A Meta-Analysis of the Relationship between *In Vivo* Brain Volume and Intelligence

Michael A. McDaniel
Virginia Commonwealth University

Nhung T. Nguyen
Lamar University

Paper presented at the Third Annual Conference of the International Society of Intelligence Research. Nashville, TN. December 5, 2002. Correspondence may be addressed to Michael A. McDaniel, Virginia Commonwealth University, Department of Management, PO Box 84400, Richmond, VA 23284-4000. E-mail: mamcdani@vcu.edu.

The authors would like to thank the many researchers in this area who provided data and commentary on this research. We appreciate the use of publication bias software developed by Michael Borenstein and Hannah Rothstein. The assistance of Sue Duval in conducting and verifying the publication bias analyses was greatly appreciated. Nathan Hartman and Smitha S. Nair assisted in the literature search. Deborah L. Whetzel helped in project and manuscript organization.

Abstract

A meta-analysis of the relationship between *in vivo* brain volume and intelligence was conducted. Based on 31 studies across 1,566 people, the population correlation was estimated at .31. Based on smaller subsets of these data, there is preliminary evidence of age and sex moderators of the relationship. Publication bias analyses indicated that our estimated population correlation might be biased upward. However, range restriction in the sample correlations may cause our estimate to be biased downward. Even if one considers moderators, and caveats regarding publication bias and range restriction, it is clear that brain volume is positively correlated with intelligence.

A Meta-Analysis of the Relationship between

In Vivo Brain Volume and Intelligence

Introduction

In 1836, Frederick Tiedmann wrote that there exists “an indisputable connection between the size of the brain and the mental energy displayed by the individual man” (as cited in Hamilton, 1935). Since that time, the quest for the biological basis of intelligence has been pursued. Various narrative reviews (e.g., Rushton & Ankney, 1996, 2000; Vernon, Wickett, Bazana, & Stelmack, 2000) and a meta-analysis (Nguyen & McDaniel, 2000) have documented a non-trivial positive relationship between brain volume and intelligence in non-clinical samples. In this literature, there are two general categories of brain volume measures. The first category consists of measures of the size of the head, such as the circumference of the head. The second category consists of measures of the *in vivo* brain volume, typically assessed through an MRI scan. For head measures, Vernon et al. (2000) reported the population correlation between head size and intelligence to be .19. Nguyen and McDaniel (2000) reported population correlations from .17 to .26 for three different sub-categories of head size measures. Studies assessing the correlation between actual brain volume and intelligence are more rare. Vernon et al. (2000) reported data on 15 such correlations and obtained a population correlation of .33. Nguyen and McDaniel (2000) reported the same population correlation based on 14 correlations.

Since the two reviews in 2000, more data relating brain volume and intelligence have become available. One purpose of this meta-analysis is to update our knowledge concerning the magnitude of the correlation between brain volume and intelligence. A second purpose of the analysis was to test the robustness of the findings by conducting a variety of sensitivity analyses.

Method

Literature review. The literature review began with a review of all known past reviews of this literature. Psychinfo and Medline searches were conducted as well as citation index searches of popular past reviews. Studies containing relevant data were reviewed to identify citations to other relevant studies. Often, we found studies in which the authors collected MRI-assessed brain volume data and intelligence data on a non-clinical sample but did not report the correlation between these measures. This does not mean that such studies were flawed. Rather, the correlation between brain volume and intelligence was not the focus of the study and the publication standards for the journal did not require a correlation matrix among all variables. For such studies, we wrote the authors and requested the correlation.

After preliminary findings were obtained, we e-mailed 51 authors who had published in the area of brain volume and intelligence, or who had provided commentaries on such literature, or who are known to have an interest in the relation between brain volume and intelligence. We provided these researchers with our preliminary findings and requested that they scan our references to determine if we missed any relevant research. We also asked these researchers if they knew of any data sets containing both MRI-assessed brain volume and intelligence that might be relevant to our study.

