

## **Test of Nyborg's General Trait Covariance (GTC) Model for Hormonally Guided Development by Means of Structural Equation Modeling**

MARTIN REUTER<sup>1\*</sup>, PETRA NETTER<sup>1</sup>, JÜRGEN HENNIG<sup>1</sup>,  
CHANGIZ MOHIYEDDINI<sup>2</sup> and HELMUTH NYBORG<sup>3</sup>

<sup>1</sup>*Department of Psychology, University of Giessen, Germany*

<sup>2</sup>*Department of Psychology, University of Zurich, Switzerland*

<sup>3</sup>*Department of Psychology, University of Aarhus, Denmark*

### *Abstract*

*Nyborg's General Trait Covariance (GTC) model for hormonally guided development investigates the influence of gonadal hormones and fluid intelligence on body build, achievement, and socioeconomic variables. According to the model, testosterone should be negatively related to height, fat/muscle ratio, intelligence, income, and education. It is conceived that this influence should be determined to a great extent by mutual relationships between these variables. The model was tested by means of structural equation modeling (SEM) in a sample of 4375 males who had served in the United States Armed Forces. The results largely confirm Nyborg's androtype model but in addition reflect the relationships between the variables included in a quantitative causal manner. It could be shown that testosterone has a negative influence on crystallized intelligence and that this effect is mainly mediated by the negative influence of testosterone on education. An additional multiple group analysis testing for structural invariance across age groups revealed that the mediating role of education is more pronounced in old veterans. Copyright © 2002 John Wiley & Sons, Ltd.*

### **INTRODUCTION**

In 1979 Nyborg published his General Trait Covariance (GTC) model for hormonally guided development which he elaborated over the past two decades (Nyborg, 1979, 1983, 1984, 1987, 1988, 1990, 1994, 1997; Nyborg & Jensen, 2000, 2001). The model allows predictions about the individual covariation of body build, brain functions, performance, education, and personality development by classifying individuals according to their

\*Correspondence to: Martin Reuter, Justus-Liebig-Universität Giessen, FB06 Psychologie, Otto-Behaghel-Str. 10F, D-35394 Giessen, Germany. E-mail: martin.reuter@psychol.uni-giessen.de

*Received 11 January 2002*

*Accepted 9 October 2002*

gonadal hormone status (androtyping for males and oestrotyping for females). The hormone production on the other hand is influenced by genes and to a much smaller extent by environment. Nevertheless, the GTC model represents a synthesis of constitutional and environmental based theories for it is suggested that biological and constitutional variables with a strong genetic determination covary with social and environmental factors. The technique of somatotyping had already been used by constitutionists such as Sheldon (Sheldon, Stevens, & Tucker, 1940) or Kretschmer (1921) but their typologies were mainly based on morphology although endocrine influences on temperament, mental disease, and education were assumed. As opposed to Kretschmer and Sheldon, the types in Nyborg's theory were defined by the gonadal hormone status. High testosterone levels mean maximum sexual differentiation and high sexual identity at the cost of less than optimal intellectual and personality development (Nyborg, 1997).

According to Nyborg's theory high levels of testosterone in males lead to early onset of puberty, increased sociability, decreased shyness, and make a male more popular among peers. Sexual identity develops in a more masculine direction associated with earlier awakening of sexual interests and more liability to develop aggressions (Dabbs & Morris, 1990). Furthermore, males with high testosterone levels are less interested in education, which according to this theory has negative consequences for scholastic achievement. A similar association between precocious puberty and negative educational outcome, which is mediated by psychosocial variables, was also reported by Sandberg and Barrick (1995). Moreover there exists evidence that precocious puberty impairs cognitive development (Rovet, 1983). Besides the hypothesis that testosterone has a negative influence on academic achievement by impairing the development of cognitive abilities or by a shift in interests towards non-school-related activities like sexual behaviour, the mediating role of aggressive and destructive behaviour between intelligence and testosterone has been described in the literature (e.g. Olweus, Mattson, Schalling, & Low, 1988). High levels of testosterone increase the propensity to engage in aggressive–destructive behaviour and make persons more impatient and irritable, which is not beneficial for educational outcomes and the development of cognitive abilities.

The inverse is the case in males with low testosterone levels. Their sexual interest develops later and they spend more time and effort in intellectual activities, resulting in higher cognitive achievement. In addition, a person's physique covaries with his intelligence and testosterone status. The predictions made by the GTC model could be confirmed by empirical data showing that extraordinarily creative scientists and noncreative high-IQ males tend to have low testosterone levels and an above average fat/muscle ratio (Nyborg, 1997). The statement that testosterone influences occupational choice was corroborated by Dabbs et al. (1990), who reported higher testosterone levels in football players and in actors than in ministers. The findings were interpreted in terms of dominance and antisocial tendencies, with the conclusion that these variables can affect occupational preference in a subtle way.

It had already been reported by Nyborg (1994) that testosterone and body mass index are negatively correlated and that intelligent persons tend to have a higher BMI since they tend to be slightly taller (familial disposition) and have an above average fat/muscle ratio due to lower anabolic effects caused by low testosterone levels. Negative correlations between testosterone levels and BMI have also been reported by other authors (e.g. Ukkola et al., 2001) and it is even suggested that this lack of testosterone predisposes to adiposity and increases the risk of type 2 diabetes mellitus (Tsai, Boyko, Leonetti, & Fujimoto, 2000). Furthermore, there is evidence in the literature that constitutional growth delay or

constitutional short stature are associated with lower IQ scores (Holmes, Thompson, & Hayford, 1984; Holmes, Karlsson, & Thompson, 1985; Gold, 1978).

So far the relationships proposed by the GTC model had only been tested in males by bivariate correlational analyses. Netter, Toll, Rohrmann, Hennig, and Nyborg (2000) tested the GTC model in a multivariate nonparametric approach by using configural frequency analysis (CFA) (Krauth, 1993; Krauth & Lienert, 1973). They could indeed identify types of configurations relating testosterone levels to physical variables, socioeconomic status, and intelligence. The results showed that the variable age is an important mediator in Nyborg's model of androtypes because some of the types detected in the sample of the younger subjects could not be confirmed in the subsample of the older ones. Nevertheless, disadvantages of the nonparametric approach are that no causal relations between variables can be established and that no estimates on the strength of the associations between the variables in the model can be made. Furthermore, transformation of metric variables into a lower scale level by dichotomizing the dimensions is inevitably associated with a loss of information.

