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Subjective well-being. Sex-specific effects of genetic and environmental factors

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Abstract

Genetic and environmental factors in subjective well-being were investigated. Special attention was paid to sex-specific effects. A classical twin design was applied, based upon data from 5140 young adult twins (same and opposite-sex). Structural equation modelling was used to estimate effects from additive genes, genetic interaction, common environment and non-shared environment. The best fitting model involved an additive genetic factor and non-shared environment. There were only marginal sex differences in mean levels of subjective well-being, and no differences in variance. Yet, there were sex differences in the degree of heritability (0.54 for women and 0.46 for men). Additionally, the correlation between the male and female genetic factors was 0.64, suggesting that, in part, different sets of genes may influence variation in subjective well-being for men and women. A dual mechanism process of genetic influence upon well-being is proposed, comprising the notion of interaction between genotypes and cultural value systems. © 2001 Elsevier Science Ltd. All rights reserved.

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What is happiness, and where does it originate? Such questions represent core issues in research on well-being, a field that has flourished in recent years (Diener, Suh, Lucas & Smith, 1999; Kahneman, Diener & Schwarz, 1999; Kwan, Bond & Singelis, 1997; Veenhoven, 1996). Under this general theme, several constructs have been studied, including quality of life, happiness, life satisfaction, subjective well-being, optimism and hope. Although not identical in content, these different constructs have in common a focus on positive characteristics and experiences, rather than the mere absence of psychological problems.

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Subjective well-being (SWB) is typically conceived of as comprising a cognitive component of life satisfaction and two affective components, namely the presence of positive affect and absence of negative affect (Diener & Suh, 1997; Diener, Suh, & Oishi, 1997; Pavot, Diener, Colvin & Sandvik, 1991). A number of correlates and potential causes of subjective well-being have been identified, for example friendship and social activity (Harlow & Cantor, 1996), marriage (Mastekaasa, 1993, 1994), perceived health (Brief, Butcher, George & Link, 1993; Harris, Pedersen, Stacey & McClearn, 1992), flow-experiences (Csikszentmihalyi & LeFevre, 1989), and goal accomplishments (Brunstein, 1993; Diener & Fujita, 1995). In contrast, factors such as material goods and income contribute very little to subjective well-being (Diener et al., 1999; Veenhoven, 1994). However, one common limitation of many studies so far is the regular cross-sectional design typically being applied. In a comprehensive state-of-the-art review, Diener et al. (1999) suggest causality to be one central topic for future research, and call for designs that apply more sophisticated methodologies in order to identify the causes and effects of SWB.

Recent studies have shown that SWB operates in both a top-down and a bottom-up fashion. That is, well-being partly results from a general tendency to hold positive life-views and partly the cumulative effect of specific events (e.g. Brief et al., 1993; Feist, Bodner, Jacobs & Miles, 1995; Headey, Veenhoven & Wearing, 1991; Mallard, Lance & Michalos, 1997; Vittersø, 1998). Moreover, Suh, Diener, and Fujita (1996) have shown that people tend to stabilise on a certain level of SWB, regardless of the daily events they face. Even after dramatic life events, such as winning a lottery or going through serious illness, within a few months people tend to return to their typical level of SWB.

The notion of a top-down process, and the finding of a return-to-the-baseline tendency, imply a general and stable disposition of holding a favourable, or not so favourable, life view.

To the extent that SWB operates as a relatively stable personality characteristic, it seems reasonable to hypothesise that genetic factors affect this characteristic. Given that genetic effects have been found for personality traits such as extraversion and neuroticism, and psychological problems such as anxiety and depression (Bergeman, Chipuer, Plomin & Pedersen 1993; Plomin, DeFries, McClearn & Rutter, 1997), it appears reasonable to expect a similar mechanism for SWB.