Analysis Approach. We used meta-analysis procedures in the tradition of Hedges and Olkin (1985) as implemented in the Comprehensive Meta-Analysis Program

(Borenstein & Rothstein, 2000). We also conducted analyses associated with psychometric meta-analysis (Hunter & Schmidt, 1990). For conducting publication bias analysis, we used Rosenthal's (1979) fail-safe N test, Begg and Mazumdar' (1994) rank correlation test, Egger, Smith, Schneider, and Minder's (1997) test of the intercept, and Duval and Tweedie's (2000) trim and fill analysis. These publication bias analyses were conducted with a beta-test version of the publication bias module of the Comprehensive Meta-analysis Program (Borenstein & Rothstein, 2000). The trim and fill analyses were verified by Sue Duval using independent software (personal communication to Michael A. McDaniel, November 28, 2002). Analyses related to restriction of range were computed using a standard psychometric formula for restriction of range.

Decision rules. We included all correlations between brain volume and intelligence we could locate as of November 22, 2002. All brain volume measures were *in vivo* measures with most being MRI-assessed brain volumes measures. If multiple measures of brain volume were available from a given sample, we used the measure that best assessed total brain volume. Intelligence measures were primarily full-scale IQ measures or the Ravens Progressive Matrices Test. If multiple measures of intelligence were available, we chose the measure that was a full-scale IQ measure, the Ravens, or in their absence, the measure that best approximated the full-scale IQ measure. Only one correlation between brain volume and intelligence for a given sample was reported. All sample members were non-clinical. Data from Yeo, Turkheimer, Raz, and Bigler (1987) were included although the subjects were not explicitly normal. We divided our analyses into one primary analysis and nine additional analyses.

Results

Table 1 presents a listing of studies used in the analysis and the results of our primary analysis. Each row of the table, except the last row, summarizes the data from one sample. The rows are sorted by the magnitude of the correlation. The first column provides a citation to the source of the data. The second column in the table is the sample size. The third column is the correlation coefficient. The fourth and fifth columns are the lower and upper bounds of the 95 percent confidence interval for the correlation. The graphic to the right displays the confidence interval for each correlation and the location of the correlation (i.e., the point estimate) within the confidence interval. Based on a random effects model meta-analysis, the bottom row shows the estimated population correlation to be .313 with a 95% confidence interval ranging from .244 to .379.

We then conducted eight sensitivity analyses. The goal of our sensitivity analyses was to determine if changes to the analysis would alter the conclusions. Our sensitivity analyses consisted of examining a subset of better studies that best fit our decision rules, evaluating two potential moderators, conducting four types of publication bias analyses, and examining the effect of the likely range restriction in this data set.

In the psychometric meta-analytic tradition, one determines the amount of variance in the observed distribution and the percentage of that variance attributable to random sampling error. The remaining variance is due to statistical artifacts and substantive variance (i.e., moderators). Four sources of artifactual variance include 1) differences across studies in measurement error in the assessment of brain volume and intelligence, 2) differences across studies in the degree of range restriction or range enhancement in the study variables, 3) differences across studies in the way in which *in vivo* brain volume measures were collected, and 4) differences across studies in the manner in which intelligence was measured. Potential substantive sources of variance would include the moderating effects of age, race, and sex. In this study, only 51 percent of the variance across studies was attributable to random sampling error. In psychometric meta-analysis (Hunter & Schmidt, 1990), a search for moderator analysis is warranted if less than 75% of the observed variance due to artifactual sources. We were able to evaluate the potential moderating effects of age and sex. Too little data were available by race subgroups to examine race as a moderator.

In our analysis we will refer to the meta-analytically-derived mean as the population correlation (D). We recognize that our estimated population mean will be downwardly biased due to the effects of measurement error and range restriction.

The results for the analysis of the full distribution of 31 coefficients described earlier and results for various sub-distributions are presented in Table 2. The analysis labeled “selected coefficients” is based on 25 coefficients. For this analysis, six correlations were dropped because the samples were not explicitly non-clinical, brain volume was measured in an uncommon manner, or intelligence was measured less optimally relative to other studies. Note that this does not mean that the studies were done poorly. Rather, it means that the studies were a poorer fit for our decision rules relative to other papers. The correlation from this analysis (D = .325) was not much different from the analysis of all 31 coefficients (D = .313). Next, we next partitioned the data by age. The estimated population correlation for children and adolescents was .254 in contrast to .339 for adults; thus, an age moderator is suggested. However, the few number of coefficients in the children and adolescent sample limits the confidence that one can place in the finding.