Therefore the aim of the present study is to test the GTC model for male hormonally guided development by structural equation modelling (SEM) in order to (i) quantify influences between variables, (ii) test whether the relationships are based on mediator effects and (iii) test whether age modifies the structure of the model or the size of relationships between the variables.

## METHOD

### Sample

The Centers for Disease Control (1988) provided an archival data set on 4375 American armed forces veterans between 31 and 49 years of age ( $M = 38.35$ ;  $SD = 2.52$ ). The original purpose in obtaining these data was to assess the long term effects of these veterans' military service about 17 years after induction in the military. Approximately half of the sample had served in the Vietnam war but did not differ in socio-economic status or the variables included in the GTC model from the half who had served elsewhere.  $n = 3579$  were White,  $n = 516$  Black,  $n = 198$  Hispanic,  $n = 34$  Asian, and  $n = 48$  Native.

### Structural equation modeling

First a structural equation model for Nyborg's androtype model was derived from theoretical considerations and then tested in a sample of 4375 subjects. Structural equation modeling would then require a trim of the initial model in order to reject spurious associations and to recognize new important relations between variables that at first were neglected (model building). The trimming as well as the model building strategy is performed to optimize the model fit on the one hand and to allow only paths that could be theoretically justified on the other hand.

The next step was to test the model for age effects, i.e. to check whether the proposed relations between variables vary across different age groups. In analogy with the above cited study by Netter et al. (2000) the total sample was divided according to the median ( $\leq 38$ ;  $> 38$  years) into two subsamples, young ( $n = 1939$ ) and old ( $n = 2436$ )

subjects. The model was then tested in the two age-subsamples. In addition, a multiple group analysis for differences in the model structure between young and old subjects was conducted.

### Variables in the model (observed variables)

In accordance with Nyborg's androtype theory, six variables from a large data pool were selected to illustrate proposals made by the GTC model. The variables were two intelligence measures, Block Design (BDWAIS) and Information (INFWAIS), from the Wechsler Adult Intelligence Scales Revised (WAIS-R), which, according to Muldoon, Ryan, Matthews, and Manuck (1997), represent fluid intelligence (BDWAIS) and crystallized intelligence (INFWAIS) *sensu* Horn and Cattell (1966), the body mass index (BMI) as a measure of physique, plasma testosterone measured by RIA and obtained in the morning (TESTO) as the endocrine parameter, income (INCOME), and education (EDUC) (i.e. highest year of formal education; the minimum score of EDUC was 1, indicating that some veterans, maybe from lower social class, who had hardly ever attended school entered the army to fight in the Vietnam war) as representations of social status.

Concerning the model specification two exogenous variables, TESTO and BDWAIS, and four endogenous variables, BMI, EDUC, INCOME, and INFWAIS, were proposed. Descriptive statistics, tests of univariate normality of the six observed variables, and their covariance and correlation matrixes are presented in Tables 1 and 2, respectively.

Testosterone and Block Design were set to be exogenous variables because of their high degree of genetic determination (Rotter, Wong, Lifrak, & Parker, 1985; Carroll, 1984). Although environmental factors like physical exercise, sports, and stress could influence testosterone levels, and stimulating surroundings could influence fluid intelligence, it was assumed that these moderating influences are only of minor relevance. According to Rotter

Table 1. Means, standard deviations, skewness, and kurtosis of observed variables and tests of univariate normality (total sample)

	<i>M</i>	STD	Min	Max	Skewness	Kurtosis
EDUC	13.29	2.30	1	18	0.05	0.30**
INCOME	3.90	2.26	1	7	-0.04	-0.41**
INFWAIS	18.96	5.30	3	29	-0.35**	-0.56**
BMI	26.88	4.48	15.74	66.80	1.48**	6.17**
BDWAIS	30.02	9.85	0	51	-0.24**	-0.51**
TESTO	679.67	235.54	53	1950	0.83**	1.50**

\*\**p*-Value < 0.01.

Table 2. Covariance matrix (bright) and correlation matrix (dark) obtained in the total sample (*N* = 4375)

	EDUC	INCOME	INFWAIS	BMI	BDWAIS	TESTO
EDUC	5.29	0.360	0.545	0.005	0.270	-0.104
INCOME	1.87	5.11	0.322	0.049	0.224	-0.112
INFWAIS	6.65	3.86	28.10	0.010	0.453	-0.087
BMI	0.05	0.50	0.23	20.10	-0.025	-0.343
BDWAIS	6.11	5.00	23.69	-1.10	97.05	-0.041
TESTO	-56.44	-59.59	-108.02	-362.19	-94.50	55 480.33

et al. (1985), the heritability of androgens is 65%. However, dimensions of social status, fat/muscle ratio, and crystallized intelligence, in contrast, are supposed to be more susceptible to modifications by environmental factors and therefore were set to be endogenous (dependent) variables. Especially with respect to differences in the amount of heritability between fluid and crystallized intelligence, there is a controversy in the literature (Cattell, 1971; Horn, 1985). Nevertheless, Cattell (1971) himself, who brought up the concept of fluid and crystallized intelligence, argued 'that fluid ability represents the biological substrate of intelligence, which is then "invested" in the particular knowledge and skills taught by a particular culture to form crystallized ability. Therefore, a measure of inborn ability should have higher heritability than one of acquired knowledge' (Mackintosh, 1998). The debate on the heritability of crystallized and fluid intelligence has certainly not come to a commonly accepted solution. By defining fluid intelligence as an exogenous and crystallized intelligence as an endogenous variable in the structural equation model we just wanted to meet the assumptions underlying Nyborg's GTC model, which is more adequately represented by Cattell's theory. We did not wish to interfere with the heredity debate ourselves.

