Intelligence 50 (2015) 159–163

Contents lists available at ScienceDirect

Intelligence

The more *g*-loaded, the more heritable, evolvable, and phenotypically variable: Homology with humans in chimpanzee cognitive abilities

Michael A. Woodley of Menie [a,b,1], Heitor B.F. Fernandes [c,*,1], William D. Hopkins [d,e]

[a] Center Leo Apostel for Interdisciplinary Studies, Vrije Universiteit Brussel, 1050 Brussels, Belgium
[b] Department of Psychology, Technische Universität Chemnitz, 09126 Chemnitz, Germany
[c] Departments of Psychology and Genetics, Federal University of Rio Grande do Sul, 90035-003 Porto Alegre, Rio Grande do Sul, Brazil
[d] Neuroscience Institute and Language Research Center, Georgia State University, Atlanta, GA 30302, USA
[e] Division of Developmental and Cognitive Neuroscience, Yerkes National Primate Research Center, Atlanta, GA 30322, USA

ARTICLE INFO

Article history:
Received 14 October 2014
Received in revised form 1 April 2015
Accepted 2 April 2015
Available online xxxx

Keywords:
General intelligence
Primate intelligence
Heritability
CVA
Chimpanzee

ABSTRACT

Expanding on a recent study that identified a heritable general intelligence factor (*g*) among individual chimpanzees from a battery of cognitive tasks, we hypothesized that the more *g*-loaded cognitive abilities would also be more heritable addition to presenting greater additive genetic variance and interindividual phenotypic variability. This pattern was confirmed with multiple analytical approaches, and is comparable to that found in humans, indicating fundamental homology. Finally, tool use presented the highest heritability, the largest amount of additive genetic variance and phenotypic variance, consistent with previous findings indicating that it is associated with high interspecies variance and has evolved rapidly in comparative primate studies.

1. Introduction

1.1. Jensen effects

Many studies involving humans have demonstrated that the vector of *g* loadings of cognitive tests is a strong positive predictor of the magnitude of a tests' correlation with numerous variables, such as brain size, reaction time, scholastic and workplace performance, and inbreeding-depression effects, and also of phenotypic and genetic characteristics associated with performance on the tests, such as the magnitude of population differences in cognitive performance (Jensen, 1980, 1998; Rushton & Jensen, 2010). The affinity that many biological variables exhibit for *g* is known as the 'Jensen effect' (Rushton, 1999). This effect indicates that *g* is a biologically grounded variable rather than a purely statistical regularity among test scores (Rushton, 1999; Rushton & Jensen, 2010). Importantly, however, examination of possible Jensen effects in nonhuman animals is almost nonexistent, even though there are many studies of nonhuman general intelligence (e.g. Galsworthy, Arden, & Chabris, 2014; Reader, Hager, & Laland, 2011).

Various studies involving humans have found Jensen effects on subtest heritabilities. In Western samples, correlations between the vector of subtest *g* loadings and heritability values range in magnitude from .27 to .77 across studies (Jensen, 1987; Kan, Wicherts, Dolan, & van der Maas, 2013; Pedersen, Plomin, Nesselroade, & McClearn, 1992; Rijsdijk, Vernon, & Boomsma, 2002; Rushton & Jensen, 2010), reaching unity if psychometric meta-analytical corrections are applied (Rushton & Jensen, 2010). In a bare-bones meta-analysis of six Japanese samples a correlation of .38 was found (te Nijenhuis, Kura, & Hur, 2014).