We then cumulated the data separately by sex and examined an additional distribution composed of mixed sex samples or samples where the sex of the sample members was unknown. The correlation was .306 for males and .401 for females. One would expect the correlation for the mixed and unknown sex samples to fall between these values, but it did not (D = .259). Although the sex analyses appear to show a sex moderator, the partitioning of the data into three sets limits the number of coefficients in each analysis. We suggest that conclusions regarding the sex moderator be viewed as tentative.

Additional sensitivity analyses were conducted to assess the potential existence of publication bias in the meta-analysis. Publication bias exists if the meta-analytic review does not contain all existing data and the missing data are not representative of the reported studies. This situation might occur if studies with statistically insignificant correlations are less likely to be published than studies with statistically significant results. Such an occurrence may result from editorial review practices or from study authors declining to submit papers for publication that report statistically insignificant results.

Numerous methods have been proposed to identify publication bias. Rosenthal (1979) offered a “fail-safe N” approach to publication bias. Rosenthal’s procedure estimates the number of studies with a zero correlation that would be needed to nullify the effect (i.e., cause the effect to be statistically insignificant). For our data, there would need to be 1,085 missing studies with a correlation of zero to make relationship between brain volume and intelligence to lack statistical significance. Another way of expressing this finding is that there needs to be 35 missing studies for every study we located.

Additional publication bias analyses involve the funnel plot (Light & Pillemer, 1984). To form a funnel plot, one graphs the correlations such that the most precise correlations (those from samples with the largest sample sizes , those with the least standard error) are at the top of the funnel and the less precise studies (those from the studies with the smallest sample sizes, largest standard error) are at the bottom of the funnel. Figure 1 shows a typical funnel chart. The circles represent the correlations from individual studies. This funnel chart shows a symmetric funnel that would be interpreted subjectively as showing no publication bias. Often the funnel shows that the correlations are missing from the lower left area of the chart. This is typically interpreted as evidence of publication bias with the missing studies representing smaller sample studies with lower magnitude correlations. Such studies are thought to have a lower probability of being accepted for publication and a lower probability of being submitted for publication.

Note that under this scenario, the small sample studies tend to be published only if they yield a correlation of a large magnitude. However, large sample studies tend to be published regardless of the magnitude of the correlation. Based on this observation, Begg and Mazundar (1994) suggested that a rank order correlation between the correlation coefficient and the standard error as a test of the extent of potential publication bias. For the collection of 31 studies, the rank corder correlation was .324 ($p < .006$) suggesting that publication bias is present.

Egger, Smith, Schneider, and Minder (1997) proposed a related analysis. They argued that one can evaluate the extent of any bias by using precision (the inverse of the standard error) to predict the standardized effect size (the correlation divided by the standrd error). In the regression equation, the size of the treatment effect is reflected in the slope of the regression line and the bias is assessed by the interecpt. For our 31 points of data, the intercept beta weight is 2.071 (confidence interval 0.606 to 3.536; $p < .007$). This analysis would also suggest the presence of publication bias in this literature.

Although the above two analyses suggest that publication bias might be present in this data set, neither provide information concerning the extent to which such bias distorts the estimated population mean correlation between brain volume and intelligence. For such an analysis, we used a procedure termed “trim and fill” (Duval & Tweedie, 2000). This analysis rests on the assumption that asymmetry in a funnel plot is evidence of publication bias. The approach is conceptually simple. First, the procedure trims off the asymmetric correlations (those correlations on the right of the funnel that do not have a close counterpart on the left side of the funnel). Second, using this trimmed distribution, the center of the funnel is determined. Third, the trimmed correlations are returned to the distribution. Fourth, the assumed missing counterparts to the asymmetric correlations are then added into the left side of the funnel creating a symmetric “filled” funnel. A meta-analysis is then conducted on this filled distribution. To the extent that the meta-analysis result for the filled distribution differs from the meta-analysis result of the original distribution, one has cause for concern regarding publication bias. There is no claim that the filled distribution gives the correct population mean. In our data, there was a slight discrepancy (.011 difference in the estimated population mean) between two software programs for the trim-and-fill results. We report the data from the software that has had the most rigorous testing. The trim and fill analysis suggested that eight studies are missing. The “filled” distribution gives an estimated population correlation of 0.254 with a confidence interval of 0.182 to 0.325. Whereas, the filled distribution population estimate of .254 differs somewhat from the original distribution population estimate (.313) there is some cause for concern about publication bias in this literature.