Although testosterone concentrations in the present sample were only measured once in adulthood, it could be assumed that these hormone concentrations are good indicators for the testosterone levels in puberty and adolescence, because of (i) the strong genetic determination and (ii) the intraindividual stability reported by longitudinal studies (Zmuda et al., 1997). Because of an observed almost zero correlation ( $r = -0.03$ ) between testosterone and fluid intelligence the two dimensions were supposed to be influenced by different genes and therefore the introduction of a common latent variable into the model with impact on the gonadal hormones as well as on fluid intelligence was rejected.

According to the GTC model we assume an influence of testosterone and of BDWAIS on each endogenous variable. It has already been reported by Nyborg (1994) that testosterone and body mass index are negatively correlated and that intelligent people tend to have a higher BMI. Furthermore, high testosterone is said to lead to an earlier onset of puberty and more interest in girls than in school, and this would result in lower education and consequently lower income. More activities outside school as a consequence of high sexual drive should lead to less acquisition of knowledge and therefore lower performance in tests of crystallized intelligence. A lack of knowledge, on the other hand, leads to lower income. The assumption that high inherited intelligence leads to higher education, more crystallized intelligence, and more income is fairly trivial. Nevertheless, it remains interesting to compare the strength of direct versus indirect influences of environmental and organismic variables (e.g. the influence of BDWAIS on income via education compared to the direct influence of BDWAIS on income).

All these explained relationships can be specified by a recursive path model although there is the possibility that later model trimming and model building could turn the model into a nonrecursive model; i.e. it was not excluded that there are causal bi-directional effects within the endogenous variables or that their disturbances (all causes of an endogenous variable that are omitted from the model (Kline, 1998, p. 53)) are correlated. Once the model is tested it will be considered if additional paths which had not been assumed in advance will improve the model even if they are nonrecursive.

In LISREL matrix syntax (Jöreskog & Sörbom, 1993) the two exogenous variables ( $X$ ) are called  $\xi$  (xis) and the endogenous variables ( $y$ ) are called  $\eta$  (etas). The  $B$  (beta) matrix is the matrix of the structure coefficients of the etas, the  $\Gamma$  (gamma) matrix is the matrix of the relations (path coefficients) between the xis and the etas and the  $\Psi$  (psi) matrix is the

matrix of the variances/covariances of the disturbances  $\zeta$  (zetas) of the etas. The model can be written in matrix notation. The coefficient matrices are B and  $\Gamma$ .

$$B = \begin{bmatrix} 0 & 0 & 0 & 0 \\ \beta_{21} & 0 & \beta_{23} & \beta_{24} \\ \beta_{31} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \eta = \begin{bmatrix} \eta_1 \\ \eta_2 \\ \eta_3 \\ \eta_4 \end{bmatrix} \quad \zeta = \begin{bmatrix} \zeta_1 \\ \zeta_2 \\ \zeta_3 \\ \zeta_4 \end{bmatrix} \quad \Gamma = \begin{bmatrix} \gamma_{11} & \gamma_{12} \\ \gamma_{21} & \gamma_{22} \\ \gamma_{31} & \gamma_{32} \\ \gamma_{41} & \gamma_{42} \end{bmatrix} \quad \xi = \begin{bmatrix} \xi_1 \\ \xi_2 \end{bmatrix}$$

with  $\eta_1 = \text{EDUC}$ ,  $\eta_2 = \text{INCOME}$ ,  $\eta_3 = \text{INFWAIS}$ ,  $\eta_4 = \text{BMI}$ ,  $\xi_1 = \text{BDWAIS}$ , and  $\xi_2 = \text{TESTO}$ .

### Data screening

Data screening revealed that the assumption of multivariate normality was violated because already skewness and kurtosis in the univariate analysis contradicted normality. Therefore the variables were normalized by means of the PRELIS normalization algorithm (Jöreskog & Sörbom, 1996) before further analyses were conducted. PRELIS scales the normal scores to have the same mean and variance as its original variable. Normalization of INCOME still resulted in significant kurtosis. Nevertheless, the absolute value ( $-0.405$ ) was small enough to regard the slight non-normality as not problematic (Chou & Bentler, 1995; Hu, Bentler, & Kano, 1992; West, Finch, & Curran, 1995). Further data inspection showed that the variance of TESTO was many times larger than the variance of the other variables. Since such differences in the size of variances can cause problems in structural equation modelling (and other methods relying on iterative processes) (Kline, 1998, p. 85), a linear transformation of TESTO was conducted by dividing the raw scores by 20 and thereby achieving 400-fold reduction in variance. With all these modifications the raw data were deemed ready for analysis, because most prerequisites of structural equation modeling were warranted. The resulting covariance matrix is represented in Table 3.

### Method of estimation and fit indices

Path analysis was performed by the method of maximum likelihood (ML) estimation (see, e.g. Bollen, 1989, pp. 107–111). The most widely used fitting function for general structural equation models is  $F_{ML}$  and an important characteristic of it is that with few exceptions  $F_{ML}$  is scale invariant and scale free (Swaminathan & Algina, 1978). This implies that the structural parameters and estimates are maintained equivalent after linear transformation as done in our sample with the variable TESTO. According to the

Table 3. Covariance matrix (bright) and correlation matrix (dark) after normalization of all variables and linear transformation of TESTO ( $N = 4375$ )

	EDUC	INCOME	INFWAIS	BMI	BDWAIS	TESTO
EDUC	5.29	0.348	0.557	0.028	0.276	-0.100
INCOME	1.22	2.32	0.311	0.076	0.216	-0.098
INFWAIS	6.79	2.51	28.10	0.015	0.452	-0.085
BMI	0.29	0.52	0.36	20.10	-0.012	-0.368
BDWAIS	6.25	3.24	23.61	-0.51	97.05	-0.034
TESTO	-2.70	-1.76	-5.29	-19.45	-3.93	138.70

recommendations in the literature (Hoyle & Panter, 1995) the following model fit indices will be reported besides the  $\chi^2$  statistic: the goodness-of-fit index (GFI), the adjusted goodness-of-fit index (AGFI), the standardized root-mean-square residual (SRMR), the comparative fit index (CFI), and the root mean error of approximation (RMSEA). A significant  $\chi^2$  value relative to the degrees of freedom indicates that the observed and estimated matrices are different. Therefore a nonsignificant  $\chi^2$  test indicates a good model fit. The GFI is based on a ratio of the sum of the squared differences between the observed and reproduced matrices to the observed variances. GFI = 1.00 indicates that the two matrices are identical (perfect fit). The AGFI adjusts the GFI index for the degrees of freedom given of a model relative to the number of variables. GFI, AGFI, and CFI should not be smaller than 0.90 (Kline, 1998, p. 131). The CFI compares the researcher's model with a null model (the observed variables are assumed to be uncorrelated). The SRMR, which represents a standardized summary of the average covariance residuals, should not be greater than 0.08, and the RMSEA, which tests whether the error of approximation is tolerable, should be smaller than 0.05 and not greater than 0.08 (Jöreskog, 1993).

