The nature of nurture: the role of gene-environment interplay in the development of intelligence

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CHAPTER 6

THE RELATIONS AMONG *g* LOADING, HERITABILITY, AND CULTURAL LOADING: DO CURRENT THEORIES OF INTELLIGENCE EXPLAIN THEM?

Abstract

A positive correlation between intelligence subtests' g *loadings and their heritability coefficients (*h^2*) has been interpreted in support of a real* g*, i.e., a unitary, largely genetically influenced (biological) variable that mediates between genes and all cognitive abilities measured by these subtests. This correlation may exist, but does not provide sufficient evidence for such substantive interpretation, because it is accompanied by an association that* g *theories do not account for: the highest heritabilities and* g *loadings are for the most culturally loaded tests (see Chapter 3). Sampling theories and reciprocal interaction theories do not explain this joint effect either, but combinations of these theories are promising.*

6.1 The Jensen Effect for Heritability

A correlation between intelligence subtests' factor loadings on the general factor of intelligence and their heritability coefficients has figured prominently in theoretical discussions concerning the scientific status of this factor. These discussions include related questions as whether the general factor (*g*, for short) is unitary or not (e.g, Petrill, 1997), whether it represents a realistic variable or is merely a statistical entity (e.g. Gray & Thompson, 2004; Rushton & Jensen 2010a), and whether specific cognitive abilities 'feed into' a general cognitive ability ('bottom up') or vice versa ('top down') (Plomin et al., 2008, p.183). According to Gray and Thompson (2004, p. 476), for instance, a correlation between *g* factor loadings and their heritability coefficients (h^2) "favours a biological over a purely statistical explanation of *g*".

Rushton and Jensen (2010a) made a similar point, and interpreted this correlation, which they dubbed a 'Jensen effect for heritability' as follows:

> "A Jensen Effect for heritability provides biological evidence for a true genetic *g*, as opposed to the mere statistical reality of *g*. It makes problematic theories of intelligence that do not include a general factor as an underlying biological variable […] such as the model proposed by Dickens and Flynn (2001), and the mutualism model by van der Maas, Dolan, Grasman, Wicherts, Huizenga, and Raijmakers (2006)." (Rushton & Jensen, 2010a, p. 213)

We take this to mean that a positive correlation between *g* loadings and heritability coefficients supports theories of intelligence that include *g* as a (single) variable that mediates between genes and all cognitive abilities measured by IQ tests. We denote these theories *g* theories. By implication, the correlation poses a problem for theories in which the general factor of intelligence is viewed as purely a statistical entity (Dickens and Flynn, 2001; van der Maas et al., 2006; Thomson, 1951; Bartholomew, Deary, & Lawn, 2009). We denote these theories alternative theories.

Indeed, it is important theoretically (but also empirically) to distinguish between, on the one hand, the general factor of intelligence as a statistical entity and, on the other hand, its interpretation as a realistic (e.g. psychological or biological) common cause of individual differences. First, because different processes can produce very similar factorial structures of intelligence tests (Anderson, 2001; Bartholomew, Deary, & Lawn, 2009; Dickens, 2008; Thomson, 1951; van der Maas et al. 2006), factor analysis in itself cannot determine whether the statistical general factor actually represents a realistic variable. Second, both *g* theories and alternative theories can explain the facts that the statistical general factor is highly heritable and has biological correlates, hence these facts do not differentiate either. In this light, the Jensen effect for heritability is regarded as a key

argument that *g* represents a real, rather than a statistical variable. Putting aside the issue whether there is empirical evidence for this effect, the question that arises immediately is *why* this effect should provide such evidence.

6.2 Does a Jensen Effect for Heritability Provide Evidence for a Real *g*?

Consider the (fully standardized) regression model displayed in Figure 6.1 (left panel). Here, intelligence subtest scores (IQ scores) are regressed on the latent (unobserved, unmeasured[13]) variable *g* (with regression weights λ_i, for $i = 1... n$); *g* in turn is regressed on latent (unobserved, unmeasured) genetic (A) and environmental (E) influences (with regression weights *h*, respectively *e*). First suppose that the more specific variables that influence IQ subtests are purely environmental (so that each h_i^2 is zero). In this situation, if the factor *g* represents a real, genetically influenced, common cause of individual differences in IQ, the subtests' regression weights on *g* (λ_i, '*g* loadings' in factor analytic terms) and the regression weights on the genetic influences (i.e., the *g* loadings multiplied by *h*: $h\lambda_i$) are collinear. In this case, the subtests' heritability coefficients will take the value of $h^2\lambda_i^2$. The rank correlation between the subtests' *g* loadings (λ_i) and their heritability coefficients ($h^2\lambda_i^2$) will be perfect (+1). Next, suppose that the more specific factors of intelligence are heritable as well, due to specific genetic variance. The correlation between *g* loadings (λ_i) and heritability coefficients (which now take the value of $h^2\lambda_i^2 + [1- \lambda_i^2]h_i^2$) will be attenuated. However, we may still expect a positive correlation if the common genetic variance, h^2, is relatively large compared to the subtests' specific genetic variances, h_i^2.