* Corresponding author.
E-mail address: heitor.barcellos@ufrgs.br (H.B.F. Fernandes).
[1] The two first authors contributed equally to this study.

http://dx.doi.org/10.1016/j.intell.2015.04.002
0160-2896/

Humans are not the only species for which *g*-loading estimates and heritability data exist on cognitive ability measures. A recent study (Hopkins, Russell, & Schaeffer, 2014) found that among a sample of 99 chimpanzees tested using a broad intelligence battery, the results of employing several analytical methods converged on the existence of a *g* factor, with *g* loadings ranging from .054 to .723, while heritabilities ranged from .00 to .74 across the 13 subtests. Heritability was evaluated using the program SOLAR (Sequential Oligogenic Linkage Analysis Routines; Almasy & Blangero, 1998), which uses a variance-components approach to estimate additive genetic variance. One strength is that this approach employs all kinship information, including full sibship, half sibship, parent–offspring and more distant relationships. With SOLAR, maximum-likelihood estimation can be applied to a mixed-effects model that incorporates additive genetic effects (matrix of genetic relationships among all subject pairs in the pedigree times the proportion of phenotypic variance attributable to genetic variation), the shared environmental effects (matrix of shared environmental variables times the proportion of variation attributable to those shared environmental effects), and a term for the unique environmental variation and error.

One Jensen effect that has been identified both in humans and more recently in nonhuman primates is the strong correlation between *g* loadings and the size of differences in cognitive abilities among populations (or among species, in nonhuman primates), a phenomenon known as Spearman's hypothesis (Jensen, 1998). Many studies have corroborated that effect in humans (for a review see Fernandes, Woodley, & te Nijenhuis, 2014). An analysis of 69 primate species with five cognitive abilities has shown almost perfect correlations between *g* loadings and the size of the differences among species (Fernandes et al., 2014).

1.2. Genetic and phenotypic diversity

An underexplored question in the intelligence literature is whether the magnitude of the heritability and the amount of additive genetic variance in cognitive abilities are predictors of their phenotypic diversity. As they are indicators of *genetic diversity* (Hughes, Inouye, Johnson, Underwood, & Vellend, 2008), heritability and the amount of additive genetic variance should be positively associated with the magnitude of the *phenotypic diversity* of the cognitive abilities in animals. This appears to be the case with humans (Spitz, 1988). Additionally, traits closely related to fitness, as is hypothesized to be be case for g, are associated with larger mutational target sizes, which increase their sensitivity to *de novo* pleiotropic mutations which in turn increase the genetic variability associated with these traits, and also the evolutionary responsiveness of the trait to selection (Houle, 2000; Miller, 2000; Penke, Denissen, & Miller, 2007; see Pavlicev, Cheverud, & Wagner, 2010; Stearns, 1992, for other putative causes leading to high genetic variability in traits that are importantly connected to fitness). As more *g*-loaded abilities appear to be under stronger selection in the primate phylogeny (Fernandes et al., 2014), we expect that *g* loadings will correlate positively not only with genetic diversity but also with phenotypic diversity.

1.3. Aims and predictions

Following on from previous research, in which Spearman's hypothesis was demonstrated to generalize to comparisons involving primate species (Fernandes et al., 2014), here we reanalyse data from Hopkins et al. (2014) in an effort to examine whether the finding that heritability is a Jensen effect generalizes to chimpanzees, and whether individual chimpanzees differ from one another to a greater degree on more *g*-loaded abilities. Therefore we attempt to investigate whether these properties are *homologous* (i.e. features common to different species that are derived from common ancestry) between humans and chimpanzees. We also expand on these previous two studies via examination of phenomena that are still unexplored in studies of Jensen effects in human intelligence: using the database from Hopkins et al. (2014) it is possible to calculate coefficients of additive genetic variance (CVA), which constitute a mean-standardized and scale-invariant index of genetic variance in a trait (Houle, 1992). Unlike heritabilities, CVAs are independent of environmental variation effects upon individuals, and they are evolutionarily informative as high CVAs are typical for fitness-related traits, especially those under directional selection and those influenced by many genes (Miller & Penke, 2007). Thus we predict that there should *also* be a Jensen effect on CVA, which would indicate that more *g*-loaded abilities have been subjected to stronger recent selection pressures than more specific and 'modularized' cognitive abilities. A positive and strong association between *g* loadings and the CVAs of the respective cognitive abilities would be consistent with the findings of Fernandes et al. (2014) in the primate phylogeny, and with the tentative demonstration that *g* has undergone positive selection in the genus *Pan* (Reader et al., 2011). Finally, we expect that heritabilities and CVAs will be positively associated with phenotypic variability in Hopkins et al.'s (2014) chimpanzee sample, for the reasons outlined in Section 1.2.

