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A Dynamical Model of General Intelligence: The Positive Manifold of Intelligence by Mutualism

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Scores on cognitive tasks used in intelligence tests correlate positively with each other, that is, they display a positive manifold of correlations. The positive manifold is often explained by positing a dominant latent variable, the *g* factor, associated with a single quantitative cognitive or biological process or capacity. In this article, a new explanation of the positive manifold based on a dynamical model is proposed, in which reciprocal causation or mutualism plays a central role. It is shown that the positive manifold emerges purely by positive beneficial interactions between cognitive processes during development. A single underlying *g* factor plays no role in the model. The model offers explanations of important findings in intelligence research, such as the hierarchical factor structure of intelligence, the low predictability of intelligence from early childhood performance, the integration/differentiation effect, the increase in heritability of *g*, and the Jensen effect, and is consistent with current explanations of the Flynn effect.

Keywords: intelligence, *g* factor, dynamical systems, mutualism, reciprocal causation

In the study of intelligence, one empirical phenomenon is well established: Test scores on cognitive tasks show a positive manifold, that is, they are invariably positively intercorrelated, albeit to varying degrees. This implies that people who score well on one cognitive test are likely to score well on other cognitive tests. The positive manifold is a robust phenomenon. For instance, it does not depend on the exact nature of the cognitive task, and it is observed in a variety of populations (Jensen, 1998). It is in the light of this empirical phenomenon that many differential psychologists accept the idea of an underlying factor of general intelligence, commonly denoted as *g*, at least as a working hypothesis (Carroll, 1993; Gottfredson, 1998; Jensen, 1998; Thorndike, 1994). As Bartholomew states, "If a set of test scores tends to be positively correlated among themselves there is a *prima facie* case for believing that those correlations are induced by a common dependence on a latent variable" (Bartholomew, 2004, p. 62).

It is important to distinguish between *g* as a psychometric and *g* as a psychological construct (Thorndike, 1994). From a psychometric point of view, the *g* factor is the outcome of factor analysis of the correlation matrices of cognitive test scores. In the simplest case, such analyses produce a dominant first-order general factor (see Figure 1a), or a dominant first eigenvalue. In more advanced versions of factor analyses of a wide variety of IQ tests (e.g., confirmatory factor analysis, exploratory or confirmatory hierarchical factor analysis), *g* is conceptualized as a higher order factor or as a general first order factor in the presence of more specific group factors (Carroll, 1993; Gustafsson, 1984; Mackintosh, 1998). In addition, in hierarchical models, *g* may be identified with first-order factors such as fluid intelligence (Horn & Noll, 1994). From a psychometric (i.e., factor analytic) point of view, *g* is considered to be well established (cf. Bartholomew, 2004). In this view, *g* is a summary measure or index of the positive manifold. As a psychological construct, *g* is much more controversial. This controversy centers on the question of the origin or nature of *g* (Sternberg & Grigorenko, 2002). Can psychometric *g* be identified with some psychological or biological variable? Does *g explain* differences amongst individuals in performance on diverse mental tests (Gottfredson, 1998)?

A century ago, Spearman (1904, 1927) introduced the notion of mental energy as the main cause or origin of *g*. Many current explanations are of this 'single quantitative latent factor' type. We denote this the *g* explanation. For instance, it has been argued that individual differences in *g* are due to individual differences in an underlying cognitive factor, such as speed or efficiency of information processing, working memory, or the capacity to handle cognitive complexity (for reviews, see Deary, 2002; Detterman, 2002; Jensen, 1998). Alternatively, *g* is identified with underlying biologically related factors such as brain size, neural efficiency or pruning, or neural plasticity (Detterman, 2002; Garlick, 2002; Gray & Thompson, 2004). Although there is ample evidence that these factors play a major role in intelligence, none of these factors is generally accepted as the unitary cause of *g* (Ackerman, Beier, & Boyle, 2005; Luciano et al., 2005).

The large body of research on *g* may give the impression that the *g* explanation is the only possible explanation of the positive

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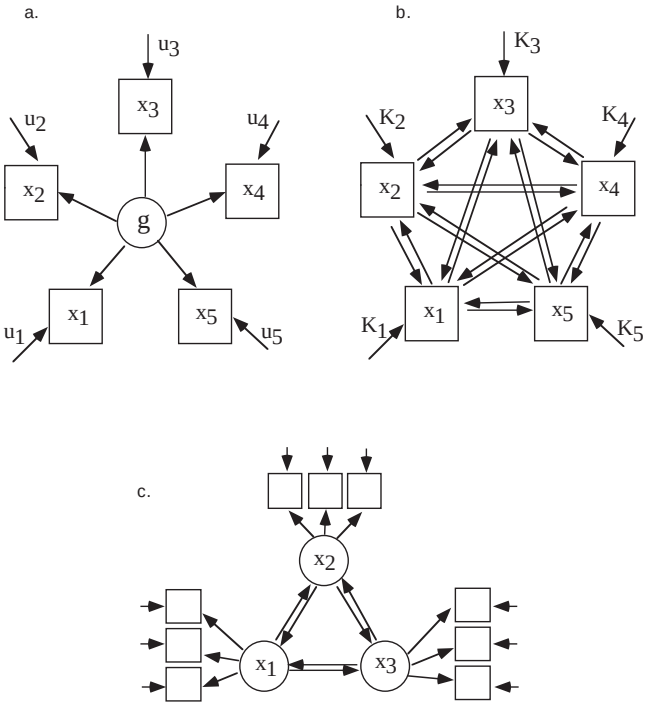

Figure 1. Three models of the positive manifold: (a) the standard g model, (b) the mutualism model, and (c) the extended mutualism model. Squares and circles denote manifest and latent variables, respectively. Symbols *x* denote processes, *u* unique variances, and *K* resources (see text).

manifold. However, to establish the *g* factor, the analysis of correlations is insufficient (Borsboom, Mellenbergh, & van Heerden, 2004). In the words of Bartholomew (2004), "What we have done is to demonstrate that what we have observed is what we would have expected if an underlying variable, called *g*, did exist. It leaves open the possibility that some other mechanism could have produced the correlation" (p. 73). Thus we must exclude alternative mechanisms, which do not include a general factor as an underlying quantitative variable but which do give rise to the positive manifold.

Thorndike (1927) and Thomson (1951) proposed one such alternative mechanism, namely, sampling. In this sampling theory, carrying out cognitive tasks requires the use of many lower order uncorrelated modules or neural processes (so-called bonds). They hypothesized that the samples of modules or bonds used for different cognitive tests partly overlap, causing a positive correlation between the test scores. In this view, the positive manifold is due to a measurement problem in the sense that it is very difficult to obtain independent measures of the lower order processes. Jensen (1998) and Eysenck (1987) identified three problems with this sampling theory. First, whereas some complex mental tests, as predicted by sampling theory, highly load on the *g* factor, some very narrowly defined tests also display high *g* loadings. Second, some seemingly completely unrelated tests, such as visual and memory scan tasks, are consistently highly correlated, whereas related tests, such as forward and backward digit span, are only modestly correlated. Third, in some cases brain damage leads to very specific impairments, whereas sampling theory predicts general impairments. These three facts are difficult to explain with sampling theory, which as a consequence has not gained much acceptance.[1] Thus, the *g* explanation remains very dominant in the current literature (see Jensen, 1998, p. 107).

The aim of this article is to outline a third possibility, a new explanation of the positive manifold. This explanation is based on a mathematically formulated developmental model with mutualism or positive beneficial relationships between cognitive processes. This explanation identifies a plausible mechanism that gives rise to the positive manifold but that does not include *g* as a latent quantitative variable. At the very least, this demonstrates that a latent variable, which is well established psychometrically (i.e., in factor analyses), need not correspond to an actual quantitative variable, such as speed of processing or brain size. This model also suggests explanations of other important empirical phenomena in intelligence research. We present and discuss possible explanations for the low predictability of intelligence from early childhood performance, the hierarchical factor structure of intelligence, the increase in heritability of *g*, the Jensen effect, the differentiation effect, and the Flynn effect.

The article is organized as follows. We first introduce a biological metaphor concerning ecosystems to explain the idea underlying our explanation. We then briefly introduce the models that biologists use to study such complex ecosystems. Following from these models, we introduce a model for intelligence based on mutualism or reciprocal causal interactions. In order to investigate this simple mathematical model, we introduce three scenarios for the explanation of data from intelligence research. Computer simulations confirm that mutualism gives rise to, and thus embodies an explanation of, the positive manifold. In the remaining sections we investigate other aspects of psychometric *g*, such as the development of intelligence, the differentiation effect, the heritability of intelligence, and the Flynn effect. We end with a discussion of possible extensions, open questions, and possible objections.

The Ecosystem Metaphor

Suppose we investigate the ecosystems of several small lakes in a specific area. We set out to determine how well these ecosystems function, for instance regarding biodiversity or sensitivity to external perturbations.[2] Our research strategy consists of collecting data concerning many different aspects of the lakes, such as measures of the quality of water and number and diversity of flora and fauna. Suppose we observe a positive manifold in these measures, and a dominant general factor in the subsequent factor analyses of the data. This would imply that "good" lakes are better,

[1] The position of Jensen is perhaps inconsistent. In his discussion of the question whether g is unitary (Detterman, 2002; Jensen, 1998, p. 260; Kranzler & Jensen, 1991), he uses arguments similar to sampling theory, which he rejected earlier. With regard to the unitary question, he argues that the *g* factor is just a psychometric construct and may not relate to a single underlying process or capacity. It is our impression, however, that most differential psychologists believe that *g* is more than a statistical construct. A recent genetic formulation of sampling theory can be found in B. Anderson (2001).

[2] In ecology, there is little consensus on measures for comparing ecosystems (but see Kennedy et al., 2002, and Brooks & Kennedy, 2004, on biodiversity barometers). Furthermore, real ecosystems include many competitive relations and will not show a simple positive manifold. For our present argument, we discard these issues.

to varying degrees, than "bad" lakes in all measured aspects. Thus, we may ask: Is there a type of *g* factor for lakes? Is there an equivalent of mental energy, a 'speed' factor, or a 'plasticity' factor for ecosystems?[3] We do not exclude this possibility. However, what is more important to our present proposal is that biologists have other explanations of these phenomena in ecosystems.

In mathematical biology, ecosystems are often modeled with coupled differential or difference equations. Famous are the Lotka-Volterra type models for prey–predator population dynamics (May, 1973; Murray, 2002). The basic Lotka-Volterra prey–predator model concerns two species with population sizes x_1 (prey) and x_2 (predator). Prey grows according to the logistic map, with birth factor *a*, and constrained by environmental resources *K*. Furthermore, predation by x_2 decreases x_1 by a factor *M*, the effectiveness of predation. x_2 has a constant death rate *c*, and its reproduction is proportional with factor *b* to amount of prey and *M*. The idea is that number of prey x_1 increases when number of predators x_2 is low and decreases when predators are numerous, whereas number of predators increases when prey are numerous and decreases when prey are rare. This leads to oscillations in both x_1 and x_2. The dynamics are specified in two equations that define the change dx/dt of the population sizes x_1 and x_2 over time:

$$\frac{dx_1}{dt} = ax_1(1 - x_1/K) - Mx_2x_1, \quad (1)$$

$$\frac{dx_2}{dt} = (Mbx_1 - c)x_2. \quad (2)$$

This elementary system generates a number of fascinating and complex phenomena, as described in any basic text on population dynamics (and illustrated with many java applets on the internet). Current more realistic models can be very complex and detailed, providing accurate description of, and predictions concerning, the dynamics of large ecosystems (e.g., Kondoh, 2003; Prakash & de Roos, 2004). In these models, interactions in the form of competition and cooperation are essential and will give rise to correlations between different aspects or parts of the ecosystem.

