The Nature of Intelligence: Novartis Foundation Symposium 233. Volume 233
Edited by Gregory R. Bock, Jamie A. Goode and Kate Webb
 ISBN: 0-471-49434-8

The *g* factor: psychometrics and biology

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Abstract. General ability, defined as psychometric *g*, arises from the empirical fact that scores on various cognitive tests are positively correlated in the population. The *g* factor is highly stable across different factor analytic algorithms, across different test batteries and across different populations. Because all cognitive tests, from the simplest to the most complex, regardless of their informational content, are *g*-loaded to varying degrees, *g* cannot be described in terms of the tests' content, or even in psychological terms. It is actually a property of the brain. The loadings of various tests on *g*, from tests of sensory discrimination and reaction time to those of highly complex problem solving, predict those tests' degree of correlation with a number of non-psychometric variables: the test's heritability, inbreeding depression, coefficient of assortative mating, brain size, reaction time, brain nerve conduction velocity, brain glucose metabolic rate and features of brain evoked potentials. Although some of the brain's cognitive functions are modular, the *g* factor reflects the all-positive correlations among virtually all cognitive functions that show individual differences. I hypothesize that the brain contains no module for general problem solving. Correlations between individuals' performances in various cognitive tasks result from quantitative individual differences in physiological conditions that do not constitute the brain's modular and other neural design features but do influence their speed and efficiency of information processing.

2000 The nature of intelligence. Wiley, Chichester (Novartis Foundation Symposium 233) p 37–57

The *g* factor: psychometrics and biology

The concept of general mental ability was first hypothesized in a scientific context by Sir Francis Galton (1869). It was later empirically investigated by Charles Spearman (1904, 1927), who invented factor analysis as a method for identifying general ability by analysis of the correlations among a number of tests of diverse mental abilities in any group of individuals whose test scores range widely. Spearman labelled this general factor simply as *g*. In discussing individual differences in mental ability, he eschewed the term 'intelligence', regarding it as a generic term for the many aspects of cognition, such as stimulus apprehension, attention, perception, discrimination, generalization, conditioning, learning,

short-term and long-term memory, language, thinking, reasoning, relation eduction, inference and problem solving. A virtually unlimited variety of tasks or tests involving one or more of these cognitive functions can be devised to assess individual differences in level of performance.

It is an empirical fact that individual differences in performance on virtually all such cognitive tests, however diverse the abilities they tap, are positively correlated to some degree. The exceptions are due to statistical artefacts that affect test intercorrelations: measurement error, sampling error and restriction of the range-of-talent. The all-positive correlations among tests mean that individuals who score above the population mean on any given test tend, on average, to score above the mean on all of the other tests, and those who score below average on any given test tend to score, on average, below the mean on all of the others. The existence of the *g* factor depends on this condition and reflects it quantitatively for any collection of diverse mental tests administered to a representative sample of the population.

Psychometric variance

Consider a test composed of n elements (i.e. items or subtests), i, j, etc. administered to a number of individuals. The total variance (V_T) of all the individuals' scores on this test consists of the sum of all the separate item variances (ΣV_i) plus twice the sum of all the item covariances ($2\Sigma r_{ij}\sqrt{(V_i V_j)}$), that is,

$$V_T = \Sigma V_i + 2\Sigma r_{ij}\sqrt{(V_i V_j)} \quad (1)$$

Because the number of correlations among the n elements is $n(n-1)/2$, the sum of the item covariances increases more rapidly as a function of n than the sum of the item variances. In standard test batteries, such as the Wechsler, the Stanford–Binet and the British IQ scales, which have large numbers of items, the item covariances account for about 90% of the total variance. Hence most of a typical test's variance attributable to individual differences in performance results from the correlations, or common variance, among its various elements.

It is also possible mathematically, by means of factor analysis, to express these elements' common variance, not in terms of the various elements themselves, but in terms of one or more linearly independent (i.e. uncorrelated) hypothetical sources of variance (Carroll 1993, 1997, Jensen 1998).

Factor analysis

Factor analysis comprises several closely related algorithms for transforming a matrix of correlations among a number of observed variables into a matrix of

latent (i.e. hypothetical) variables, called *common factors*, each of which represents a linearly independent source of variance that is common to at least three or more of the variables in the analysis. A factor matrix shows the correlations of each of the observed variables with each of the latent variables, or factors. These correlations are called the *factor loadings*. The number of significant factors is typically much smaller than the number of variables. Yet an algorithm applied to the factor loadings can usually reproduce the original correlation matrix within some negligible margin of error. In terms of factor analysis, then, the total variance (V_T) of scores on a test is composed of the variance contributed by the *g* factor that is common to all of the observed variables + the variance associated with *group* factors F1, F2, etc. (so-called because each one is common only to certain groups of variables that share some variance independent of *g*), + all the variance components [*s*] that are specific to each observed variable, + the variance due to measurement error [*e*], thus:

$$V_T = Vg + V_{F1} + V_{F2} + \ldots V_{Fn} + V_s + V_e \quad (2)$$

The V_T in Equation 1 is identical to V_T in equation 2. The second term in equation 1 is equal to the sum of all the common factor variances in equation 2, while the first term in equation 1 is equal to the sum of V_s and V_e in equation 2. From this we see that the second term in equation 1 (which constitutes the test's so-called 'true-score' variance) comprises different sources of common variance, which the orthogonalized hierarchical factor-analytic model divides up into *g*, and a number of other common factors independent of *g* and of each other. Factor analysis is usually performed on the standardized covariances (i.e. Pearson correlation coefficients) rather than on the raw covariances. This type of hierarchical analysis is shown graphically in Fig. 1. Table 1 is the corresponding factor matrix showing the loadings of each variable on each of the orthogonal (i.e. uncorrelated) factors. This represents only one of several different algorithms or factor models for estimating *g* (and other factors) in a given correlation matrix. Provided the mental tests in the analysis are numerous and diverse in the kinds of knowledge and cognitive skills they call for, the obtained *g* factors are highly congruent (i.e. correlations >0.95) across the different methods of analysis (Jensen & Weng 1994). Estimates of *g* are also highly similar across different batteries of numerous and diverse tests, and tests' *g*-loadings remain virtually the same whether extracted from the tests' intercorrelations obtained entirely within families (thereby excluding the effects of all of the shared 'family background' variables) or from unrelated individuals in the general population (Jensen 1998, p 170). In a wide range of different test batteries, depending on the cognitive diversity of their subtests and the range-of-talent in the subject sample, the *g* factor generally accounts for anywhere from about 30–60% of the total variance