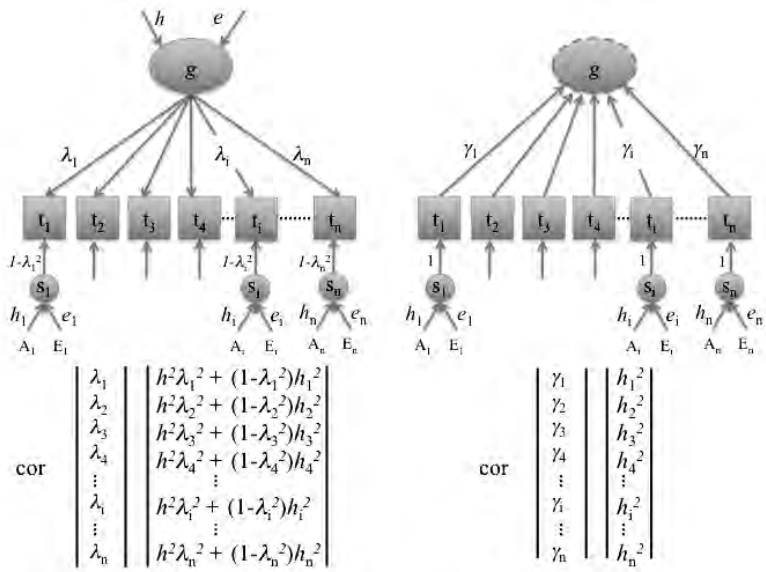

Figure 6.1 If *g* is a realistic, genetically influenced variable (left panel) and the only genetic influence on the subtests, the ranks of the subtests' factor loadings and heritability coefficients are collinear, because both vectors only depend on *l*. The rank correlation is then +1. If specific are also heritable this correlation will be attenuated. If *g* is merely a statistical summary of heritable indicators (right panel), a relation between the subtests' regression weights and heritability coefficients is not expected, unless additional assumptions are made.

[13] *g* is a higher order factor. IQ tests *estimate g* to a certain extent, because the cognitive abilities measured by IQ tests (on the zero or first order level) are influenced by *g*. The tests do not *measure g* directly.

Now suppose that the common factor is merely a summary of diverse heritable cognitive abilities and does not exist in reality. For instance, suppose that *g* is a latent variable of the formative kind (see Figure 6.1, right panel; Bollen & Lennox, 1991; Borsboom; Mellenberg, & van Heerden, 2003; Diamantopoulos, Riefler, & Roth, 2008), which means that individual differences in cognitive abilities (as measured by IQ tests) determine individual differences in the statistical variable general intelligence rather than vice versa. In this case, unless we make additional assumptions, there is no reason to expect an association between heritability coefficients (h_i^2) and summation weights ('*g* loadings', g_i). The claim that the Jensen effect for heritability provides evidence for a realistic *g* is thus derived from the argument that "what is observed is what would have been expected if an underlying *g* did in fact exist" (Rushton & Jensen, 2010a. p. 214, see Plomin et al., 2008, p.185, for a similar argument, and Bartholomew, 2004, p. 73 for the logic of inferences concerning *g*).

To test whether a positive correlation between *g* loading and heritability coefficient is significant does not provide the means to statistically test whether the relationships between genes and IQ scores are actually mediated by *g* (Dolan & Hamaker, 2001, see also Appendix B). First, it might be the case that general genetic influences and general environmental influences follow independent pathways (e.g. Plomin, 2003). Second, the possibility that the Jensen effect for heritability is due to some other process than a general mechanism (see, for example, van der Maas et al., 2006) cannot be ruled out. Putting aside this issue of testing, we agree that if a correlation between heritabilities and *g* loadings exists, this correlation requires explanation. Will any theory of general intelligence be substantive, it must be able to account for it. In addition, such theory must not conflict with other empirical data.

Our research has shown that - next to the Jensen-effect - there is a relationship between heritabilities and cultural load, and one between cultural load and *g* loading (see Chapter 3). In this chapter we evaluate whether, how, and to what extend the joint effects, which we proposed to call the Cattell-Jensen effect, poses problems for theories of intelligence. First, we evaluate *g* theories. We split these up in complexity theory and investment theories. Next, we consider alternative theories, which we further categorize into sampling theories and reciprocal interaction theories.

6.3 The Relations Among *g* Loading, Heritability, and Cultural Influences in *g* Theories

Concerning the explanatory status of common factors and *g* loadings, Jensen rightly stated:

> "Factors, including *g*, are not themselves explanatory constructs. They are constructs which themselves require explanation." (Jensen, 1987, p. 95)

> "The salient characteristics of the most highly *g*-loaded tests are not essential or definitional, but are empirical phenomena in need of theoretical explanation in their own right" (Jensen, 1998, p.92).

Nonetheless, *g* theorists are rather unspecific about the exact nature of *g* (for a critique, see Demetriou, 2002; Ackerman & Lohman, 2003), and how *g* affects test scores. For example, although Rushton & Jensen (2010a) maintain that *g* is an underlying biological variable, they do not indicate what this biological variable *g* represents. Yet, *g* theorists do have offered accounts for the finding that some tests have high *g* loadings (or, as it is sometimes expressed, are 'highly *g* loaded'). We can categorize these accounts as follows. Either they are unspecific about the role of *g* in the acquisition of cultural knowledge (e.g. Jensen, 1973; 1987), or they involve an account in terms of, or similar to, Cattell's investment theory (Carroll, 1993, p. 658; Cattell, 1987; Jensen, 1998, 2001). We denote the latter investment theories, and the former complexity theory.

6.3.1 Complexity theory

Complexity theory offers the following account for the relation between *g* and *g* loadings:

> "Probably the most undisputed fact about *g* is that the *g* loadings of cognitive tasks are an increasing monotonic function of the perceived complexity of the task." (Jensen, 1987, p. 111).