Our dynamical explanation of the positive manifold of cognitive tasks is based on this type of interaction in multivariate dynamical systems (cf. van Geert, 1991). We argue that the positive manifold may be a by-product of the positive interactions between the different cognitive processes of the system. In our proposal, all processes of the system are initially undeveloped and uncorrelated. During the development of the system, the dynamical interactions give rise to correlations among the processes of the system.

A Simple Dynamical Model for the Positive Manifold of Intelligence

The model consists of two parts, a dynamical part and an interaction part. The dynamical part represents the development of cognitive processes. Developmental curves generally first show a strong increase and then reach some kind of asymptote (i.e., intelligence does not grow unboundedly). This type of growth pattern can be modeled in many ways. A popular model is the logistic growth model (first part of Equation 1), which was introduced as model for psychological growth by van Geert (1991) and is also used by Eaves, Kirk, Martin, and Russell (1999) and Molenaar and Raijmakers (1999) in simulations of genetic effects in development. The logistic model can be seen as an elementary model of growth, which can be extended in many ways. We also chose a very simple interaction model, which is a multivariate extension of the second part of Equation 1. Interactions are determined by a matrix of weights. Figure 1b gives a schematic view of the model. We will introduce the model step by step.

The first step is the assumption that intelligence is based on underlying cognitive processes, such as perceptual, memory, decision, and reasoning processes. There is at present little consensus about the specific basic processes underlying intelligent behavior (Deary, 2002, p. 153). It is possible to adopt one theoretical proposal, like the Sternberg model or the minimal cognitive architecture model of M. Anderson (1992) and develop the mutualism model in these terms. The circles and squares used in Figure 1c could be labeled with components proposed by Sternberg (1988). However, our account of the positive manifold does not hinge on a particular cognitive architecture or brain model. We use the term cognitive processes in a general sense, including such notions as modules, capacities, abilities, or components of the (neuro)cognitive system, as they are routinely measured in intelligence tests. Thus, as "species" in our model, we consider *W* processes x_i (i in $1 \ldots W$), which represent basic cognitive (perceptual, memory, decision, etc.) processes. The processes are denoted by a vector **x**. The model requires some quantification of the processes. Of course, a simple quantification as used in biology (number of animals) is not possible. The standard way to quantify psychological processes is by a test score. We assume that for each process in our model, such a score is available. Given the existence of a large number of intelligence tests and a large number of cognitive tasks, this is feasible in principle.

Our choice for the logistic growth model is based on new dynamical system approaches to the study of psychological development (e.g., Port & van Gelder, 1995; Thelen & Smith, 1994; van der Maas & Hopkins, 1998; van Geert, 2003). Key concepts in this approach are emergence, autocatalytic growth, phase transitions, and self-organization. The basic logistic model that we apply does not incorporate all these phenomena (although it can; see van Geert, 1991). It does, however, incorporate auto-catalytic growth based on the notion that the development of cognitive processes is largely an autonomous self-regulating process (Molenaar, Boomsma, & Dolan, 1993).

The logistic model includes three types of parameters: initial values, growth parameters, and carrying capacities (or limited resources). The initial values **x0** are not important in our application of the model. They will be set to uncorrelated and arbitrary low values. They are important for certain applications of the logistic model in which sensitivity to initial conditions plays an important role (e.g., models with large values of the growth parameters, e.g., van Geert, 1991, or modeling involving strong competition; Sprott, 2004).

The *W* growth parameters in **a** determine the steepness of the logistic growth function associated with each *x*. We can assume the

[3] As an alternative example we may consider humans with severe psychological problems or the crises in the poorest developing countries. Concerning the latter, we might ask whether they are due to one underlying cause (in case of developing countries, IQ, according to Lynn & Vanhanen, 2002) or the interaction between many relevant variables (Diamond, 1997).

growth parameters to be correlated or uncorrelated. As we demonstrate below they need *not* be correlated to explain the positive manifold. If they are assumed to be correlated (e.g., due to shared environment in the early phase of development), other empirical phenomena can be explained too. The W parameters in **K** determine the asymptotes of the logistic growth processes and are often interpreted as limiting resources or carrying capacity for growth. We assume that the growth of each cognitive process uses limited resources. We conceptualize these resources mainly in terms of biological constraints, such as neuronal speed and size of neural systems associated with each of the cognitive processes (Garlick, 2002; Jensen, 1998). For instance, the growth of short-term memory reaches a maximum in late childhood presumably because of limitations of the underlying neural system (e.g., Gathercole, 1999). As with the growth parameters, we may assume the limited resources of the processes to be correlated or uncorrelated. The positive manifold does not require correlated resources, although correlated resources may explain additional phenomena.

The next major step is the assumption that these cognitive processes have mutual beneficial or facilitating relations. Each process supports the development of other processes. This view of relations in developing complex systems is in accordance with modern views of dynamical systems (for discussion, see Wagner, 1999). These positive relations can be direct (bidirectional or reciprocal) or indirect (via other processes). Reciprocal causal relations are well known in the psychological literature. For instance, better short-term memory helps to develop better cognitive strategies, and better strategies make it possible to increase the efficiency of short-term memory (Siegler & Alibali, 2005). There are many examples of positive influences of language on cognition, and visa versa. Examples are syntactic bootstrapping (Fisher, Hall, Rakowitz, & Gleitman, 1994), and semantic bootstrapping (Pinker, 1994). Similar examples are the relations between cognition and meta-cognition (Sternberg, 1998), between action and perception (Gibson, 1986), and between performance and motivation (Dweck, 1986). Clearly, these positive reciprocal relations are not limited to the intellectual domain. For instance, abstract thinking may help to find creative solutions for interpersonal social or emotional problems (Gottfredson, 2002), whereas good control over emotional and social life are beneficial to academic success (Strahan, 2003). Ideally such positive influences are demonstrated in experimental research, in which the independent variable is manipulated experimentally.[4] It is of course possible that there are no facilitating interactions between certain processes, or even competitive or debilitating interactions. A simple example of the latter is the time constraint on cognitive expertise. Becoming an expert in say, chess, may not allow other specializations. Below we demonstrate that the model can include a good degree of zero or competitive interaction without affecting the fundamental result of the positive manifold of correlations. In short, we propose to view the cognitive system as a developing ecosystem (or society) with primarily cooperative relations between cognitive processes. Note that this model does not make use of latent variables.[5]

Given these assumptions we can formulate the model mathematically:

$$\frac{dx_i}{dt} = a_i x_i(1 - x_i/K_i) + a_i \sum_{\substack{j=1 \\ j \neq i}}^{W} M_{ij} x_j x_i / K_i \quad \text{for } i, j = 1 \ldots W. \quad (3)$$

Variables x_i represent the W cognitive processes. Parameters a_i are growth parameters, determining the steepness of the logistic growth function associated with each x_i. Parameters K_i represent the limited resources of the logistic growth processes. The matrix **M** contains weights M_{ij}, used to specify the generally positive, possibly asymmetric, relations between pairs of processes in development. Parameters **x0**, **a**, **K**, are random parameters (i.e., they differ over subjects), whereas the matrix **M** contains population parameters (i.e., they are equal for all subjects). In the biological literature, this type of model is known as the Lotka-Volterra competition model. However, given that in our application the M_{ij} are generally positive, it is more appropriately called a mutualism model (Murray, 2002). This is the simplest instance of a model for mutualism, but it suffices for our present purposes.[6] The dynamics of the mutualism model are fairly simple. For appropriate starting values **x0** and appropriate values of the parameters **a**, **K**, and **M**, each x_i converges to a constant level or a steady state. Large values of **M** imply that the x_i grow without bound, which is of course unrealistic and should be avoided in simulations (see Appendix and Murray, 2002).[7] Figure 2a shows the results of a typical run of the model (the development of one individual). Starting from $x0_i$ each process follows a logistic curve until an asymptote (larger than K_i) is reached. With all $M_{ij} = 0$, the asymptotes equal K_i. In the Appendix, we provide the formulas for the stable states and the associated expected covariance matrix.[8] These can be used to investigate the behavior of the model. For instance, the stable states of the model are independent of both **x0** and **a**, implying that individual differences in the initial phase of development do not predict later differences. Below we discuss this in more detail. We first focus on the positive manifold, as this is the most important empirical fact in the study of intelligence.

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[4] But this is not strictly necessary. Under certain assumptions, cross-lagged effects in the multivariate longitudinal panel design and in multivariate time series can be used to study causality (Pearl, 2000; Rogosa, 1980; Wadsworth, DeFries, Fulker, Olson, & Pennington, 1995).

[5] In his classification of stereotypical influence patterns that may describe correlation data, Cattell (1965) called this model structure the general reticule (see McArdle, 1984). Cattell never investigated this structure.

[6] In theoretical biology, this model is applied to the study of food webs (cf. Bruno, Stachowicz, & Bertness, 2003) and biodiversity (cf. Kennedy et al., 2002). Recently proposed extensions are models with adaptive **M** (Ackland & Gallagher, 2004; Kondoh, 2003), multiple resources (Kooi, Kuijper & Kooijman, 2004), environmental effects on **K** (van Nes & Scheffer, 2004), strong competition with new species (Sprott, 2004), and stochastics (Mao, Sabanis, & Renshaw, 2003). This work can be very helpful in extending the mutualism model for the positive manifold in intelligence. van Geert (1991) investigated a number of applications to psychological development.

[7] May (1975) described this unbounded growth as an "orgy of mutual benefaction" (p. 95). Recently, it has been shown that stochastic versions of the Lotka-Volterra mutualism model do not have this problem. The addition of tiny amounts of environmental noise prevents the unbounded growth of the x values (Mao et al., 2003).

[8] The formula for the covariance matrix resembles Submodel 3B of LISREL (Jöreskog & Sörbom, 1993). We may use LISREL to fit the mutualism model directly to real data. Clearly, identification requires some further restrictions on **M**. However, fitting the mutualism model to real data, although interesting, is not immediately relevant to our main message in this article.

