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

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CHAPTER 7

DISCUSSION

Abstract

Knowledge tests display higher g *loadings and heritability coefficients than cognitive processing tests. In isolation, mainstream theories of intelligence fail to explain this effect. To account for it, we present an integrated model of intelligence. We propose the following. The development of cognitive processes is initially largely autonomous and self-regulating, but benefits from knowledge acquisition (learning), whereas knowledge (including skills and solving strategies) is the result of cognitive processing. Growth in cognitive processing and knowledge (cognitive abilities) is constrained by limited resources, which show genetically and environmentally influenced individual differences. As a result, some individuals develop as being more intelligent than others. Society selects on intelligence. Its demands influence the interactions among cognitive abilities, hence their intercorrelations, and so their factor loadings. Computer simulations showed that if society promotes the acquisition of knowledge rather than training of cognitive processes, knowledge tests demonstrate higher* g *loadings and heritability coefficients than cognitive processing tests.*

7.1 Better Theory of Intelligence is Warranted

As pointed out in the introduction, to attach a meaning to intelligence other than its interpretation as a rank order on psychometric tests, both the inter-individual differences perspective and the developmental (intra-individual differences) perspective are required. The former is required to account for the covariance structure among the cognitive abilities measured by intelligence tests; the latter to account for cognitive growth. As most models of intelligence (factor models) only concern inter-individual differences in cognitive abilities and do not incorporate cognitive growth, the development of an integrated theory is warranted. The first aim of this thesis was to reinvigorate the development of such theory. Of course, like any theory of intelligence, such integrated theory should be able to account for the most robust and salient empirical findings in intelligence research. These include, for example, the positive correlations among IQ test scores (hence the general factor of intelligence), the heritability of intelligence, the increasing heritability of intelligence throughout development, and the fact that subtests' estimated heritability coefficients correlate with their loadings on the general factor. While working on a model that can account for all of these effects, we noticed that certain empirical findings in intelligence research are not fully addressed in the literature. The second aim of this thesis was to review these findings in more detail.

From the previous chapters we can conclude that in general the dynamical character of intelligence is not fully appreciated in intelligence research. This concerns the dynamic interplay between genes and environment in particular. In this last chapter we focus on this issue, but the message is broader: The mutualism model of intelligence (van der Maas et al. 2006) provides an excellent framework to study effects in intelligence research (in isolation or combined), and to check old and new hypotheses. In Table 7.1 we summarized the most well-known of these findings. The table also includes findings discussed in the previous chapters. We first collate these again below. Next, we provide an example of how the mutualism model can help researchers to study combined effects. We aim to explain one of the most intriguing empirical effects: The more culturally loaded an intelligence subtest is, the higher its heritability estimate is. That is, the better culturally dependent, specific knowledge differentiates between people, the better individual differences in test scores reflect genetic differences. Together with this effect, we aim to explain the fact that the test with the highest cultural load and heritability coefficients are also the most *g* loaded. We implement our hypotheses in the mutualism model and run a number of series of simulations with this model. The results will be discussed.

Table 7.1 Knowns and Unknowns in intelligence research

Factor analytical & Measurement
Positive manifold
Factor indeterminacy
Group factors: e.g. Gf, Gc, Verbal, Perceptual, Image Rotation, etc.
g factor convergence across batteries
High correlation between Gf and *g*
High *g* loading of Raven's
High *g* loadings of knowledge (crystallized intelligence) tests
Lack of unidimensial measures
No direct measure of *g* (*g* is a higher order factor)
Cultural specifity of measurement
Behavior genetics
Heritability of IQ and the factors of intelligence
Gene-environment correlations
Gene-environment interactions
No genes/QTL/SNPs identified
Jensen effect for heritability
Relation heritability, *g* loading and cultural loading
Inbreeding effects
Developmental
Cognitive development
Increasing stability of IQ
Increase of heritability with age (decrease of shared environmentality)
Domain-specific aging trends
Lack of transfer of training
Poor health & pre/perinatal effects on IQ
Specific effects of brain damage
Stage transitions
Correlates
Physical/physiological measures, e.g. brain size
Imaging results
Jensen effects for physical/physiological measures, e.g. brain size
Social economic status
Educational attainment & school performance
Job performance and income
Social outcomes & health
Group differences
Racial and ethnic differences
Jensen effect for Black-White differences
Black-white differences are most pronounced on culture loaded tests and least so on culture reduced tests
Jensen effect for inbreeding effects
Flynn Effect (+ lack of invariance across subtests)