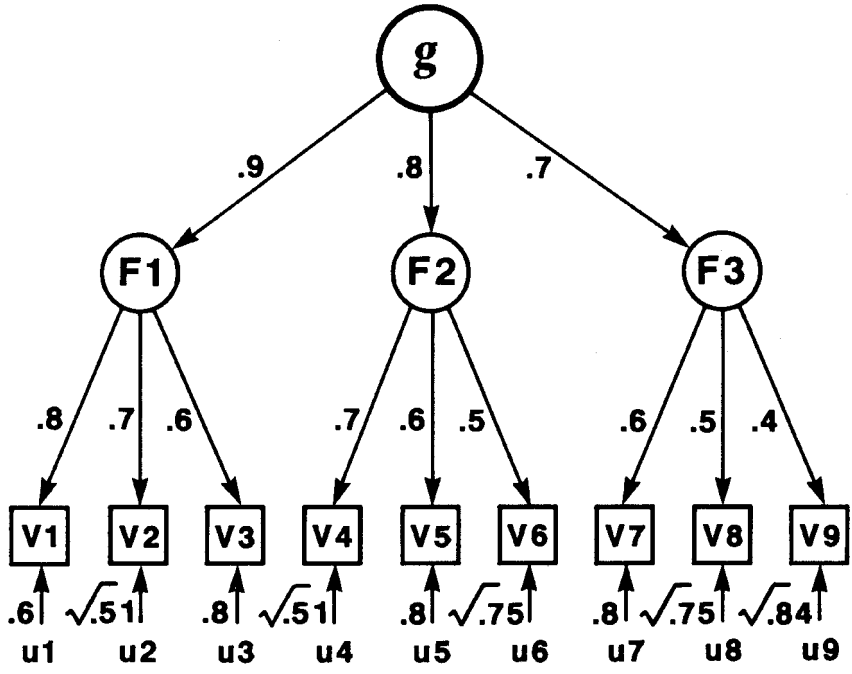

FIG. 1. A hierarchical factor model in which the group factors (**F**) are correlated, giving rise to the higher-order factor *g*. Variables (**V**) are correlated with *g* only via their correlations with the group factors. The correlation coefficients are shown alongside the arrows. The **u** is a variable's 'uniqueness', i.e. its correlation with whatever it does not have in common (i.e. specificity + error) with any of the other eight variables in the analysis. Reproduced from Jensen & Weng (1994) with permission.

in test scores. Most psychometric tests have higher loadings on *g* than on any independent group factors.

Unlike the group factors, which can usually be described in terms of the types of tests (e.g. verbal, spatial, numerical, memory) most highly loaded on them, the higher-order *g* on which virtually all objectively scored cognitive tests are loaded cannot be described in terms of the test's visible characteristics or even the hypothesized mental operations called for by the test. Extremely dissimilar tests requiring very different cognitive skills can have identical *g* loadings. It appears that *g* itself is not really an ability but rather something in the brain that causes all cognitive abilities, however diverse, to be positively correlated to some degree. The *g*-loadings of various tests is a perfectly continuous variable ranging from about +0.10 to about +0.90.

TABLE 1 An orthogonalized hierarchical factor matrix

	Factor loadings			
	Second order	*First order*		
Variable	*g*	F_1	F_2	F_3
V_1	0.72	0.35		
V_2	0.63	0.31		
V_3	0.54	0.26		
V_4	0.56		0.42	
V_5	0.48		0.36	
V_6	0.40		0.30	
V_7	0.42			0.43
V_8	0.35			0.36
V_9	0.28			0.29
% variance[a]	25.4	3.1	4.4	4.4

[a] Per cent of total variance accounted for by each factor.

Besides *g*, which is common to all of the variables, there are three distinct classes of variables here (group factors F_1, F_2, F_3), e.g. verbal, quantitative and spatial reasoning. The original correlation matrix can be reconstituted (usually within a small margin of error) by adding the products of their factor loadings, e.g., the correlation between V_1 and V_2 is $(0.72\times0.63)+(0.35\times0.31)=0.56$. Altogether 37.3% of the total variance in all nine variables is accounted for by common factors, of which *g* is the largest, accounting for 25.4% of the total variance and 68% of the common factor variance. The remaining 62.7% of the total variance (consisting of specificity and error) is unique to each of the variables so does not contribute to their intercorrelations.

Non-psychometric correlates of *g*

Although the *g* factor is necessarily revealed by psychometric methods, it is not exclusively a psychometric construct, nor is it a methodological artefact of the way psychometric tests are constructed or of the particular factor-analytic algorithms used to extract *g*. The extra-psychometric reality of *g* is indicated by the many significant correlations that *g* has with a wide variety of variables, both physical and behavioural, that have no intrinsic or conceptual relationship to psychometrics or factor analysis. In this respect, *g* seems to differ from other psychometric factors (Jensen 1993, 1994).