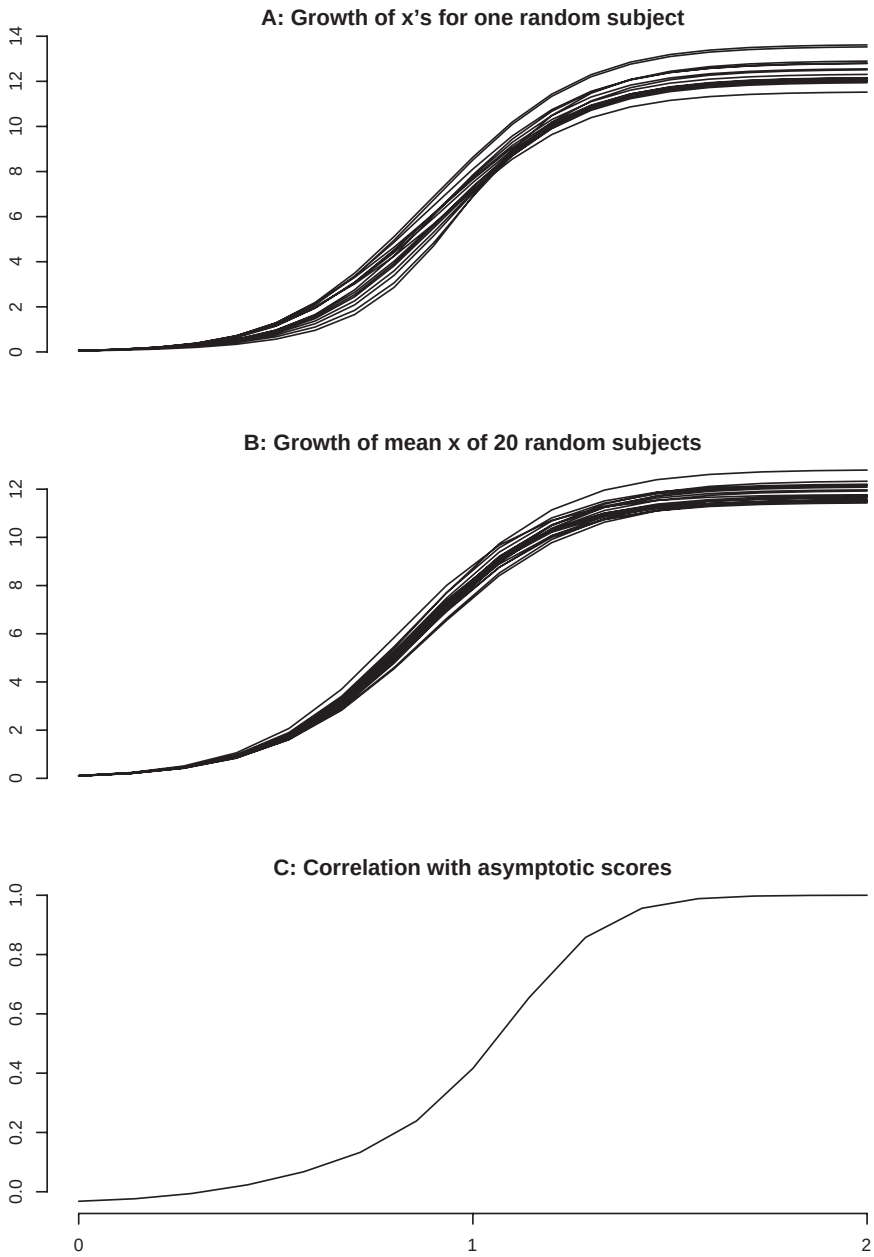

Figure 2. (a) Simulation based on Equation 3 with 16 processes. Mean (*SD*) of a_i, K_i, and $x0_i$ are respectively: 6 (.5), 3 (.5), .05 (.01). All M_{ij} equal .05. On account of the positive interactions, the asymptotes clearly exceed the K_i values. (b) Growth of the average of the 16 processes (an index of psychometric *g*) for 20 random subjects. (c) Correlation between average scores at different times in development with asymptotic scores. The model predicts that the stability of intelligence scores is initially very low but increases steadily during development.

Explaining the Positive Manifold

There are at least two independent ways in which the positive manifold may arise in the mutualism model: by specifying correlated parameters (especially **K**) or by positive **M**. When the resource parameters **K** are correlated we enter a *g* like scenario: Above-average scores on many different tasks are explained by one above-average biological or cognitive resource or set of correlated resources. Obviously, in such a case we expect a positive manifold. However, if all model parameters are uncorrelated, and **M** is positive (and equal over subjects), there is no such thing in the model as a single underlying factor. We hypothesize that beneficial relations between processes also result in a positive manifold and consequently give rise to a single dominant factor in a factor analysis.

To study these two mechanisms we investigate three scenarios. In the first scenario, the model parameters are uncorrelated and all $M_{ij} = 0$; hence, no positive manifold is expected. In the second scenario (*g*), the **K** parameters are correlated and all $M_{ij} = 0$. In the third scenario (mutualism), the **K** parameters are uncorrelated and

all $M_{ij} > 0$. The last two scenarios should both give rise to a positive manifold. If Scenario 3 does indeed produce a positive manifold, this demonstrates that mutualism suffices to explain this important finding in intelligence research.

For each scenario, we simulated and analyzed data. The main result below can also be derived analytically (see Appendix; the derivation of the positive manifold). However, the results of simulations are easier to obtain and to understand. Also many other results could only be established by means of simulation. We ran the model N times with new samples of the parameter values, to obtain data of N subjects. Data were obtained for one point in time, after the steady state was reached. Each simulation resulted in a data set of measurements of W variables of N subjects, for example, the kind of data that are used in factor-analytic studies of intelligence. Unless stated otherwise, W and N are set to 16 and 1,000, respectively. We did not add measurement error, but adding measurement error does not change the results qualitatively. In analyzing the data, we followed the common practice of inspecting the correlations between observed variables, plotting the eigenvalues of the correlation matrix and fitting the common factor models.

Scenario 1: No Positive Manifold

In this scenario, $M_{ij} = 0$ for all runs (subjects). The parameters $x0_i$, a_i, and K_i for each process i are sampled from uncorrelated normal distributions with means (standard deviations in parentheses) equal to 6 (.5), 3 (.5), and .05 (.01). These values are quite arbitrary; other values could have been used. The parameters are uncorrelated, so there is no mutualism, and we expect a zero-factor solution to characterize the data. In Figure 3, we report the main findings of this scenario. The histogram shows that the correlations between the observed variables **x** are indeed close to zero. The eigenvalues in the next panel also suggest a zero-order structure. If there is a dominant factor underlying the data, we expect a dominant first eigenvalue (much larger than one) and much smaller subsequent eigenvalues. The last panel shows the p values of the fit of factor models with 0 to 4 factors for the observed variables and the parameters. The zero-factor model of the observed variables (**x**) cannot reasonably be rejected, that is, the associated p value is larger than .05.[9]

Scenario 2: g

In this scenario, we simulated the standard explanation of g in terms of a latent factor influencing all processes. This was accomplished by sampling the resources K_i for each run (subject) from a correlated multivariate distribution ($r = .5$) with $\mathbf{M} = 0$. Figure 4 summarizes the results. The correlations between the observed variables (**x**) are all positive ($\approx .5$) and the eigenvalues suggest a dominant factor; the zero-factor model for the observed variables (and for **K**) is rejected, whereas the one-factor model fits the data well. All variables x_i load highly on this factor, as shown in the fourth panel.

Scenario 3: Mutualism

The only difference with Scenario 1 concerns **M**. All M_{ij} are fixed at .05. Parameters **K** (as well as **a** and **x0**) are drawn from an uncorrelated multivariate normal distribution. As shown in the first panel of Figure 5, the observed correlations are again close to .5.

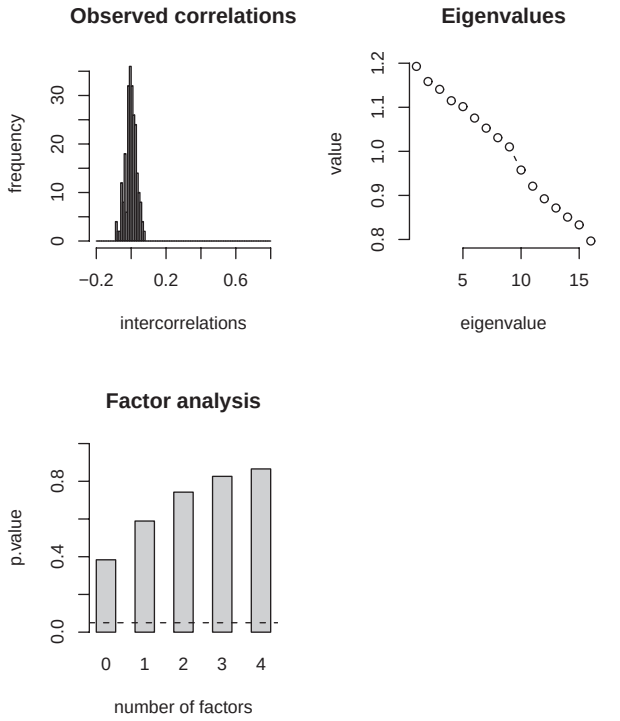

Figure 3. Scenario 1: All M_{ij} are zero and parameters are uncorrelated. The first panel shows the correlations between the observed variables, the second shows the eigenvalues, and the third shows p values of the fit of factor models with 0 to 4 factors. A zero factor model fits the data well. The dashed line shows the $\alpha = .05$ level.

The pattern of eigenvalues is very similar to that of Scenario 2.[10] A single common-factor model fits the observed data well, and all variables have large positive loadings on this factor. This shows that mutualism may give rise to the positive manifold and a single dominant common factor in factor analysis of the data generated with this model. This result was found to be very robust. That is, if all M_{ij} are drawn from a normal distribution with a positive mean (for instance with $M = .05$, $SD = .03$, with about 5% negative M_{ij}), we find very similar results (only the variance of the distribution of observed correlation increases). Also **M** can be sparse, with many $M_{ij} = 0$, or the M_{ij} can be made subject dependent (i.e., random) without changing the results qualitatively.

[9] The figure shows the result of one simulation with 1,000 runs. Each scenario was simulated many times, but the results were very stable and consistent with the analytical solution in the Appendix. For this reason, we decided not to report means over simulations. The results in the figures represent average results very well.

[10] These results and inspection of the formula for the covariance matrix (see Appendix) shows that the mutualism model and the standard factor models are statistically equivalent (i.e., implying the same covariance matrixes with equal numbers of parameters) in some particular cases. For instance, the model in Scenario 3 and the one factor model with equal factor loadings are equivalent, but other variants of the mutualism model are not equivalent to any factor model (for instance, a model in which the beneficial relations between processes are organized in a circle). Model equivalence in structural equation modeling is a complex issue (Molenaar, 2003; Raykov & Penev, 1999) and the present case requires further study.

Summarizing, we have shown two independent mechanisms for the creation of a positive manifold in the correlations between a set of observed variables: (a) by using correlated parameters (Scenario 2) and (b) by mutualism (Scenario 3). Scenario 2 is compatible with a *g*-type explanation, as here the resources of all processes are correlated. The explanation demonstrated in Scenario 3 is, to our knowledge, new.[11] This explanation does not involve or require a latent quantitative variable such as *g*, even though factor analysis of data of the mutualism model does produce a single common factor.

An interesting question arises as to what causes the individual differences in Scenario 3. A *g* factor in the sense of a latent quantitative variable is absent in this scenario, but the positive manifold does emerge, which implies that people, who are good at one test, are good at other tests as well. What then explains these individual differences, given that *g*, on account of its absence, cannot be invoked? It cannot be **M** itself, since **M** is equal over subjects. The answer lies in the resources **K** (weighted by **M**). Although all **K** are sampled from the same uncorrelated multivariate normal distribution, small differences in the average of **K** between model runs (subjects) exist. By mutualism, these differences influence all processes, which results in the positive manifold. Therefore, the weighted sum of **K** is a good predictor of the individual differences in cognitive performance. A formal treatment of this point is given in the Appendix (see *The Relation Between g and* **K**).

Note that the average or sum of uncorrelated **K** is not a common factor, in the factor analytic sense (i.e., a single underlying variable). For instance, one person's high performance on cognitive tasks may be due to exceptionally high K_i for certain processes (e.g., memory processes), another person's high performance may be due to high K_i for completely other processes (e.g., language processes), and a third person's high performance might be due to the general absence of low K_i. This differs fundamentally from the *g* explanation of individual differences in which similar performance is due to similar *g* value. This *g* explanation leads to the expectation that we should be able to find some (presumably biological) factor equal to *g* (Bartholomew, 2004). Failure to find such a factor supports alternative approaches, for example, the mutualism model.