7.2 The Role of Gene Environment Interplay in the Dynamic Development of Intelligence

The dynamical interplay between genes and environment is largely ignored in the statistical (cross sectional) modeling of heritable intelligence. This makes that the further interpretation of the statistical results requires caution. This is so for at least two reasons. First, whereas the hypothesized underlying variables in statistical models are generally modeled as having linear effects, dynamical systems - such as humans - often display behavior that is nonlinear. If nonlinearity is present, a linear statistical model does thus not represent the true data generating mechanism. Second, dynamical gene-environment interplay can lead to gene-environment correlation, which is usually not modeled in statistical models of intelligence. Both the presence of nonlinearity and gene-environment correlation affect heritability estimates. How or to what extent heritability estimates of intelligence are affected is unknown and requires research.

Chapter 2 dealt with the problem of nonlinearity. We noted that the genetic and environmental variables in behavior genetic models are generally not measured, but merely inferred, using genetically informative research designs (e.g. twin and adoption studies), and that these genetic and environmental variables are modeled as having linear relationships with the observed variables, e.g. cognitive abilities measured by IQ tests. However, in reality cognitive development is typically nonlinear and is characterized by stage transitions (see Table 7.1). By means of computer simulations, we investigated the effects on estimated heritabilities and environmentalities in statistical linear behavior genetic modeling in case the underlying mechanisms were nonlinear. In doing so, we were unable to use the mutualism model of van der Maas et al. (2006), because this model does not contain nonlinear terms. We could have developed a nonlinear version of the mutualism model and use this to investigate the effects of nonlinear effects, but this would have required first a full systematic analysis of its dynamical behavior, which was beyond the scope of the thesis. Instead of the mutualism model we used the two-cell model of van Oss & van Ooyen (1996). The main advantage of using the two cell-model was that its dynamics were rich and already known. Although originally the two cells or units represent an inhibitory and an excitatory neuron, they can be interpreted as representing inhibitory and excitatory networks constituting a neural system (van Oss & van Ooyen, 1996), for example a system that underlies working memory. We implemented heritable individual differences in the parameters of the model, ran a series of simulations, and investigated whether the estimated genetic and environmental structure resembled the implemented structure.

In the results of our simulations we observed effects that are in line with empirical findings in intelligence research. First, when nonlinearity was present, estimated heritabilities appeared to increase over time (see Table 7.1), so that the estimated relative contribution of environmental influences decreased. The heritabilities were overestimated, sometimes by as much as 25%. Second, more particular, the estimated relative effects of shared environmental influences decreased (often to values of zero) rather than those of nonshared influences (see Table 7.1). Third, in the behavior genetic modeling the ultimate underlying causes were hard to detect, which fits the fact that to date the search for specific environmental and genetic influences on intelligence has been largely unsuccessful (see Table 7.1). We concluded that within the linear statistical framework, nonlinear development can be considered as constituting a source of phenotypic variance on its own, next to the genetic and environmental sources (see also Molenaar et al., 1993). As in reality cognitive growth is characterized by stage transitions, we hope new mutualism models will be developed that can account for such transitions. We also hope that such model will be used to investigate the role of nonlinear development as a third source of variance in intelligence. Since the presence of stage transitions can lead to an increasing overestimation of heritability coefficients over time, this provides an additional or alternative explanation to the mainstream explanation of the increase of heritability. This increase is usually explained in terms of increasing gene-environment correlation.

Increasing gene-environment correlations also constituted our explanation of the results discussed in Chapter 3, in which we summarized data from 23 independent behavior genetic studies into intelligence. These data showed the contra-intuitive result that the most cultural dependent intelligence tests (i.e. knowledge tests) demonstrated higher heritabilities than the least cultural dependent tests (i.e. cognitive processing tests). These cultural dependent knowledge tests also showed the relatively highest *g* loadings (see Table 7.1). In isolation, mainstream theories of intelligence fail to explain this combined effect (see Chapter 6). Some of them even predict the opposite. We hypothesized that if society puts demands on acquired knowledge rather than cognitive processing, gene-environment correlations would become higher for individual differences in knowledge than individual differences in cognitive processing. In standard behavior genetic modeling gene-environment correlations will be subsumed under the genetic variance, leading to higher heritability estimates.

