible resolutions to this quandary. One might be to emphasize the unusual anatomical constraints on bone-encased organs such as brains. Most other organs can grow or shrink dramatically over time in response to physiological demands (Piersma & Lindstrom, 1997; Piersma & Drent, 2003), such that high organ CVPs (e.g. for lungs, hearts, and livers) may reflect temporary individual differences in organ use more than stable heritable sizes. However, this cannot explain the persistently high CVAs for these organ sizes across individuals and evolutionary time. Also, while brains cannot be larger than the skulls that encase them, they can be smaller: 'enlarged ventricles' (i.e. shrunken brains) are symptomatic of many physical illnesses, mental illnesses, and brain injuries. The potential discrepancy between skull size and brain size is precisely why MRI imaging of live brain volume yields higher correlations with intelligence than external skull measurements do.

A related anatomical constraint is that the human brain is the largest bone-encased structure that must fit through the mother's birth canal. When women die in childbirth, the baby's head is often too large. This problem of 'cephalopelvic disproportion' is a major predictor of serious birth difficulty (Ferguson & Sistrom, 2000), and is fairly common (e.g. affecting 6.9% of nulliparous women in Zaire, such that they require emergency C-sections, Liselele, Boulvain, Tshibangu, & Meuris,

2000). A modern 6.9% rate of emergency C-sections due to cephalopelvic disproportion suggests a similar rate of death in prehistoric childbirth. This obstetric constraint could have imposed much of the stabilizing selection on brain size, severely limiting the brain's potential CVA compared to that of other organ volumes; it could have also imposed much of the directional selection for larger female body size and pelvic diameter (Correia, Balseiro, & De Areia, 2005a; Correia, Balseiro, & De Areia, 2005b; Guegen, Teriokhin, & Thomas, 2000; Tague, 2000), which increased markedly in the last 3 million years. By this birth-constraint account, the human brain's modest CVA reveals that it has been under strong stabilizing selection not to be too large — at least throughout recent evolutionary history (e.g. since the emergence of anatomically modern *Homo sapiens* about 150,000 years ago, when fossil evidence suggests that brain size and female pelvis size reached almost their current average). Further research could clarify these relationships between brain size, skull size, pelvis size, and obstetric constraints during human evolution.

A second possible resolution might accept the common evolutionary psychology view that the human mind is a massively modular set of domain-specific adaptations optimized for reliable, efficient, low-variance, low-heritability performance (e.g. Gardner, 1983; Kanazawa, 2004; Pinker, 1997; Tooby & Cosmides, 1990, 2005). This view implies that stabilizing selection would favor tightly canalized (mutation-resistant) development of all component brain systems (i.e. all psychological adaptations) and all interconnections amongst them. Presumably, the sum total of such stabilized systems – the whole brain itself – should also look tightly stabilized by selection, such that all normal humans should have brain sizes and intelligence levels very close to a population-typical optimum. These models would predict low variance, low heritability, and low CVA for human brain size and intelligence. By this functional-efficiency account, the human brain's low CVA reveals that it has been under strong stabilizing selection for reliable performance, much like the human eye, ever since our species emerged 150,000 years ago with roughly its present brain size.

However, the functional-efficiency view from evolutionary psychology tends to dismiss the *g* factor as biologically trivial, adaptively irrelevant, or a by-product of evolutionarily novel challenges in modern society (e.g. Diamond, 1999; Gould, 1991; Kanazawa, 2004). The human mind's species-typical cognitive architecture may be massively modular and awesomely efficient, but at the level of individual differences, it shows substantial pleiotropic genetic variation (Kovas & Plomin, 2006) that is manifested in highly heritable brain size ($h^2 \sim .9$), highly heritable intelligence in mature adults ($h^2 \sim .7$), and a substantial positive correlation ($r \sim .4$) between them, which is largely genetic in nature (Pennington, 2000; Posthuma et al., 2002, 2003). Thus, the functional-efficiency explanation of the brain's low CVA is hard to reconcile with intelligence research since Galton and Spearman, and with the high heritability of brain size.