The method of correlated vectors

Because every psychometric test reflects, besides *g*, its specificity and usually at least one group factor, the correlation between any single psychometric test and some non-psychometric variable is not informative as to precisely which source of

TABLE 2 Example of the method of correlated vectors based on the evoked potential habituation index (EPHI) and the *g* factor loadings of the Wechsler Adult Intelligence Scale (WAIS)

	g factor loadings				*subtest × EPHI correlations*			
	uncorrected		*corrected*[a]		*uncorrected*		*corrected*[a]	
WAIS subtest	*g*	*Rank*	*g′*	*Rank′*	*r*	*Rank*	*r′*	*Rank′*
Information	0.71	10	0.74	10	0.41	8	0.43	7
Comprehension	0.49	5	0.55	5	0.39	7	0.44	8
Arithmetic	0.57	7	0.64	7.5	0.32	5	0.37	4
Similarities	0.59	9	0.64	7.5	0.50	11	0.53	10
Digit span	0.32	2	0.38	2	0.03	1	0.04	1
Vocabulary	0.77	11	0.80	11	0.45	10	0.46	9
Digit symbol	0.26	1	0.27	1	0.17	2	0.18	2
Picture completion	0.46	3.5	0.50	3	0.21	3	0.23	3
Block design	0.50	6	0.54	4	0.38	6	0.41	6
Picture arrangement	0.58	8	0.71	9	0.44	9	0.54	11
Object assembly	0.46	3.5	0.57	6	0.31	4	0.38	5

[a] Corrected for attenuation (unreliability).
From Jensen (1998, p 590).

variance these two measurements may have in common. An efficient, practicable and statistically rigorous way to discover whether a given variable is importantly related to psychometric *g* is the method of correlated vectors, which can show whether the relative sizes of a set of diverse tests' *g*-loadings predicts the degree to which those tests are correlated with some external variable. The method is most easily explained by an example. Schafer (1984, 1985) measured the habituation of the amplitude of brain potentials (EP) evoked by repeated auditory stimuli (50 'clicks' at short random intervals averaging 2 sec) in 50 young adults with IQs ranging from 98 to 142. The index of habituation of the evoked potential (EPHI) is the average amplitude of the EP over the first set of 25 clicks minus the average EP amplitude over the second set of 25 clicks. The EPHI correlated +0.59 with Full Scale IQ on the Wechsler Adult Intelligence Scale (WAIS). But what is the locus of this correlation in the factor structure of the WAIS? The method of correlated vectors, illustrated in Table 2, indicates that the column vector of the WAIS subtests' *g*-loadings is positively and significantly correlated with the column vector of the subtests' correlations with the EPHI, as shown in the scatter diagram in Fig. 2. The *g*-loadings and

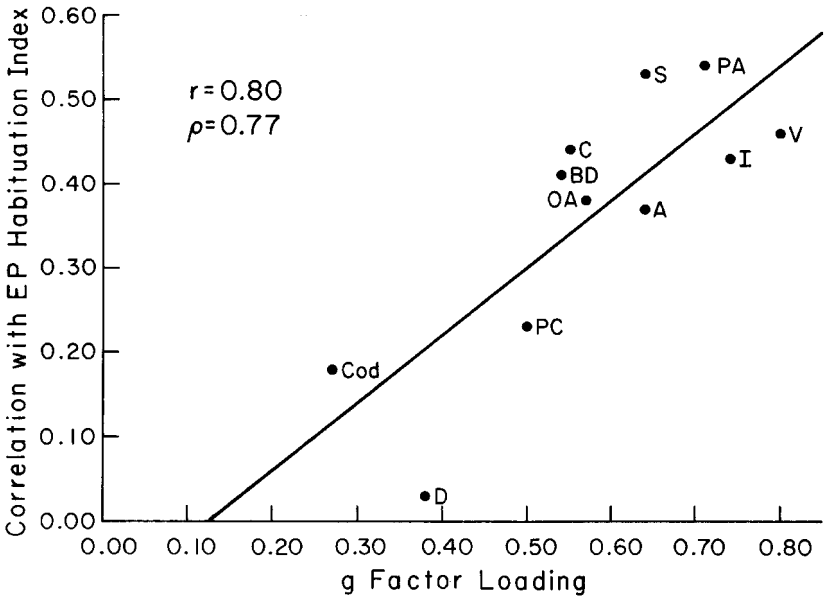

FIG. 2. Scatter diagram showing the Pearson correlation (r) and the Spearman rank-order correlation (ρ) between the correlations of each of the 11 subtests of the Wechsler Adult Intelligence Scale with the evoked potential (EP) habituation index (on the vertical axis) and the subtests' loadings on the *g* factor. The subtests are: A, arithmetic; BD, block designs; C, comprehension; Cod, coding; D, digit span; I, information; OA, object assembly; PA, picture arrangement; PC, picture completion; S, similarities; V, vocabulary. Reproduced from Jensen (1998) with permission.

correlations are corrected for attenuation to rule out any correlation between the vectors because of correlated errors of measurement. Spearman's rank-order correlation, which minimizes the effects of outliers, is used to test the statistical significance of the correlation between the vectors. (For the statistical rationale and variations of this method, see Jensen 1998, p 589.) Finally, when *g* is statistically partialled out of the WAIS subtests' correlations with the EPHI, all of the partialled correlations diminish to near-zero, as does the overall correlation between the Full Scale IQ and EPHI.

The same method of correlated vectors based on the *g*-loadings of many different psychometric tests has revealed the predominant relationship of *g* to various non-psychometric variables in studies from different laboratories around the world (all of them referenced in Jensen 1998). Typical vector correlations are shown in parentheses:

- Scholastic performance (0.80).
- Occupational level (0.75).