On the basis of the results from Chapter 2, we can also formulate an alternative or additional hypothesis. As was shown, stage transitions in cognitive development can result in overestimation of the true heritability. The development of certain cognitive abilities may be characterized by more transitions than the development of other cognitive abilities, so that the increase in heritability due to nonlinear effects will differ across abilities. For example, verbal comprehension, usually measured by knowledge tests, may develop stage wise, whereas nonverbal

cognitive abilities, usually measured by cognitive processing tests, develop gradually (whether this is the case should be investigated empirically). Hence, the heritability coefficients of the verbal tests can be overestimated, while those of nonverbal tests are correct or less affected. Whether this alternative explanation of increasing heritability can account for the high *g* loadings of verbal knowledge tests (see Table 7.1) is unknown, but can be investigated with a nonlinear model of general intelligence. Simulations with a mutualism model that includes nonlinear terms may help to discriminate between the two explanations.

Both explanations of the increase in heritability are also relevant in the discussion of racial and ethnic group differences. Black-White differences in IQ exist, for example (see Table 7.1). Chapter 4 showed that they are the most pronounced on verbal knowledge tests (see Table 7.1). If verbal comprehension develops nonlinearly, small differences in initial conditions, e.g. small language deficits in early development or differences in language use (vocabulary), can have large consequences for the further acquisition of knowledge, hence for development of intelligence and scholastic and academic achievement, especially when selection to educational systems is based largely on knowledge and when transmission of knowledge (teaching) takes place verbally. In principle, Black-White mean group differences in later IQ can thus be the result of small initial differences in verbal comprehension or language use, for example. To date, the actual causes are unknown, however, although some researchers (Rushton & Jensen, 2010a) have argued that certain empirical results imply Black-White differences are genetic.

As mentioned Mean Black-White differences in IQ are the most pronounced on verbal knowledge tests. These tests also happen to demonstrate the highest heritabilities and *g* loadings (see above). This effect has been interpreted as a genetic effect (Rushton & Jensen, 2010a). In Chapter 4 we demonstrated that the reasoning behind this interpretation was invalid. We showed analytically that group differences can be the most pronounced on the most heritable and the most *g* loaded tests even when the origin is purely environmental. Moreover, as Chapter 3 showed, the most heritable, most *g* loaded tests are also the most cultural loaded. The finding that Black-White differences are the most pronounced on the most culturally loaded subtests are problematic for Rushton and Jensen's (2010a) biological *g* theory, because this theory predicts the opposite: the mean group differences are most pronounced on the least culturally loaded tests.

In line with Cattell's investment theory of fluid and crystallized intelligence, cultural loaded knowledge tests are often referred to as crystallized tests. Crystallized tests often load highly on a factor that is separate from nonknowledge tests (cognitive processing tests). For this reason, such factor (abbreviated Gc) is often interpreted as 'crystallized intelligence'. In addition, crystallized intelligence is interpreted as the result of the 'investment' of fluid intelligence (reasoning). From our review of the fluid-crystallized theory (Chapter 5), we argued that 1) crystallized intelligence is purely a statistical summary, and that 2) the investment hypothesis of fluid intelligence does not explain why crystallized tests should load on a factor other than the factor fluid intelligence (Gf); 3) a second influence is necessary to dislocate the factor crystallized intelligence from the factor fluid intelligence. Also from the review, we concluded that this second causal influence is most likely verbal comprehension or education. Supported by results from factor analyses, we concluded that once cultural, educational and age differences (hence developmental differences) are taken into account, factor Gc is equivalent to verbal comprehension. We maintained that if researchers find a separate Gc factor, this is due to sample heterogeneity with respect to developmental differences. Likely, such sample heterogeneity also leads to the separation of *g* and Gf. In homogeneous samples they will be equivalent (see Table 7.1), as we found.

Chapter 6 was a theoretical chapter. We addressed in detail whether and to what extent one of the most intriguing empirical findings poses a problem for current theories of general intelligence That is, we asked ourselves whether *g* theories, investment theories, sampling theories, and reciprocal interaction theories explain the finding that the most cultural dependent cognitive abilities (crystallized abilities) are the most *g* loaded and most heritable. We concluded that (in isolation) they do not. By implication, the reviewed theories do not explain how Black-White differences become the most pronounced on the most culturally dependent, most heritable, most *g* loaded subtests. To explain this effect better theory is required. An adequate theory of general intelligence certainly needs to incorporate the developmental perspective.