Examining whether *g* loadings, heritability, additive genetic variance, and phenotypic variance are positively interrelated in chimpanzees is an important step towards determining whether the human *g*-nexus (i.e. the nomological net of psychological findings indicating the centrality of *g* in predicting phenotypic and genetic characteristics associated with cognitive abilities; Jensen, 1998) generalizes to other primates. Additionally, further studies on *g* in chimpanzees would provide invaluable data and test many contemporary theories stemming from ethologists and also evolutionary psychologists who propose that humans and other animals are essentially different in ways that make the organization of their cognitions incomparable (e.g. Barkow, Cosmides, & Tooby, 1992; Herrmann & Tomasello, 2012; Macphail, 1985).

2. Methods

The subjects and the collected data used here are the same as those reported in Hopkins et al. (2014). There were 99 captive chimpanzees (*Pan troglodytes*) housed at the Yerkes National Primate Research Center. Each chimpanzee was tested on 13 tasks designed to broadly assess social and physical cognition (see Table 1). Normal probability plots (a special case of Q–Q probability plots; Chambers, Cleveland, Kleiner, & Tukey, 1983) and skewness and kurtosis tests (Kim, 2013;

Table 1
g loading, heritability (h^2), coefficient of additive genetic variance (CVA), and phenotypic variability (CV) of the 13 cognitive tasks.

Cognitive task	*g* loading	h^2	CVA	CV	Mean performance (SD)
Spatial memory	.270	.268	45.474	87.841	.623 (.547)
Object permanence	.659	.295	31.635	58.244	.615 (.358)
Rotation	.466	.000	0.000	75.441	.488 (.368)
Transposition	.538	.479	48.965	70.749	.661 (.468)
Relative numbers	.472	.000	0.000	46.383	.665 (.308)
Causality-noise	.203	.000	0.000	54.972	.543 (.299)
Causality-visual	.237	.000	0.000	50.701	.628 (.318)
Tool use	.516	.740	263.795	306.656	.305 (.935)
Tool properties	.191	.105	18.398	56.776	.701 (.398)
Comprehension	.468	.268	74.991	144.857	.419 (.607)
Production	.607	.501	150.058	212.003	.352 (.746)
Attention state	.515	.314	73.773	131.654	.393 (.517)
Gaze following	.048	.020	20.484	144.842	.474 (.687)

Lewis-Beck, Bryman, & Liao, 2013) indicated that the distribution of performance on the 13 tasks did not deviate from normality. In the present study, we used a different technique to assess *g* in the chimpanzee sample than that employed in Hopkins et al. (2014), we calculated the additive genetic variance and the phenotypic variance for performance on each cognitive task, and then proceeded to test the various predictions regarding Jensen effects proposed in Section 1.3 within the chimpanzee sample.

First, as Principal Axis Factor analysis (PAF) eliminates error variance and retains only truly shared variance among traits (Costello & Osborne, 2005), we reanalyzed the raw data from Hopkins et al. (2014) using PAF in order to obtain a purer set of *g* loadings for the analyses. Similarly to Hopkins et al. (2014) and to general practice (Jensen & Weng, 1994), we used the loadings of the first unrotated factor on the cognitive abilities as the *g* loadings, as rotations are designed to break general factors into components (Darton, 1980). We estimated the heritabilities of the cognitive abilities with SOLAR (Almasy & Blangero, 1998) as in Hopkins et al. (2014), thus using a variance-components approach to estimate the additive genetic variance associated with each ability.

Second, as in other primate intelligence studies in which performance on tasks was compared within and between species (e.g. Herrmann, Call, Hernández-Lloreda, Hare, & Tomasello, 2007), to compare the phenotypic variability (i.e. the dispersion) of different cognitive abilities, we computed the coefficient of (phenotypic) variation (usually referred to as CV or CVP, which is the standard deviation/mean ratio, multiplied by 100; Sokal & Rohlf, 1995) for each ability. This is because CVs are a mean-standardized and scale-invariant measure of trait variability, and are thus ideal for comparing the variability of different traits, especially if their means are considerably different, as was the case for the performance means on the 13 cognitive tasks employed in this study (see Table 1). The CVAs were also computed for each cognitive task, by computing the product of its CV and the square root of its heritability (Houle, 1992).