### RESULTS

#### Test of the model in the total sample

Figure 1 illustrates the trimmed path model derived after eliminating nonsignificant paths from the initial model (see LISREL matrix syntax given before) in the total sample of males. The model fit was very good, GFI = 1.00, AGFI = 1.00, CFI = 1.00, SRMR = 0.0068, RMSEA < 0.003, and even the goodness-of-fit  $\chi^2$  statistic is nonsignificant

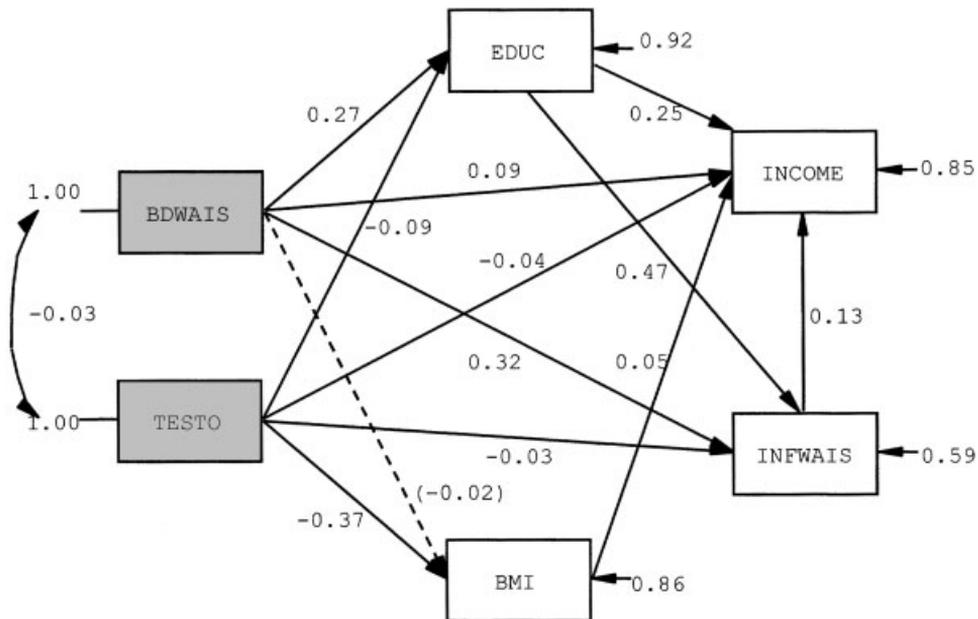


Figure 1. Path diagram for total sample (standardized solution); dotted arrows indicate nonsignificant paths, which were eliminated from the model.

Table 4. Decomposition of standardized effects for Nyborg's model of androtypes

Causal variables	Endogenous variables			
	EDUC	INCOME	INFWAIS	BMI
<b>BDWAIS</b>				
Direct effect	0.27	0.09	0.32	-0.02*
Indirect via EDUC		0.07	0.13	
Indirect via INFWAIS		0.04		
Indirect via BMI		<0.01		
Indirect via EDUC and INFWAIS		0.01		
Total Effect	0.27	0.21	0.45	-0.02*
<b>TESTO</b>				
Direct effect	-0.09	-0.04	-0.03	-0.37
Indirect via EDUC		-0.02	-0.04	
Indirect via INFWAIS		<-10.01		
Indirect via BMI		-0.02		
Indirect via EDUC and INFWAIS		<-10.01		
Total Effect	-0.09	-0.09	-0.07	-0.37

< -|0.01|, absolute magnitude of a negative coefficient <0.01.

\*Nonsignificant path, which was eliminated from the model.

( $\chi^2 = 3.11$ ,  $df = 3$ ,  $p = 0.374$ ), a result that is not very likely with large samples. All but one parameters have significant  $t$ -values. The path from BDWAIS to BMI, which just failed to be significant ( $t = -1.71$ ), was eliminated. Fluid intelligence does not seem to be related to this constitutional variable.

Eight percent of the variance of education, 15% of the variance of income, 41% of the variance of fluid intelligence, and 14% of the variance of the body mass index were explained by the model. All direct and indirect effects of testosterone and fluid intelligence on education, income, crystallized intelligence, and body mass index are presented in Table 4. The results show that the direct effects of testosterone on income and crystallized intelligence are smaller than the indirect effects, whereas education and BMI are only directly influenced by the hormone. The same is the case for the direct and indirect effects of fluid intelligence on the endogenous variables. Education and BMI are only directly influenced, whereas great proportions of the variance of income and crystallized intelligence are explained by indirect effects.

### Testing the model in different age samples

As mentioned above, the nonparametric approach for the evaluation of Nyborg's androtype model by configural frequency analysis (CFA) (Netter et al., 2000) had revealed that some CFA types identified in young subjects could not be detected in the subgroup of older subjects, indicating that age was a salient mediator variable in the androtype model. Therefore it was decided to test our structural equation model in the same two subsamples of old and young subjects. Median dichotomization ( $\leq 38$ ;  $> 38$  years) resulted in a sample of 1939 young and 2436 old veterans. In order to warrant the prerequisite of multivariate normality each subsample was normalized separately before the covariance matrices were calculated. The variable TESTO was submitted to linear transformation to correct for differences in the relative magnitude of observed variances as done for the total sample before. The covariance matrix of the young sample is presented in Table 5.