However, research on heritability and environmental effects for SWB is scarce. Based on correlations in twin data, Lykken and Tellegen (1996) have estimated that genes explain 40% of the variance in SWB, and when considering only the stable variance of SWB over time the heritability is estimated to be 80%. Moreover, they found a substantial correlation between monozygotic (MZ) twins, but the corresponding correlation between dizygotic (DZ) twins was negligible. This finding was interpreted to indicate that the genetic effect is non-additive rather than additive. That is, the genetic component of SWB seems not to be a simple summed effect of a set of specific genes, rather, interactions arising from a constellation of genes are the mechanisms of genetic expression. However, their interpretation that non-additive effects (genetic interaction) represents the major source of total genetic variance in SWB is somewhat at odds with findings of additive components as the primary genetic factors in personality traits, psychological problems and attitudes (e.g. Plomin et al., 1997).

The other published studies addressing heritability and SWB are typically based on samples of elderly people. Using data from the Swedish Adoption/Twin Study of Aging (SATSA) Harris et al. (1992) found that genes explains 48% of the variance in life satisfaction among people older than 65. Furthermore, Bergeman, Plomin, Pedersen and McClearn (1991) also analysed the

SATSA sample, including all respondents 50 years and older, and found a common genetic factor influencing both life satisfaction, depression and perceived support. These important findings, however, raise the question of whether similar or different mechanisms operate among young adults.

An important general issue in research on SWB concerns the potentially different etiologies of the phenomenon for women and men. Although mean levels of SWB do not seem to vary much between men and women (Diener et al., 1999; Veenhoven, 1996), several studies have shown sex differences in the predictors and potential mechanisms involved in generating and sustaining SWB. For example, the effect of life events upon SWB seems to be different for men and women (Forest, 1996; French, Gekoski & Knox, 1995), the relationship between personality traits and SWB differs across sexes (Vittersø, 1998), and there are sex differences in the association between marital satisfaction and relationship awareness (Acitelli, 1992).

Despite the accumulating knowledge regarding different sources of happiness for men and women, there appear to be no published studies focussing on potential differences in heritability for SWB across sexes. Given that there are genetic and environmental components in subjective well-being, it is possible that the effects of these components differ in magnitude in females and males. Moreover, it is also possible that the genes influencing SWB are partly different across sexes. Such findings have been reported for other psychological phenomena, including neuroticism, anxiety and reading performance (Alarcon, DeFries & Fulker, 1995; Eaves, Heath, Neale, Hewitt & Martin, 1998; Tambs, Harris & Magnus, 1995). Furthermore, given that SWB represents an evaluation of one's life according to one's own standards and values (e.g. Pavot & Diener, 1993), and that partly different values and ideals prevail among women and men (e.g. Larsen & Cutler, 1996), it may be hypothesised that partly different genetic factors influence SWB for women and men. That is, if physical and psychological features — which are partly genetic — are valued differently for men and women, there is reason to expect partly sex-specific sources of happiness. Yet, in general it seems fair to say that, at present, the ways in which potential sex-specific genetic factors operate are poorly understood.

The aims of the present study were to: estimate the contribution of additive genetic factors, genetic interaction, common environment, and non-shared environment, to variation in subjective well-being in young adults; to investigate sex differences in the relative magnitude of genetic and environmental effects; and to investigate whether the same or partly different genes were involved in generating SWB for men and women.

1. Methods

1.2. Sample

The data were collected in Norway in 1992. Based on the Norwegian Birth Registry, comprising all births since 1 January 1967, a total of 3996 like- and unlike-sexed twin pairs aged 18–25 were identified and were mailed a questionnaire. Responses were obtained from 2570 (of which 2562 had valid responses on the measures studied) complete pairs (64%) and 724 twins for which their co-twin did not respond, altogether 5864 individuals (74%). Zygosity was determined on the basis of seven questions previously validated to categorise correctly more than 97% of the twins

in another Norwegian study (Magnus, Berg & Nance, 1983). For a more elaborate description of the sample and classification procedure see Harris, Tambs and Magnus (1995).

In order to check for volunteer bias, we tested for differences between initial responders and responders to the reminder. If non-responders were in fact systematically different from responders, a tendency of differences between initial responders and reminder-needing responders would be expected. However, there was no difference between these two groups in mean level of SWB, and, more importantly, there was no difference in the variance of SWB. Secondly, we did a similar test between the twins for which both individuals responded (complete pairs) and the single twin responders. There was a small, but significant, difference in mean SWB, however, again there was no difference in the variance of SWB. Since our study focuses on variances and covariances of SWB, rather than levels of SWB, these findings represent empirical indications that volunteer bias does not seem to be a threat to the validity of the results.