In the next section we aim to reinvigorate the development of such theory. We hope to so by giving an account for the empirical relations among intelligence subtests' *g* loadings, their heritability coefficients, and their cultural load**.** To this end, we integrate various hypotheses from current theories of general intelligence and implement them in one (mutualism) model.

7.3 An Integrated Model of Intelligence

In developing a new theory, it helps to make a distinction between the factors as statistical entities and their interpretation as realistic common causes of individual differences in cognitive abilities. When doing so, two kinds of theories of general intelligence can be distinguished (see Chapter 6). The one kind, which we denote *g* theories, regards the general factor as representing a realistic (e.g. biological) variable. The positive manifold is regarded as the result of the influence of a general, but unobserved, influence, *g*. Examples of *g* theories are Spearman's (1904) two-factor theory, Jensen's (1968) Level I and level II theory, Cattell's (1963) original investment theory of fluid and crystallized intelligence, and Rushton and Jensen's (2010a) biological *g* theory. The other kind, which we denote alternative theories, do not deny the general factor as a robust statistical phenomenon, but they do not include a variable *g* as an explanatory construct. That is, in these theories the general factor does not explain the positive manifold, rather the positive manifold explains the general factor. Examples of alternative theories are Thomson's (1919; 1951) sampling theory and its elaborations (e.g. Bartholomew et al, 2009b), van der Maas et al.'s (2006) mutualism theory, Dickens' (2008) multivariate version of Dickens and Flynn's (2001) social multiplier theory, and the genetic theories of Anderson (2001) and Penke, Denissen, and Miller (2007).

Like we can distinguish among multiple explanations of the positive manifold, we can also distinguish among multiple explanations of *g* loadings (i.e., regression weights on the statistical general factor). Usually, *g* loadings are explained in terms of complexity. This is the case in Spearman's (1904) and Jensen's (1968) *g* theory, but also in certain alternative theories, for example in Thomson's sampling theory (see Jensen, 1998, for a discussion). We denote these theories complexity theories. Complexity theories predict that the more complex a item or test is the larger its loading on the general factor. Alternatively, *g* loadings have been explained in terms of environmental demands (Dickens & Flynn; Dickens, 2001). Here, the prediction is that the larger the demands of society are, the larger the *g* loading will be. Finally, mutualism theory explains *g* loadings in terms of developmental interactions (van der Maas et al., 2006). The highest *g* loadings are for the cognitive abilities that influence other cognitive abilities the most, and for the cognitive abilities that receive the most influences from other cognitive abilities.

Explanations of *g* loadings are not necessarily mutually exclusive. It is possible that society puts more demands on complex problem solving than noncomplex rote memory or perceptual speed (as in the social multiplier theory), and that the former is practiced more during the course of development than the latter, and so has larger influences on other cognitive abilities during development (as in the mutualism theory). As a result the complex tests display higher *g* loadings (as in complexity theory).

With respect to cognitive development, we can make yet another distinction between theories of intelligence. Sampling theories and *g* theories do not incorporate the developmental perspective, whereas mutualism theory and investment theory do. However, mutualism is not specific enough concerning knowledge acquisition, while investment theory often predicted effects opposite to the empirical effects reviewed in this thesis. Nevertheless, we believe that the essence of investment theory remains tenable. This is the assumption that knowledge acquisition is not only influenced by learning experiences, but also by genetic influences, via cognitive processing. This assumption can be easily implemented in the mutualism model. The assumption that the learning environment is an important mediator of these genetic effects can also be implemented. These assumptions may sound trivial, but since current theories do not fully account for the effect we want to explain, we contend it is important to be explicit about which hypotheses we retain and which we do not.

Our integrated model thus borrows from the investment theory of fluid and crystallized intelligence, but its basis is the mutualism model of van der Maas et al. (2006). The model also

incorporates the idea of a social multiplier effect (Dickens & Flynn, 2001). That is, more intelligent people end up in more cognitive stimulating environments. In the next section, we present the model in more detail and aim to explain how knowledge, the result of heritable cognitive processing and environmental influenced learning experiences, displays higher heritability than these causes themselves. We contend that the answer lies in a dynamical interplay between heritable abilities and the demands of society.