The last set of sensitivity analyses concerned the effects of range restriction on these data. Although one study (Willerman et al., 1991) in our analysis was designed to have range enhancement on intelligence (subjects were selected from lower and higher IQ groups), it appears likely that many of the studies have restriction of range in intelligence. For example, a sample of college students can be expected to have a more narrow of range of intelligence than the general population because of the cognitive screening used in college admissions and the self-selection of those with low intelligence into non-collegiate pursuits. Few of the studies included in our analysis reported data needed to correct the correlations for range restriction. Using the correlation of .313 as the range-restriction-attenuated correlation, we estimated what the unattenuated population correlation would be if the samples in our study had varying levels of range restriction (95%, 90%, 85%, 80%, 75%. The results shown in Table 3 give unattenauted population estimates ranging from .325 to .371.

Discussion

Our best estimate of the correlation between brain volume and intelligence is .31. A variety of sensitivity analyses suggested that this estimate might be somewhat higher or somewhat lower, depending on the analysis conducted. However, regardless of the adjustments, the correlation between brain volume and intelligence is always positive and not far from our initial estimate of .31. It is very clear that brain volume and intelligence are related. In this discussion, we examine the various sensitivity analyses.

Sensitivity Analyses

Our first sensitivity analysis dropped six coefficients from studies considered to be somewhat flawed. For this analysis, the estimated population correlation was .325 that is not substantially different in magnitude from the value of .313 in the primary analysis. Although others could use different decision rules concerning the inclusion and exclusion of studies, our decision rules did not result in much change in the estimated population correlation. Whereas all correlations, but one, are positive, it is difficult to imagine a set of decision rules that would not yield a positive correlation between brain volume and intelligence.

The second sensitivity analysis was a moderator analysis contrasting adults with children and adolescents. The estimated population correlation for children and adolescents was .254 compared with .339 for adults. Although these results can be offered as preliminary evidence for age as a moderator, the partitioning of the data results in an analysis with small numbers of studies and small cumulative sample sizes, particularly for the children and adolescent analysis. Firm conclusions should be based on future analyses as more data become available.

A sex moderator was evaluated in the third sensitivity analysis. Most of the samples contained both males and females. These mixed-sex samples yielded an estimated population correlation of .259. The male-only samples yielded an estimated population correlation of .306 while the female-only samples yielded an estimated correlation of .401. We would expect that the estimated population correlation for the mixed-sex samples would fall between the estimates for the male and females samples. It did not. If not due to sampling error, something about mixing males and females into the same sample appears to be lowering the correlation. The analysis of these sex distributions could be interpreted as evidence for a sex moderator of the relationship between brain volume and intelligence. As with the age analyses, firm conclusions should await more data.

The fourth sensitivity analysis was the calculation of Rosenthal's (1979) fail-safe N. There would need to exist 1,085 studies with a correlation of zero to reduce our estimated population correlation so that it would not be statistically different from zero. This number of missing studies is not credible. Thus, we conclude that brain volume and intelligence have a positive correlation beyond any reasonable doubt.

The next three sensitivity analyses used more recent publication bias approaches. All indicated that publication bias was likely. The trim and fill analysis yielded and estimated correlation .07 below the primary analysis estimate of .313. Even if one were to accept the trim and fill estimated population correlation of .243 as factual, the conclusion that brain volume and intelligence are related is not in dispute.

We have concerns about the accuracy of the trim and fill approach to publication bias for our data set. The symmetry assumption of the trim and fill method appears to assume that there are no moderators influencing the magnitude of the correlations. Our results offer preliminary evidence of age and sex moderators. Likewise, we calculated that sampling error variance accounts for only about half of the observed variance in our distribution. Although some of this unexplained variance might be due to statistical artifacts, the magnitude of the unexplained variance suggests the presence of moderators. Thus, we suggest that one interpret the trim and fill results with some level of caution. Terrin, Schmid, Lau, and Olkin (2002) have asserted problems with the trim and fill procedure when the effects sizes (here, correlations) are heterogeneous (e.g., the relationship has moderators). On the other hand, in agreement with the trim and fill results, both the Egger et al. (1997)

and the Begg and Mazumdar (1994) analyses provided evidence of publication bias. Thus, publication bias in these data cannot be ruled out.