So, one may infer a relation between complexity and heritabilities of subtests (see also Beaujean, 2005; Plomin et al. 2008, p.183): on the one hand *g* loadings are indicative of complexity; on the other hand they are predictive of heritability (see Figure 6.2). Hence, we expect most complex tasks (e.g. Raven's Progressive Matrices, the so-called the prime 'marker of *g*'; see Jensen, 1998, p. 38) to be most *g* loaded and most heritable, and the culturally loaded knowledge tests (such as information and vocabulary, see Chapter 3) to be less *g* loaded (and given the Jensen-Effect for heritability, less heritable) because *g* theorists do not regard them as complex:

> "Information tests consisting of questions like 'Who was the first President of the United States?', 'Whose picture is on a penny?', and so on, make poor test items mainly for two reasons: (a) they do not get at complex mental process, and (b) they cannot be steeply graded in difficulty level without introducing items of information to which there is a relatively low probability of exposure, in which case social status and educational differences become practically impossible to avoid. The same holds true for vocabulary tests […] The difficulty levels differ only because of frequency of exposure. Such items based on information and vocabulary are rightly regarded as more culturally loaded than items which vary in difficulty because of the complexity of the mental processes involved." (Jensen, 1973, p. 184)

Indeed, in his older work Jensen predicted that culture loaded tests show low heritabilities and culture reduced tests high heritabilities (Jensen, 1973, p.194-195; see also Rushton & Jensen, 2005; Rushton, 1995). As is clear from Chapter 3, the empirical evidence does not support these predictions: the largest heritabilities (and *g* loadings) are for the most culturally loaded, noncomplex, knowledge tests. We conclude that complexity theory does not explain the observed relations among *g* loading, cultural load, and heritability.

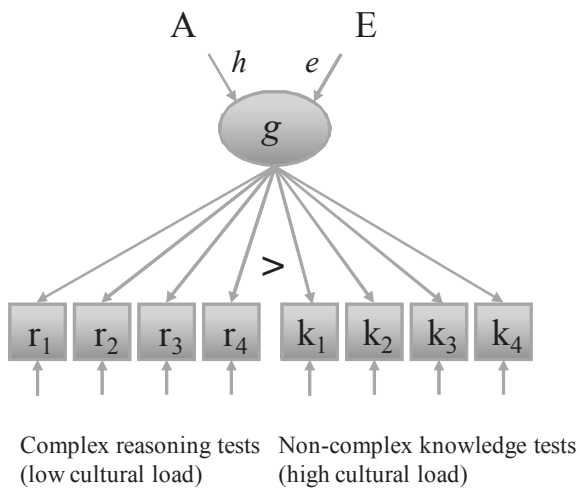

Figure 6.2 In complexity theory, the most culturally loaded tests (i.e. knowledge tests) have low *g* loadings and low heritabilities, because they are not complex.

6.3.2 Investment theories

In investment theories, *g* and *g* loadings are still related to complexity, but additionally, assumptions are made about the role of *g* in the acquisition of knowledge. Solving the items on a knowledge tests is not complex, but the acquisition of the required knowledge is.

Investment theories are derived from or inspired by Cattell's theory of fluid and crystallized intelligence (e.g. Cattell, 1987). Key in this theory is that knowledge is acquired through exposure to information, and that compared to people with low levels of *g*, people with high levels require less exposure to acquire the same amount of knowledge. Stated otherwise, compared to less intelligent people, more intelligent people are able to acquire more knowledge in the same amount of time.

In Cattell's original theory, fluid intelligence is a realistic variable, or, in his words, a 'source trait'. It is a single reasoning capacity and described as follows:

> "Fluid intelligence [..] is an expression of the level of complexity of relationships which an individual can perceive and act upon when he does not have recourse to answers to such complex issues already stored in memory" (Cattell, 1987, p. 96).

Fluid intelligence (abbreviated gf or Gf) is considered to be related to the maturation of the brain and to be highly genetically influenced. Crystallized intelligence (abbreviated gc or Gc), in contrast, is a not a realistic variable (Cattell, 1987; Kan, Kievit, Wicherts, Dolan & van der Maas, 2011; see Chapter 5), but rather a summary statistic, or, in Cattell's words, a 'surface trait', best conceptualized as a formative variable (Bollen & Lennox, 1991; Diamantopoulos et al., 2008). Crystallized intelligence is the result of 'investment' of fluid intelligence during one's learning experiences throughout the lifespan.

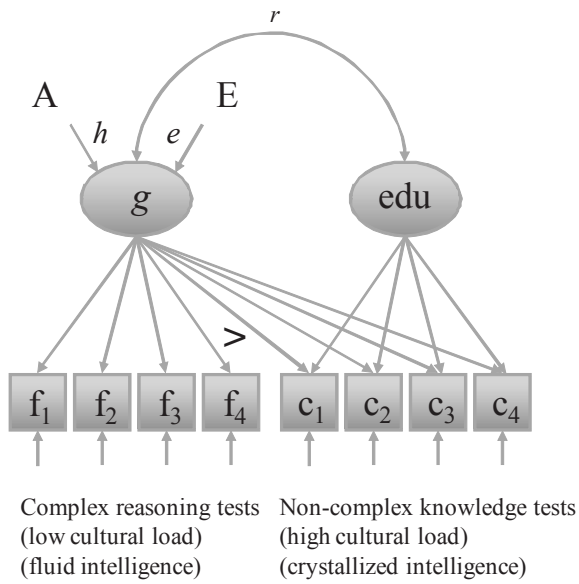

Figure 6.3 In investment theories, the culturally loaded knowledge tests (crystallized tests) may have large *g* loadings, but should show smaller heritability estimates than fluid tests, because of the intrusion of environmental influences.

In his later work, Jensen seems to subscribe to investment theory in stating that "the psychological distinction that Cattell makes between [fluid and crystallized intelligence] are valid" (Jensen, 1998, p. 124). However, because Jensen equates gf with *g* (Jensen, 1998, p.124), he considers crystallized intelligence to be the result of investment of *g* (see Carroll, 1993, p. 658, for a similar point of view). In other words, one may read investment theory as a theory that includes a general factor as a realistic, genetically influenced common cause of variance, that is, as a *g* theory (Rushton & Jensen, 2010a).