7.3.1 Hypotheses

We make the usual distinctions between crystallized abilities (knowledge, including skills and solving strategies) and fluid cognitive abilities (variables that represent differences in cognitive processing). In line with the mutualism theory, we do not consider fluid intelligence to constitute a single, realistic latent variable, but rather as the result of mutual beneficial interactions among multiple basic cognitive processes. In line with the investment hypothesis, we assume relatively large influences of fluid abilities on crystallized abilities. Since transfer of training is generally absent (e.g. Cattell, 1987; see Table 7.1), we assume that crystallized abilities do not influence each other directly. In line with mutualism, we assume that due to the process of learning, cognitive processing benefits from the development of crystallized abilities. The development of working memory, for example, benefits from actively learning the knowledge and skills that are demanded by the cognitive environment. In line with Dickens and Flynn (2001), we assume that more intelligent people end up in more cognitive stimulating environments which provide opportunities to further train cognitive skills and to acquire knowledge. In line with mutualism, the growth of such cognitive abilities is constrained by limiting capacities. Although we believe genetic sampling is present in reality (any two limiting capacities can share some genetic determinants), we also believe that this assumption is not necessary to account for the effects under investigation. The same holds for sampling on test level. With test sampling we mean that, for example, solving an item of an intelligence tests always requires previous acquired knowledge, as well as cognitive processing, but that items call upon acquired knowledge and cognitive processing to different degrees. With test sampling, we also mean that certain processes are called upon across different tests, for instance that working memory is involved in both verbal and spatial tasks. Figure 7.1 shows a graphical representation of the fully integrated model.

7.3.2 Explorations of the integrated model

Incorporating the assumptions above in the mutualism model (van der Maas, 2006), preliminary results have shown that multiple variants of this setup can lead to the observation that *g* loading and heritabilities correlate positively, crystallized abilities being the most heritable and having the highest loadings on the general factor. The variants differed with respect to the presence or absence of details of the setup (see Appendix B). The variants fall in two categories. In the first category, the systems have reached their equilibrium points, hence approximate their heritable limited capacities. In the second category they have not.

Variants of the first category only differed from the simulations of van der Maas et al. (2006) in that the mutualism matrix is made more specific (see Appendix B). We specified this matrix in accordance with the present model. For explanation of the simulation procedure and programming codes, we refer to van der Maas et al. (2006) and the R mutualism package. Both are available at http://hvandermaas.socsci.uva.nl/Homepage_Han_van_der_Maas/Home.html. Here one can also find all programming codes of the simulations that are discussed in this thesis. Here, we merely mention our main conclusions.

For the effect to appear that crystallized abilities display the largest heritabilities, the presence of genetic sampling (weak genetic intercorrelations) was required. In addition, it had to be assumed that the cognitive environment selects on general intelligence, i.e., both crystallized and fluid abilities, rather than either crystallized or fluid intelligence. Last, large genetic effects on the limiting capacities as well as large environmental effects on cognitive abilities were required.

Because in simulations of the first category all systems were in equilibrium, this may be interpreted to mean that the associated abilities were fully developed for every person in the sample.

We consider this as not very plausible. We therefore also studied the situation in which the systems were not in equilibrium. This setup is consistent with the situation in which the subjects in a given sample are heterogeneous with respect to the amount of practice. This implies the presence of individual differences in the abilities that stem from differences in development. We contend that further increase in intelligence is possible, and that crystallized abilities are usually more practiced than fluid abilities, and thus reflect better the genetically influenced limited capacities. In this setup, we could relax the assumption that environment selects on general intelligence. That is, for the Jensen effect for heritability to appear, we could assume that cognitive environment selects on knowledge and skills (hence, on crystallized intelligence) rather than general intelligence, which we consider more plausible. This assumption was not necessary, however. Neither necessary was the assumption of genetic sampling. Crystallized abilities had the largest *g* loadings and heritabilities, provided environmental influences had a large effect.

Because the simulations of the second category differ substantially from the simulations in van der Maas et al. (2006) in a number of aspects, we discuss below one of the variants in more detail (any other variants are described briefly in Appendix B). As the simulations could not be based on the equilibrium covariance matrix, we simulated data using the mutualism model itself (basic simulations with the mutualism R package are discussed in van der Maas et al).