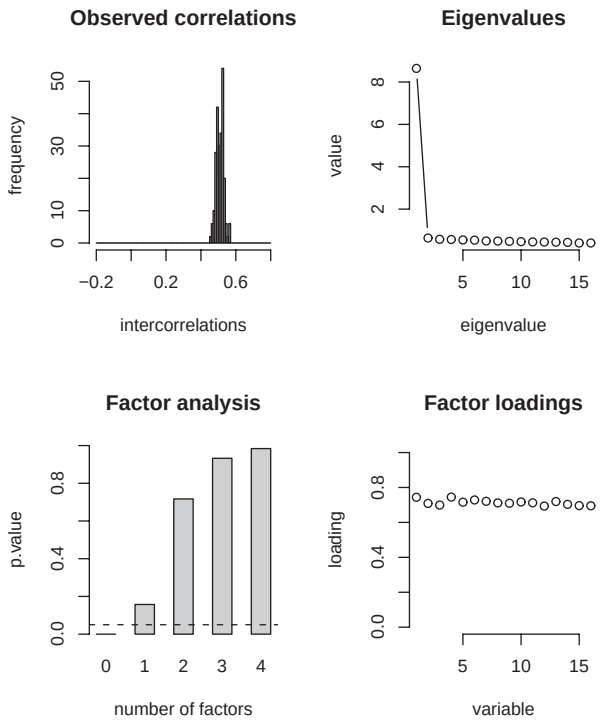

Figure 4. Scenario 2: Resources are correlated but M_{ij} are zero. The correlations between observed variables are positive, the eigenvalues suggest one factor, and a one-factor model fits the data well. All variables x_i load high on this factor.

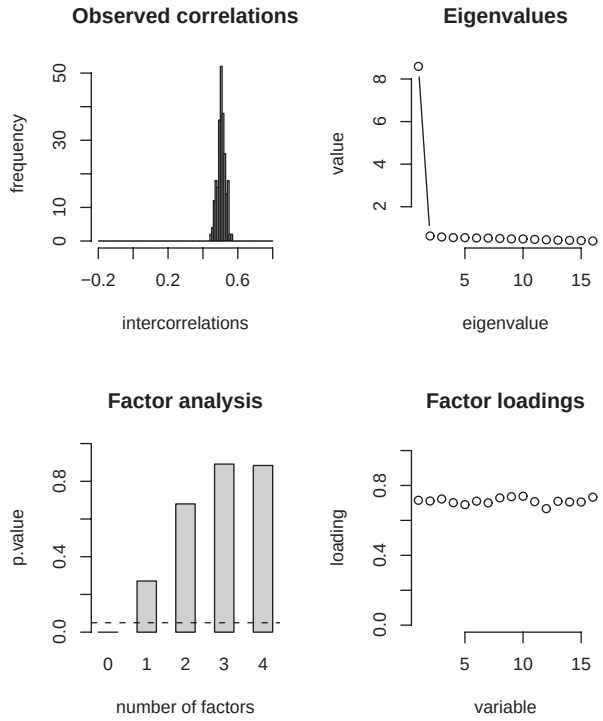

Figure 5. Scenario 3: Resources (and other parameters) are uncorrelated but all $M_{ij} = .05$. The correlations are positive, the eigenvalues suggest one factor, and a one-factor model fits the data. All observed variables load high on this factor.

Other Empirical Phenomena in the Study of General Intelligence

The positive manifold is by far the most important phenomenon to explain in any model of intelligence. The explanation in terms of mutualism, that is, facilitating developmental relations between processes, in fact, does not depend on the precise set-up of our model. The logistic growth model is not essential, although posi-

[11] There are important similarities with Detterman's (1987) complex system account of intelligence, in which mental ability is viewed as a complex system of independent but interrelated parts. However, Detterman's proposal is more akin to sampling theory. Basic processes are uncorrelated and "system wholeness" is a function of level of measurement. More complex measures will include more parts of the system and will therefore suggest a higher degree of wholeness (see summarizing point 3 of Detterman, 1987).

tivity of interaction is. The logistic growth model is, however, important for the explanation of other empirical results. In the next sections, we investigate a number of empirical phenomena.

Hierarchical Factors

Scenario 3 gives rise to a correlation matrix that fits a factor model, with a single first-order factor. Real psychometric cognitive abilities data, however, require several correlated first-order factors and thus allows for a higher order general (*g*) factor (e.g., Mackintosh, 1998). In hierarchical factor models, observed variables are correlated with *g* only via their correlations with first-order group factors that are nested under *g* (e.g., Carroll, 1993). Fortunately, it is not difficult to generate such correlation structures with the mutualism model. The most debatable assumption in Scenario 3 is that all **M** are set equal to the same arbitrary small positive value. Certainly, the elements M_{ij} are not necessarily equal. **M** may also be sparse and may include negative values. For instance, some processes might compete for the same resources, and some processes in language acquisition might even require certain constraints (Newport, 1990). It appears that more complex **M** lead to more complicated factor structures akin to factor structures found for real data.

To investigate what happens when the M_{ij} are not all equal and even partly negative, we sampled the M_{ij} from a normal distribution with a mean of .05 and standard deviations varying from .02 to .06. Note that for larger standard deviations, the probability of negative M_{ij} is quite high (e.g., given a standard deviation of .04, the probability of negative M_{ij} is .10; given a standard deviation of .06, this probability is .20). In Figure 6 we show histograms of the correlations between the variables *x* for increasing standard deviations of the M_{ij}. The mean correlation stays roughly the same, but the variance increases. The number of factors required to describe the data also increases. The correlation matrices obtained from the factor scores of these factors again show a positive manifold, which can be described with one or more correlated factors, that is, a typical hierarchical factor structure.

A more controlled way to obtain a hierarchical structure is by specifying a matrix **M** in which the M_{ij} are high (strong interaction = .08) between variables associated with the same group factor and low (weak interaction = .02) between variables of different group factors (see Table 1). To demonstrate this, we simulated data for four group factors, each associated with four variables x_i. Figure 7 displays the correlations, the eigenvalues, the *p* values of the factor models, and the correlations of each factor of a four-factor model with the variables x_i. Again the correlations suggest a general *g* factor as in Scenario 3. However, the one-, two- and three-factor models are rejected ($p < .05$). In contrast, the four-factor model fits the data well ($p > .05$). The correlations of the four factors with the observed variables clearly display the expected pattern.

Further analysis shows that these four factors are intercorrelated following Promax rotation (Lawley & Maxwell, 1971). The single common factor model fits the covariance matrix of the four common factors well. A full hierarchical confirmatory factor model with four group factors and one general factor also fits the data well; $\chi^2(100, N = 1{,}000) = 93.2$, $p = .67$. Indeed, under certain choices of **M**, the covariance structures associated with the hierarchical factor model and the mutualism model are technically equivalent (see Footnote 10). By weakening the weak interactions in the matrix **M** (leading to a block diagonal matrix structure), the dependency between the four group factors can be reduced. In such a case, the model resembles a multiple intelligence model with independent factors (cf. Gardner, 1983; Horn & Masunaga, 2000) in which psychometric *g* does not exist.

In addition, Cattell's (1971) distinction between fluid intelligence (*gf*) and crystallized intelligence (*gc*) can be accommodated in the mutualism model. Crystallized intelligence is thought to develop by the interaction of fluid intelligence and cultural experience (e.g., Hunt, 1997). Consistent with this, measures of *gc* tend to improve in adulthood, whereas measures of *gf* reach their highest levels in early adulthood (McArdle, Hamagami, Meredith, & Bradway, 2000). Whether the *gc–gf* theory rejects the *g* theory (Carroll, 1997; Hunt, 1997) is not directly relevant here. It is well known that *gf* and *g* correlate highly (Jensen, 1998) and, more importantly, in the present context, *gf* and *gc* are both *g*-like factors, that is, higher order common factors that explain correlations between sets of observed variables. The possible circumstances in which such common factors may arise in the mutualism

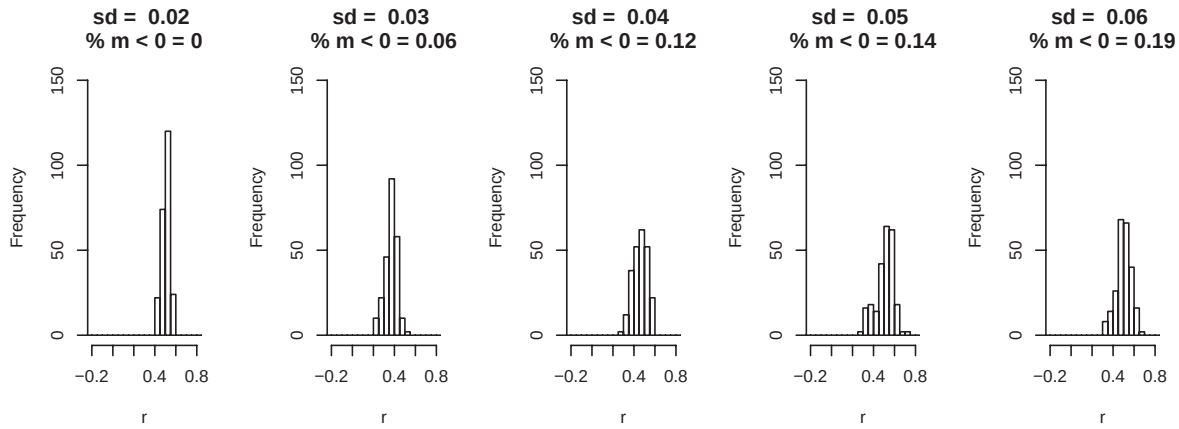

Figure 6. Histograms of the correlations between the processes **x** as a function of the standard deviation of the connection weights in **M**. The mean of **M** is fixed at .05. The percentage of negative weights increases with increasing standard deviation. However, the positive manifold remains present.

Table 1
M Matrix for Hierarchical Factor Data

.00	*.08*	*.08*	*.08*	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
.08	*.00*	*.08*	*.08*	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
.08	*.08*	*.00*	*.08*	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
.08	*.08*	*.08*	*.00*	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
.02	.02	.02	.02	*.00*	*.08*	*.08*	*.08*	.02	.02	.02	.02	.02	.02	.02	.02
.02	.02	.02	.02	*.08*	*.00*	*.08*	*.08*	.02	.02	.02	.02	.02	.02	.02	.02
.02	.02	.02	.02	*.08*	*.08*	*.00*	*.08*	.02	.02	.02	.02	.02	.02	.02	.02
.02	.02	.02	.02	*.08*	*.08*	*.08*	*.00*	.02	.02	.02	.02	.02	.02	.02	.02
.02	.02	.02	.02	.02	.02	.02	.02	*.00*	*.08*	*.08*	*.08*	.02	.02	.02	.02
.02	.02	.02	.02	.02	.02	.02	.02	*.08*	*.00*	*.08*	*.08*	.02	.02	.02	.02
.02	.02	.02	.02	.02	.02	.02	.02	*.08*	*.08*	*.00*	*.08*	.02	.02	.02	.02
.02	.02	.02	.02	.02	.02	.02	.02	*.08*	*.08*	*.08*	*.00*	.02	.02	.02	.02
.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	*.00*	*.08*	*.08*	*.08*
.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	*.08*	*.00*	*.08*	*.08*
.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	*.08*	*.08*	*.00*	*.08*
.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	*.08*	*.08*	*.08*	*.00*

Note. The group factors are associated with the blocks of higher *M* values (italic).

model have been outlined above. However, it is important to specify the manner in which the mutualism model can account for the asymmetry in *gf* and *gc*, as specified in Cattell's investment hypothesis (Cattell, 1971).