- Assortative mating correlation between spouses' test scores (0.95).
- The genetic heritability of test scores (0.70).
- Inbreeding depression of test scores in offspring of cousin mating (0.80).
- Heterosis — outbreeding elevation of test scores in the offspring of interracial mating (0.50).
- Reaction time on various elementary cognitive tasks (ECTs) (0.80).
- Intra-individual variability in RT on ECTs (0.75).
- Head size as a correlated proxy for brain size (0.65).
- Habituation of the amplitude of brain evoked potentials (0.80).
- Complexity of waveform of brain evoked potentials (0.95).
- Brain intracellular pH level; lower acidity → higher *g* (0.63).
- Cortical glucose metabolic rate during mental activity (−0.79).

In addition, there are numerous studies that have shown significant and substantial correlations of certain sensory and brain variables simply with IQ, which is always highly *g*-loaded but may also contain other factors: visual, auditory and tactile discrimination; brain volume measured *in vivo* by magnetic resonance imaging (MRI); EEG coherence; event related desynchronization of brain waves; frontal lobe alpha brain wave frequency; and many other physical variables less obviously related to brain functions (Jensen & Sinha 1993). Hypothesizing that the physiological basis of *g* results in part from individual differences in nerve conduction velocity (NCV), Reed & Jensen (1992) demonstrated a relationship between non-verbal IQ (Raven's matrices) and NCV in a brain tract from the retina to the visual cortex. The result, shown in Fig. 3, was recently replicated (A. Andres-Pueyo, R. M. Boastre & A. Rodrigues-Fornells, unpublished paper, 9th Biennial Convention of the International Society for the Study of Individual Differences, 6 July 1999). Of course, to serve as reliable clues for developing a physical theory of *g*, the results for all of the physical variables listed above require replications.

Toward a theory of *g*

Although the present findings provide clues for possibly explaining the physical basis of *g*, we are still far from having a full-fledged theory of *g*, which must consist of more than just a collection of correlations. Understanding and explaining these correlations beyond psychometrics, that is, at a causal level, calls for the involvement of molecular genetics, the brain sciences (including animal models) and evolutionary psychology.

The task ahead may seem less daunting if we keep in mind the conceptual distinction between intelligence and *g* (or other psychometric factors). Intelligence involves the brain's neural structures or design features, circuitry

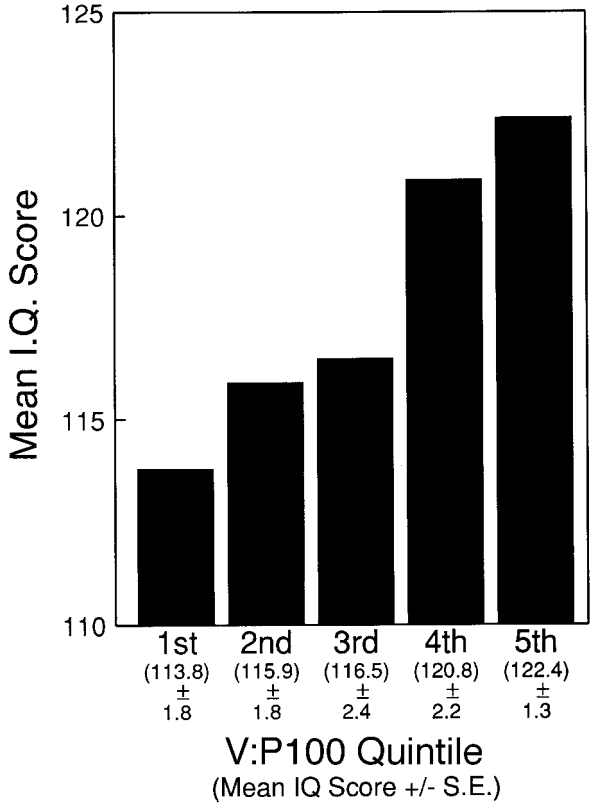

FIG. 3. IQ means (in parentheses) ± standard errors of each quintile of the distribution of nerve conduction velocity (NCV) measured in the visual tract in 147 male college students. This sample comprises only the top one-third of the IQ distribution in the general population. Individual values of velocity (V:P100) were based on the P100 latency of the visual evoked potential. The measures of NCV in this sample range from the slowest at 1.75 m/s to the fastest at 2.22 m/s. The Pearson *r* between NCV and IQ is 0.26 ($P < 0.002$); corrected for restriction of IQ range in this college sample, $_cr = 0.37$. Reproduced from Reed & Jensen (1992) with permission.

and specialized modules that enable various behavioural capacities that are common to all biologically normal members of a given species — capacities such as learning, memory, language and reasoning in humans. The *g* factor results from some condition(s) of the brain that causes correlation between individual differences in the speed and efficiency of operation of these diverse capacities and probably governs the asymptote of their growth or development under optimal environmental conditions. These two conceptually distinct aspects of brain

function most likely have different physiological bases. Considering the great anatomical similarities between primate brains in their non-quantitative structural features, it seems unlikely that there are individual differences in the design features and operating principles of biologically normal brains within the same species. It seems more likely that the source of individual differences, hence *g*, lies in some quantitative features of the brain that affect many of its diverse cognitive processing mechanisms in common (Jensen 1997). A crude analogy would be like comparing different makes of cars that differ in quantitative performance indices such as horsepower, top speed and fuel efficiency. All the autos have internal combustion engines (i.e. the same operating principles), but these can differ quantitatively in number of cylinders and different cubic capacities, running on gas of different octane grades, and these variables are all positively correlated across the different cars — hence individual differences in overall performance.