From investment theories it follows that people who possess higher degrees of fluid intelligence (or alternatively *g*) tend to acquire more knowledge and skills in all kinds of domains, which are the crystallized abilities. Crystallized intelligence is the total of the crystallized abilities. According to Cattell (1987), one might expect lower loadings for crystallized ability tests (crystallized tests, for short) than fluid intelligence tests (fluid tests, for short), because of "the intrusion of intermediate influences in the specific learnings" (p. 140). However, as he continued, a second common influence, assumed to be exposure to information via education, may render the loadings of crystallized tests on the *g* factor as large as the loadings of fluid tests (see Figure 6.3).[14]. In investment theories crystallized tests can thus have the largest *g* loadings. What about their heritabilities?

Cattell believed that

> "the population heritability is noticeably lower for crystallized intelligence than fluid intelligence, which is precisely what we should expect from the investment theory [...]". (Cattell, 1987, p. 324)

He expected this "on the hypothesis that gf is physiologically determined whereas gc is a product of environmentally varying, experientially determined investments of gf." (Cattell, 1963, pp. 3-4, see Carroll, 1993, p.374, for a similar point of view).

Exposure to information through education itself is clearly an environmental factor (but may be influenced by genetic factors as a result of gene-environment interplay, e.g. see Scarr & McCartney, 1985; Chapter 7). So, crystallized abilities (e.g. vocabulary and general information) may have large *g* loadings in investment theories, but, compared to the markers of *g* or gf (e.g. Raven's progressive matrices), they should have lower heritabilities, because of the intrusion of environmental influences (see Figure 6.3). As shown in Chapter 3, Cattell's expectation is not supported by empirical evidence.

Although researchers, including Jensen (1998), have mentioned the high heritability of crystallized intelligence, it is unclear whether this observation should be taken as evidence against investment theories, or as an erroneous expectation of Cattell (Mackintosh, 1998). In any case, no investment theory (Carroll, 1993; Cattell, 1987; Jensen; 1998) predicts that the most cultural influenced tests should be subject to the highest genetic influences. We conclude that invest theories do not explain the observed relation between *g* loading, cultural load, and heritability.

6.3.3 Conclusion

Both complexity theory and investment theories predict that heritabilities of the culturally loaded knowledge tests (crystallized tests) are lower (or at least not higher) than the heritabilities of the most complex processing tests (complex fluid tests). As is clear from Chapter 3, these predictions are not supported by empirical evidence. This is not to say that future *g* theories cannot account for the relations among *g* loading, cultural influences, and heritabilities at all or in principle, but that the existing *g* theories do not fully account for them. These theories are likely to require additional assumptions.

As mentioned above, a positive correlation between *g* loadings and heritabilities has been considered to be problematic for alternative theories of general intelligence (Rushton & Jensen,

[14] We note that the literature gives another reason for crystallized tests having larger loadings on the first principal factor than fluid tests, namely the composition of the test battery. Crystallized tests tend to have large loadings on this factor if the battery contains relatively many crystallized tests (Ashton & Lee, 2005).

2010a). Indeed, if *g* is a formative variable (Bollen & Lennox, 1991), and unless additional assumptions are made, there is no reason to expect this correlation (see above). At first glance *g* theories seemed thus more parsimonious, but, in view of the above, in order to explain the relations among *g* loading, heritability, and cultural influences jointly, *g* theories are likely to require additional assumptions as well. One question is thus how plausible the assumptions in theories of general intelligence are. In the next sections we consider the assumptions in alternative theories of general intelligence.

6.4 The Relations Among *g* Loading, Heritability, and Cultural Influences in Other Theories

We divide alternative theories in sampling theories and reciprocal interaction theories. In sampling theories *g* is essentially due to a measurement problem. If we could measure the underlying variables of intelligence independently, the positive manifold would disappear, and a *g* factor would not be present. In reciprocal interaction theories the *g* factor would remain.

6.4.1 Sampling theories

As early as 1903, hence before the publication of the original *g* theory (Spearman, 1904), Thorndike had observed positive, but imperfect correlations among cognitive tasks. He explained this observation as follows: Whenever people aim to solve a problem, they 'tap' from a pool of learned stimulus-response associations, which are hardwired as neural connections. Thorndike called these connections 'bonds'. Tests tap (sample) from some of the same bonds, so across tests there will be an overlap in the tapping. This overlap causes the positive intercorrelations among test scores.

Based on this principle, Thomson (1919; 1927; 1951) developed an alternative to *g* theory, which is now called sampling theory (Jensen, 1998) or the bonds model of intelligence (Bartholomew et al, 2009). Like Thorndike, Thomson (1919) accounted for the positive manifold (hence the statistical general factor) by assuming a simultaneous sampling from (statistically) independent variables. In Thomson's theorizing, and elaborations of his bonds model (e.g. Bartholomew, Deary, & Lawn, 2009; Thomson, 1951) (henceforth sampling theories), the brain consists of a (large) number of bonds. These bonds are called upon when completing an IQ test or item. The nature of the bonds usually remains implicit, but their underpinnings are usually sought in neural terms (Bartholomew et al., 2009; Jensen, 1998).

