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

Kan, K.-J.

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SUMMARY IN ENGLISH / SAMENVATTING IN HET ENGELS

Better Theory

This thesis stressed the importance of the development of better theory in intelligence research. In addition, it illustrated that taking explicit scientific philosophical standpoints (e.g. realist or nonrealist) with respect to intelligence, and the variables that relate to it, helps to understand empirical results. Without adequate theory and without researchers' philosophical standpoints, it is extremely difficult to make sense of the intelligence literature, which can hinder scientific progress in intelligence research.

In the introduction we explained why it is so difficult to make sense of the intelligence literature. First, we pointed out that intelligence relates to how well systems process information and that - when we restrict ourselves to human intelligence - the level of intelligence is usually determined using psychometric tests via ordinal scaling. Next, it was made clear that without further commitment to any specific theory that relates intelligence to a quantitative property (or multiple quantitative properties), intelligence is nothing more than a rank order on intelligence test scores (Bartholomew, 2004), whether these comprise raw scores or scores on variable extracted from those raw scores. The problem that arises next, is that different rank orders can be made, so that based on the same data one researcher can (legitimately) conclude that intelligence grows, hence changes throughout development, while another researcher can (also legitimately) conclude that intelligence is stable over time and does not change at all (see Figure 1.1). What conclusion is drawn in practice largely depends on the scientific perspective of the researcher. With scientific perspective we mean an intra-individual perspective, which is common in developmental psychology, or an interindividual perspective, which is common in differential psychology. We stressed that the causes of inter-individual differences can be entirely different from the causes of intra-individual differences. In order to understand, explain, and model intelligence well, we need both perspectives. The interindividual perspective is needed to explain the covariance structure of intelligence test scores, the intraindividual perspective to explain cognitive growth. We noted that the majority of studies into intelligence lacks the developmental perspective and that discussions and theories are mainly concerned with (statistical) descriptions of individual differences according to factor models.

Aims

The aims of the thesis were twofold. The first was to reinvigorate the development of an adequate theory of intelligence by providing a model that accounts for both cognitive growth and (heritable) interindividual differences in intelligence. This model had to be able to explain salient findings in intelligence research, such as a correlation between intelligence subtests' heritability coefficients and their loadings on the (statistical) general factor of intelligence. While developing such model, we encountered theoretical issues that were not fully addressed in the literature. They concern mostly the interpretation of the heritability of intelligence. The second aim was to address these issues in more detail.

Working Hypothesis

Throughout the thesis we provisionally accepted the Cattell-Horn-Carroll (CHC) model as a working hypothesis. Taking a realist scientific philosophical position concerning the variables of intelligence, we hypothesized that the second order factors in this model (including Gf and Gc) represent individual differences in unique cognitive systems, which are constrained by genetically and environmentally influenced capacities. We did not posit a substantive underlying general factor, because we believed positive intercorrelations among the cognitive systems are due to reciprocal interactions among those systems that occur throughout cognitive development, as in the mutualism model (see Figure 1.8). We also assumed that in principle individual differences in these systems can

be measured by intelligence tests, although we maintained that in practice intelligence tests are likely not uni-dimensional (see Figure 7.1).

Summary per Chapter

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

Chapter 2 dealt with the problem of nonlinearity. We noted that the genetic and environmental variables in behavior genetic models are generally not measured, but merely inferred, using genetically informative research designs (e.g. twin and adoption studies), and that these genetic and environmental variables are modeled as having linear relationships with the observed variables, e.g. cognitive abilities measured by IQ tests. However, in reality cognitive development is typically nonlinear and is characterized by stage transitions (see Table 1). By means of computer simulations, we investigated the effects on estimated heritabilities and environmentalities in statistical linear behavior genetic modeling in case the underlying mechanisms were nonlinear. In doing so, we used the two-cell model of van Oss & van Ooyen (1996). Although originally the two cells or units represent an inhibitory and an excitatory neuron, they were interpreted as representing inhibitory and excitatory networks constituting a neural system (van Oss & van Ooyen, 1996), for example a system that underlies working memory. We implemented heritable individual differences in the parameters of the model, ran a series of simulations, and investigated whether the estimated genetic and environmental structure resembled the implemented structure.

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

Gene-environment correlations also constituted our explanation of the results discussed in Chapter 3, in which we summarized data from 23 independent behavior genetic studies into intelligence. These data showed the contra-intuitive result that the most cultural dependent intelligence tests (i.e. knowledge tests) demonstrated higher heritabilities than the least cultural dependent tests (i.e. cognitive processing tests). Notably, these cultural dependent knowledge tests also showed the relatively highest *g* loadings (see Table 1). In isolation, mainstream theories of intelligence fail to explain this combined effect (see Chapter 6). Some of them even predict the opposite. We hypothesized that if society puts demands on acquired knowledge rather than cognitive

processing, gene-environment correlations would become higher for individual differences in knowledge than individual differences in cognitive processing. In standard behavior genetic modeling gene-environment correlations will be subsumed under the genetic variance, leading to higher heritability estimates.