Our last sensitivity analysis concerned the impact of range restriction. Our simple simulation of potential range restriction effects offers estimates of the unattenuated population correlation after adjustment for range restriction. It is certain that our estimated population correlation is also downwardly biased by measurement error. However, the reliability of full-scale intelligence measures is quite high. Likewise, when the reliability of brain volume measures are reported, they are also quite high. Thus, measurement error is unlikely to have attenuated our estimate of the population correlation substantially. What attenuation that does exist is primarily driven by range restriction on intelligence in the study samples.

Data Reporting and Availability Issues

We have concerns regarding the reporting practices of research in this area. Few studies report means and standard deviations and a zero-order correlation matrix among the variables. The lack of reported standard deviations makes it impossible to estimate precisely range restriction effects in these data. The lack of a correlation matrix results in our excluding data from this analysis and thus increases publication bias concerns. Although we greatly appreciate the actions of several authors who provided us with unpublished correlations, many authors did not respond to our inquiries. We find this particularly aggravating when the authors who withhold results from us are those who received public funds to conduct their research or are employees of federal agencies or state agencies, such as public universities. Results of studies based on public funding should not be withheld from the public.

Given the preliminary evidence of age and sex moderators in these data, more research reporting results separately by age and sex is warranted. We were unable to examine potential race moderators due to the relative lack of non-Caucasians in the samples and the failure to report correlations separately by race.

Additional research

In addition to more research with better reporting, two additional areas deserve greater research attention. The first area is an examination of the brain volume and intelligence relationship at a more refined level of analysis than total brain volume. For example, although the Staff (2002) results indicated a negative correlation between brain volume and intelligence, the fraction of brain volume that was gray matter was correlated .35 with intelligence. Likewise MacLullich et al., (2002) examined the relationship between regional brain volumes (e.g., left and right hippocampus, left and right frontal lobe, left and right temporal lobe) with intelligence. We had considered including in this meta-analysis an analysis of regional brain volumes with intelligence but the studies are too few to yield a credible analysis. The second area worthy of increased attention is the genetic contribution to the brain volume and intelligence relationship. The research in this area is both recent and rapidly growing (Molloy, Rapoport, & Giedd, 2002; Pennington et al., 2002; Posthuma, et al., 2002; Schoenemann, Budinger, Sarich, & Wang, 2000; Thompson, et al. 2001). These two research areas will help us to better understand the causal relationship between brain volume and intelligence.

Conclusion

Based on a collection of 31 effect sizes, the estimated population correlation between brain volume and intelligence is .31. We conducted multiple sensitivity analyses and always found the estimated population correlation to be positive and not far from the estimated value of .31. Thus, it is clear that the relationship between brain volume and intelligence is positive.

References

*Aylward, E. H., Minshew, N. J., Field, K., Sparks, B. F., & Singh, N. (2002). Effects of age on brain volume and head circumference in autism. *Neurology, 59*, 175-183.

*Andreasen, N. C., Flaum, M., Swayze II, V., O'Leary, D. S., Alliger, R., Cohen, G., Ehrhardt, J., & Yuh, W. T. C. (1993). Intelligence and brain structure in normal individuals. *American Journal of Psychiatry, 150*, 130-1134.

Begg, C.B. & Mazumdar, M. (1994). Operating characteristics of a rank correlation test for publication bias. *Biometrics, 50*, 1088-1101.

Borenstein, M. & Rothstein, H.R. (2000). Comprehensive Meta-Analysis. Englewood NJ: Biostat, Inc.

*Castellanos, F. X., Giedd, J. N., Eckburg, P., Marsh, W. L., Vaittuzis, A. C., Kaysen, D., Hamburger, S. D., & Rapoport, J. L. (1994). Quantitative morphology of the caudate nucleus in attention deficit hyperactivity disorder. *American Journal of Psychiatry, 151*, 1791-1796.