Why is *g* related to brain size? This relationship *per se* is well established and may account for as much as 20% of the *g* variance, but its basis is still conjectural. Is it total number of neurons in those cortical regions that serve cognitive functions? Amount of dendritic arborization? Degree of myelination of axons, which affects nerve conduction velocity? Number of glial cells, which nutritionally support the myelin? Why is *g* inversely related to glucose metabolic rate in the active brain? Does the implied efficiency involve differences in brain chemistry, such as different concentrations of neurotransmitters (e.g. acetylcholine, glutamate, aspartate) or inhibitors that commonly affect chemical receptors in various cognitive neural systems or modules? Why do *g* factor scores show a curvilinear (inverted U) relationship to testosterone levels in males (Nyborg & Jensen 1999)? Do other hormones also affect *g*?

The first steps in the reductionist study of the basis of *g* call for securing beyond question the physical correlates of *g* already mentioned as well as other possible correlates yet to be discovered. The next steps will necessarily measure as many of these brain variables as possible in the same group of individuals. Analysis of the correlations among individual differences in these variables might be able to identify the one variable, or the few variables, that account for most of the heritable variance in *g*.

Is it all too fantastic to predict that there will be found a general factor in the correlations among some small number of brain variables — histological, biochemical, physiological — and that this general factor will prove to be coincident with psychometric *g*? I am betting on it. Such an outcome would be a major advance toward the kind of theory of *g* originally envisaged by Spearman (1927), who wrote that the final understanding of *g* '. . . must come from the most profound and detailed direct study of the human brain in its purely physical and chemical aspects' (p 403).

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DISCUSSION

Hinde: I wanted to ask a question about causation. You showed that assortative mating was correlated with the *g* factor, and you said that people were choosing partners with similar genes. I understand that there's also a strong correlation between the length of the ear lobe between partners, implying perhaps that partners choose each other on the lengths of their ear lobes! What does one conclude from all this? Is the ear lobe part of *g*?

Jensen: On some of these physical features connected with IQ or *g* you can't find any causal connections, but you can be interested in what might be called the cultural anthropology or sociology of some of these correlations. For example, IQ is correlated with a host of physical variables that certainly have no causal connection with IQ, such as height. Height is positively correlated about 0.2 with an IQ in the population. This can be shown not to be a functional correlation, but it comes about through assortative mating. Both height and IQ are valued in our society, and these are both selected together in mate choice.

Therefore the genes for both of them show up in the progeny of people who are tall and intelligent, but they're not functionally related. You can show this by the fact that within families there's no correlation between height and intelligence, whereas the correlation is 0.2 in the population. If you take the taller siblings in families, the average IQ of those siblings will be the same as that of the shorter siblings. Now this isn't true of some traits, such as myopia. If you take the more myopic children in a family they will have higher IQs than the non-myopes in the same family. This is a kind of pleiotropic relationship rather than just a simple genetic correlation. There are other examples: the ability to curl the tongue is correlated with IQ. A single gene makes this possible. No one knows why that should be correlated with IQ. I wrote a whole chapter (Jensen & Sinha 1993) on physical correlates of IQs — it is a rather amazing collection of characteristics. Some of them are functionally related and some are not. Brain size is one that is functionally related, and shows up within families as well as between families: there's a correlation of about 0.4 between brain size and IQ.

Harnad: I am a tremendous admirer of your work, and so what I am about to say, although it is critical, is only about its limits rather than its limitations. You made an excellent description of the extraction of *g*, but what was left out of the description is how *g* is interpreted. There is a huge hermeneutic component to psychometric analysis. The empirical part is the calculation of the correlations in the extraction of the factors; the hermeneutic part is in interpreting the factors, figuring out what on earth they may mean. Of course, all you have to go by is patterns of correlation. Yet, I think one of the themes of this symposium is causation, and causation with dimension. I want to suggest that in the extension of the psychometric paradigm, which is a correlation-plus-hermeneutics paradigm, you get your factors and then you try to look back at the clusters of things that load on factors and guess what might be behind them — that's where the inference comes in. Do we get beyond hermeneutics when we add to the psychometric battery, a biometric battery? I want to suggest you don't: you are still stuck in the same paradigm. It is not just *psycho*metric anymore to be sure, but you're still in the business of looking at correlations and trying to guess hermeneutically what might be behind them, whereas what you really want is to find the underlying causal mechanism. To find this, you have to break out of the hermeneutic circle, because it won't be given to you by the loadings on *g*.

Jensen: The loadings of these physiological variables on *g* affords better clues than sheer guesswork as to where to look for causal mechanisms. For example, you can do a preliminary analysis to see whether some relationship is functional or not, looking to see whether you get within- or just between-family correlations, or both between and within. If you don't get any within-family correlations between two variables, they're not functionally related, so you can dismiss them. However, it may still be of interest to the cultural

anthropologists and sociologists as to how those things got together, as in the case of height and intelligence.

Flynn: Are you saying that if you take a group of people who are one standard deviation above the mean in *g*, their degree of assortative mating will be greater than people one standard deviation below the mean in *g*? That is, the actual correlation between the spouses' IQ rises as *g* rises. Is this your point?

Jensen: That's not what I'm saying, but it happens to be true. There is a higher degree of assortative mating above the mean in the bell curve that there is below. The bell curve is not a perfectly normal Gaussian curve: it has a bump at the low end for types of mental defect and also an excess at the upper end. One explanation for this deviation at the upper end is that there is a higher degree of assortative mating in the upper half of the curve, which increases the genetic variance and pushes more offspring into the upper end of the curve.

Flynn: That is very interesting, because it seems to be another case of high IQ seeking out an enriched environment. After all, one's spouse is a great part of one's intellectual environment. Apparently, the higher you go up the IQ scale the greater the match with your spouse's IQ.

Jensen: Society itself helps a lot with that in the educational system, because graduate students don't often marry high school dropouts — they hardly ever meet them.

Flynn: Ulrich Neisser, in his review of your book *The g factor*, points out that when you compare reaction times in people with higher and lower IQs, the maximum responses are very similar — it tends to be more the variance that separates the two. Is that correct, and if so what do you make of it?