Table 5. Covariance matrix (bright) and correlation matrix (dark) of the young sample after normalization of all variables and linear transformation of TESTO ( $n = 1939$ )

	EDUC	INCOME	INFWAIS	BMI	BDWAIS	TESTO
EDUC	4.71	0.31	0.49	0.02	0.24	-0.08
INCOME	0.98	2.14	0.27	0.07	0.20	-0.09
INFWAIS	5.60	2.09	27.47	0.01	0.42	-0.08
BMI	0.17	0.47	0.27	19.77	-0.04	-0.37
BDWAIS	5.07	2.80	21.29	-1.57	94.72	-0.01
TESTO	-2.08	-1.56	-4.97	-20.03	-1.57	150.49

The path diagram of the young sample is depicted in Figure 2. In the sample of the young subjects the fit indices were also very good ( $GFI = 1.00$ ,  $AGFI = 0.99$ ,  $CFI = 1.00$ ,  $SRMR = 0.012$ , and  $RMSEA = 0.018$ ) and again even the goodness-of-fit  $\chi^2$  statistic is nonsignificant ( $\chi^2 = 6.56$ ,  $df = 4$ ,  $p = 0.161$ ). As in the total sample, the path from BDWAIS to BMI was eliminated because it was not significant ( $t = -1.94$ ). In addition, the path from TESTO to INCOME had to be excluded ( $t = -1.67$ ). This means the result obtained in the total sample, that there is no significant association between fluid intelligence and BMI, could be confirmed in the sample of the young veterans. Moreover, in young veterans testosterone only influences income indirectly via an influence on

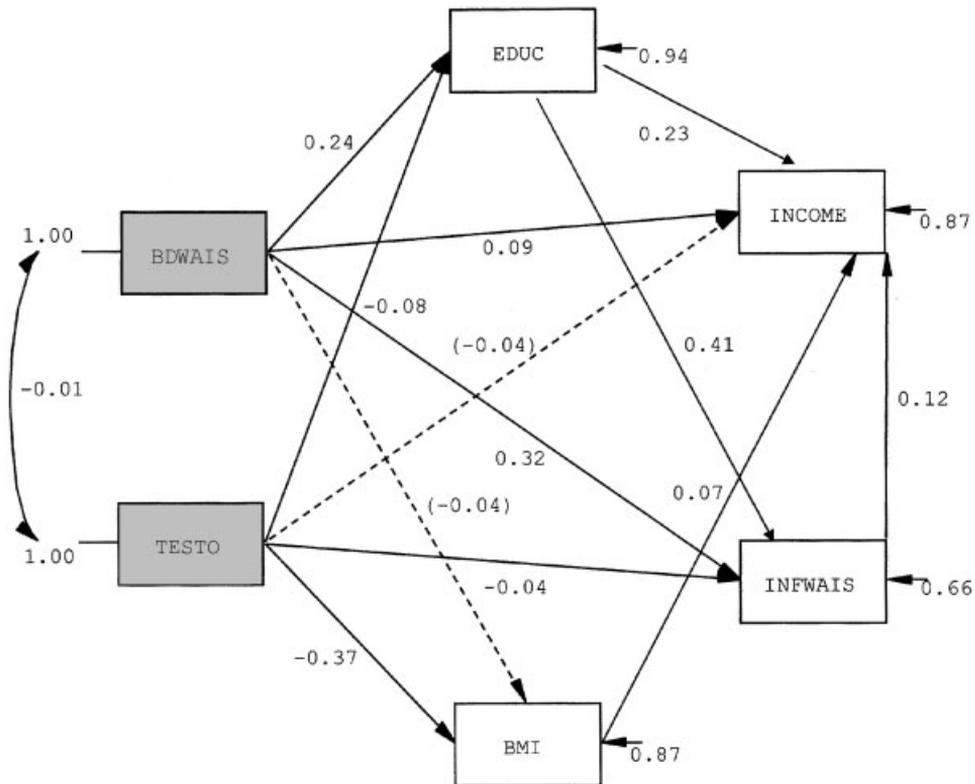


Figure 2. Path model of the young sample (standardized solution); dotted arrows indicate nonsignificant paths, which were eliminated from the model.

Table 6. Covariance matrix (bright) and correlation matrix (dark) of the old sample after normalization of all variables and linear transformation of TESTO ( $n = 2436$ )

	EDUC	INCOME	INFWAIS	BMI	BDWAIS	TESTO
EDUC	5.56	0.35	0.59	0.02	0.31	-0.08
INCOME	1.28	2.37	0.32	0.06	0.24	-0.07
INFWAIS	7.40	2.61	28.10	0.01	0.48	-0.07
BMI	0.21	0.43	0.12	20.24	0.01	-0.36
BDWAIS	7.18	3.59	25.40	0.34	98.95	-0.05
TESTO	-2.12	-1.16	-3.86	-17.93	-5.73	123.04

education. The proportions of explained variance in the sample of the young veterans were smaller than in the total sample. The explained variances for the young as compared with the total sample were 34% of INFWAIS in comparison with 41%, 13% of INCOME in comparison with 15%, 6% of EDUC in comparison with 8%, and 13% of BMI in comparison with 14%.

In the sample of the old veterans the same normalization and transformation procedures as described above were applied. The normalized covariance matrix in the sample of the older subjects is presented in Table 6. The model established for the sample of the older subjects (see Figure 3) also has very good fit indices (GFI = 1.00, AGFI = 1.00, CFI = 1.00, SRMR = 0.0062, RMSEA < 0.001) and again even the goodness-of-fit  $\chi^2$  statistic is nonsignificant ( $\chi^2 = 2.04$ ,  $df = 5$ ,  $p = 0.844$ ). Nevertheless the model is more parsimonious than the model of the total sample and the model of the younger subjects.