1.3. Measurement

Subjective well-being was measured by a short version of a SWB-scale developed by Moum, Naess, Sorensen and Tambs (1990), which has been shown to have good psychometric properties. The present SWB-index was constructed using a sum-score of four items: (a) ''When you think about your life at present, would you say you are mostly satisfied with your life, or mostly dissatisfied?'' Six response categories were used, ranging from 1 = 'extremely satisfied' to 6 = 'very dissatisfied'. (b) ''Are you usually happy or dejected?'' The five response categories ranged from 1 = 'dejected' to 5 = 'happy'. (c) ''Do you mostly feel strong and fit or tired and worn out?'' The four response categories ranged from 1 = 'very strong and fit', to 4 = 'tired and worn out'. (d) ''Over the last month, have you suffered from nervousness (felt irritable, anxious, tense, or restless)?'' The four response categories ranged from 1 = 'almost all the time', to 4 = 'never'. Item (a) and (c) were reversed prior to construction of the index, so that a high score reflects a high degree of SWB. The index was based on the mean score of the four items.

The index comprised both a cognitive aspect (i.e. life satisfaction), as well as positive affect (i.e. happy, strong) and negative affect (i.e. tired, nervous), and thus conforms to generally accepted operationalisations of global subjective well-being (see e.g. Diener et al., 1999). Moreover, the index correlated 0.995 with a corresponding index based on standardised items, and 0.998 with a factor-score index. A principal component analysis of the four items yielded a clear uni-dimensional solution (eigenvalue: 2.1), with all items loading above 0.70 on this factor. When testing for a second factor, the eigenvalue was only 0.7, thus leaving us with one single dimension. Cronbach's α for the index was 0.71. As an additional validation the SWB index was included in a pilot-study among students at the University of Oslo ($n = 99$), and correlated 0.73 ($r = 0.88$, when latent variables were modelled) with the widely used Satisfaction With Life Scale (Pavot & Diener, 1993; Pavot et al., 1991).

Since the present study partly focuses on sex-differences, it was also important to check whether the psychometric properties were invariant across sex. There were no sex-differences in variance, and the internal reliability was 0.71 for women and men alike. Moreover, a two-group confirmatory factor analysis in which the factor loadings were constrained to be equal across sex, yielded good fit, relaxing of these constraints did not improve the fit, and it was concluded that there were no significant sex-differences in factor structure.

1.4. Analyses

Structural equation modelling (SEM) was used to estimate the genetic and environmental parameters and to test the relative fits of alternative models. These models are widely used to analyse twin data and are described in detail elsewhere (e.g. Neale & Cardon, 1992). Briefly, the associations between scores for identical and fraternal twins can be represented by a set of structural equations specifying how the genetic and environmental factors contribute to the twin covariances. The free parameters of the model are the paths from the hypothesised latent variables (e.g. the additive genetic factor, or the common environment factor) to the observed phenotypic measure of SWB. The degree of biological relatedness is represented as constraints imposed on these parameters as a function of zygosity. Estimation of the relative contribution of the different latent factors allows us to determine whether (1) the phenotypic measures of SWB are familial or random, and (2), if familial, the relative contribution of genetic and common environmental effects to familial aggregation. By resolving the covariance equations the model yields estimates of genetic and environmental variance parameters.

For example, Fig. 1 depicts a basic twin covariance model and includes five general sources of influence: additive genetic effects (A), genetic dominance (D), genetic epistasis (I), common environmental effects (C) and non-shared environmental effects (E).