*De Meyer, M. K., Gilmor, R. L., Hendrie, H. C., & De Meyer, W. E. (1988). Magnetic resonance brain images in schizophrenic and normal subjects: Influence of diagnosis and education. *Schizophrenia Bulletin, 14*, 21-32.

Duval, S. & Tweedie, R. (2000) Trim and fill: A simple funnel plot based method of testing and adjusting for publication bias in meta-analysis. *Biometrics, 56*, 276-284.

*Egan, V., Wickett, J. C., & Vernon, P. A. (1995). Brain size and intelligence: Erratum, addendum, and correction. *Personality and Individual Differences, 19*, 113-115.

Egger, M, Smith, G, Schneider, M, & Minder C. (1997). Bias in meta-analysis detected by a simple, graphical test. *British Medical Journal, 315*, 629-634.

*Flashman, L. A., Andreasen, N. C., Flaum, M., & Swayze, V. W. (1998). Intelligence and regional brain volumes in normal controls. *Intelligence, 25*, 149-160.

*Gur, R. C., Turetsky, B. I., Matsui, M., Lan, M., Bilker, W., Hughett, P., & Gur, R. E. (1999). Sex differences in brain gray and white matter in healthy young adults: Correlations with cognitive performance. *Journal of the Neuroscience, 19*, 4065-4072.

Hamilton, J. A. (1935*). The association between brain size and maze ability in the white rat*. Unpublished doctoral dissertation. University of California, Berkeley.

*Harvey, I., Persaud, R., Ron, M. A., Baker, G., & Murray, R. M. (1994). Volumetric MRI measurements in bipolars compared with schizophrenics and healthy controls. *Psychological Medicine, 24,* 689-699.

Hedges L. & Olkin, I (1985). *Statistical methods for meta-analysis*. London: Academic Press.

Hunter, J. E. & Schmidt, F.L. (1990*). Methods of meta-analysis: Correcting error and bias in research findings*. Newbury Park, CA: Sage.

*Jones, P. B., Harvey, I., Lewis, S.W., Toone, B.K., et.al. (1994). Cerebral ventricle dimensions as risk factors for schizophrenia and affected psychosis: an epidemiological approach to analysis. *Psychological Medicine, 24*, 995-1011.

*Jorm, A. F., Creasey, H., Broe, G. A., Sulway, M. R., Kos, S. C., & Dent, O. F. (1997). The advantage of being broad-minded: brain diameter and neuropsychological test performance in elderly war veterans. *Personality and Individual Differences, 23*, 371-377.

*Kareken, D. A., Gur, R. C., Mozley, P. D., Mozley, L. H., Saykin, A. J., Shtasel, D. L., & Gur, R. E. (1995). Cognitive functioning and neuroanatomic volume measures in schizophrenia. *Neuropsychology, 9*, 211-219.

Light, R. & Pillemer, D. (1984). *Summing up: The science of reviewing research*. Cambridge, MA: Harvard University Press.

*MacLullich, A. M. J., Ferguson, K. L., Deary, I. J., Seckl, J. R., Starr, J. M., & Wardlaw, J. M. (2002). Intracranial capacity and brain volumes are associated with cognition in elderly men. *Neurology, 59*, 169-174.

Molloy, E., Rapoport, J. L., & Giedd, J. N. (2002). Letter to the editor: Nongenetic factors influence the association between brain morphometry and intelligence. *Nature Neuroscience, under review*.

Nguyen, N.T. & McDaniel, M.A. (December, 2000*). Brain size and intelligence: A meta-analysis*. Paper presented at the First Annual Conference of the International Society of Intelligence Research. Cleveland, OH.

*Nosarti, C., Mazin, H. S., Al-Asady1, S. F., Stewart1, A. L., Rifkin, L., & Murray, R. M. (2002). Adolescents who were born very preterm have decreased brain volumes. *Brain, 125*, 1616-1623. Data supplemented by personal communication from Chiara Nosarti to Michael A. McDaniel November 6, 2002 and November 8, 2002.