We note a certain shifts in the bonds model, which is not without consequences for presenting sampling theories as a true alternative for *g* theories. At one point in Thomson's theorizing, the sampled variables represented (statistically) independent group and specific factors of intelligence (Thomson, 1919). The subtests determine which variables (i.e., factors of intelligence) are called upon. We will refer to this kind of sampling as 'test sampling'. In other models (Thomson, 1951; Bartholomew et al. 2009), the sampled variables are not interpreted as the factors of intelligence, but as their underlying 'elements'. Here it is not assumed that *tests* sample from the factors of intelligence, rather it is assumed that *people* sample from the underlying elements when they attempt to solve a test item. We will refer to this type of sampling as modern sampling theory.

Recently, alternative theories of general intelligence have been developed that can be regarded as sampling theories in which the underlying elements represent genes or genetic mutations (Anderson, 2001; Penke et al., 2007). In this case, tests do not sample from cognitive abilities or capacities (factors of intelligence), nor do people sample from the underlying elements when attempting to solve an item, rather *cognitive capacities* sample from the genes. We refer to these theories as genetic sampling theories.

Next we examine how sampling is related to the general factor of intelligence and *g* loading. Furthermore, we ask ourselves whether sampling theories explain the relations among *g* loading, heritability, and cultural influences.

Test sampling In test sampling theory, the sampled variables represent independent factors of intelligence. Here, 'independent' means statistically independent across members of the population. This implies that the number of variables is essentially the same for each member of the population in question. The variables may represent elementary cognitive processes (Jensen, 1998), for example. Each cognitive tests measures a number (sample) of these variables (e.g. elementary processes). Factor loadings represent the (mean) fraction of overlap among group factors. In test sampling theory the positive manifold is thus essentially a measurement problem, because tests are multi-dimensional. If we would and could construct unidimensional tests (measuring each factor, e.g. elementary process, apart) the positive manifold would disappear, hence the *g* factor as well.

Test sampling theory does not give an explanation for a relation between factor loadings and heritability. Nevertheless, under certain specific assumptions the effect can be present. It will be present when it is assumed that the most heritable factors are called upon the most frequently, for example. We conclude that the theory does not give an account for the Jensen effect, but also that it does not conflict with it. Test sampling theory is silent about knowledge acquisition and the role of culture.

Modern sampling In modern sampling theory the independent variables (bonds) are interpreted as the underlying elements. People may differ in the *values* of these elements as well as in the *number* of elements (Bartholomew et al., 2009). As mentioned, in modern sampling tests do not sample from group factors, rather people sample from the elements when attempting a test item. Some items call upon a small number of the elements, whereas other items call upon many of them.

In some versions of modern sampling theory, one may encounter difficulties in interpreting them as true alternatives of *g*. These difficulties concern the level of analysis (see also Burt 1940). First, in interpreting bonds at the level of neurons or synapses (e.g. Thomson, 1951) it is hard to conceive of *particular* neurons showing *inter-individual* differences. It implies that neurons can be labeled (e.g. Neuron A, Neuron B, etc.) and identified in each member of the population (or subpopulation); across people there is a distribution of values of a certain property of Neuron A, a distribution of values of a certain property of Neuron B, etc., and these values are statistically independent. Second, if the properties are all of the same kind and their values are additive (e.g. when they represent mass, or amount of myelin), we actually have a single, quantitative inter-individual variable (the total or mean value of this property, for example neuronal mass). It is thus important to state what the elements or bonds represent, whether they are identifiable across individuals, and if their values can be truly added, or are merely regressed on them.

In modern sampling theory, factor loadings represent the (mean) fraction of elements used in the test (Bartholomew et al. 2009, p. 576). In our interpretation of certain bonds models, the factors loadings of subtest i (λ_i) on the estimated variable k (hence λ_{ik}) just need a subscript for the individual, i, (hence λ_{ijk}), and perhaps even for the item, l, (λ_{ijkl}). Intelligence tests scores are eventually manifestations of variable k, for instance a capacity (e.g. neuronal mass) that constrains working memory.

Like test sampling theory, modern sampling theories do not give an account for the Jensen-effect. Neither do they make explicit the role of environment or the impact of cultural influences.

Genetic sampling Multiple genes can affect one phenotype. This is called polygeneity. In addition, a single gene can influence multiple phenotypes. This is called pleiotropy. Given polygenetic and pleiotropic influences on diverse cognitive capacities are present, these capacities can share genetic influences. It is thus possible that genetic intercorrelations between cognitive processes are present. This can be due to genes that have a general effect (Kovas & Plomin, 2006), for example. The intercorrelations on the genetic level will give rise to intercorrelations on the phenotypic level. When general genetic effects are present, that is, when certain genes influence all cognitive processes, one can conceive of the general factor as representing a genetic factor: the total of general genetic effects.

The presence of a statistical general genetic factor does not necessarily mean that general effects are present in reality. A genetic factor can be the result of what we call genetic sampling, which we define as follows: *genetic sampling means that any two cognitive processes always share*

some of their genetic determinants (genes or genetic mutations), but there are no genes that influence all cognitive processes. In genetic sampling theories (Anderson, 2001; Penke et al., 2007) the sampled elements thus represent genes. Here, it is easier to conceive of the elements showing individual differences than in certain interpretations of modern sampling theories. Genes can be labeled (e.g. gene A, gene B, etc.), and in principle they are identifiable in each member of the population (or subpopulation). Across people, there is a distribution of values of gene A, a distribution of values of gene B, etc.; in genetic sampling theories these values are statistically independent. In quantitative genetics, genetic values are additive, so also in genetic sampling theory we (can) have a single, quantitative interindividual variable again (e.g. the total genetic value of 'genes that influence working memory capacity'). Whether genetic sampling is a true alternative theory is thus debatable.