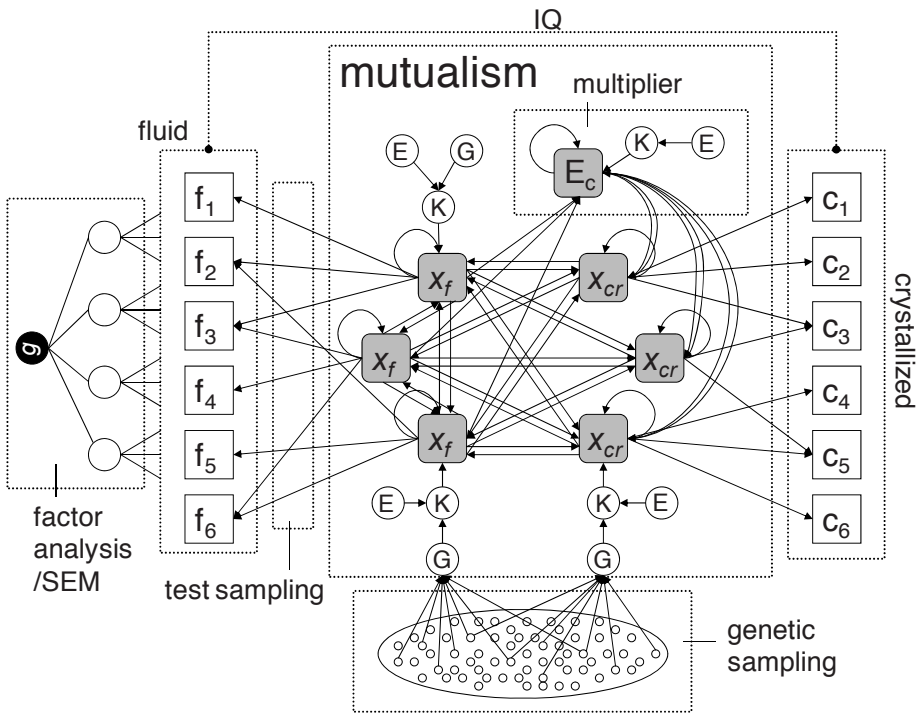

Figure 7.1 The fully integrated model. Correlations among fluid abilities are the result of mutual beneficial interaction between basic cognitive processes during development. Crystallized abilities (knowledge and skills) are the result of cognitive processing. The development of crystallized abilities has a beneficial effect on the development of fluid abilities. Some individuals turn out to be more intelligent, because they posses higher levels of cognitive abilities. More intelligent individuals will be more likely to end up in cognitive environments conducive to the further development of crystallized abilities (which is beneficial to the further development of fluid abilities). The growth of the cognitive abilities will be constrained by genetically and environmentally influenced limited capacities. These capacities are possibly weakly intercorrelated due to genetic sampling. Sampling on test level may also be present. General intelligence, as the outcome of factor analysis, is an index measure of cognitive functioning.

A simulation example One way to accomplish that crystallized abilities are practiced more than fluid abilities in the mutualism model is to assume different time scales for the development of crystallized and fluid abilities. We can accomplish this by assigning differences in growth rates (i.e., the parameters *a*, in van der Maas et al. 2006; see also Appendix B); crystallized abilities having higher growth rates than fluid abilities.

We simulated data for 17 observed variables (8 fluid abilities, 8 crystallized abilities, and 1 environmental variable) for N = 500 'monozygotic (MZ) twin pairs raised apart'. The mutualism matrix is taken as in Figure 7.2, with an additional, but arbitrary, amount of noise (mean 0; SD .02). Following van der Maas et al. (2006), starting values were sampled from a (17 dimensional) uncorrelated multivariate normal distribution with means equal to .05 and SD's of .01. Growth parameters were sampled from a 17 dimensional uncorrelated multivariate normal distribution with arbitrary means of 6 (SD = .5) for fluid abilities and 9 (SD = .5) for crystallized abilities and the environmental variable.