*Nosarti, C. (2002). *Personal communication to Michael A. McDaniel on November 6,2002 and November 8, 2002.*

*Pennington, B. F., Filipek, P. A., Lefly, D., Chhabildas, N., Kennedy, D. N., Simon, J. H., Filley, C. M., Galaburda, A., & Defries, J. C. (2000). A twin MRI study of size variations in the human brain. *Journal of Cognitive Neuroscience, 12*, 223-232.

Posthuma, D., Geus, E. J. C. D., Baare, W. F. C., Pol, H. E. H., Kahn, R. S., & Boomsma, D. I. (2002). The association between brain volume and intelligence is of genetic origin. *Nature Neuroscience, 5*, 83-84.

*Raz, N., Torres, I. J., Spencer, W. D., Millman, D., Baertschi, J. C., & Sarpel, G. (1993). Neuroanatomical correlates of age-sensitive and age-invariant cognitive abilities: An in vivo MRI investigation. *Intelligence, 17*, 407-422.

*Reiss, A. L., Abrams, M. T., Singer, H. S., Ross, J. L., & Denckla, M. B. (1996). Brain development, gender and IQ in children. *Brain, 119*, 1763-1774. Data supplemented by personal communication from A.L. Reiss to Michael A. McDaniel October 17, 2002.

Rosenthal, R. (1979). The “file drawer problem” and tolerance for null results. *Psychological Bulletin, 86*, 638-641.

Rushton, J. P., & Ankey, C. D. (1996). Brain size and cognitive ability: correlations with age, sex, race, social class, and race. *Psychonomic Bulletin & Review, 3*, 21-36.

Rushton, J. P., & Ankney, C. D. (2000). Size matters: a review and new analyses of racial differences in cranial capacity and intelligence that refute Kamin and Omari. *Personality and Individual Differences, 29*, 591-620.

*Schoenemann, P. T., Budinger, T. F., Sarich, V. M., & Wang, W. S. Y. (2000). Brain size does not predict general cognitive ability within families. *Proc Natl Acad Sci, 97*, 4932-4937. Supplemented for data received through personal communication from P.Thomas Schoenemann to Michael A. McDaniel on November 14, 2002.

*Staff, R. T. (2002). *Personal communication to Michael A. McDaniel on November 11, 2002.*

*Tan, U., Tan, M., Polat, P., Ceylan, Y., Suma, S., & Okur, A. (1999). Magnetic resonance imaging brain size/IQ relations in Turkish university students. *Intelligence, 27*, 83-92.

Tiedmann, F. (1836*). Sur l’encÁphale du NÙgree comparÁ × celui de l’EuropÙen et celui de l’orangoutang*. Philosphical Transactions of London.

Terrin, N., Schmid, C.H., Lau, J. & Olkin, I. (2002, May*). Adjusting for publication bias in the presence of heterogeneity*. Paper presented at Meta-Analysis Symposium, Mathematical Sciences Research Institute. Berkeley.

*Thompson, P. M., Cannon, T. D., Narr, K. L., Erp, T. v., Poutanen, V.-P., Huttunen, M., Lonnqvist, J., Standertskjold-Norenstam, C.-G., Kaprio, J., Khaledy, M., Dail, R., Zoumalan, C. I., & W.Toga, A. (2001). Genetic influences on brain structure. *Nature Neuroscience, 4*, 1253-1258.

Vernon, P. A., Wickett, J. C., Bazana, P. G., & Stelmack, R. M. (2000). The neuropsychology and psychophysiology of human intelligence. In R. J. Sternberg (Ed.), *Handbook of intelligence* (pp. 245-264). New York: Cambridge University Press.

*Wickett, J., Vernon, P. A., & Lee, D. H. (1994). *In vivo* brain size. head perimeter, and intelligence in a sample of healthy adult females. *Personality and Individual Differences 16,* 831-838.

*Willerman, L., Rutledge, J. N., & Bigler, E. D. (1991). *In vivo* brain size and intelligence. *Intelligence, 15*, 223-228.

*Yeo, R. A., Turkheimer, E., Raz, N., & Bigler, E. D. (1987). Volumetric asymmetries of the human brain: Intellectual correlates. *Brain and Cognition, 6*, 15-23.