The presence of genetic sampling implies that if cognitive abilities are (solely) intercorrelated due to genetic effects, the rank ordering of people on Full scale IQ will be an estimation of the rank ordering on the total genetic value of the genes that influence these cognitive abilities. Across individuals, this total genetic value predicts the levels of the cognitive abilities to a certain degree, some better than others. Factor loadings will represent the mean fraction of genes that influence a cognitive ability. Given that mutations are generally harmful, and lower genetic values, one way to conceive *g* is 'mutation load'. The more genetic mutations one carries, the lower one's genetic values, which probably means that cognitive development is disrupted to higher degrees.

Unless the pathways of genetic and environmental influenced are modeled explicitly, the relation between heritability coefficients and *g* loadings (loadings on the phenotypic psychometric *g*) is *a priori* unknown (see also Chapter 4). Genetic sampling theories are silent about the role of culture.

Conclusion In test sampling theories and modern sampling theories, factor loadings represent the (mean) fraction of bonds used in the test (Bartholomew et al., 2009). Differences in these loadings are usually explained from the tests or items' levels of complexity (Jensen, 1998): Complex items will call upon many of the bonds, hence show larger overlaps (hence larger intercorrelations, hence larger *g* loadings) than noncomplex items. If the bonds' values are heritable, sampling theories resemble Jensen's complexity theory, which, as we have shown, cannot explain the mutual relationships among heritability, *g* loading, and cultural load. There is only one important difference. If *g* loadings represent fractions of statistically independent, heritable bonds, there will be no relation between subtests *g* loadings and heritabilities. This is because a sum of two statistically independent variables will have a heritability that falls somewhere in between the heritability of the two variables. A Jensen effect might be present however, namely if the most heritable bonds are called upon the most frequently. We conclude that sampling theories can incorporate a correlation between heritabilities and *g* loadings, by making additional assumptions, but that they do not give an account for it.

Sampling may occur at every level, so genetic sampling, test sampling and modern sampling can be combined. Sampling does not account for a correlation between *g* loading and cultural load. This is because sampling theories do not make explicit the role of cultural and environmental influences that affect intelligence during its development. However, we do believe that sampling may play a role in explaining the Jensen effect (see below, and Chapter 7).

6.4.2 Reciprocal interaction theories

In reciprocal interaction theories (Dickens & Flynn, 2001, Dickens, 2008; van der Maas et. al, 2006), the statistical *g* factor is the result of mutual beneficial interactions between cognitive processes or abilities. These theories do not include *g* as a realistic latent variable. The general factor is not a measurement problem, as in sampling theories.

The mutualism theory In the mutualism theory of van der Maas et al. (2006), general intelligence is assumed to be the result of mutual, largely beneficial, interactions among basic cognitive processes (such as perceptual, memory, and reasoning processes), which occur throughout development (see Figure 6.4). One assumption is that the growth of each cognitive process depends on limited resources, which are conceptualized in terms of biological constraints, such as neuronal speed and the

size of neural systems associated with each of the cognitive processes. Another assumption is that the development of cognitive processes is largely an autonomous, self-regulating process. By incorporating these assumptions in a multivariate dynamical systems model (henceforth, the mutualism model), it was shown that mutual, largely beneficial, interactions among variables can yield a statistical common factor.

The mutualism model is formulated mathematically as follows:

$$\frac{dx_i}{dt} = a_i x_i (1 - x_i / K_i) + a_i \sum_{\substack{j=1, \\ j \neq i}}^{W} M_{ij} x_j x_i / K_i \qquad \text{for } i, j = 1 \ldots W.$$

The K's represent W limited resources of the growth processes of an individual system. Parameters a are growth parameters, determining the steepness of the (logistic) growth function associated with each variable x. Weights M_{ij} determines the influence of x_i on x_j. In the population, the parameters a and K are considered to be parameters that differ over individuals, whereas the weights $M_{ij}$are considered to be equal for all individuals (i.e., they are population parameters, like factor loadings in factor models of intelligence).

When all individual systems are equilibrium, the covariance matrix of the x's equals:

$$\Sigma = [\mathbf{I} - \mathbf{M}]^{-1} \Psi [\mathbf{I} - \mathbf{M}]^{-\mathrm{T}}$$

where $\mathbf{\Psi}$ is a diagonal matrix containing the variances of the K's, and $\mathbf{I}$ is the identity matrix. Superscript -1 denotes matrix inversion, and superscript -T denotes inversion and transposition.

In simulating the development of general intelligence, van der Maas et al. (2006) modeled all underlying variables (a's, K's, and starting values) as initially uncorrelated. During the development of individual systems the values of the observed variables (x's) increased while becoming intercorrelated due to their mutual beneficial interactions. Once the systems were all in equilibrium, a factor analysis on these variables yielded a 'general factor'. This factor is a purely statistical entity, however, as it does not represent any common, underlying psychological or biological variable; it is simply a summary index of the positive correlations among the observed variables. When genetic and environmental influences were introduced in the model (via the limited resources, K), the general factor was found to be heritable, thus even when all genetic and environmental influences on the limited resources were assumed to be uncorrelated. Notably, the common factor appeared *more* genetically influenced (i.e., had a higher heritability) than the limited resources themselves.

These simulations did not produce the Jensen effect, but following the introduction of very small positive correlations among the genetic influences on the limited resources K, the Jensen effect was observed (van der Maas et al., 2006). Using the equation for the covariance matrix, van der Maas et al. simulated data for 16 K's with normally distributed M_{ij} (mean = .05). Heritabilities of the K's were normally distributed with mean .5 and SD .02). Their genetic intercorrelations were assumed to be equal (SD = 0), whereas their environmental intercorrelations were fixed at zero. Generally, the correlations between factor loadings on the first principal factor and observed heritabilities were positive (see Figure 9 in van der Maas et al.). The variance in **M** was introduced to investigate the robustness of the Jensen effect, and is not a necessary or essential aspect in this setup. The essential assumption was the presence of nonzero genetic intercorrelations. If the interaction weights in **M** are uniform and the K's are heritable to different degrees, the introduction of any nonzero genetic intercorrelation, no matter how small, will yield a vector correlation between heritabilities and g loadings of 1.