There are three independent ways to model this hypothesis. First, we can model the asymmetry in the relation between *gf* and *gc* by specifying asymmetries between the processes associated with *gf* and *gc* in **M**. We could assume that *gf* processes strongly influence *gc* processes, whereas *gc* has no influence on *gf*. When x_i associated with *gc* also compete with each other (negative M_{ij}), the dynamics of the model resemble biological models with competition, parasites, prey, and predators. This gives rise to more complex and more interesting dynamical behavior. However, as long as the majority of interactions are positive, competition by no means rules out the positive manifold. In the section on differentiation, we discuss a simple way to implement such a mechanism. Second, the asymmetry may be modeled via **K**. In the model of van Geert (1991, 1994) the carrying capacity (K_i) of one process is made dependent on the growth (**x**) of other processes (so-called bootstrap dynamics). In such a model, the dependent process will not start to grow before the basic process has reached a certain level. Third, we could simply assume that the growth speeds (**a**) of the processes associated with crystallized intelligence are (much) lower than the growth speeds of fluid processes.

Preliminary simulations have shown that all three mechanisms may explain the segregation in development of fluid and crystallized intelligence. Further work should be directed toward a theoretical analysis to determine which of these three mechanisms best represents the investment hypothesis.

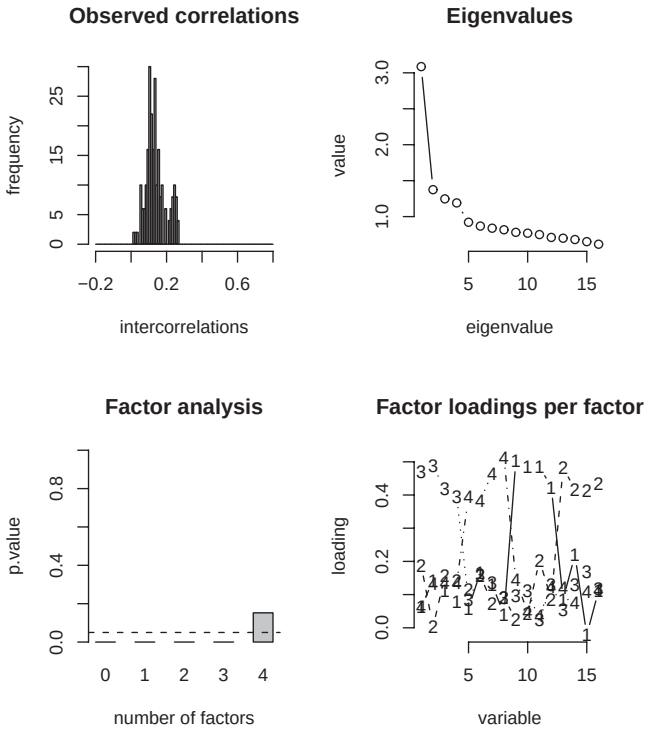

Figure 7. Resources (and other parameters) are uncorrelated, the M_{ij} resemble a hierarchical structure with four group factors and a general factor. The zero-, one-, two-, and three-factor models are rejected ($p < .05$). The four-factor model is accepted. The loadings of the four factors show the expected pattern.

The Development of Intelligence

The *g* theories are rather unspecific concerning development. For instance, the subject index of the book of Jensen (1998) does not contain references to age or development. Ackerman and Lohman (2003) even state, "As near as we can tell, *g* theories have failed to provide any account of development across the lifespan" (p. 278). Yet a number of developmental effects are often discussed, and in the present and the next section we investigate how well the mutualism model accounts for these effects.

One important effect is the low predictive validity of test performance during infancy and early childhood. In a classic longitudinal study, Bayley (1949) found essentially no correlation between test performance in the first 3 years and test performance at 18 years. This correlation rises during childhood to attain a quite high level between 11 and 18 years. This finding has been replicated many times. There may well be a low correlation between infant measures and later intelligence, but these correlations are

certainly much lower than those observed later in development (Honzik, 1983; McCall & Carriger, 1993; Slater, 1997). In the mutualism model, performance in the initial phase of development is determined by the starting values (**x0**) and the growth parameters (**a**). The stable asymptotic states of the x_i (e.g., adulthood performance), however, do not depend on the growth parameters and the initial values (see equation for the stable states in the appendix). This implies a low or even zero correlation between initial and later performance.

But what about the much higher correlations between test scores later in development (e.g., late childhood) and test performance at 18 years? These correlations are substantial (i.e., in the range of .40 to .80) and imply that growth speed and asymptotic values are correlated. That is, high-intelligent subjects develop faster than low-intelligent subjects. At first sight, this seems to contradict one of our model assumptions. In all simulations and analyses we assumed the growth parameters (**a**) and limited resources (**K**) to be uncorrelated. Also the asymptotic stable states have been shown to be independent of **a**. However, the actual growth speed does not only depend on **a** but also on **K** and **M**. Equation 3 specifies the change in **x** in time. This change depends on **a**, **K**, and **M**. In general, higher **K** and higher **M** will increase the growth speed, causing a correlation between actual growth speed and asymptotic performance. Figures 2b and 2c demonstrate this. Figure 2b shows the growth of the mean of **x** (as an index of general intelligence) of 20 subjects. Inspection of the developmental curves suggests an increase in stability of intelligence. Figure 2c supports this. The correlation between test performance and asymptotic performance increases over time. Note that this is a property of the logistic equation. If we use Scenario 2 (*g* scenario), we find the same increase in correlation.

In the next section, we discuss a more complicated developmental effect, the change in the positive manifold or the strength of psychometric *g* during development.

Differentiation Effects

There is some evidence that the positive manifold is not uniform in the population. Two possibly related differentiation effects have been much researched and debated (Deary et al., 1996). The age differentiation effect concerns a decline of *g* with age (e.g., Tideman & Gustafsson, 2004). The ability differentiation effect refers to stronger *g* in low-IQ groups (e.g., Facon, 2004; Jensen, 2003).

Neither effect has been replicated consistently (for reviews, see Carroll, 1993; Facon, 2004; Hartmann & Teasdale, 2004; Reinert, 1970). Especially for age differentiation, the results are very mixed. Reinert (1970) reviewed more than 50 studies. Some of these studies support differentiation, some support integration, some support a pattern of integration followed by differentiation, and many studies did not find any pattern of integration or differentiation. Carroll (1993) reanalyzed 12 data sets and concluded that in "nearly all instances the same number of factors was extracted at each age" (p. 679) and "age-differentiation is a phenomenon whose existence is hard to demonstrate" (p. 681). Newer studies are also inconclusive (Bickley, Keith, & Wolfle, 1995; Juan-Espinosa, Garcia, Colom, & Abad, 2000; Rietveld, Dolan, van Baal, & Boomsma, 2003; Rose, Feldman, & Jankowski, 2004, 2005; Tideman & Gustafsson, 2004).

From a theoretical point of view, a strong *g* in infancy is implausible. Given most conceptualizations of intelligence (e.g., Bartholomew, 2004), and also most theories of psychometric *g*, a newborn cannot be viewed as intelligent. Such conceptualizations refer to understanding, problem solving, goal-directed adaptive behavior, rational thinking, and cognitive ability. If we consider the subtests of the dominant IQ tests, such as the Wechsler Adult Intelligence Scale, it is clear that most of these subtests do not have equivalent or analogous variants that are applicable to infants. Thus, intelligence, which standard psychometric intelligence tests purport to measure, takes some time to develop in children. Consequently, we expect that it takes some time for the positive manifold, and thus the psychometric *g* factor, to emerge. In the empirical literature there is some support for this initial phase of integration (see Reichard, 1944; Reinert, 1970; Rose, Feldman, & Jankowski, 2005). For instance, Rose, Feldman, and Jankowski (2005) tested infants of 7 and 12 months old, and find correlations between subtests that are much lower than those usually found for older children. In the next section, we discuss the increase of heritability of *g* with age. This also suggests that the strength of *g* increases in the first phase of development.[12]

It is important to realize that current models of psychometric *g* do not offer straightforward explanations of differentiation. Standard *g* theories are not specific on the development of psychometric *g*. They seem to imply an increase in the strength of *g* over time (i.e., integration instead of differentiation). This, at least, is what happens in the simulation of Scenario 2 (see below). Alternatively, it could be argued that *g* (as a genetic biological variable) is fixed over development, but then the emergence of psychometric *g* still requires explanation, as does differentiation (for further discussion, see Ackerman & Lohman, 2003). Spearman (1927) invoked the law of diminishing returns to explain differentiation. However, as Detterman (1991) clarified, this law does not explain differentiation. Newer theories refer to the idea of investment (Cattell, 1971; Hunt, 2005) and to the idea of *g* as constraint (M. Anderson, 1992, 2001; Detterman, 1987). These theories explain how more specialized abilities emerge within individuals and how these abilities are related to more basic processes. These ideas are interesting, but they are not formulated in such detail that we can derive clear predictions of age differentiation in the population. More importantly, these theories probably also predict an initial phase of integration (the emergence of *g*) and invoke additional mechanisms to explain differentiation.[13]

[12] Why are the data of a century of research so inconclusive? In our opinion, methodological problems provide at least part of the answer. Testing for integration or differentiation is difficult. The main problems concern selection effects (in both cross-sectional and longitudinal studies; see Rabbitt, Diggle, Holland, & McInnes, 2004), the composition of test batteries (which changes with age), validity problems (especially in infancy and early childhood), measurement bias (such as that caused by ceiling effects), and the choice between several measures of differentiation. We have to be careful not to overinterpret the data (Carlstedt, 2001; Facon, 2004; Fogarty & Stankov, 1995; Reinert, 1970).

[13] There is probably only one theory that gives a straightforward explanation of differentiation, and that is the sampling theory. According to Thurstone (1938) differentiation may take place because younger children use a smaller set of basic processes for different cognitive tasks than older children, who also have at their disposal specialized processes. This is a viable idea, but it is not in conflict with our proposal, as it represents an explanation of differentiation in terms of a measurement problem. We made the simplistic assumption that we can measure each process with one task. If we relax this assumption, and choose a more complex measurement model, we can incorporate this explanation.

The mutualism model allows for both integration and differentiation. We first examine the change in the strength of psychometric *g* in Scenarios 2 and 3. For different time points we compute the first eigenvalue as an indication of psychometric *g*. The first eigenvalue corresponds to the average correlation between the cognitive processes and is therefore a simple straightforward measure of psychometric *g*.[14] As a measure of intelligence, we simply take the mean **x** score of subjects. The first plot in Figure 8 shows that Scenario 2 (the *g* model) only produces integration. The first eigenvalue only increases, following the developmental trend of the mean **x**. The other lines show the standardized regression (beta) coefficients for the subjects, specific random **a**, **K**, and **x0**. They indicate the amount of variance in the **x** that can be explained with the variance in **a**, **K**, and **x0**. At first, the growth parameters and the initial values explain most of the variance in **x**, but after some time, **K** takes over. In this scenario the positive manifold is caused by the correlated **K** values. Therefore the beta coefficient associated with **K** and the first eigenvalue co-vary.

The second plot corresponds to Scenario 3 (mutualism). As in earlier simulations, all M_{ij} are set to .05, and the a_i, K_i, and $x0_i$ are sampled from uncorrelated normal distributions. In this scenario the first eigenvalue first increases and then decreases (integration followed by differentiation). This is interesting, as no additional mechanism was invoked to create differentiation. Inspection of the beta coefficients associated with **a**, **K,** and **x0**, reveal that the peak in the curve of the first eigenvalue can be explained by the fact that the influence of **a** declines before the influence of **K** increases. Further simulations show that this is a robust effect that occurs given a range of parameter settings.[15] Simulations also show that the peak occurs later in the low-intelligent group than in the high-intelligent group. This gives rise to a complicated pattern of ability differentiation.