Identical (MZ) twins share all their genes, and fraternal (DZ) twins share, on average, 50% of their segregating genes, therefore the latent factor, A, is specified to be perfectly correlated in MZ twins, and correlated 0.5 in DZ twins. Dominance refers to intra-locus genetic interaction, and D-factors will be perfectly correlated in MZ-twins and correlated at 0.25 in DZ-twins. Epistasis represents inter-locus genetic interaction. In the extreme form, epistasis is termed emergenesis, implying the emergent effect from the total constellation of several genes. Emergenesis can be

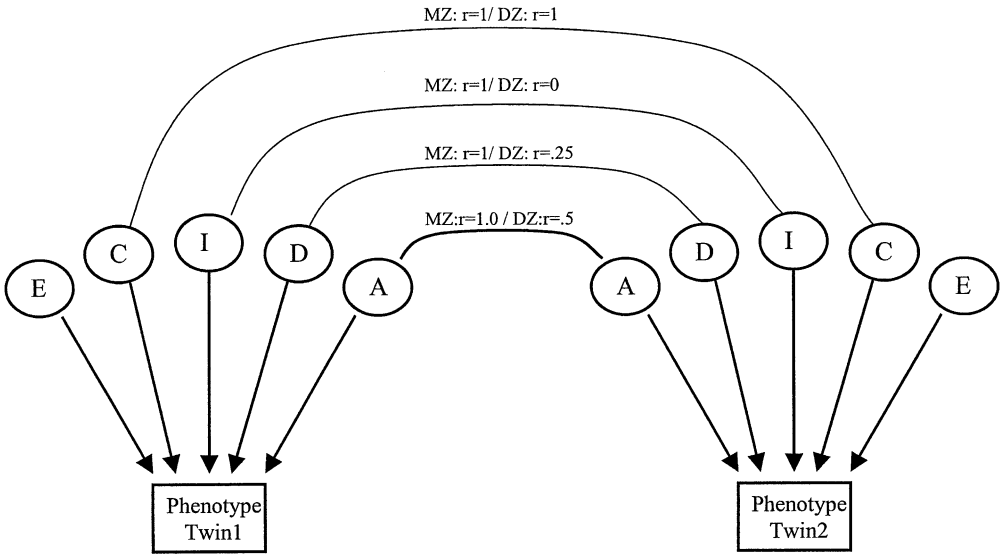


Fig. 1. Basic twin model comprising latent additive genetic factors (A), dominance (D), epistasis (I), common environment (C) and non-shared environment (E).

conceived of as the 'phone-number' situation, in which a change in a single digit can totally change the content of the number. Thus, the epistasis (I) factor is perfectly correlated in MZ-twins, and uncorrelated in DZ-twins. (For elaborations of non-additive genetic effects, see e.g. Beer, Arnold & Loehlin, 1998; Lykken, McGue, Tellegen & Bouchard, 1992; Lykken & Tellegen, 1996; Plomin, Corley, Caspi, Fulker & DeFries, 1998).

C refers to environmental effects that by definition are shared by twins and is, therefore, perfectly correlated between twins in both zygosity groups. E represents non-shared environmental effects that do not contribute to pair resemblance, and these effects are uncorrelated for all pairs. Moreover, as depicted here, the five components (i.e. A, D, I, C and E) are mutually orthogonal.

Some limitations in the testing of such models should be noted. Due to issues of identification and confounding, only three kinds of latent factors should be included in a single analysis, implying that an ACIE model would not be testable. Therefore, alternative models are tested separately (e.g. ACE and AIE), and the relative fits are compared to find the model which best explains the data. Moreover, although classical twin analyses may provide evidence that non-additive genetic effects are important, resolution of the specific type of interaction (I or D) involved requires other kinds of data than available in the present study.

Earlier studies of personality have typically focussed on the effects of dominance, rather than those of epistasis. However, the one published SWB study addressing genetic interaction (i.e. Lykken & Tellegen, 1996) has argued in favour of emergenesis. Several other recent studies of personality also provide evidence of epistatic interactions (Eaves et al., 1998; Plomin et al., 1998). Thus, we decided to focus on models in which the interaction factor was defined as an epistatic (I) latent variable. Yet, it should be noted that analyses were also conducted in which the I factor was replaced with a D factor, and the results were highly similar, both with regard to the effects of the different factors and the fits of the models.