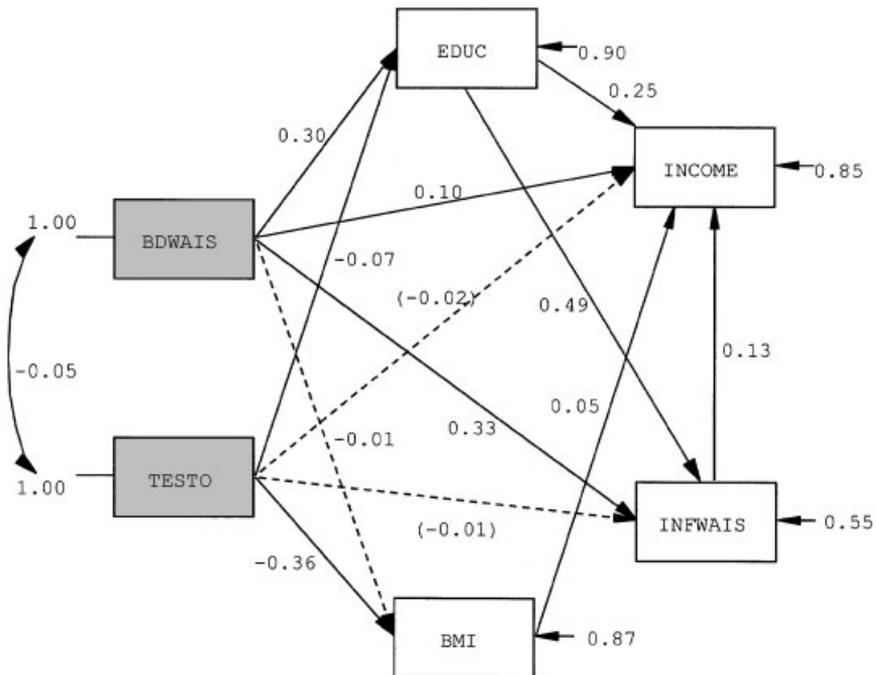


Figure 3. Path model of the old sample (standardized solution); dotted arrows indicate nonsignificant paths, which were eliminated from the model.

Three paths have nonsignificant  $t$ -values (path from TESTO to INCOME,  $t = -0.086$ ; path from TESTO to INFWAIS,  $t = -0.058$  and path from BDWAIS to BMI,  $t = -0.59$ ), and therefore they were eliminated. Results show that testosterone has no direct influence on crystallized intelligence in the sample of the old veterans as compared with the sample of the young veterans. The model trimming by eliminating all nonsignificant paths does not improve the  $\chi^2$  statistic significantly ( $p(\chi^2\Delta^2) = 0.703$ ;  $\Delta df = 3$ ). Usually, if two models are in nested sequence, the model with the fewest parameters will provide the worst fit to the data (i.e. be associated with the highest  $\chi^2$  value and the largest degrees of freedom) (Kelloway, 1998). Nevertheless, the deletion of nonsignificant paths could result in a better overall fit, although analysing a more parsimonious model means more conservative testing. This is the case in the present analysis. The chi-square increase is small relative to the increase in degrees of freedom, and the  $p$ -value therefore goes up instead of down. We accepted the trimmed model according to the common strategy of choosing the most parsimonious model if the overall fit indices of two models in nested sequence do not differ significantly.

The proportions of explained variance resemble those of the total sample and the young sample (45% of INFWAIS, 15% of INCOME, 13% of BMI, and 10% of EDUC). The proportion of explained variance of INFWAIS is largest in the sample of the old subjects.

### Multiple group analysis

In order to test if the relationships between variables obtained in the old and young sample were significantly different a multiple group path analysis<sup>1</sup> was conducted with equality constraints on the path coefficients across both groups. There were no constraints imposed on the error variances ( $\Psi$  matrix), which would only be necessary if it is intended to test whether the model has comparable predictive power across the groups. The multiple group analysis yielded perfect global fit indices (CFI = 1.00, RMSEA < 0.001) and a nonsignificant goodness-of-fit  $\chi^2$  statistic ( $\chi^2 = 23.36$ ,  $df = 24$ ,  $p = 0.499$ ). The  $\chi^2$  of the model with its path coefficients constrained to equality was then contrasted against the  $\chi^2$  of the unconstrained model ( $\chi^2 = 8.27$ ,  $df = 14$ ,  $p = 0.875$ ).

The difference test was not significant ( $p(\Delta\chi^2) = 0.129$ ;  $\Delta df = 10$ ) indicating that the free estimation of the constrained parameters in each sample would not significantly improve the model. Further analyses revealed that the direct effects from BDWAIS on EDUC (modification index  $\chi^2 = 7.27$ ) and from EDUC on INFWAIS (modification index  $\chi^2 = 3.03$ ) were significantly or almost significantly greater in the group of the old veterans. No other path coefficients differ significantly across groups.

## DISCUSSION

Nyborg's General Trait Covariance (GTC) model for hormonally guided development, which until now was only corroborated by descriptive observations, correlative analyses, univariate analyses of variance, and a single multivariate but nonparametric approach (Netter et al., 2000), was tested by means of structural equation modeling. It was the aim to establish a path model based on Nyborg's theoretical considerations which allows us (i)

<sup>1</sup>The multiple group analysis was calculated with the model obtained in the young sample to warrant the comparability of all possible paths across both samples. The model of the old sample was nested in the one of the young veterans.

to test the assumptions of the androtype model, (ii) to quantify the direct and indirect influences of testosterone on body build, socioeconomic variables (such as education and income), and crystallized intelligence, and (iii) to verify the findings of Netter et al. (2000) that age is a salient mediator of these relationships, by testing whether the structure of the model varies across different age groups.

A structural equation model reflecting the GTC model was established and tested in a sample of 4375 male US military veterans. The data fit the model and reveal that by means of testosterone levels and fluid intelligence, which both have a strong genetic determination (Rotter et al., 1985; Carroll, 1984), 41% of the variance of crystallized intelligence, 15% of the variance of income, 14% of the variance of the body mass index, and 8% of the variance of education could be explained. The total effects of testosterone and of fluid intelligence on education, income, and crystallized intelligence were all significant. In addition to partly confirming the relationships postulated in Nyborg's model and known from correlational analyses (Sandberg & Barrick, 1995; Rovet, 1983; Olweus et al., 1988; Dabbs et al., 1990; Ukkola et al., 2001; Tsai et al., 2000; Holmes et al., 1984, 1985; Gold, 1978), the model revealed that most of the influence of testosterone on crystallized intelligence is exerted by indirect effects. It turned out that some of the indirect effects on the endogenous variables were larger than the direct effects. However, a negative relationship between intelligence and BMI proposed by Nyborg (1994) could not be confirmed. BMI also does not mediate the effects of testosterone on education or on crystallized intelligence, and there is also no direct effect from BMI to education as suggested by Nyborg's model.