M

	Fluid abilities (x_f)								Crystallized abilities (x_c)								Cognitive environment (x_e)
x_f	.00	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.00
	.02	.00	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.00
	.02	.02	.00	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.00
	.02	.02	.02	.00	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.00
	.02	.02	.02	.02	.00	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.00
	.02	.02	.02	.02	.02	.00	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.00
	.02	.02	.02	.02	.02	.02	.00	.02	.02	.02	.02	.02	.02	.02	.02	.02	.00
	.02	.02	.02	.02	.02	.02	.02	.00	.02	.02	.02	.02	.02	.02	.02	.02	.00
x_c	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
	.04	.04	.04	.04	.04	.04	.04	.04	.00	.00	.00	.00	.00	.00	.00	.00	d
x_e	d	d	d	d	d	d	d	d	d	d	d	d	d	d	d	d	00

Figure 7.2 Matrix of mutualistic weights used in the example simulation study (see text). It contains the interactions (weights) among fluid and crystallized abilities and the cognitive environment.

The parameter values of the limited capacities (the parameters K in van der Maas et al., 2006; see also Appendix B) were determined as follows. N values were sampled from a 16 dimensional multivariate normal distribution with means 0 and SD's of 0.5 (these SD's equal the arbitrary SD's in van der Maas et al., 2006), with intercorrelations r_g. These parameter values represent the genetic values of the limited capacities associated with the cognitive abilities. They were taken identical for each twin (so we obtained 2*N values for each capacity associated with the cognitive abilities). Across simulations r_g varied (from 0 to .5), but within each simulation r_g was fixed (i.e., did not vary over cases). The genetic value for the limited capacity associated with the cognitive environmental variable was fixed at zero for every individual. In addition, 2*N values were sampled from a 17 dimensional uncorrelated multivariate normal distribution with means 0 and standard deviations .5. These represent the environmental values of the limited capacities.

We added the genetic and environmental values together with weights $\sqrt{(h^2)}$ and $\sqrt{(1- h^2)}$ respectively, where h^2 was set at an relatively high, but arbitrary value of 0.7. This implies that the capacities associated with the cognitive variables had an expected heritability of 0.7, and that the capacity associated with the environmental variable had an expected heritability of 0. Next, we

rescaled the values to accomplish they had a mean of 3. In this way, the mean and SD of the parameters equaled the arbitrary settings of van der Maas et al. (2006).

To obtain data of N twins, we ran the mutualism model $2*N$ times with the twin parameter values. Data were obtained for one point in time (set arbitrarily at $t = 1$), after the beginning of development ($t = 0$), but before the systems reached their end states (which was around $t = 2.5$). Each simulation resulted in a data set of measurements of the 17 variables of the $2*N$ subjects. The observed heritabilities were estimated by calculating the MZ twin correlation for each of the 16 cognitive abilities. The values on the cognitive abilities of the first members of the twin pairs were submitted to factor analysis. That is, we followed the common practice of inspecting the intercorrelations of the observed variables, plotting the correlation matrix' eigenvalues, and fitting the common factor model.

Figure 7.3 shows the results of a typical simulation run in which d (the interaction weights between the cognitive environment and cognitive abilities; see Figure 7.2) is 0.2, and in which the limited capacities were all completely independent (i.e., $r_g = 0$). Thus, genetic or environmental intercorrelations among the underlying limited capacities were absent. Correlations among the observed cognitive abilities were all positive, the first eigenvalue was dominant and a common factor model fitted the data. Heritabilities and g loadings showed a positive correlation. Only when d was positive and relatively large did the crystallized abilities display the highest g loadings and heritabilities.