Table 1. Meta-analysis of Brain Volume and Intelligence

Citation	NTotal	Effect	Lower	Upper
Staff	106	-.080	-.267	.112
Aylward et al.	83	.040	-.177	.254
Nosarti,	20	.070	-.384	.497
Yeo et al.	41	.070	-.243	.370
Jorm et	167	.110	-.043	.258
De Meyer et	21	.120	-.329	.525
Schoenemann (2000)	72	.210	-.023	.421
Flashman et al.	90	.250	.045	.435
Tan et al.	49	.280	-.001	.520
Jones et al.	67	.300	.064	.504
Kareken et al. (1995)	68	.300	.066	.502
Egan et al.	40	.310	-.002	.567
Pennington	36	.310	-.021	.580
Willerman et al.	20	.330	-.132	.674
Castellanos, et	46	.330	.044	.566
Wickett et al.	68	.350	.122	.543
Reiss et al. (1996)	57	.370	.121	.575
Thompson et al.	20	.370	-.087	.698
Nosarti et al. (2002)	42	.370	.074	.606
Gur et al.	40	.390	.089	.626
MacLullich et al.	97	.390	.207	.547
Wickett et al.	40	.400	.101	.633
Gur et al.	40	.400	.101	.633
Andreasen et al.	37	.400	.087	.641
Raz et	29	.430	.075	.688
Andreasen et	30	.440	.095	.691
Thompson et	20	.450	.009	.744
Willerman et al.	20	.510	.087	.777
Reiss et al. (1996)	12	.520	-.077	.842
Tan et al.	54	.620	.422	.761
Harvey et al.	34	.690	.459	.834
Combined	**1566**	**.313**	**.244**	**.379**

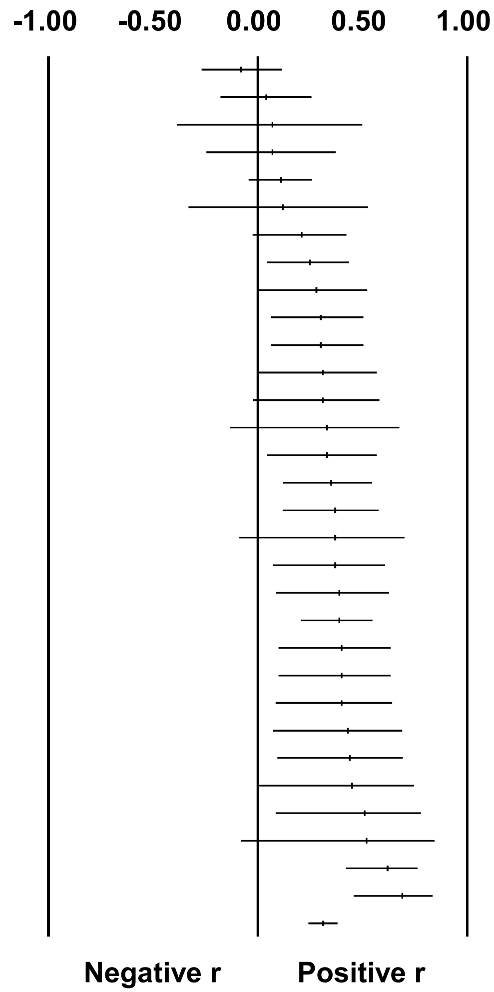

Table 2. Summary statistics for the distribution of all coefficients, the distribution of selected coefficients, and the age and sex sub-distributions.

Analysis	Total N	# of studies	D	95% CI	
All coefficients	1,566	31	.313	.244	.379
Selected coefficients	1,216	25	.325	.254	.392
Children and adolescents[1]	380	7	.254	.138	.363
Adults [1]	1,103	23	.339	.258	.416
Males	576	10	.306	.219	.389
Females	313	7	.401	.280	.509
Mixed or unknown sex	677	14	.259	.134	.376

Data from Aylward et al. (2002) is not included in the age analysis because the sample included both children and adults.

Table 3. Estimated Unattenuated Mean Population Correlations for Varying Levels of Range Restriction

	Range restriction level (s/F)					
	1.0	.95	.90	.85	.80	.75
Unattenuated coefficient	0.313	0.325	0.337	0.349	0.36	0.371

The expression s/F refers to the ratio of the sample standard deviation for intelligence to the population standard deviation for intelligence.

Figure 1. Hypothetical Funnel Plot Showing Symmetry