The initial model derived from the total sample could be confirmed in the group of the young and of the old veterans as well, indicating that the findings by Netter et al. (2000) that age is an important mediator in the model have to be interpreted with caution. An explanation for the different results of the present study and the study by Netter et al. (2000) with respect to the influence of age in the GTC model is obviously the completely different mathematical foundation of the methods used. Configurational nonparametric methods are completely different from the SEM approach with respect to scale level, prerequisites and data model. Nevertheless, SEM certainly suits the GTC model better because the theory explicitly stresses the covariant influences between its parameters and path analysis is based on covariances. Furthermore, SEM is able to detect causal relationships between variables which CFA is not. Nevertheless, model trimming revealed more parsimonious models in both age groups than in the total sample. A direct influence from TESTO to INCOME could not be detected neither in old or in young veterans. Obviously this association is only significant in the total sample because of its greater statistical power. Moreover, in the sample of the old veterans testosterone has no direct influence on crystallized intelligence, whereas in the young sample this direct relationship was observed. In old veterans crystallized intelligence is only indirectly influenced, via education.

Also the multiple-group analysis indicated that the importance of education increases for older subjects. On the one hand the influence of fluid intelligence on education is stronger, and on the other hand the influence of fluid intelligence on crystallized intelligence is increased through education. The reasons for this mediator effect of education on crystallized intelligence remain unclear. A possible explanation would be that it is a cohort effect which would imply that in the cohort of the younger sample other factors apart from school education contribute to the development of crystallized intelligence. This raises another problem of the study presented. Nyborg's theory assumes developmental processes to be responsible for the influence of fluid intelligence and

testosterone on crystallized intelligence and income. The data presented is not longitudinal and therefore developmental processes could not virtually be proved, but given the fact that the exogenous variables testosterone and fluid intelligence are rather stable over time (because they are to a great extent genetically determined (Rotter et al., 1985; Carroll, 1984)) it could be assumed that the associations detected by the SEM model in adulthood are evidence for a covariation over time between testosterone and fluid intelligence on the one hand and environmental factors on the other hand. Not all possible environmental factors are included in the model as endogenous variables and only some possible mediators such as aggression or early onset of puberty are mentioned in the introduction (although their influence could not be investigated due to limitation of the data available) because they are implied by testosterone. However, SEM allows to quantify the amount of variance of the endogenous variables explained by the model and the disturbances ( $\zeta$ ) can be interpreted as the variance explained by variables not included in the model. This unexplained variance is in terms of multivariate statistics not very high (e.g. 59% for crystallized intelligence) but nevertheless indicates that further investigations are necessary to completely reveal the process by which the outcome variables are influenced.

In summary, it may be stated that Nyborg's GTC model could be largely confirmed by structural equation modeling with respect to the overall fit of the statistical model. Only the proposed association between BMI and intelligence could not be corroborated. Yet, it has to be pointed out that many direct associations between the exogenous and the endogenous variables are far from being impressive.

Although the influence of testosterone on education, income, and crystallized intelligence is small in absolute size, the effect seems to be stable (Dabbs & Morris, 1990) and therefore should not be ignored when studying the covariant development of body build, intelligence, and social and economic achievement. Bivariate relationships reported in the literature between testosterone and BMI (Ukkola et al., 2001; Tsai et al., 2000) and between testosterone and intelligence (Nyborg, 1997) could be confirmed. However, relationships between body build and intelligence (Holmes et al., 1984, 1985; Gold, 1978) could not be corroborated. The GTC model combines biological and environmental factors for predicting cognitive development and achievement and therefore could be recognized as an important contribution to personality psychology, which could be considered as an improvement to traditional constitutional theories (e.g. Kretschmer, 1921; Sheldon et al., 1940), which based their typologies mainly on morphology neglecting environmental variables and which never related biological variables to intelligence.

## REFERENCES

- Bollen, K. A. (1989). *Structural equations with latent variables*. New York: Wiley.
- Carroll, J. B. (1984). Raymond B. Cattell's contribution to the theory of cognitive abilities. *Multivariate Behavioral Research*, 19, 300–306.
- Cattell, R. B. (1971). *Abilities: Their structure, growth and action*. Boston, MA: Houghton-Mifflin.
- Centers for Disease Control. (1988). Health status of Vietnam veterans. *Journal of the American Medical Association*, 259, 2701–2719.
- Chou, C. P., & Bentler, P. M. (1995). Estimates and tests in structural equation modeling. In R. H. Hoyle (Ed.), *Structural equation modeling* (pp. 37–55). Thousand Oaks, CA: Sage.
- Dabbs, J. M., Jr., de La Rue D., & Williams, P. M. (1990). Testosterone and occupational choice: actors, ministers, and other men. *Journal of Personality and Social Psychology*, 59(6), 1261–1265.