In the simulations of van der Maas et al. (2006) only very weak genetic intercorrelations were thus required. Here, we interpret these weak intercorrelations in terms of genetic sampling (Anderson, 2001; Penke et al., 2007), which we took to mean that any two cognitive processes always share some of their genetic determinants, but that there are no genes that influence all cognitive processes (genetic sampling may be interpreted as a weak form of pleiotropy). We conclude that a correlation between g loadings and heritabilities can be reasonably explained without including a

general, largely genetically influenced, mediating variable or the assumption of general genetic effects. We stress we do not argue for or against the presence of general genetic effects in reality. Rather, we note that a correlation between heritabilities and *g* loadings does not necessarily imply that general effects are present.

Mutualism provides a plausible explanation of the positive manifold. As shown in van der Maas et al. (2006), mutualism can also account for the hierarchical factor structure of intelligence (e.g. Carroll, 1993) and developmental effects, such as integration and differentiation effects (Deary et al., 1996). In addition, it can account for the (high) heritability of *g* (e.g. Plomin et al. 2008), and the increase of heritability of *g* during the lifespan (Bartels et al., 2002; Haworth et al., 2009; Plomin et al., 2008). It is also consistent with the finding that more specific factors of intelligence (spatial ability, verbal ability, memory, and processing speed) are genetically independent initially, but become increasingly genetically interrelated as development unfolds (Hoekstra, Bartels & Boomsma, 2007). Such developmental effects are not readily explained by *g* theories or sampling theories. We conclude that mutualism explains more empirical findings (including behavior genetic results) within intelligence research than *g* theories and sampling theories.

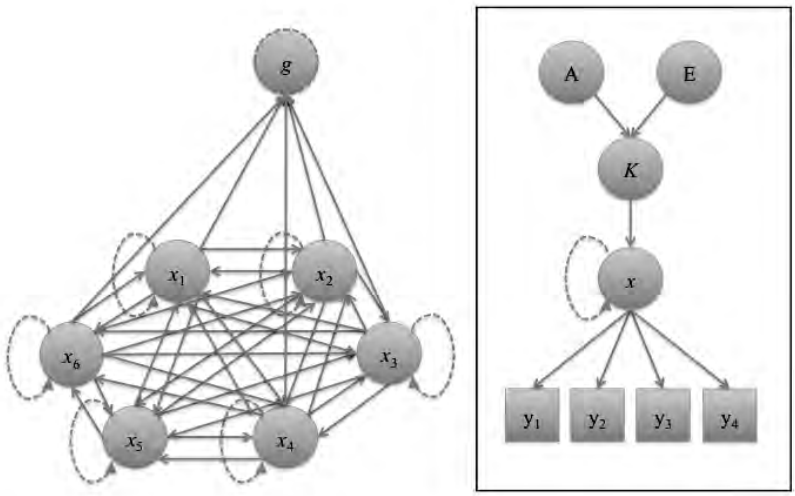

Figure 6.4 The mutualism model. The *g* factor is the result of mutual beneficial interactions among cognitive processes (*x*'s) during development. The growth of these processes is constrained by limited resources (*K*), which are influenced by genetic (A) and environmental variables (E). Variables *y* are indicators of individual differences in *x*.

Mathematically, the mutualism model bears strong similarities with Dickens' model (Dickens, 2008; unpublished). In fact, the latter can be regarded as a mutualism model in which the role of the environment is made explicit.

Dickens' model Though not published in a peer-reviewed journal, we acknowledge Dickens' model (Dickens, 2008; unpublished) as an important alternative theory of general intelligence, and discuss it here, because Dickens provides another account for the Jensen effect without assuming a realistic *g*. In addition, we believe that Dickens (unpublished, p. 7) gives an important perspective on the relations among *g* loading, heritability, and cultural influences by suggesting that "those skills that are emphasized in practice will have a tendency to be the ones that will have the highest heritability." (see also Chapter 7).

Dickens' model extends the theory of Dickens & Flynn (2001), but the basic assumption is the same, namely that people who possess higher levels of any cognitive ability are more likely to end up in environments conducive to the development of their cognitive abilities. In Dickens' model it is assumed that people differ in cognitive abilities as a result of both environmental and genetic

differences. More specifically, individual differences in cognitive abilities are assumed to be heritable, but not due to a common dependence on a single underlying biological, heritable variable. In essence, Dickens' model is a model of (evocative, passive and active) gene-environment correlation (Scarr & McCartney, 1983). Current levels of ability are the result of a feedback process in which environmental influences are influenced by genetic influences (via the abilities), whereas environmental demands cause the heritable cognitive abilities to become correlated (see Figure 6.5).

Dickens was able to account for salient findings of behavior genetics and intelligence research, including the Jensen effect for heritability, by making a few additional assumptions (Dickens, unpublished, p. 9). The most relevant assumptions are that (1) abilities that are valued the most important within the cognitive environment will be practiced most often in that environment, and (2) people who excel in the most valued abilities will have the greatest chance to end up in cognitive demanding environments. By incorporating these assumptions into his model, Dickens demonstrated that the most valued abilities became the most highly *g* loaded.

Dickens' (2008; unpublished) account of the Jensen effect differs from that of van der Maas et al. (2006). A Jensen effect for heritability appeared in Dickens simulations by assuming that cognitive abilities that are the least important are more ubiquitous in non-cognitive activities, and, as a result, are more open to environmental influences outside the cognitive environmental system. We believe that this assumption is plausible, but future research is necessary to establish whether this is indeed the case.