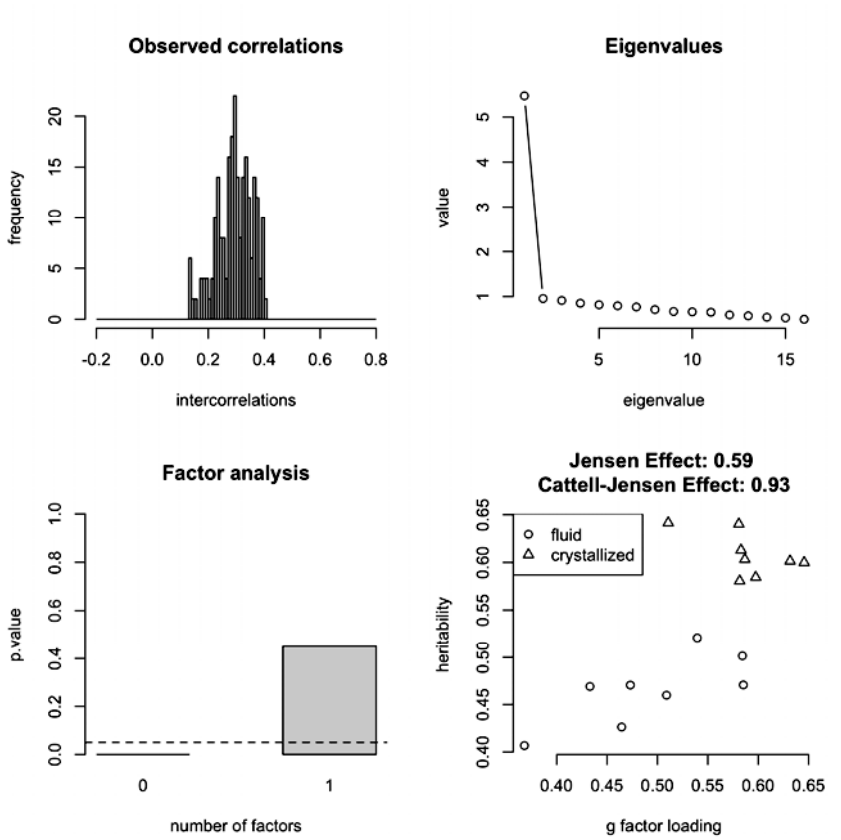

Figure 7.3 The results of a typical simulation run with the mutualism model, using the design matrix from Figure 7.2, and assuming completely independent limiting capacities. A dominant first eigenvalue is present and a common factor model fits the data. Crystallized tests have the largest loadings on this factor. They are also the most heritable.

7.4 Overall Conclusions

Current theories of intelligence in isolation do not account for the empirical relations among intelligence subtests' loadings on the general factor, heritability coefficients, and their cultural load. One of the purposes of this chapter was to account for these relations. To this end, we integrated hypotheses form different theories of intelligence. Another purpose of this chapter was to show that the mutualism model of intelligence can be used to study (combined) effects and new and old hypothesis. As an example we implemented the hypotheses above in the mutualism model. From our simulation studies, we concluded that the relations among *g* loading, heritability, and cultural load will be positive when the society fosters crystallized abilities rather than fluid abilities.

The simulations also showed that the general factor of intelligence does not have to represent a realistic (e.g. biological) common cause of individual differences. In our integrated model it is a merely a statistical entity, 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. A distinction between *g* as a statistically entity, and *g* as interpreted as a realistic, common cause of individual differences, is not only important theoretically, but also empirically. Consider genetic association and linkage studies of intelligence, for example. So far, the search for genes for general intelligence has met with relatively little success (Deary, Johnson, & Houlihan, 2009; Plomin and Spinath, 2004; Chabris et al., *in press*). The alternative theories of Dickens and Flynn (2001; Dickens, 2008) and of van der Maas et al. (2006), in which the general factor of intelligence is a statistical entity originating in reciprocal beneficial interactions among cognitive processes or abilities, are able to provide a plausible explanation of this lack of success. In these theories, there are no direct genetic influences general to all abilities. If general intelligence is indeed the outcome of such interactions, the search for specific 'genetic influences on *g*' is a questionable undertaking (Dolan, Kan, van der Maas, 2008; van der Sluis, Kan & Dolan, 2010), in particular when (1) the genetic determinants of these processes are under mutation selection balance, which means that the process of natural selection reduces genetic variance but cannot deplete all of it because new variation - due to mutations - is continually reintroduced (Penke et al., 2007), and (2) the interactions are nonlinear (e.g. Molenaar, Boomsma, & Dolan, 1993; Kan, Ploeger, Raijmakers, Dolan & van der Maas, 2011).

Cross-sectional factor analysis cannot discriminate between *g* theories and alternative theories of general intelligence. In addition, both *g* theories and alternative theories can explain the facts that the general intelligence is (highly) heritable and has (strong) biological correlates. So these facts will not discriminate between different theories either. Developmental analysis on the other hand, may have the potential to discriminate between theories of intelligence. For example, group factors of intelligence are more or less genetically independent initially, but become increasingly genetically interrelated as development unfolds (Hoekstra et al., 2007). This observation is consistent with mutualism, but is not readily explained by *g* theories or sampling theories. Developmental effects are thus important in the understanding of individual differences. We hope future (behavior genetic studies) studies into intelligence will use longitudinal modeling. Together with formal, dynamical system modeling, this will enrich intelligence research.