Third, the method of correlated vectors (MCV; Jensen, 1998) was employed in testing all predictions, as is common practice in human studies investigating the relationship between *g* loadings and heritability (e.g. Rushton & Jensen, 2010). This involves calculating the Pearson product–moment correlation between (a) the column vector of a property of the cognitive tasks (such as their *g* loadings), and (b) the column vector of a second property of the respective cognitive tasks (such as heritability estimates, CVs, or CVAs).

Finally, we subjected the variables to multivariate MCV, which involves taking the common factor among the four vectors (*g* loadings, heritabilities, CVs and CVAs), and then correlating each vector with the common factor (Rushton, 1998). Such a "multivector common factor" represents the common-factor variance among the *g* vector in addition to the vectors of other biological/genetic variables (Woodley, te Nijenhuis, & Murphy, 2014). The factor loading of the multivector common factor upon each of its constituent vectors represents an omnibus test of the Jensen effect for a given variable, as they constitute the correlation between that vector and the set of all vectors.

With four indicators, and an *N* of 13 datapoints apiece, it is impossible to recover a meaningful factor structure for the multivector common factor using conventional factor analysis, unless the factor structure is defined a *priori* (Figueredo, Cox, & Rhine, 1995; Gorsuch, 1983). This is because factor loadings produced with Principal Axis Factoring and Principal Components Analysis (PCA) in small samples are less reliable than those computed with unit-weighted factoring, as increasing standard errors of PCA or PAF factor loadings with decreasing sample size prevents one from discriminating between the factor loadings of the different variables (Bobko, Roth, & Buster, 2007; Figueredo et al., 1995; Gorsuch, 1983; Schmidt, 1971). To that end, we constructed a *unit-weighted factor* by simply standardizing each indicator (i.e. computing its *z*-score) and then summing across the vectors in order to generate a composite score, as is recommended (Figueredo et al., 1995; Gorsuch, 1983). The unit-weighted factor loadings were then obtained by correlating each indicator with the common factor.

Table 2
Vector correlations between *g* loadings, heritabilities, CVAs, and CVs of cognitive tasks.

	(1)	(2)	(3)
(1) *g* loading	–		
(2) Heritability (h^2)	.61*	–	
(3) CVA	.43	.90*	–
(4) Phenotypic variability (CV)	.26	.75*	.94*

* Significant correlations ($p < .05$; $N = 13$).

3. Results

The descriptive statistics for each cognitive measure used in the analyses are presented in Table 1. The *g*-loading scores varied from .048 to .607 (see Supplement 1 for correlations and covariances among the tasks, which also indicate a positive manifold), and parallel analysis (Horn, 1965) indicated that only one factor should be extracted in this PAF analysis, as in Hopkins et al.'s (2014) Principal Components Analysis (PCA) results. The heritability estimates ranged from 0 to .740. The coefficients of additive genetic variance ranged from 0 to 263.80 and the phenotypic variability ranged from 46.38 to 306.66.

The results for the vector analyses are presented in Table 2. As can be seen, correlations among *g* loading, heritability, CVA and CV ranged from $r = .26$ to $r = .94$, and most were statistically significant.

Lastly, the multivector MCV analysis confirmed that these vectors were strongly and significantly related to one another, sharing variance with the multivector common factor (see Table 3). The factor loadings ranged from .67 to .95. Reproducing this analysis with PAF produces similar loadings for all vectors (.94 for h^2, .97 for CVA, .94 for CV, all significant) except for the loading of the factor on *g* loadings (.46, $p > .05$). It is important to keep in mind, however, that PAF loadings are known to present large standard errors in small samples, and thus unit-weighted factor loadings are to be preferred due to their reliability in such cases (Bobko et al., 2007; Figueredo et al., 1995; Gorsuch, 1983; Schmidt, 1971).