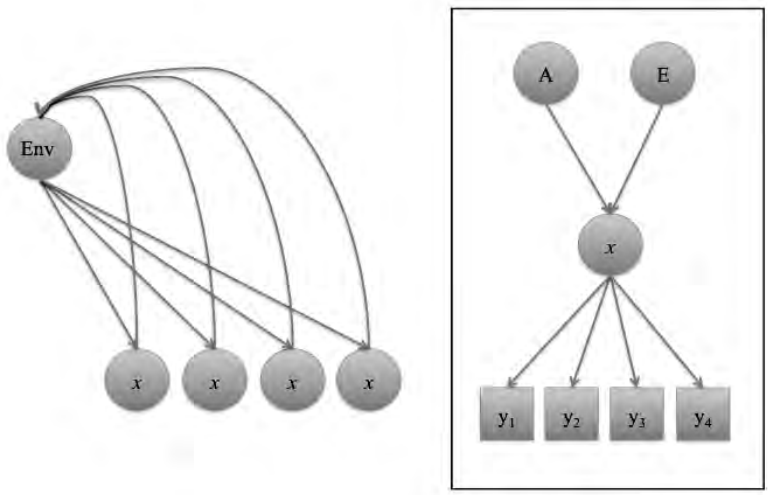

Figure 6.5 Dickens' model (based on Dickens & Flynn, 2001). Environment is influenced by heritable cognitive abilities, whereas environmental demands cause the abilities to become correlated.

Conclusion As shown in Chapter 3, the most culturally loaded tests (generally verbal knowledge tests) have the highest *g* loadings. It is conceivable that in society in general, and in educational settings in particular, verbal skills and knowledge are most highly valued, and that these are subject to more training and exercise than, say, perceptual speed. Following Dickens' model, the most highly valued skills (verbal skills and knowledge) will show the highest *g* loadings, and, given the Jensen effect, also the highest heritabilities.

As mentioned, Dickens' model can be accommodated within the mutualism model by making the role of the environment explicit, i.e., by introducing mediation by the environment of the mutual interactions among the cognitive processes. Therefore, we believe that mutualism, in combination with genetic sampling, is promising in accounting for the relations among *g* loading, heritability, and cultural load when incorporating assumptions from the theory of Dickens & Flynn (2001) and Dickens' (2008; unpublished) model.

6.5 Discussion

In this chapter, we reviewed the issue whether the relation between *g* loadings and heritabilities supports the interpretation of *g* as a realistic (biological), largely genetic, mediating variable (e.g. Gray & Thompson, 2004; Rushton & Jensen, 2010a). We subscribe to Rushton and Jensen's statement:

> "[T]here is no absolute claim that *g* effects have been proven; only that what is observed is what would have been expected if an underlying *g* did in fact exist." (Rushton & Jensen, 2010a, p.214)

Indeed, if a real, largely genetic influenced, mediating variable is present, we would expect a Jensen effect for heritability. This effect may exist, but in view of the following, the Jensen effect for heritability effect is not convincing evidence for a realistic, biological *g* as Rushton and Jensen put forward.

First, in *g* theories theory large genetic influences and large cultural influences are juxtaposed, but cultural load and heritability are not mutually exclusive. Indeed, empirical evidence has shown that that large genetic and large cultural influences on intelligence can go together: Chapter 3 revealed that the highest heritability coefficients and *g* loadings are of the most culturally loaded (knowledge) tests. Current *g* theories do not account for this joint effect. Sampling theories (Anderson, 2001; Bartholomew et al., 2009; Penke et al. 2007; Thomson, 1951) do not account for it either, because they do not make explicit the role of environment in the development of intelligence. Furthermore, some interpretations of certain sampling theories (Bartholomew et al., 2009) must be regarded as *g* theories, in which factor loadings are person specific. Reciprocal interaction theories (Dickens, 2008; van der Maas et al., 2006) in isolation do not account for the relations among *g* loading, heritability, and cultural load either (but see below and Chapter 7).

We believe that integration of (elements from) alternative theories provide a promising perspective. First, the mutualism theory (van der Maas et al., 2006) accounts for developmental effects that are not accounted for by other theories of general intelligence. Second, assuming genetic sampling (Anderson, 2001; Penke et al. 2007), a Jensen effect for heritability is present in the mutualism model. Third, Dickens's (2008; unpublished) model, can be incorporated in the mutualism model, which can explain that the most practiced abilities are the most *g* loaded. Fourth, elements of the investment theory of fluid and crystallized intelligence can be incorporated. We believe that mutualism can account for the joint effect if it is assumed that the cognitive environment puts demands primarily on crystallized abilities.

We also contend that the general factor of intelligence does not represent a realistic (biological) common cause of individual differences, but is a merely a statistical entity, best conceptualized as an index measure, or formative variable (Bollen & Lennox, 1991; Borsboom et al., 2003), i.e., an informative summary descriptive. However, as such, general intelligence certainly has utility. Specifically, we consider it to be similar to 'general health'. Like general health is an informative summary descriptive of physical functioning, '*g*' is an informative summary descriptive of cognitive functioning.

In the introduction of this chapter we mentioned that factor analysis does not discriminate between *g* theories and alternative theories, and that both *g* theories and alternative theories can explain the facts that the general intelligence is (highly) heritable and has (strong) biological correlates. As shown, the Jensen effect for heritability does not help discriminating among theories either. In order to explain the relations among *g* loading, heritability, and cultural load, we need better theory. In order to discriminate among these theories we need true differential predictions.