- Dabbs, J. M., Jr., & Morris, R. (1990). Testosterone, social class, and antisocial behavior in a sample of 4462 men. *Psychological Science, 1*, 209–211.
- Gold, R. F. (1978). Constitutional growth delay and learning problems. *Journal of Learning Disabilities, 11*(7), 427–429.
- Holmes, C. S., Karlsson, J. A., & Thompson, R. G. (1985). Social and school competencies in children with short stature: longitudinal patterns. *Journal of Developmental and Behavioral Pediatrics, 6*(5), 263–267.
- Holmes, C. S., Thompson, R. G., & Hayford, J. T. (1984). Factors related to grade retention in children with short stature. *Child Care, Health and Development, 10*(4), 199–210.
- Horn, J. L. (1985). Remodeling old models of intelligence. In B. B. Wolman (Ed.), *Handbook of intelligence: Theories, measurements, and applications* (pp. 267–300). New York: Wiley.
- Horn, J. L., & Cattell, R. B. (1966). Refinement and test of the theory of fluid and crystallized general intelligences. *Journal of Educational Psychology, 57*(5), 253–270.
- Hoyle, R., & Panter, A. (1995). Writing about structural equation models. In R. H. Hoyle (Ed.), *Structural equation modeling, concepts, issues and applications* (pp. 158–176). Thousand Oaks, CA: Sage.
- Hu, L. T., Bentler, P. M., & Kano, Y. (1992). Can test statistics in covariance structure analysis be trusted? *Psychological Bulletin, 112*, 351–362.
- Jöreskog, K. G. (1993). Testing structural equations models. In K. A. Bollen, & S. J. Long (Eds.), *Testing structural equations models* (pp. 295–316). Newbury Park, CA: Sage.
- Jöreskog, K. G., & Sörbom, D. (1993). *LISREL8 user's guide*. Chicago, IL: Scientific Software International.
- Jöreskog, K. G., & Sörbom, D. (1996). *PRELIS 2 user's reference guide: A program for multivariate data screening and data summarization; a preprocessor for LISREL*. Chicago, IL: Scientific Software International.
- Kelloway, E. K. (1998). *Using LISREL for structural equation modeling: A researcher's guide*. Thousand Oaks, CA: Sage.
- Kline, R. B. (1998). *Principles and practice of structural equation modeling*. New York: Guilford.
- Krauth, J. (1993). *Einführung in die Konfigurationsfrequenzanalyse (KFA)*. Weinheim: Beltz.
- Krauth, J., & Lienert, G. A. (1973). *KFA—Die Konfigurationsfrequenzanalyse und ihre Anwendung in Psychologie und Medizin*. Freiburg: Alber.
- Kretschmer, E. (1921). *Körperbau und Charakter*. Berlin: Springer.
- Mackintosh, N. J. (1998). *IQ and human intelligence*. Oxford: Oxford University Press.
- Muldoon, M. F., Ryan, C. M., Matthews, K. A., & Manuck, S. B. (1997). Serum cholesterol and intellectual performance. *Psychosomatic Medicine, 59*(4), 382–387.
- Netter, P., Toll, C., Rohrmann, S., Hennig, J., & Nyborg, H. (2000). Configural frequency analysis of factors associated with testosterone levels in Vietnam veterans. *Psychologische Beiträge, 42*, 504–514.
- Nyborg, H. (1979, June). Sex chromosome abnormalities and cognitive performance V: female sex hormone and discontinuous cognitive development. In *Cognitive studies*. Symposium conducted at the Fifth Biennial Meeting of the International Society for the Study of Behavioural Development, Lund.
- Nyborg, H. (1983). Spatial ability in men and women: review and theory. *Advances in Human Research and Theory, 5*, 39–140.
- Nyborg, H. (1984). Performance and intelligence in hormonally different groups. In G. Vries, J. Bruin, H. Uylings, & M. Corner (Eds.), *Sex differences in the brain: Progress in brain research* (pp. 491–508). Amsterdam: Elsevier.
- Nyborg, H. (1987). Individual differences or different individuals? That is the question. *The Behavioral and Brain Sciences, 10*, 34–35.
- Nyborg, H. (1988). Sex hormones and covariant body, brain and behavioural development. *Neuroendocrinology Letters (Abstracts), 10*, 217.
- Nyborg, H. (1990). Good, bad and ugly questions about heredity. *Behavioral and Brain Sciences, 13*, 142–143.
- Nyborg, H. (1994). *Hormones, sex, and society: The science of psychology*. Westport, CT: Praeger.
- Nyborg, H. (Ed.). (1997). *The scientific study of human nature: Tribute to Hans J. Eysenck at eighty*. Amsterdam: Pergamon–Elsevier.

- Nyborg, H., & Jensen, A. R. (2000). Testosterone levels as modifiers of psychometric *g*. *Personality and Individual Differences*, 28(3), 601–607.
- Nyborg, H., & Jensen, A. R. (2001). Occupation and income related to psychometric *g*. *Intelligence*, 29(1), 45–55.
- Olweus, D., Mattson, A., Schalling, D., & Low, H. (1988). Circulating testosterone levels and aggression in adolescent males: a causal analysis. *Psychosomatic Medicine*, 50(3), 261–272.
- Rotter, J. I., Wong, E. L., Lifrak, E. T., & Parker, L. N. (1985). A genetic component to the variation of dehydroepiandrosterone sulfate. *Metabolism Clinical and Experimental*, 34(8), 731–736.
- Rovet, J. F. (1983). Cognitive and neuropsychological test performance of persons with abnormalities of adolescent development: a test of Waber's hypothesis. *Child Development*, 54(4), 941–950.
- Sandberg, D. E., & Barrick, C. (1995). Endocrine disorders in childhood: a selective survey of intellectual and educational sequelae. *School Psychology Review*, 24(2), 146–170.
- Sheldon, W. H., Stevens, S. S., & Tucker, W. B. (1940). *The varieties of human physique*. New York: Harper.
- Swaminathan, H., & Algina, J. (1978). Scale freeness in factor analysis. *Psychometrika*, 43(4), 581–583.
- Tsai, E. C., Boyko, E. J., Leonetti, D. L., & Fujimoto, W. Y. (2000). Low serum testosterone level as a predictor of increased visceral fat in Japanese–American men. *International Journal of Obesity and Related Metabolic Disorders*, 24(4), 485–491.
- Ukkola, O., Gagnon, J., Rankinen, T., Thompson, P. A., Hong, Y., Leon, A. S., Rao, D. C., Skinner, J. S., Wilmore, J. H., & Bouchard, C. (2001). Age, body mass index, race and other determinants of steroid hormone variability: the HERITAGE Family Study. *European Journal of Endocrinology*, 145(1), 1–9.
- West, S. G., Finch, J. F., & Curran, P. J. (1995). Structural equation models with non-normal variables: problems and remedies. In R. H. Hoyle (Ed.), *Structural equation modeling* (pp. 56–75). Thousand Oaks, CA: Sage.
- Zmuda, J. M., Cauley, J. A., Kriska, A., Glynn, N. W., Gutai, J. P., & Kuller, L. H. (1997). Longitudinal relation between endogenous testosterone and cardiovascular disease risk factors in middle-aged men: a 13-year follow-up of former multiple risk factor intervention trial participants. *Journal of American Epidemiology*, 146(8), 609–617.

Copyright of European Journal of Personality is the property of John Wiley & Sons Inc. and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.

Copyright of European Journal of Personality is the property of John Wiley & Sons, Inc. and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.