For comparability, the statistical effects presented in Tables 2 and 3 were also examined using the *g* loadings reported by Hopkins et al. (2014), based on a PCA. As that set of *g* loadings and the set presented here in Table 2 (based on a PAF) correlate at $r = .99$ ($p < .05$), the vector correlations displayed on Table 2 involving the vector of *g* loadings were reduced in magnitude by only .00–.04 when using the PCA-based *g* loadings. The multivector common factor loading upon the vector of PCA-based *g* loadings was reduced by .03 compared to the common factor loading upon the vector of PAF-based *g* loadings.

4. Discussion

This study examined whether in chimpanzees the *g* loadings of cognitive abilities, their heritabilities, CVAs, and CVs are positively interrelated. Several predictions were tested: more *g*-loaded cognitive abilities should be more heritable in chimpanzees as has been found in multiple studies with humans, individual chimpanzees were expected to differ more among themselves in more *g*-loaded abilities as is the case with humans – both of which would indicate homology between humans and chimpanzees, Additionally, heritability and additive genetic variance were predicted to be positively associated with phenotypic diversity, as they are indicators of genetic diversity. Moreover, CVA should be higher in more *g*-loaded traits, as *g* has been tentatively demonstrated to have undergone positive selection in *Pan*. The correlations among those vectors were moderate to strong (see: Cohen, 1988; Kotrlik & Williams, 2003) and were all positive (Table 2). Moreover, the loadings of the multivector common factor (comprising *g* loadings, heritabilities, CVAs and CVs) on each indicator were of high magnitude (Table 3), thus they were concordant with the bivariate correlations, corroborating all predictions for the specified Jensen effects.

The observed relationship between *g* loadings and CVs (.26; Table 2) however was smaller than expected (i.e. .46) if trait heritability were assumed to be a complete mediator of the relationship and also smaller than expected (i.e. .40) if CVA were assumed to be a complete mediator of the relationship. The suggests that environmental influences on the ontogenetic development of cognitive abilities may attenuate the relationship between *g* loading and phenotypic variability.

Additionally, it should be noted that, with regard to the multivector common factor, stronger factor loadings were found on the indicators of genetic diversity than on the phenotypic properties of the cognitive tasks, reinforcing the proposal that genetic components are central to the *g*-nexus.

It is also important to note that tool use exhibited the highest interspecies variance and *g* loading in recent comparative (i.e. cross-species) studies involving primates (Fernandes et al., 2014; Reader et al., 2011). In the present analysis it was also found to exhibit the highest heritability, phenotypic variability (CV), and CVA of the thirteen abilities, in addition to exhibiting a high *g* loading. This is consistent with the theory presented by Fernandes et al. (2014) that the need for ecological control is among the most domain-general, and therefore *g*-loaded classes of cognitive problems. Being able to manipulate ecology furthermore has the potential to generate positive evolutionary feedback operating on *g* via niche construction (Odling-Smee, Laland, & Feldman, 2003). There are more than 370 independently reported instances of tool-using behavior in chimpanzees (Reader et al., 2011), far more than in any other nonhuman primate; it is therefore likely that, just as in humans (e.g. Cochran & Harpending, 2009), selection for highly *g*-loaded and highly heritable tool-using behaviors might have been an instrumental driving force in the evolution of chimpanzees.

Thus findings from the study of human intelligence, nonhuman intelligence at the individual level, and comparative intelligence between taxa continue to be largely comparable, consistent with the prediction that the *g*-nexus should generalize to all taxa for which *g* is a central component of cognition, indicating profound homology.

Table 3
Loadings of the vectors of *g* loadings, heritabilities, CVAs and CVs on the multivector common factor using unit-weight factor analysis.

Indicator	Factor loadings
g loading	.67*
Heritability (h^2)	.95*
CVA	.95*
Phenotypic variability (CV)	.86*

* Significant factor loadings ($p < .05$; $N = 13$).