On the basis of the results from Chapter 2, we can also formulate an alternative or additional hypothesis. As was shown, stage transitions in cognitive development can result in overestimation of the true heritability. The development of certain cognitive abilities may be characterized by more transitions than the development of other cognitive abilities, so that the increase in heritability due to nonlinear effects will differ across abilities. For example, verbal comprehension, usually measured by knowledge tests, may develop stage wise, whereas nonverbal cognitive abilities, usually measured by cognitive processing tests, develop gradually (whether this is the case should be investigated empirically). Hence, the heritability coefficients of the verbal tests can be overestimated, while those of nonverbal tests are correct or less affected. Whether this alternative explanation of increasing heritability can account for the high *g* loadings of verbal knowledge tests (see Table 7.1) is unknown, but can be investigated with a nonlinear model of general intelligence. Future simulations with a mutualism model that includes nonlinear terms may help to discriminate between the two explanations.

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

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

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

Likely, such sample heterogeneity also leads to the separation of *g* and Gf. In homogeneous samples they will be equivalent (see Table 7.1), as we found.

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

In Chapter 7 we first summarized the previous chapters. Next we presented an integrated model of general intelligence. It was a mutualism model (van der Maas et al., 2006) that incorporated the main idea of investment theory (individual differences in cognitive processes - fluid abilities - give rise to differences in knowledge and skills - crystallized abilities) and Dickens & Flynn's (2001; Dickens, 2008) social multiplier. In line with the mutualism theory (van der Maas et al., 2006), we assumed that cognitive processing benefits from knowledge acquisition. In the integrated theory, an underlying *g* (Spearman, 1904; Carroll, 1993; Jensen, 1998) was absent. Genetic correlations among limiting capacities can be present, but were taken to be the result of what we denoted genetic sampling (Thompson, 1951; Bartholomew et al. 2009; Anderson, 2001; Penke et al., 2007; see Chapter 6) and not as due to general genetic effects (Kovas & Plomin, 2006). The integrated theory accounted for the fact that individual differences are the most pronounced on the most culturally dependent subtests, which are the most heritable and the most *g* loaded.

General Conclusions and Discussion

The main aim of this thesis was to reinvigorate the development of an adequate theory of intelligence by providing a model that accounts for both cognitive growth and (heritable) interindividual differences in intelligence. The model had to be able to explain salient findings in intelligence research, such as a correlation between intelligence subtests' heritability coefficients and their loadings on the (statistical) general factor of intelligence (current theories of intelligence in isolation do not account for those relationships). To this end, we aimed to integrate hypotheses from different theories of intelligence.

In the last chapter we presented an integrated model of general intelligence. The integrated model accounted for the fact that individual differences are the most pronounced on the most culturally dependent subtests, which are the most heritable and the most *g* loaded. The effect was due to differences in gene-environment effects across cognitive abilities. From our simulation studies with the integrated model, we concluded that the relations among *g* loading, heritability, and cultural load will be positive when the society fosters crystallized abilities rather than fluid abilities.

The simulations also showed that the general factor of intelligence does not have to represent a realistic (e.g. biological) common cause of individual differences. In our integrated model it is a merely a statistical entity, i.e., an informative summary descriptive. However, as such, general intelligence certainly has utility. Specifically, we consider it to be similar to 'general health'. Like general health is an informative summary descriptive of physical functioning, '*g*' is an informative summary descriptive of cognitive functioning.

Nevertheless, a distinction between *g* as a statistically entity, and *g* as interpreted as a realistic, common cause of individual differences, is not only important theoretically, but also empirically. Consider genetic association and linkage studies of intelligence, for example. So far, the search for genes for general intelligence has met with relatively little success (Deary, Johnson, & Houlihan, 2009; Plomin and Spinath, 2004; Chabris et al., *in press*). The alternative theories of Dickens and Flynn (2001; Dickens, 2008) and of van der Maas et al. (2006), in which the general factor of intelligence is a statistical entity originating in reciprocal beneficial interactions among cognitive processes or abilities, are able to provide a plausible explanation of this lack of success. In these theories, there are no direct genetic influences general to all abilities. If general intelligence is

indeed the outcome of such interactions, the search for specific 'genetic influences on *g*' is a questionable undertaking (Dolan, Kan, van der Maas, 2008; van der Sluis, Kan & Dolan, 2010), in particular when (1) the genetic determinants of these processes are under mutation selection balance, which means that the process of natural selection reduces genetic variance but cannot deplete all of it because new variation - due to mutations - is continually reintroduced (Penke et al., 2007), and (2) the interactions are nonlinear (e.g. Molenaar, Boomsma, & Dolan, 1993; Kan, Ploeger, Raijmakers, Dolan & van der Maas, 2010).

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

The main points of this thesis are as follows. First, although it is still not possible to determine whether a realistic, underlying *g* is present or not, we can conclude that current *g* theories are inadequate in explaining certain salient empirical findings. Next to the individual differences perspective they have, they need a developmental perspective. The role of the dynamic interplay between genetic and environmental variables that occurs during development needs to be explicated. Second, formal modeling is important in intelligence research. Using the mutualism model can help researchers to study combined effects. The main advantage of the mutualism model is that it is able to combine the intraindividual differences and interindividual differences scientific perspectives on intelligence, as we advanced.