One may question the time scale of this integration/differentiation pattern. In the simulation, the largest part of development is characterized by integration. As we discussed above, there is little clear evidence for age differentiation, but none for a long initial period of integration either. In view of the methodological problems mentioned above, the safest conclusion is that an initial relatively short period of integration is followed by a stable *g*. This pattern can be achieved in the mutualism model by increasing the level of mutualism. The third plot demonstrates this. All M_{ij} are set equal to .065 instead of .05, which lead to a very short period of integration followed by a stable *g* factor. Note that the integration process is already finished before mean **x** starts to rise.

These simulations show that the mutualism model gives rise to rather complex patterns of integration/differentiation without invoking any additional mechanism. Nevertheless, we did consider possibilities to incorporate other mechanisms as put forward in the investment and the constraint models of differentiation. The basic idea in these models is that during development more and more specialized cognitive processes (related to *gc*) emerge. A simple implementation of this idea is to let the standard deviation of the M_{ij} increase with time while the mean of the M_{ij} remains constant. Thus, during development certain mutualistic interactions become stronger, whereas other interactions decrease in strength or may even turn into competitive relations. This means that some mutualistic interactions are optimized (e.g., the interaction between strategy selection and memory use), whereas other abilities/processes compete for the same resources (such as time). From Figure 6 we already learned that the increase in the standard deviation of **M** indeed leads to a more complicated factor structure. The last plot of Figure 8 again demonstrates this. It shows the growth of the cognitive processes of one individual, the change in the first eigenvalue in the population, and the change in the number of factors required to explain the data. As expected, with time, more and more factors are required to describe the data. Note that the first eigenvalue and the number of factors do not necessarily give the same impression of integration/differentiation. Interestingly, the growth curves of the processes x_i show a diverging pattern in which some processes start to decline as soon as the main developmental period is finished. Evidence for such declines in cognitive functioning in adulthood are reviewed in McArdle, Ferrer-Caja, Hamagami, and Woodcock (2002).

In summary, the present simulations show that complex interrelated integration/differentiation effects (as indicated by the first eigenvalue) arise without any changes to the model. The timing of these effects can be altered with changes in parameter values, especially the strength of mutualism. Differentiation in terms of an increase in the number of factors can be achieved by assuming that the mutualism is first rather uniform and later evolves toward a more complex structure. The constraint and investment models of differentiation are somewhat more sophisticated than this simple implementation. However, in view of the lack of a clearly established empirical pattern, we do not consider further complications of the model.

Heritability of Intelligence

In his account of the *g* factor, Jensen (1998) discussed the heritability of *g*. Twin studies have indicated that the heritability of cognitive tests, IQ, and *g* vary between .5 and .8 (Jensen, 1998; Plomin & Spinath, 2004). Both Plomin (2001) and Jensen (1998) argued that the main factor in the heritability of intelligence is *g*. One argument of Jensen is particularly intriguing. For a number of data sets, he computed the correlations between the vector of heritabilities and the vector of *g* loadings of cognitive tests (i.e., the method of correlated vectors). These correlations are larger than .5, which is taken to suggest a high influence of the genetic component of *g*. This result is sometimes called the Jensen effect.[16]

Can we explain this result with the mutualism model? Eaves et al. (1999) and Molenaar and Raijmakers (1999) have shown that the use of the logistic model for the simulation of phenotypic differences between genetically equal twins may lead to nonintuitive results. First, note that other results obtained with the method of correlated vectors (for instance, concerning speed of processing)

[14] Alternatively, we could test for the number of factors required to describe the data. However, we already know (from Figures 4 and 5) that Scenario 2 and Scenario 3 (with all M_{ij} equal) always require only one factor.

[15] Rietveld, Dolan, van Baal, and Boomsma (2003) and Thompson, Detterman, and Plomin (1993), suggested that shared family environment early in development and at lower IQ levels might cause differentiation effects. We tested this by using correlated growth parameters. This indeed leads to a strengthening of the integration/differentiation pattern shown in second plot of Figure 9.

[16] The method of correlated vectors has been criticized by Dolan (2000) and Lubke, Dolan, and Kelderman (2001), yet multivariate genetic analyses (e.g., Plomin & Spinath, 2004) seem to support Jensen's results (see also Luo, Thompson, & Detterman, 2003).

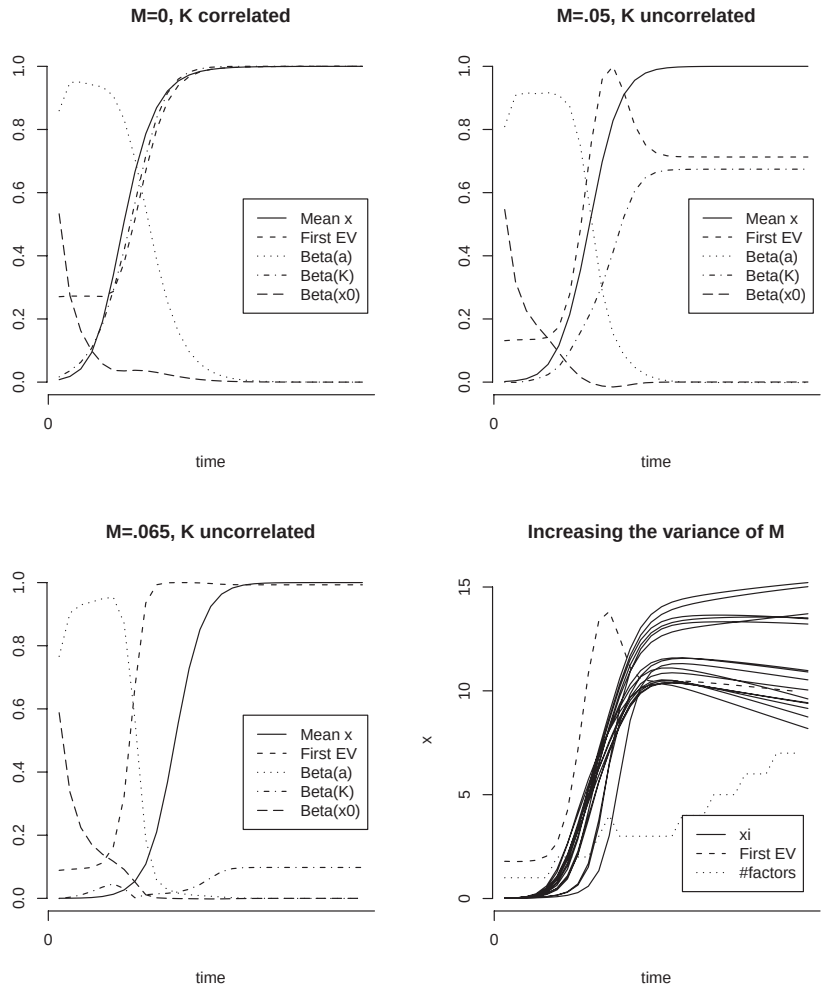

Figure 8. The top panels show the development of *g* (first eigenvalue [EV]) in Scenarios 2 and 3. The plots also show the mean trend of **x**, the standardized regression coefficients of the regression of **x** on **a**, **K**, and **x0**. The latter indicate how much variance in **x** can be explained by the subject-dependent parameters. The bottom left plot depicts the change in the first eigenvalue for a higher value of **M**. As shown by the first eigenvalue, integration is limited to the very early phase of development. The first eigenvalue reaches its maximum before the mean of **x** starts to rise. The last plot illustrates what happens when the variance of the mutualistic interactions is increased during development. The number of factors required to describe the data increases, and some processes decline later in development.

are explained easily with the mutualism model (see the discussion of Figure 10, presented below). For each variable in the mutualism model, the correlation with the other variables will co-vary with the loadings of the first factor of these variables. In fact, this is a property of positive manifold data, whether generated by the mutualism model or by the *g* model. Second, high heritability for cognitive tests and *g* can be achieved readily. Genetic and environmental influences can be introduced in the model via **K**. A simple additive model is $K_i = c_i\, G_i + (1 - c_i)\, E_i$, where **G** and **E** are the genetic and environmental factors with weights **c** and 1− **c**, respectively. Assuming that all G_i and all E_i are uncorrelated and sampled from distributions with the same means and standard deviations, heritability h^2 equals c^2. Thus, different uncorrelated genetic influences or constraints on **K** may cause a high heritability of *g*. Notably, this explanation is consistent with the finding in quantitative trait locus (QTL) research that there are no single-gene influences that strongly correlate with IQ or *g* (see, Plomin & Spinath, 2004). Furthermore, this setup of the model also explains the increase in heritability during development (Bartels, Rietveld, van Baal, & Boomsma, 2002; Fulker, DeFries, & Plomin, 1988). Initially the values of the variables are determined by the random initial values **x0** and growth parameters **a**. The influence of the genetic part of **K** is very low in this phase. Only later in development, when variables reach their asymptotic values determined by **K**, the genetic part of **K** comes into play (see Figure 8).

However, this does not explain the positive correlation between the heritability and *g* loading of the cognitive tests (the Jensen effect). It appears that the mechanism of mutualism only leads to such a correlation if we introduce small positive correlations between the genetic resources G_i, on the basis of evidence for heritability of *g*. However, introducing correlations between model parameters undermines the idea of the mutualism model (see

Scenario 2). Yet, simulation of the mutualism model shows that the Jensen effect only requires very weak genetic intercorrelations between the G_i. We used the equation for the covariance matrix (see Appendix) to simulate data with normally distributed M_{ij} (mean = .05), normally distributed h_i^2 (mean = .5, sd = .02), and uniform genetic intercorrelations (sd = 0). The environmental intercorrelations were fixed at zero. We varied the levels of genetic intercorrelation and the standard deviations of the M_{ij} to investigate the robustness of the results. Figure 9 shows the correlation between the heritability and *g* loading of the cognitive tests for different values of the genetic intercorrelations of the genetic resources G_i (*x* axis), and different values of the standard deviation of the values in **M**. For most values, the vector correlations are clearly positive, which is consistent with the Jensen effect.[17]

In sum, the mutualism model explains most results obtained with the method of correlated vectors. Also the increase in heritability during development can be explained by assuming that uncorrelated limited resources are (partly) genetically based, whereas the growth parameters are not, or are genetically based to a lesser degree. Explaining the Jensen effect requires an additional assumption concerning very small, but nonzero, genetic intercorrelations of the resources K_i.

Flynn Effect and the IQ Paradox

Another intriguing phenomenon in intelligence is the so-called Flynn effect (Flynn, 1984). This effect refers to the massive IQ gains over time, in the order of one standard deviation per generation. Explanations of this effect include nutrition, family size, education, environmental complexity, and test-taking strategies (Wicherts et al., 2004). Dickens and Flynn (2001) discuss the Flynn effect in relation to the high heritability and the low contribution of environmental factors to *g* and pose the so-called IQ paradox: How can high heritability and presumed large environmental influence co-exist?

Dickens and Flynn (2001) present a model based on strong reciprocal causation between phenotypic IQ and environment leading to a gene–environment correlation that masks the potency of the environment. The idea is that people shape or select their environment depending on their IQ. Higher IQ leads one into better environments, causing still higher IQ, and so forth. For instance, in an adoption study of Braungart, Fulker, and Plomin (1992), the heritability of Home Observation for Measurement of the Environment (HOME) scores (an environmental factor) was estimated at 40%. Whereas a number of authors have proposed this idea of reciprocal causation (e.g., Ceci, 1990; Jensen, 1998; Scarr & McCartney, 1983), Dickens and Flynn (2001) used a formal model to show that this idea may explain the IQ paradox.