Acknowledgments

This research was supported by NIH grants NS-42867, NS-73134, and HD-60563 to W.D.H. We would like to acknowledge Conor V. Dolan, two anonymous reviewers and also Aurelio José Figueredo for discussions that substantially improved this manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.intell.2015.04.002.

References

Almasy, L., & Blangero, J. (1998). Multipoint quantitative-trait linkage analysis in general pedigrees. *The American Journal of Human Genetics*, *62*, 1198–1211.

Barkow, J., Cosmides, L., & Tooby, J. (Eds.). (1992). *The adapted mind: Evolutionary psychology and the generation of culture*. New York: Oxford University Press.

Bobko, P., Roth, P. L., & Buster, M. A. (2007). The usefulness of unit weights in creating composite scores: A literature review, application to content validity, and meta-analysis. *Organizational Research Methods*, *10*, 689–709.

Chambers, J., Cleveland, W., Kleiner, B., & Tukey, P. (1983). *Graphical methods for data analysis*. Monterey, CA: Wadsworth.

Cochran, G., & Harpending, H. (2009). *The 10,000 year explosion: How civilization accelerated human evolution*. New York: Perseus.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates.

Costello, A. B., & Osborne, J. W. (2005). Best practices in exploratory factor analysis: Four recommendations for getting the most from your analysis. *Practical Assessment, Research & Evaluation*, *10*, 1–9.

Darton, R. A. (1980). Rotation in factor analysis. *The Statistician*, *29*, 167–194.

Fernandes, H. B., Woodley, M. A., & te Nijenhuis, J. (2014). Differences in cognitive abilities among primates are concentrated on *G*: Phenotypic and phylogenetic comparisons with two meta-analytical databases. *Intelligence*, *46*, 311–322.

Figueredo, A. J., Cox, R. L., & Rhine, R. J. (1995). A generalizability analysis of subjective personality assessments in the stumptail macaque and the zebra finch. *Multivariate Behavioral Research*, *30*, 167–197.

Galsworthy, M. J., Arden, R., & Chabris, C. F. (2014). Animal models of general cognitive ability for genetic research into cognitive functioning. In D. Finkel, & C. A. Reynolds (Eds.), *Behavior genetics of cognition across the lifespan* (pp. 257–278). New York: Springer.

Gorsuch, R. L. (1983). *Factor analysis*. Hillside, NJ: L. Erlbaum.

Herrmann, E., Call, J., Hernández-Lloreda, M. V., Hare, B., & Tomasello, M. (2007). Humans have evolved specialized skills of social cognition: The cultural intelligence hypothesis. *Science*, *317*, 1360–1366.

Herrmann, E., & Tomasello, M. (2012). Human cultural cognition. In J. C. Mitani, J. Call, P. M. Kappeler, R. Palombit, & J. B. Silk (Eds.), *The evolution of primate societies* (pp. 701–714). Chicago: University of Chicago Press.

Hopkins, W. D., Russell, J. L., & Schaeffer, J. (2014). Chimpanzee intelligence is heritable. *Current Biology*, *24*, 1649–1652.

Horn, J. L. (1965). A rationale and test for the number of factors in factor analysis. *Psychometrika*, *30*, 179–185.

Houle, D. (1992). Comparing evolvability and variability of quantitative traits. *Genetics*, *130*, 195–204.

Houle, D. (2000). Is there a g factor for fitness? In G. R. Bock, J. A. Goode, & K. Webb (Eds.), *The nature of intelligence* (pp. 149–170). Chichester: Wiley Ltd.

Hughes, A. R., Inouye, B. D., Johnson, M. T., Underwood, N., & Vellend, M. (2008). Ecological consequences of genetic diversity. *Ecology Letters*, *11*, 609–623.

Jensen, A. R. (1980). *Bias in mental testing*. New York: Free Press.

Jensen, A. R. (1987). Individual differences in mental ability. In J. A. Glover, & R. R. Ronning (Eds.), *Historical foundations of educational psychology* (pp. 61–88). New York: Plenum.

Jensen, A. R. (1998). *The g factor: The science of mental ability*. Westport, CT: Praeger.