Jensen: Yes that's correct. Even comparing Berkeley students with mentally retarded people in institutions, their fastest reaction times do not differ all that much, but the retarded people produce many more slower reaction times. A more important correlate of *g* than reaction time is the intra-individual variance in reaction time: brighter people show less variation from trial-to-trial of a reaction time test than less bright people.

Flynn: Do you have any physiological explanation of that?

Jensen: No, but there are hypotheses, such as the theory that there's simply more noise in the nervous systems of lower IQ people, and that this variation from trial-to-trial in reaction time tests reflects neural noise, whatever that may mean. This should be investigated, because it's a more striking correlate of IQ than is reaction time itself.

Humphrey: I want to come back to the question of correlation and causation. It's tempting of course to assume that a relation between two variables is causal when we can see how it would work, but to assume it's a mere correlation when we can't see it. So, when we find that IQ correlates with brain size or head size, we think that's probably because large brains do indeed cause high IQ. But when we find IQ

correlates with height or ear lobe size we don't think the relationship is causal — instead we postulate, for example, that bright men want to marry tall girls. But we should be careful. Because even in the case of IQ and brain size, the relationship may not be what we think it is. In fact there is very good reason to suppose that brain size really can't be the cause of IQ — at least in any straightforward way. John Skoyles (1999) in a recent paper has drawn attention to the fact that people with brains as small as 800–900 cm^3 can have more or less normal IQ.

Jensen: That will always happen when there is a correlation as low as 0.4. In fact, one of my former graduate students has been studying midgets. He was interested in the brain size-intelligence correlation, so he's gone to Ecuador where there are true midgets who are perfectly proportioned and have head sizes similar to a three-year-old child. He has collected some 80 of these individuals, and they have perfectly normal intelligence when given IQ tests (Kranzler et al 1998). This shows that variation in head size itself is not a crucial factor in intelligence: it's neither necessary nor sufficient to have a large head for above average IQ.

Humphrey: This has evolutionary implications. It suggests that our *Homo erectus* ancestors, who had brains of about 750–800 cm^3, may well have had the capacity for an IQ or *g* equivalent to that of modern humans. We should perhaps therefore be thinking of explanations of the doubling of brain size since then, other than that it was just needed to increase general cognitive abilities.

Jensen: If you read some of my writings on this, I claim that the correlation between brain size and IQ is still a mystery: we don't know what there is about brain size that makes it correlated with IQ, but it certainly is — you can't deny a correlation of 0.4. Many different studies have been done on this now. It is an interesting scientific question as to whether there is a causal relationship or not. It may be a sociological kind of correlation, or it may actually be functional one. I would suspect a functional explanation in the case of brain size, because a larger brain size is not evolutionarily a good thing in its own right, unless it confers advantages such as increased behavioural capacity.

Humphrey: Among other things it confers strong resistance to dementia: if you are in the lower quintile for brain size you have three times the risk of Alzheimer's disease (Schofield et al 1997).

Deary: Would that affect fitness? Alzheimer's disease usually occurs so long after the age of reproductive activity that I can't imagine it having a fitness effect.

Humphrey: Fitness effects can occur after reproductive age. Grandmothers, for example, are increasingly being seen as important for the fitness of their grandchildren and perhaps even their great-grandchildren. For that matter, men can remain reproductive well into the age when they are beginning to suffer from all sorts of brain deterioration of the kind for which larger brain size provides protection.

Deary: Was the average lifespan of people during the time this would have been selective getting to the age where Alzheimer's is common?

Humphrey: I don't think we can be sure of that at all. I have discussed the issue in a recent paper (Humphrey 1999).

Houle: I'm interested in the point you've made about within- versus between-family correlations because it seems to me that you are drawing an incorrect conclusion. Assortative mating involving pairs of traits, such as height or brain size, for example, even if they are not causally related to each other at all, will cause genetic associations between these traits through linkage disequilibrium. This effect will be stronger for loci that are closely linked to each other. This will cause within-family correlation. The conclusion I would draw when you have assortative mating and find a lack of within-family correlation, is that the assortative mating is actually not on the genetic component of the traits being considered, but on the environmental deviations from the breeding value.

Jensen: That's possible, but I have been told by geneticists that the linkage disequilibrium would not account for within-family correlations beyond the first generation. This is something that washes out very quickly. In the general population, if you have a large sample and look for these correlations, very little of it would be caused by linkage. It would be more pleiotropic, meaning that one gene has two or more apparently unrelated effects.

Houle: It depends on the assumptions you make. If you assume very simple genetics — for example, one gene influencing each trait — they are very unlikely to be closely linked. This would, to a large extent, get rid of this effect, but not entirely. Since traits such as brain function and height are the product of many genes some loci are bound to be closely linked, so any association would decay slowly for these loci; it's very unlikely that you would be able to wash that out completely. The thing about assortative mating is that it occurs every generation so those correlations are constantly being reinforced: they won't be large, perhaps, but they won't be zero either. So if you can confidently say there's no within-family correlation, you're actually making a strong statement about the genetic relationship of genes to those traits.

Jensen: That's a good point.

Whiten: It was interesting that the hierarchical factor structures that you came up with can apparently be accommodated within just three levels, or even sometimes fewer. This becomes interesting if it represents a finding about the natural world that we might not have predicted in advance. You seem to be saying that it is not merely a mathematical or statistical feature of factor analysis. This leads to two thoughts. First, could this be an answer to the question Stevan Harnad originally asked, about what this tells us about cognition? If this is a discovery about the natural world — that this hierarchical structure exists — this could be one answer to Stevan's question. Second, if this is a finding about the natural world, is it about

intellect in particular? Is there any other set of biological data that has been looked at by this factor analytic approach, which actually produces more than three levels? Or is there just something about biological data of this kind that they naturally fall into this very economic number of levels?