How could intelligence have been under directional selection and brain size have been under stabilizing selection, if they are so closely related? The evidence is reasonably good that intelligence was under positive directional selection (more was better), at least until the last few hundred years. Recent molecular-genetic research suggests that throughout human history, brain-size-related alleles have continued to evolve, with significant allele changes in genes such as *Microcephalin* around 37,000 years ago (Evans et al., 2005), *APSM* around 5800 years ago (Mekel-Bobrov et al., 2005), and some sphingolipid-related genes within the last 1000 years (Cochran, Hardy, & Harpending, 2006). Also, intelligence appears highly valued in mate choice across all human cultures that have been studied so far (e.g. Buss, 1989; Correia, 2003; Feingold, 1992; Hatfield & Sprecher, 1995; Marlowe, 2004; Rucas et al., 2006; Shackelford, Schmitt, & Buss, 2005). So, sexual selection consistently favored higher intelligence (Crow, 1993; Darwin, 1871; Miller, 2000a) — but is unlikely to have been the only selection pressure to do so. Intelligence predicts objective performance and learning ability across all important life-domains that show reliable individual differences (Deary, 2000; Gottfredson, 1997; Jensen, 1998), so intelligence probably showed positive fitness payoffs in most evolutionarily relevant domains of survival, social living, mating, and parental investment.

Our results suggest that the evolutionary genetics of human brain size variation can impose some illuminating (if frustrating) constraints on theorizing about the evolution of human intelligence. A really good model of human mental evolution should be able to explain the following:

(1) the low CVA in brain size found in this paper, which suggests strong stabilizing selection (perhaps through obstetric constraints on skull size);
(2) the high heritability of intelligence and brain size, and the genetic correlation between them;
(3) apparent directional selection for higher intelligence, continuing even throughout recent historical time (Cochran et al., 2006; Evans et al., 2005; Mekel-Bobrov et al., 2005);
(4) why intelligence is reduced by inbreeding (Agrawal, Sinha, & Jensen, 1984; Badarudozza, 2004; Badarudozza & Afzal, 1993; Jensen, 1983), and apparently

increased to some degree by outbreeding (Mingroni, 2004; Jensen, 1998) — which suggests an important role for harmful mutations in maintaining the heritability of intelligence (Keller & Miller, in press);

(5) why intelligence (but not skull size) has remained sexually and socially attractive as a fitness indicator (Miller, 2000a; Shackelford et al., 2005);

(6) why brain size and body symmetry (a standard index of overall genetic quality and phenotypic condition) seem to be independent, uncorrelated predictors of intelligence (Prokosch et al., 2005; Thoma et al., 2005);

(7) why reductions in general phenotypic condition (starvation, sleep deprivation, sickness, intoxication) impair intelligence quickly, dramatically, and reversibly (Bartholomew et al., 1999; Belanger & Vanderploeg, 2005; Frencham, Fox, & Mayberry, 2005; Lieberman et al., 2005; Mann, Gunther, Stetter, & Ackermann, 1999; Szinnai, Schachinger, Arnaud, Linder, & Keller, 2005), whereas they reduce brain size only marginally, largely through dehydration (De Bruin et al., 2005; Duning et al., 2005; Gazdzinski, Durazzo, & Meyerhoff, 2005).

Ideally, such a model could lead to more integrative life-history theory of human intelligence (e.g. Kaplan, Hill, Lancaster, & Hurtado, 2000; Rushton, 2004) that explains both species-typical psychological adaptations and individual-differences patterns in their functioning.

Another crucial constraint concerns sex differences. Male humans grow somewhat larger brains (by about 136 cc, 11%, or $d = .30–.35$: see Table 2; plus Anderson, 2003; Ankney, 1992; Gignac, Vernon, & Wickett, 2003; Lynn, 1994, 1999; Packenberg & Gundersen, 1997; Rushton, 1992; Rushton & Ankney, 1996). These sex differences arise largely after puberty, when sex-specific fitness payoffs diverge (Lynn, 1999). Also, some recent evidence suggests that intelligence levels follow a similar developmental trajectory, resulting in a slightly higher male mean intelligence in mature adulthood (Irwing & Lynn, 2005; Nyborg, 2005; Rojahn & Naglieri, 2006). Other research however suggests no adult sex difference in *g* (Camarata & Woodcock, 2006; Colom, Juan-Espinosa, Abad, & Garcia, 2000; van der Sluis et al., 2006). If the sex difference exists, it is probably too small to have much practical significance (Rojahn & Naglieri, 2006), but it may have theoretical significance. Specifically, it would raise the possibility that a newly-recognized evolutionary process called 'sexually antagonistic co-evolution' (Rice & Chippindale, 2001) could maintain much of the genetic variation in human brain size. In this process, alleles that boost brain size might be favored in males but disfavored in females, which could result in fast, ongoing, ever-changing evolution of brain-size-related alleles — maintaining high heritability but low CVA in brain size, with minimal net change in average human brain size across the last 150,000 years.