The model may also be expanded to test for several different types of sex-specific effects (e.g. Neale & Cardon, 1992). Under the *common sex-limitation* model, the same genes contribute to variation in the phenotype under study, however, the magnitude of the genetic and environmental effects is allowed to vary across sexes. The *general sex-limitation* model allows for differences in the genetic sources and in the magnitude of estimates of variance for men and women. Testing the general sex-limitation model requires samples of MZ and DZ twin pairs of both sexes, plus DZ twins of unlike sex. Evidence for sex-specific genetic effects is indicated when the co-twin correlation for unlike-sex pairs is less than that of the like-sex DZ pairs. It is also possible that there are sex-specific environmental effects. Thus, the sex-limitation models were also specified to test whether such effects may be important.

For the modelling procedures the data were summarised into twin-cotwin variance-covariance matrices for the five groups of twins, that is MZ-male, DZ-male, MZ-female, DZ-female, and DZ-unlike-sex. A series of models was tested, comprising different combinations of A, I, C and E. Typically, when testing for genetic interaction effects, the additive genetic factor is also included in the model. However, based on the findings of Lykken and Tellegen (1996), of only negligible additive genetic effects in SWB, we also tested models including an I-factor but no A-factor. Model fit was evaluated by the χ^2 statistic, in addition to the Root Mean Square Error of Approximation (RMSEA) and Akaike's Information Criterion (AIC). To test for the significance of specific parameters, the χ^2-difference test for nested models was used.

2. Results

Descriptive statistics and intraclass correlations for the five subsamples (i.e. MZM, MZF, DZM, DZF, and DZU) are shown in Table 1.

Men scored, on average, higher than women ($P < 0.001$), and MZ twins scored higher than DZ twins ($P < 0.001$). However, neither sex nor zygosity explained more than 1% of the variance in SWB. There were no differences in variances across sex or zygosity groups.

The pattern of intraclass correlations across the sex and zygosity groups revealed greater similarity among MZs compared to DZs, and among the like-sexed compared to the unlike-sexed DZ pairs. This suggests that genetic effects are important, and that sex-specific effects may exist.

Results from the modelling analyses investigating the effects of additive genetic effects (A), genetic interaction (I), common environment (C) and non-shared environment (E) are listed in Table 2.

In total, 16 models were tested. These models fall within three general categories, that is I: *no sex-effect models* in which effects are constrained to be invariant across sex (models 1–5), II: *common sex-limitation* models allowing the magnitude of effects to differ for men and women, yet constraining the additive genetic factor and the common environment factor to be common for both sexes (models 6–10), and III: *general sex-limitation* models allowing the relative magnitude of effects to vary across sex and estimating the correlation between the female and male genetic factor (models 11a,12,15), or common environment factor (models 11b, 13, 14). Thus, the three categories of models are ordered from the most to the least constrained.

The models in category I yielded bad to moderate fits. The CE model, which did not include any genetic factor, clearly did not fit the data. The AE model is nested under the ACE model, but inclusion of the C parameter (ACE) did not yield improvements of fit as compared to the AE model. In contrast, models including non-additive genetic effects (AIE, ICE) fitted better than the AE model. However, in summary, the models assuming equal effects for men and women were not able to account for the observed data.

The models in category II relaxed the constraint specifying equal magnitudes of the parameter estimates across sex. These models tended to fit better than did the corresponding models under category I, thus suggesting sex-differences in the magnitudes of genetic and environmental effects. Again, the CE model yielded poor fit, the other models fitted the data to an acceptable degree. The ACE model yielded an effect from common environment for males but not for females. However, this model did not yield significantly better fit than did the nested AE model

Table 1
Descriptive statistics and intraclass correlations for the Subjective Well-Being index

	Mean	S.D.	*r*	No. of twins
MZ[a] — male	3.77	0.60	0.46	828
MZ — female	3.67	0.57	0.53	1054
DZ[b] — male	3.69	0.60	0.22	774
DZ — female	3.59	0.60	0.23	882
DZ — unlike-sex	3.64	0.59	0.15	1586

[a] Monozygotic.
[b] Dizygotic.