Jensen: I don't know the answer to that. I know that there have been factor analyses of up to 50 different body measurements, but I can't recall the hierarchical analysis. There is a general factor in body measurements. These measurements still exist: the British garment industry has collected about 50 body measurements on 10 000 women, and the correlation matrix exists. When I took a course on factor analysis, our final exam project was to factor analyse this huge correlation matrix which in those days took a 40 hour week to do on a desk calculator! There was a big general factor and about four or five other factors that were large enough to be significant.

Rutter: I'd like to return to the topic that John Maynard Smith posed at the beginning, in terms of the biological significance of *g*. It is still not clear to me what postulate either David Lubinski or Arthur Jensen is putting forward. The workings of the mind have to be based on the functioning of the brain. But it is not obvious what more one can conclude. If one takes James Flynn's findings on the rise in IQ over time, that rise was paralleled by a rise in head size. Similarly, in our own study of adoptees from very deprived Romanian institutions (Rutter et al 1998), their head size at the time of entering this country was well below UK norms, as was their developmental quotient, but two years later both had risen greatly. There are good reasons for inferring that the initial deficits were due to institutional deprivation and that the rise was a function of the much better rearing conditions in the adoptive homes (O'Connor et al 2000). Obviously, the improved cognition must reflect the functioning of the brain but where does that get us? You are saying that *g* is not a 'thing', so what use it is?

Jensen: It is not a 'thing', but *g* is instead the total action of this number of things. Brain size may be correlated with intelligence because there are more brain cells in more intelligent people, so this is something that can be investigated. You would be one step further ahead if you found that to be the case, or even if you found it not to be the case. Then the next thing that you could look at may be the amount of myelin in the brain: myelin controls the speed of neural conduction, and we know that cognitive capabilities increase with age and myelination increases with age; we demyelinate as we get old and cognitive functions begin to decline, and so on. One can simply go through these correlates of *g* and investigate them empirically. My view is that the only place to go with this kind of research on *g* and mental abilities is into the brain itself. We have to figure out strategies for zeroing in on those aspects of brain function that can be said to be causal of the *g* factor.

Rutter: Isn't there a danger of unwarranted biological determinism? For example, in studies of individuals with obsessive disorders there are differences in

PET scan findings, and treatment changes that. But psychological treatments cause the same changes as do pharmacological ones (Baxter et al 1992). The abnormal behaviour and the brain functioning are meaningfully associated, but it doesn't necessarily follow that the behaviour is being driven by something that is biologically more basic.

Jensen: That is true, but I don't think we can give up the enterprise of trying to get a neurological or brain account of the kinds of phenomena that I've shown here. We are at the frontier of this research on the Galtonian paradigm. I can't see anywhere else left to go.

Maynard Smith: Your talk cleared up many of my difficulties. But the thing that became quite clear from the last part of your paper is that although you expect differences in cognitive ability to be reflected in differences between brains (it would be bloody weird if that were not the case), you are not looking for a single kind of difference between brains. In other words, you really rather expect all sorts of quite different anatomical and physiological measures on brains to have some effect upon your measurements of *g*. You are therefore looking for a multicausal, multifactorial basis for differences in cognitive ability. This is entirely reasonable, but in a sense you are not really looking for something like ethanol. The point about your ethanol example is that this is one factor.

Jensen: Every analogy only goes so far.

Maynard Smith: I liked the analogy, but you weren't implying, were you, when you used that analogy that you are really looking for one thing?

Jensen: This one thing is just a component of variance, not necessarily one brain process.

Brody: I have a very simple methodological question, which derives from the comment about the role of linkage. What about analyses using genetic covariance approaches in which you contrast correlations among monozygotic (MZ) and dizygotic (DZ) twins, with an effort to see whether or not the genes that are contributing to intelligence are co-varying in that way? For example, some studies suggest that assortative mating is not genetically covariant, even though it has a high correlation with your *g* factor. The correlation between MZ twin spouses in intelligence is no higher than the correlation of DZ twin spouses. This seems to come about solely because of social homogamy effects: people just get tossed together who are somewhat equal in IQ in social settings. The data on head size are ambiguous or undecided with respect to whether or not IQ and head size are genetically covariant. On the other hand, if you found that there are genetically covariant relations underlying these correlates, is that design sufficient to move you a step forward in the way that a comparison of between- and within-family correlations may not be?

Rutter: That is an interesting finding: can you say more about the studies that is based on?

Brody: This is a general and interesting phenomenon. It is something that people in evolutionary biology might tell someone like me a lot more about. Lykken & Tellegen (1993) looked at correlations between the spouses of MZ twins and DZ twins. We know that MZ twins are more alike primarily for genetic reasons, and that if people select spouses who are genetically similar to themselves you would expect the spouses of MZ twins to be similar in a way that the spouses of DZ twins are not. It turns out that across a large range of characteristics this is not the case. They argue that attraction to others is a kind of evolutionary mechanism to create genetic diversity, and people are simply attracted to people who are not necessarily genetically similar. People who study relationships often point out that it's very hard to know why people are initially attracted. You can sometimes predict whether people will stay together or break up, by differences in political attitudes, for example. But the initial attraction seems almost like a random phenomenon.

Hinde: There is a vast literature on the attractiveness of attitude similarity. This is presumably not very much genetically determined.

Miller: The biological correlates of *g* come back to Stevan Harnad's question about the hermeneutic interpretation of what *g* means. The last 10 years of work on the biological correlates keeps us from jumping to a cognitivist interpretation of *g* that would have been popular 20 years ago, when people tended to interpret *g* as meaning that perhaps there is some sort of general purpose processing device in the human brain, or some general purpose learning device. The explanation of *g* tended to be at the psychological level, and the biological correlations expand the possibilities for interpreting what *g* really is. It is not necessarily a psychological phenomenon at all: you can measure it psychometrically, but that doesn't mean that it taps into a unitary cognitive ability, for example.