Jensen, A., & Weng, L. (1994). What is a good *g*? *Intelligence*, *18*, 231–258.

Kan, K. J., Wicherts, J. M., Dolan, C. V., & van der Maas, H. L. (2013). On the nature and nurture of intelligence and specific cognitive abilities the more heritable, the more culture dependent. *Psychological Science*, *24*, 2420–2428.

Kim, H. Y. (2013). Statistical notes for clinical researchers: Assessing normal distribution (2) using skewness and kurtosis. *Restorative Dentistry & Endodontics*, *38*, 52–54.

Kotrlik, J. W., & Williams, H. A. (2003). The incorporation of effect size in information technology, learning, information technology, learning, and performance research and performance research. *Information Technology, Learning, and Performance Journal*, *21*, 1–7.

Lewis-Beck, M. S., Bryman, A., & Liao, T. F. (2013). *Encyclopedia of social science research methods*. New York: Sage.

Macphail, E. M. (1985). Vertebrate intelligence: The null hypothesis. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *308*, 37–51.

Miller, G. F. (2000). Mental traits as fitness indicators: Expanding evolutionary psychology's adaptationism. *Annals of the New York Academy of Sciences*, *907*, 62–74.

Miller, G. F., & Penke, L. (2007). The evolution of human intelligence and the coefficient of additive genetic variance in human brain size. *Intelligence*, *35*, 97–114.

Odling-Smee, F. J., Laland, K. N., & Feldman, M. W. (2003). *Niche construction: The neglected process in evolution. Monographs in population biology*, *37*, New Jersey: Princeton University Press.

Pavlicev, M., Cheverud, J. M., & Wagner, G. P. (2010). Evolution of adaptive phenotypic variation patterns by direct selection for evolvability. *Proceedings of the Royal Society B: Biological Sciences*, *rspb*, *20102113*.

Pedersen, N. L., Plomin, R., Nesselroade, J. R., & McClearn, G. E. (1992). A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychological Science*, *3*, 346–353.

Penke, L., Denissen, J. J., & Miller, G. F. (2007). The evolutionary genetics of personality. *European Journal of Personality*, *21*, 549–587.

Reader, S. M., Hager, Y., & Laland, K. N. (2011). The evolution of primate general and cultural intelligence. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, *366*, 1017–1027.

Rijsdijk, F. V., Vernon, P. A., & Boomsma, D. I. (2002). Application of hierarchical genetic models to Raven and WAIS subtests: A Dutch twin study. *Behavior Genetics*, *32*, 199–210.

Rushton, J. P. (1998). The "Jensen Effect" and the "Spearman–Jensen Hypothesis" of Black–White IQ differences. *Intelligence*, *26*, 217–225.

Rushton, J. P. (1999). Secular gains in IQ not related to the g factor and inbreeding depression—Unlike Black–White differences: A reply to Flynn. *Personality and Individual Differences*, *26*, 381–389.

Rushton, J. P., & Jensen, A. R. (2010). The rise and fall of the Flynn effect as a reason to expect the narrowing of the Black–White gap. *Intelligence*, *38*, 213–219.

Schmidt, F. L. (1971). The relative efficiency of regression and simple unit predictor weights. *Educational and Psychological Measurement*, *31*, 699–714.

Sokal, R. R., & Rohlf, F. J. (1995). *Biometry: The principles and practice of statistics in biological research* (2nd ed.). New York: Freeman.

Spitz, H. H. (1988). Wechsler subtest patterns of mentally retarded groups: Relationship to g and to estimates of heritability. *Intelligence*, *12*, 279–297.

Stearns, S. C. (1992). *The evolution of life histories. vol. 248*, Oxford: Oxford University Press.

te Nijenhuis, J., Kura, K., & Hur, Y. M. (2014). The correlation between *g* loadings and heritability in Japan: A meta-analysis. *Intelligence*, *46*, 275–282.

Woodley, M. A., te Nijenhuis, J., & Murphy, R. (2014). Is there a dysgenic secular trend towards slowing simple reaction time? Responding to a quartet of critical commentaries. *Intelligence*, *46*, 131–147.