($\Delta\chi^2 = 3.41$, d.f. = 2, n.s.). The other three-factor models, involving non-additive genetic effects (AIE, ICE) tended to yield the best fit, however, a formal test between the AIE and AE models did not provide evidence for a significantly better fit for the AIE model ($\Delta\chi^2 = 5.41$, d.f. = 2, n.s.).

Relaxing the constraint of the same genes, or the same common environment, across sex in category III, generally lead to further improvements of the model fits. Two different ACE-models were tested, that is, model 11a involving estimation of the correlation between the A_m and A_f factors, and model 11b involving estimation of the correlation between the C_m and C_f factors. Both of these ACE models yielded acceptable fit, but the parameter estimates for the effect of C were not significant. The CE model (14) again did not fit the data. The AIE and ICE models fitted well. However, the best fitting model was the more parsimonious AE-model, comprising only additive genetic effects and non-shared environment (model 15). In this model the correlation between the female and male genetic factor was estimated to 0.64. The AE-model yielded the lowest χ^2/d.f.-ratio, the highest *P*-value, the lowest RMESA value, and finally the lowest AIC value of all tested models. Moreover, allowing for different sets of genes in males and females (model 15) provided a significantly better fit than the model which only allowed the magnitude of

Table 2
Variance estimates, male-female correlations and goodness-of-fit statistics for models of Subjective Well-Being[a]

Sex effect	Model	Males				Females				Sex-correlation		Fit measures				
		a_m^2	i_m^2	c_m^2	e_m^2	a_f^2	i_f^2	c_f^2	e_f^2	r_a	r_c	χ^2	d.f.	*P*-value	RMSEA	AIC
I	1. ACE	0.49		0.00	0.51	0.49		0.00	0.51	1.0	1.0	24.05	12	0.02	0.020	0.05
None	2. AIE	0.39	0.12		0.49	0.39	0.12		0.49	1.0		18.73	12	0.10	0.015	−5.27
	3. ICE		0.31	0.20	0.49		0.31	0.20	0.49		1.0	18.73	12	0.10	0.015	−5.27
	4. CE			0.31	0.69			0.31	0.69		1.0	102.58	13	0.00	0.052	76.58
	5. AE	0.49			0.51	0.49			0.51	1.0		24.05	13	0.03	0.018	−1.95
II	6. ACE	0.29		0.14	0.57	0.54		0.00[b]	0.46	1.0	1.0	12.99	9	0.16	0.013	−5.01
Common	7. AIE	0.38	0.08		0.54	0.40	0.15		0.45	1.0		10.99	9	0.28	0.009	−7.02
sex-	8. ICE		0.27	0.19	0.54		0.35	0.20	0.45		1.0	10.99	9	0.28	0.009	−7.02
limitation	9. CE			0.26	0.74			0.35	0.65		1.0	96.80	11	0.00	0.055	74.80
	10. AE	0.44			0.56	0.53			0.47	1.0		16.40	11	0.13	0.014	−5.60
III	11a. ACE	0.46		0.00	0.54	0.54		0.00	0.46	0.58	1.0	9.61	8	0.29	0.009	−6.39
General	11b. ACE	0.37		0.08	0.55	0.52		0.02	0.46	1.0	−1.0	11.09	8	0.20	0.012	−4.90
sex-	12. AIE	0.46	0.00		0.54	0.46	0.09		0.45	0.69		8.66	8	0.37	0.006	−7.34
limitation	13. ICE		0.23	0.23	0.54		0.32	0.23	0.45		0.69	8.66	8	0.37	0.006	−7.34
	14. CE			0.35	0.65			0.39	0.61		0.86	64.96	10	0.00	0.046	44.96
	15. AE	0.46			0.54	0.54			0.46	0.64		9.67	10	0.47	0.000	−10.33

[a] RMSEA, Root Mean Square Error of Approximation; AIC, Akalke's Information Criterion. A, Additive genetic factor; I, Genetic interaction factor; C, common environment; E, Non-shared environment. Subscripts $_m$ and $_f$ refer to males and females, respectively. Reported effects are variance estimates (e.g. the values in the a_m^2 column represent percentage of variance explained by the male additive genetic factor).