Sexually antagonistic co-evolution is especially likely on the X chromosome, because frequency-dependent, differential gene expression in the two sexes can promote stable polymorphisms much more easily on the X chromosome than on autosomes (Gibson, Chippindale, & Rice, 2002). This is why the human X chromosome has so many genes associated with sex and reproduction (Lercher, Urrutia, & Hurst, 2003; Saifi & Chandra, 1999), and why the *Drosophila* X chromosome contains an astonishing 45% of all genome-wide fitness variation, and 97% of all genome-wide sexually antagonistic variation (Gibson et al., 2002). This may also be why the human X chromosome holds such an abundance of intelligence-related alleles (Check, 2005; Correia et al., 2005a,b; Graves, Gecz, & Hameister, 2002; Inlow & Restifo, 2004; Zechner et al., 2001).

The sexually antagonistic co-evolution model leads to the predictions that many alleles affecting brain size should (1) be sex-linked (e.g. found on the X chromosome), (2) show incomplete sex-limitation (i.e. some phenotypic expression in both sexes), (3) show genomic imprinting effects (Davies, Isles, Burgoyne, & Wilkinson, 2006), and (4) create sexually opposed effects on reproductive success (negative intersexual heritability for fitness) in natural-fertility populations such as hunter-gatherers. Also, Albert and Otto (2005) point out that any X-linked trait that is costly but sexually attractive (e.g. a larger-than-average brain) would never be passed directly from an attractive father to a son (who always inherits his X chromosome from mother), whereas it would be passed to a daughter, who could suffer the net fitness cost of carrying the display trait. Eventually, given XY sex determination in mammals and the details of their model, the X-chromosome alleles that affect brain size should evolve to favor somewhat smaller (female-advantageous brains) (Albert & Otto, 2005). This leads to a further prediction: (5) any brain-size-increasing alleles found on the X chromosome should be quite evolutionarily recent. These predictions deserve further research, since sexually antagonistic co-evolution is one of the few evolutionary processes that can maintain high heritability in sexually dimorphic traits such as the human brain, and might thus explain the portion of genetic variance in human intelligence that overlaps with genetic variance in brain size.

9. Limitations and directions for further research

This study has some important limitations that should be addressed in further research.

First, this was a provisional analysis of results from a rapidly-advancing field, not a final meta-analysis of a mature research area. Cognitive neuroscience has started to accept the *g* factor in recent years, and a flood of new papers is emerging on relations between intelligence, brain size, brain structure, and brain physiology (e.g. Anderson, 2003; Haier, Jung, Yeo, Head, & Alkire, 2005; Posthuma et al., 2002). This analysis should be repeated in a few years with the larger sample of studies that are likely to emerge.

Second, the MRI subjects for most brain size studies reviewed here were convenience samples recruited by advertisement near North American and European medical schools, rather than true population-representative samples. Thus, the samples are almost all from individuals of white European descent, and are likely to suffer from some restriction of range in intelligence and brain size. This would lead to under-estimating the standard deviations, CVPs, CVAs, and heritabilities in brain size. Thus, our estimated brain size CVA of 7.8 is likely to err somewhat on the low side. In future work, a high priority should be given to collecting more data on CVPs, CVAs, and heritabilities for brain size and other intelligence-related endophenotypes (see below) in truly representative samples of adult humans, across ethnic groups.

Third, we did not 'correct' brain size estimates for body size, because (a) none of the MRI studies we reviewed included body size data, (b) although brain size scales up with body size across species (Roth & Dicke, 2005), brain size within the human species shows rather low correlations with body size (Pakkenberg & Gundersen, 1997; Witelson et al., in press), and (c) in individual development, human brain size approaches 95% of adult asymptote around age 6, long before height does (Caviness , Kennedy, Richelme, Rademacher, & Filipek, 1996; De Bellis et al., 2001). If the developmental time-course of body growth is quite distinct from that of brain growth, then it would be odd to consider one to be an allometric by-product of the other. Although brain size and body size are slightly correlated within humans, it is not clear which drives which: brain size could scale up as an allometric size-effect of body size, or body size could scale up as an energetic side-effect of brain size demands on overall metabolism (Aiello & Wells, 2002). Also, it remains unclear whether sex differences in brain size should be 'corrected' for sex differences in height (Ankney, 1992, cf. Andreasen, Flaum, Swayze, O'Leary, & Allifer, 1993; Flashman, Andreasen, Flaum, & Swayze, 1997). Thus, brain/body allometry remains a contentious topic.