The mutualism model is consistent with the solution proposed by Dickens and Flynn. The current mutualism model only incorporates mutualistic (e.g., reciprocal causal) relations between cognitive processes, but there is no reason to limit the model in this way. We already mentioned a reciprocal relation between intellectual performance and motivation, and other beneficial relations between cognitive and noncognitive processes are possible. A natural extension is to incorporate reciprocal causal relations between cognitive and environmental processes. This provides an explanation of differential generation effects. The Flynn effect is not uniform over IQ subtests (Wicherts et al., 2004), and it seems that the positive manifold changes over time. By specifying the precise relations between environmental changes and cognitive processes, it may be possible to explain these differential Flynn effects in the mutualism model.

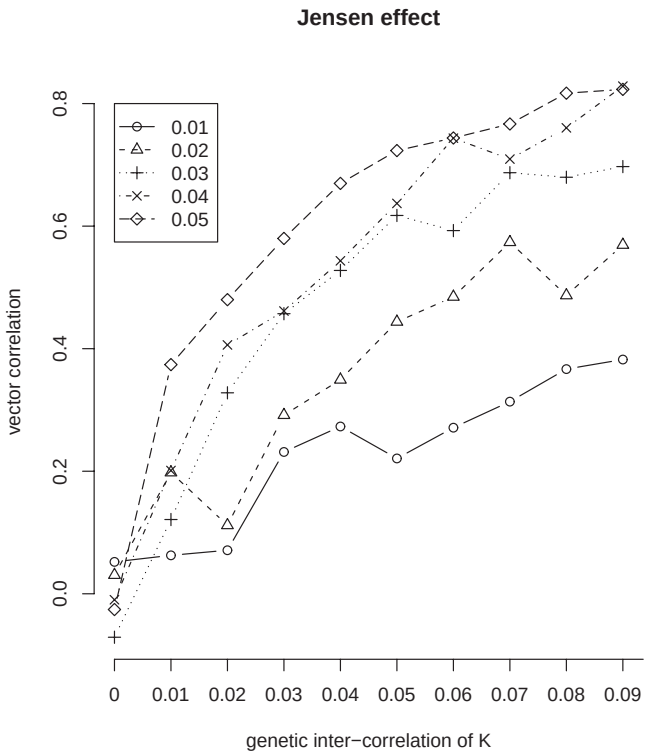

Figure 9. The correlation between heritability and *g* loadings of the processes x_i as a function of the genetic correlations between the genetic components of K_i for different values of the standard deviation of the M_{ij} values (lines). A very weak genetic correlation suffices to explain the Jensen effect.

The Interpretation of High g Loadings

An important implication of the mutualism model is that high *g* loadings of variables or (first-order) factors in a factor analysis can mean two very different things: the processes associated with these variables may either influence or be influenced by many other processes. Suppose the **M** matrix has the following form: .005, .005, ...005 in the first row, .01, .01, ...01 in the second row, with this pattern continueing to .08, .08, ...08 in the last row. So the last process (row) has a strong positive influence on all other processes, and we may expect a high *g* loading. Now, we take the transpose of this matrix **M**. In this case, the last process (column: .08, .08, ...08) is strongly influenced by all other processes, and again we may expect a high loading. Figure 10 shows the *g* loadings for the processes for both cases. The *g* loading of the 16th process is indeed highest for both cases (note that the slope of the *g* loadings is less steep in the latter case). Thus, high *g* loadings, or

[17] We also tested an alternative scenario. It is possible to make **M** different between individuals, with a genetic part equal for twins. Under certain conditions, using **M** as specified in the section on the interpretation of high *g* loadings (see below), the Jensen effect can be simulated.

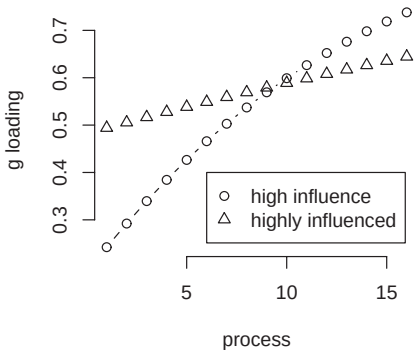

Figure 10. The *g* loadings for two different influence situations. In the first, the 16th process strongly influences all other processes, and in the second the 16th process is strongly influenced by all other processes. In both cases, the 16th process shows the highest correlation with *g*.

high correlations with *g*, are not necessarily informative about the role of processes in intelligence or the nature or origin of *g* (Detterman, 2002).

A related phenomenon is that manipulation of a single variable in complex systems is often ineffectual. Training of speed of information processing or working memory, in spite of their high correlation with *g*, may not lead to increases in general intelligence. In more complex dynamical systems interventions can even have counterintuitive effects. The lake example mentioned in the introduction is again useful. To improve the water quality (visibility) of lakes, biologists sometimes simply remove most fish from lakes (Scheffer, 1998). In some cases, this is more effective than the reduction of fertilizers, that is, the most probable cause of low water quality (Meijer, de Boois, Scheffer, Portielje, & Hosper, 1999). Similarly, sudden shifts may occur in complex foodwebs. Small changes in variables may lead to catastrophic changes (van Nes & Scheffer, 2004). van der Maas and Molenaar (1992) argue that such changes also occur in cognitive development.

Discussion

The positive manifold is arguably both the best established and the most striking phenomenon in the psychological study of intelligence. Over the past 100 years differential psychologists have sought to explain this phenomenon by invoking an underlying general intelligence factor. At present, the factor analytic (statistical) support for a general factor is considered strong (Bartholomew, 2004; Carroll, 1993; Jensen, 1998). However, the nature of the *g* factor remains unclear (Deary, 2002). An assumption that is often made is that the *g* factor represents an underlying quantitative variable. Indeed, many attempts have been made to actually identify this factor with measurable variables (e.g., speed of nerve conductance, reaction time, glucose metabolism in the brain). These studies have produced interesting correlations but have not revealed the single underlying cause of the *g* factor (Ackerman, Beier, & Boyle, 2005; Luciano et al., 2005).

The aim of the present article was to offer a new explanation for the positive manifold that does not include a *g* factor as an underlying quantitative dimension. In this explanation, we assume that in the initial phase of development, cognitive processes are uncorrelated. During development, the positive manifold emerges as a consequence of mutually beneficial interactions between these processes. Factor analysis of data generated by this dynamical process suggests the presence of a dominant factor, as demonstrated in the simulations. Interestingly, under certain circumstances, the mutualism model and the factor models are statistically equivalent, in the sense that they produce the same covariance structure (see Footnote 10). However, the mutualism or cooperation between processes is conceptually very different from the *g* explanation, in terms of a single quantitative dimension. Also, the mutualism model not only explains the positive manifold, it sheds light on various other phenomena, such as developmental, genetic, and cohort effects. These effects are amenable to explanation within the mutualism model because this model is essentially a developmental model, that is, it is concerned with the outcome of a dynamic process.

First, the model provides a plausible explanation of hierarchical factor structures. Variability in the interaction weights in **M**, provided the average of **M** is positive, leads to complex positive manifolds, as observed in real data. Second, the model explains a number of developmental effects. The low correlation between infant test performance and adulthood IQ can be explained by the fact that the asymptotic states are independent of the growth parameters and the initial values, which together determine the model's behavior in the initial phase of development. The correlation between test performance and adulthood IQ increases quickly because the limited resources and mutualism influence both the growth speed and the asymptotic states. Third, the mutualism model allows for interrelated integration/differentiation effects. In contrast to other models, differentiation can occur in the model without invoking any additional mechanism. However, when we assume an increase in the variance of mutualistic interactions during development, differentiation and the decline of a limited set of cognitive processes in adulthood can be explained. Fourth, results obtained with the method of correlated vectors do not pose a problem. They can be explained without further assumptions. Fifth, the increase in heritability of intelligence follows from the mutualism model if we are willing to assume that genetic effects are (primarily) on the limited resources **K**. Sixth, provided the genetic contributions to individual differences in **K** are minimally correlated (i.e., correlations in the order of .01 to .09; see Figure 9), we can explain the Jensen effect, that is, the correlation between factor loadings and heritabilities of subtests. This assumption of low correlations between genetic contributions to individual differences in **K** is in accordance with the low correlation between singles genes and *g* in QTL research (Plomin & Spinath, 2004). Finally, the model may be extended, with reciprocal causal relations between phenotypic intelligence and environmental factors leading to a gene–environment correlation that masks the potency of the environment. According to Dickens and Flynn (2001), this accounts for the coexistence of a high heritability of psychometric *g* and a large environmental (Flynn) effect.

It is important to note that the mutualism model is consistent with many other models and theories of psychological development. It is a nonlinear dynamical model, and, as such, related to much recent work in developmental psychology (Thelen & Bates, 2003; van der Maas & Molenaar, 1992). Especially relevant are the applications of van Geert (1991, 1994) in developmental psychology and the model of Dickens and Flynn (2001).

The positive manifold, *g*, and general intelligence are often viewed as synonymous. We have shown that positive manifold does not necessarily imply a single quantitative latent factor, but

what about in the case of general intelligence? Of course, this depends on one's definition of general intelligence. If we equate general intelligence with *g*, then the mutualism model does not support general intelligence. We prefer to associate general intelligence with the positive manifold, so that we may view the mutualism model as a model of general intelligence in the sense that it explains why people, who are good at one test, are good at other tests as well. Given this interpretation of general intelligence, there is nothing wrong with using the *g* factor as a summary or psychometric index variable (e.g., in prediction), as long as we do not assume that this variable relates to a single underlying quantitative process or capacity. In this view, the *g* factor is *not* advanced as an explanatory variable.

Several possible points of criticism of the present proposal may be raised. First, we may ask whether the mutualism model is really different from the sampling theory. In sampling theory, the elementary processes are uncorrelated. Correlations between performance on cognitive tasks exist because these tasks appeal to overlapping subsets of these elementary processes. In sampling theory, and this is true for all its variants, the positive manifold is due to a measurement problem. If we were able to measure elementary processes independently, the positive manifold would disappear. In the mutualism model, the elementary processes are uncorrelated initially but become correlated during development. A more refined measurement will not make the positive manifold disappear.

The objections raised to the sampling theory (see introduction) are also less relevant to the mutualism model, because functional independence does not imply a developmental independence. Performance on simple reaction time tasks and performance on intelligence tests, such as the Raven's Progressive Matrices test, may not have much in common. They may be functionally independent. Yet, in the *development* of reasoning processes that are important in the Raven test performance, speed of processing could well have been very important. Another example is the relation between short-term memory and many cognitive skills. In the first phase of skill acquisition, short term memory is essential, but later, when processes are automatized, short-term memory is no longer involved in performance (J. R. Anderson, 1982). Also brain damage might selectively impair performance on one type of test without impairing other, highly correlated, performances in the population, because the correlation is not based on current functional dependency or overlap in processes but rather on developmental dependency. Moreover, correlation between processes can be based on many M_{ij} through indirect pathways.

Another possible objection is that the mutualism model does not constitute a parsimonious explanation of the data. Indeed, if every element M_{ij} has to be determined from data, there is no reduction of information. We offer three responses to this. First, we do not assume the presence of a rather mysterious latent variable, such as *g*. Rather, we use a developmental model with a clear mechanism to account for the positive manifold. Second, we can constrain the structure of **M**, as in Scenario 3. Indeed, **M** can be quite sparse and still give rise to a positive manifold. Many kinds of restrictions may be placed on the elements in the matrix **M**, which can be based on theoretical considerations or the results of experimental studies. If, for instance, experimental manipulation of short-term memory is found to alter the performance on an arithmetic task, we would have reason to constrain the relevant M_{ij} to a positive value. Finally, irrespective of these considerations, if we believe that beneficial relations between important cognitive or brain processes exist, we have to take the mutualism explanation of the positive manifold seriously.