Nesse: I would like to address this question of how we can account for the correlations that we're finding between measured intelligence and various other things. When one finds intelligence as a strongly heritable trait, correlating with another strongly heritable trait such as myopia, for instance, it is tempting to assume that the association must be a pleiotropic effect or some other explanation based on genetics. On the other hand, there are other possibilities. In the case of myopia, there's a very plausible explanation for the correlation aside from genetic pleiotropy: people who are more intelligent are more likely to read earlier in life, because they're capable of it or more interested in it. It is clear that reading early in life is a precursor to myopia in those who are genetically predisposed. Thus we have an alternative mechanism that goes via intelligence to a preference for a behaviour, to a pathological state. The association turns out not to be genetic.

Jensen: Most researchers studying myopia have already dismissed that as an explanation for myopia. They find that retarded children who never take to reading or any other kind of near work have the same frequency of myopia.

Nesse: It would take us off topic to go too far into this, but the study I like the best is the one on Inuits, where on a population basis the rate of myopia was very low prior to institution of schooling, but increased rapidly afterwards (Norn 1997). It is very hard to imagine how severe myopia could be compatible with any high reproductive success on the African savannah.

Jensen: The latest opinion on this among myopia researchers is that it is caused by an interaction between some genetic predisposition and these other factors.

Nesse: Since we are on this subject, let me take the opposite point of view for a moment and turn to mechanisms. It appears that what's going on in myopia is that the eye grows to the right distance so that things focus. It is not pre-programmed, but based on feedback mechanisms. Blurry images cause the eyeball to grow until the image is in focus again, much like an automatic slide projector, but much better. And it appears that there's a strong genetic difference in how fast that happens, or whether the process stops at a certain point or not. I could imagine that in those people in whom the eyeball grew faster, or in whom that mechanism was programmed quite differently, this is related to some other brain function that could conceivably account for IQ. Another theme here is trying to see the specific mechanisms responsible for these correlations.

Houle: I'm concerned with the assumption that figuring out mechanisms is what this meeting is all about, or should be about. There are several overlapping questions here. How does the brain work and what's the relationship of brain function to *g*? What's the practical validity and the predictive usefulness of *g*? How does *g* evolve? Finally, we can ask what causes variation in *g*? These are very different questions; they're overlapping but not entirely the same.

I think that the next step forward in understanding variation in *g* is clearly not resting with any of us in this room — it is resting with the people who are going to map the genes responsible for variation in *g*. This will offer a clear explanation of what causes variation in *g*, but it's not necessarily going to tell us much about how the brain works. By the same token, evolutionary questions may or may not depend on the genetic details of what's going on here. Darwin invented the whole field of evolution before anyone worked out the mechanism of inheritance. There is more to this work than simply tracing everything down to causal mechanisms.

Rutter: In terms of causal mechanisms we need to come back to James Flynn's point, that the explanation for individual differences may or may not be the same as the explanation for changes over time. This is an empirical question. It's easy to think of examples where the causes are quite different. There are other examples where they probably are very similar.

Deary: The genetics of apolipoprotein E (ApoE4) have shown us that the individual differences in mental ability might have different causes at different ages (MacLullich et al 1998). If one has the ε4 allele of this gene one is more liable

to get dementia. This raises the possibility, firstly, of giving us a clue as to where to start looking for mechanism once we get a gene–ability association and, secondly, warning about the fact that that the genetics of intelligence might differ across time as well. Any one gene–ability association clue is liable to give us a small amount of the variance in a mental ability and it could just be the beginning of a very long series of causal mechanisms, possibly disappearing in so many biochemical processes that it is impossible to link genes to behaviours.

Rutter: At first, some investigators seemed to imply that ApoE4 might cause Alzheimer's disease directly despite the evidence that the association was only probabilistic. Individuals with the $\varepsilon 4$ allele did not necessarily develop Alzheimer's disease and those with other alleles also developed Alzheimer's disease, although they did so less frequently. It is now apparent that, in addition, ApoE4 also predicts response to head injury (Teasdale et al 1997) and to cerebrovascular accidents (McCarron et al 1998). The implication is that the genetic effect may concern brain responses to a range of environmental hazards and not just predisposition to a single disease, Alzheimer's disease. But it is not known whether the effects involve one or several different causal mechanisms.

Gangestad: It seems that one of David Houle's points is that the factors that give rise to the genetic variance in *g* may have little to do with the brain mechanisms that underlie the cognitive abilities that are captured by *g*. For instance, it's possible that mutations across the whole genome contribute to that variation, but contribute to variation in lots of other traits as well. The genes may have little to do with the actual brain mechanisms.

Deary: Spearman addressed the problem of how the brain works in general in one book, and the individual differences in another. His 1923 book was called *The nature of intelligence and the principles of cognition*. The 'nature of intelligence' isn't what the differentialists are telling us about here. It wasn't about the individual differences in mental abilities: it was actually the ordinary, average (modal) function of the brain. Unfortunately, Spearman did that from the armchair, using a philosophical approach. In contrast, his 1927 book is full of data, and it's all about the individual differences in human mental abilities. He did, though, try to tie the two of them together: the modal function and the differences. He realized as early as the 1920s that one might or might not need to know the average function of the brain before one could account for the individual differences. These issues have been laid out long and wearily, and we are admitting that we still don't seem to know the answer today. Certainly, those of us who are studying individual differences haven't waited for the biological or cognitive architecture to arrive pre-packaged: we've gone on anyway with the crumbs from the cognitivist's and biologist's tables and seen whether their parameters are any good in predicting individual differences (Deary & Caryl 1997). As I will tell you after lunch, they are not particularly good.

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