Fourth, our estimate of brain size CVA may have been affected by assortative mating for human intelligence, which tends to show spousal correlations around+.3 to +.4 (Bouchard & McGue, 1981; Buss, 1984; Plomin, DeFries, & Roberts, 1977; Phillips, Fulker, Carey, & Nagoshi, 1988; Mascie-Taylor, 1989; Nagoshi, Johnson, & Honbo, 1992; Reynolds, Baker, & Pedersen, 2000; Watkins & Meredith, 1981; Watson et al., 2004). This seems likely to result not from a preference for IQ-similarity per se, but from mutual preferences for higher IQ among both men and women, in a competitive mating market where individuals of lower mate value end up settling for each other (Hooper & Miller, submitted for publication; Penke, Todd, Lenton, & Fasolo, in press; Miller & Todd, 1998). Since assortative mating for any trait tends to amplify genetic variance in that trait, assortative mating for intelligence (and hence for brain size) could theoretically amplify both the heritability and the CVA for brain size. Future studies concerning the heritability of brain size should try to explicitly model such assortative mating effects.

Fifth, we could find only two studies on brain size heritability in non-human primates (rhesus macaques: Cheverud et al., 1990; baboons: Mahaney et al., 1993), and none that allowed CVA calculations. To gain a comparative perspective on human brain evolution, we need much more extensive data on the relevant evolutionary genetic parameters of other primate brains, as well as of other mammalian species' brains. Since humans seem to face the most severe obstetric constraint on brain size, we may be subject to stronger stabilizing selection on brain size than any other mammal. Thus, our brain size CVA may be lower, even if directional selection for intelligence has been stronger in our species.

A sixth limitation of this study points to some directions for future research. Brain size is far from a perfect index of intelligence, through they are significantly correlated. We initially thought that the CVA of human brain size would be nicely informative about the underlying CVA of human intelligence itself. The comparisons in Tables 4.1, 4.2, and 4.3 convinced us otherwise: brain size is a convenient ratio-scale endophenotype for intelligence, but it may not be a very informative one for understanding genetic variance in intelligence. Perhaps the alleles that create genetic correlations between brain size and intelligence are a rather small and special portion of the alleles that create genetic variance in intelligence generally. Future research should try to estimate CVAs for other ratio-scale measures that are genetically correlated with

intelligence, such as nerve conduction velocities (Rijsdijk & Boomsma, 1997) and reaction times for elementary cognitive tasks (Luciano et al., 2001; Neubauer, Spinath, Riemann, Angleitner, & Borkenau, 2000). Some of these genetically informative data sets may already allow good estimates of these trait means, variances, and heritabilities, so would permit CVA calculations (Penke, 2004). CVA estimates could also be derived, potentially, for other ratio-scale measures of brain function that are at least phenotypically correlated with intelligence, such as cortical concentrations of *N*-acetylaspartate, choline, and phosphorus metabolite ratios as assessed by magnetic resonance spectroscopy (Rae et al., 2003; Ross & Sachdev, 2004), specific cortical area sizes as assessed by voxel-based morphometry with MRI data (Haier et al., 2005), and the fractional anisotropy of white matter as assessed by diffusion tensor MRI (Schmithorst, Wilke, Dardzinski, & Holland, 2001). In each case, genetically informative studies would be required to estimate the heritabilities of these traits, and larger-scale studies would be required to estimate accurately their populations mean and variances.

10. Conclusion

Why should intelligence researchers care about the evolutionary genetics of brain size and *g*? Our principal aim here was to challenge the assumption, common among some intelligence researchers, evolutionary psychologists, and behavior geneticists, that genetic variance in a trait is *prima facie* evidence of its adaptive irrelevance. The new evolutionary genetics of mutation-selection balance (Prokosch et al., 2005; Keller & Miller, in press) strongly challenges that assumption, and shows how highly fitness-related traits can maintain high genetic variance, high heritability, and high genetic correlations among one another. We think this is one promising way that individual-differences research on the factor-analytic structure of genetic and phenotypic variance in human mental abilities (i.e. intelligence research) can be reconciled with adaptationist research on domain-specific, species-typical mental abilities (i.e. evolutionary psychology) (Miller, 2000c). In other words, the apparent conflict between intelligence research's unitary *g* factor and evolutionary psychology's massive modularity view is not a genuine paradox, but a levels-of-description problem that may be resolvable through evolutionary-genetic insights.

What can we conclude from the human brain showing a modest CVA of around 7.8? This is the lowest CVA we could find for any organ volume in the human body, suggesting that the brain has been under strong recent stabilizing selection with respect to overall size. This could support the functional-efficiency argument from evolutionary psychology, or reflect a birth-canal constraint. In either case, apparent stabilizing selection on human brain size is hard to reconcile with all reasonable models of directional selection for human intelligence, given the substantial positive correlation between brain size and intelligence. For the moment, we can caution that brain size may not be the most appropriate ratio-scale endophenotype for understanding the evolutionary genetics of intelligence.

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