A general objection to all three explanations of the positive manifold concerns falsification (Jensen, 1998). It is not easy to falsify explanations of the positive manifold. It is also quite possible that a full explanation of intelligence data requires elements of all three explanations. Some cognitive processes are probably much more important than others in the explanation of individual differences, reciprocal causation plays a role and the measurement problems in sampling theory can hardly be excluded. The best way to falsify the mutualism model is to find a variable that correlates perfectly with *g*. Furthermore, the present model incorporates various assumptions, which can be challenged in different ways. For instance, the model predicts that the initial values and growth parameters do not determine the asymptotic stable states. Demonstration of a correlation between early infant performance and adulthood intelligence or a demonstration of a strong *g* factor in infancy would pose a problem for the mutualism model.

A number of issues require further study. For instance, we hope to (a) extend the dynamical model using results of population biology, (b) establish statistical model equivalence between the mutualism model and factor analytic models, (c) make an inventory of causal links between relevant cognitive (and noncognitive) processes based on experimental evidence, (d) formulate the mutualism model in terms of a cognitive architecture with a choice of processes and causal links, and (e) obtain sufficient information to actually fit the mutualism model to data.

This research program appears to have good prospects. In the past few years, the mutualism model and its various extensions have been studied extensively in population biology (see Footnote 6). Additionally, model equivalence has been investigated in quite some depth (e.g., Molenaar, 2003; Raykov & Penev, 1999). Objective c requires the review of the relevant literature and may necessitate new experimental cognitive research. Understanding the function of many biological complex systems, such as genetic networks and molecular signaling pathways, requires precise identification of the interaction between individual components. The most common strategy to identify the causal links involves piecemeal perturbations of the elements involved (Krupa, 2002). Translating this to psychology, we need (preferably) experimental data about the precise relations between modules, processes, or capacities of the mind. The last two issues require specification of the processes **x** in our model. We consider both a data-driven and a theory-driven strategy for this specification. In the data-driven strategy, we can work with one of the many available data sets from intelligence research and use subtest scores as measures of the processes **x**. In the theory-driven approach, we can adopt (or develop) a cognitive model of intelligence, such as the Sternberg model (Figure 1c) or a *gf–gc* type model. Because we have at our disposal the expected covariance matrix for the basic mutualism model (see Appendix), fitting the model to data is possible in principle. However, such an undertaking would require the specification of constraints on the matrix **M** to reduce the number of parameters and render the model falsifiable. One possibility is to constrain all elements of **M** to be equal as in Scenario 3, but this may not be very plausible. The matrix displayed in Table 1 resembles hierarchical factor models, but there are many other possibilities. The most promising option is to analyze longitudinal

data with dynamic structural equation models (McArdle et al., 2000). Further technical developments are required in this area.

To summarize, in the present article, we proposed a novel explanation of the positive manifold. Positive interactions, that is, reciprocal causation, in the development of cognitive processes, gives rise to the same important phenomenon, viz. the positive manifold, that has been advanced as evidence for a dominant underlying latent variable in intelligence. A developmental model with such interactions may also explain other important effects in intelligence research or, at minimum, provide new ways to study them.

A final question is whether it is possible to apply this model, or at least this line of reasoning, to other areas in psychology, where correlational data play an important role, and the identification of latent variables is based strongly on the results of factor analyses. It would certainly be a useful exercise to conceptualize broad personality dimensions (e.g., the Big Five) as manifestations of the interaction between processes within a broadly defined psychological system. The same applies to more narrowly defined personality variables. For instance, should we view depression as a true latent variable, which determines a variety of psychological behaviors, or is it actually the upshot of system of self-reinforced negative behaviors and feelings? Such questions can perhaps be answered by developing and testing models of the kind proposed in this article.

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(*Appendix follows*)

Appendix

Stable States and Covariance Matrix for the Lotka-Volterra Mutualism Model

The model is formulated as:

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

(Equation 3)

Stable States

The system is in equilibrium when for all x_i, $dx_i/dt = 0$. This trivially takes places when $a_i = 0$ or $x_i = 0$ or when:

$$a_i x_i - a_i x_i x_i/K_i + \frac{a_i x_i}{K_i} \sum_{\substack{j=1 \\ j\neq 1}}^{n} M_{ij}x_j = 0 \Leftrightarrow$$

$$1 - x_i/K_i + \frac{1}{K_i} \sum_{\substack{j=1 \\ j\neq 1}}^{n} M_{ij}x_j = 0 \Leftrightarrow$$

$$K_i = x_i - \sum_{\substack{j=1 \\ j\neq 1}}^{n} M_{ij}x_j$$

in matrix notation ($M_{ii} = 0$):

$$\mathbf{K} = \mathbf{X}^*[\mathbf{I} - \mathbf{M}] \Leftrightarrow$$

assuming that **[I − M]** is invertible,

$$\mathbf{X}^* = [\mathbf{I} - \mathbf{M}]^{-1}\mathbf{K}.$$

Note that equilibrium **X*** is independent of **a** and initial values **x0**.

For mutualistic interactions ($M_{ij} \geq 0$), Travis and Post (1979) show that this equilibrium is stable if and only if all eigenvalues of $[\mathbf{M} - \mathbf{I}]$ have negative real parts, that is, Re $\lambda_i(\mathbf{M} - \mathbf{I}) < 0$, for $i = 1, 2, \ldots$. Travis and Post also provide an equivalent condition in terms of the values of **M**.

The Relation Between g and K

Given this equilibrium state, the covariance matrix can be written as $\Sigma = [\mathbf{I} - \mathbf{M}]^{-1}\Psi[\mathbf{I} - \mathbf{M}']^{-1}$, where Ψ is a diagonal matrix representing the variances of the uncorrelated **K**.

Given this covariance structure we can indicate how a *g* score, obtained in a PCA, arises. A PCA involves the following decomposition, $\Sigma = \mathbf{PDP}'$, where **P** is orthonormal (eigenvectors) and **D** is positive and diagonal (eigenvalues). The inverse transformation is $\mathbf{D} = \mathbf{P}'\Sigma\mathbf{P}$, and it is this inverse transformation that is used to obtain component scores (*g*) associated with the largest value (eigenvalue) in **D**. We can apply this to the covariance structure of the mutualism model.

$$\Sigma = [\mathbf{I} - \mathbf{M}]^{-1}\Psi[\mathbf{I} - \mathbf{M}']^{-1} = \mathbf{PDP}'$$

and the inverse:

$$\mathbf{P}'[\mathbf{I} - \mathbf{M}]^{-1}\Psi[\mathbf{I} - \mathbf{M}']^{-1}\mathbf{P} = \mathbf{D}.$$

Let us assume that the principal component scores associated with the largest eigenvalue approximate the *g* scores, that is, $g = \mathbf{P}_\mathbf{g}'\mathbf{x} = \mathbf{P}_\mathbf{g}'[\mathbf{I} - \mathbf{M}]^{-1}\mathbf{K}$, where $\mathbf{P}_\mathbf{g}$ is the eigenvector in **P** that is associated with the largest eigenvalue in **D**. It is then clear the *g* scores associated with the largest eigenvalue in **D** cannot be related simply to any source of individual differences K_i; they are a function of both **K** and **M**.

Derivation of the Positive Manifold

The simulations demonstrate that positive **M** results in a positive manifold, that is, positive Σ. Because $\boldsymbol{\Psi}$ is always positive, a sufficient condition for positive Σ is $[\mathbf{I} - \mathbf{M}]^{-1}$ being non-negative. The conditions for non-negative $[\mathbf{I} - \mathbf{M}]^{-1}$ are specified in Lemma 1.

Lemma 1. Let $\mathbf{M} \in \Re^{K\times K}$ be a non-negative matrix, that is, a matrix with only non-negative elements, such that $|\mathbf{I} - \mathbf{M}| \neq 0$. Then the matrix $[\mathbf{I} - \mathbf{M}]^{-1}$ is non-negative if and only if the eigenvalues λ_i of **M** satisfy

$$|\lambda_i(\text{M})| < 1, \text{ i} = 1, 2, \ldots .$$

Proof. Subtract the partial sums

$$\sum_{i=0}^{n} \mathbf{M}^i = \mathbf{I} + \mathbf{M} + \mathbf{M}^2 + \ldots + \mathbf{M}^n$$

and

$$\mathbf{M}\sum_{i=0}^{n} \mathbf{M}^i = \mathbf{M} + \mathbf{M}^2 + \ldots + \mathbf{M}^{n+1}$$

from each other to obtain $(\mathbf{I} - \mathbf{M})\sum_{i=0}^{n} \mathbf{M}^i = \mathbf{I} - \mathbf{M}^{n+1}$, or

$$\sum_{i=0}^{n} \mathbf{M}^i = (\mathbf{I} - \mathbf{M})^{-1}(\mathbf{I} - \mathbf{M}^{n+1}).$$

Hence in the limit of $n\rightarrow\infty$, if $M^n\rightarrow 0$,

$$\sum_{i=0}^{\infty} \mathbf{M}^i = (\mathbf{I} - \mathbf{M})^{-1},$$

the left hand side of which is clearly non-negative as **M** is non-negative, and so $[\mathbf{I} - \mathbf{M}]^{-1}$ is non-negative.

For sufficiency, it remains to be shown that $\lim_{n\rightarrow\infty} \mathbf{M}^n = 0 \Leftrightarrow |\lambda_i(\mathbf{M})| < 1$. Let λ be an eigenvalue of **M** and **x** be a corresponding normalized eigenvector. Clearly $\lim_{n\rightarrow\infty} \mathbf{M}^n\mathbf{x} = \lim_{n\rightarrow\infty} \lambda^n\mathbf{x} = 0 \Leftrightarrow |\lambda| < 1$. Given that any matrix norm $\|\mathbf{M}\|$ is bounded from below by the absolute value of the largest eigenvalue of **M**, a sufficient condition for $[\mathbf{I} - \mathbf{M}]^{-1}$ to be non-negative is that $\|\mathbf{M}\| < 1$ for any matrix norm.

Next we show necessity. Assume that $[\mathbf{I} - \mathbf{M}]^{-1}$ is non negative. Because **M** is non-negative, $\mathbf{M} + \varepsilon^2\mathbf{11}'$, where **1** is a vector with all entries equal to one and $\text{e} \in \Re$ is positive. Therefore, Theorem 7.44 in Schott (1997) [1] can be applied, and hence, if

$$\rho(\varepsilon) = \max_i |\lambda_i(\varepsilon)|,$$

where $\lambda_i(\varepsilon)$ are eigenvalues of $\mathbf{M} + \varepsilon^2\mathbf{11}'$ and **x(ε)** is an eigenvector corresponding to $\lambda_i(\varepsilon)$ for which $\rho(\varepsilon) = |\lambda_i(\varepsilon)|$, then

$$(\mathbf{M} + \varepsilon^2\mathbf{11}')abs[\mathbf{x}(\varepsilon)] = \rho(\varepsilon)abs[\mathbf{x}(\varepsilon)].$$

Here abs[.] indicates element wise absolute value. As above,

$$\sum_{i=0}^{n} (\mathbf{M} + \varepsilon^2\mathbf{11}')^i = [\mathbf{I} - (\mathbf{M} + \varepsilon^2\mathbf{11}')]^{-1}[\mathbf{I} - (\mathbf{M} + \varepsilon^2\mathbf{11}')^{n+1}].$$