[b] Refers to a case of negative parameter estimate (i.e. for c_f) and the subsequent constraint of zero as a lower bound. $P < 0.05$ for all parameter estimates > 0.10.

genetic and environmental influences to vary across sexes, that is model 10 ($\Delta\chi^2 = 6.73$, d.f. = 1, $P < 0.01$).

Fig. 2 shows the final model, with estimated path coefficients from the additive genetic factors and the non-shared environmental factors upon subjective well-being, and with the estimated correlation between the male and female genetic factor.

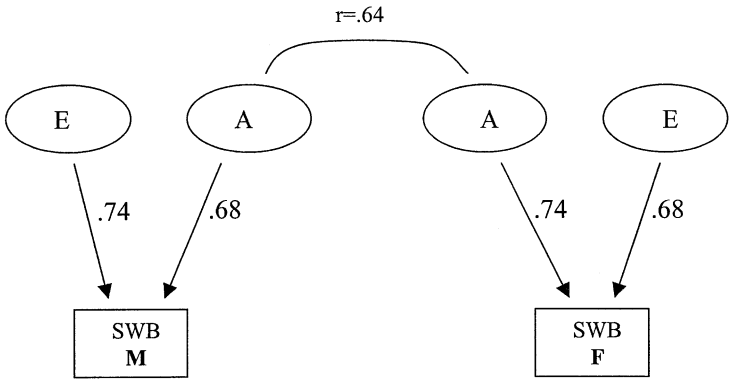


Fig. 2. Final model, comprising additive genetic factors (A) and non-shared environmental factors (E), and the male-female correlation for the genetic factor. (M, males; F, females).

3. Discussion

This study explored genetic and environmental factors in subjective well-being, and focussed specifically on sex-differences in such factors. Whereas numerous previous studies have identified different correlates and predictors of subjective well-being, little attention has been paid to the issue of genetic versus environmental contributions to human happiness. Individual variance in human phenotypes generally arise from four broad sources of influence, that is, additive genetic factors, genetic interaction, common environment and non-shared environment. We tested a number of alternative models comprising different combinations of these potential causes to investigate how these factors contribute to SWB.

For women and men alike, genetic and environmental factors contributed to variation in subjective well-being, and each of these components explained approximately half the variance. This finding is in accordance with the few previous studies reporting on SWB in twin samples (Bergeman et al., 1991; Harris et al., 1992; Lykken & Tellegen, 1996). It is important to note that the findings of heritability in SWB among elderly people seem to generalise to young adults as well. Moreover, the heritability estimates for SWB found here are similar in magnitude to those for typical personality traits (e.g. Bergeman et al., 1993; Eaves et al., 1998; Plomin, Scheier, Bergeman & Pedersen, 1992) supporting the notion of SWB as operating in a trait like fashion.

With regard to sex-specific effects, the present analyses yielded two potentially important findings. First, genes appeared to explain more of the variance in SWB in women than in men (i.e. 55 vs. 46%). Even though the difference is not very large, and could partly be attributed to sample characteristics, the finding fits with recent findings of similar differences in for example neuroticism (Eaves, Heath, Martin, Maes, Neale, Kendler, Kirk & Corey, 1999). Secondly, there appear

to be differences between the two sexes regarding the set of genes influencing SWB. This finding is congruent with previous reports of some differences in environmental predictors of SWB for women and men (e.g. Forest, 1996; French et al., 1995). It also fits with previous findings of sex-specific genetic effects on personality traits such as neuroticism (Eaves et al., 1998). It should be noted that in principle sex-specific effects (as indicated by lower correlations between opposite-sex DZ twins than between same-sex DZ twins) may be due to both genetic and environmental effects. However, given that we found no evidence of a common environment factor, the environmental explanation seems implausible in this case.

However, the notion of partly different genetic architecture involved in SWB for women and men, requires further elaboration and interpretation. This finding can be conceived of as reflecting a *dual mechanism process*. While it seems implausible that completely different genes directly influence the same characteristic in women and men, the sex-specific effects make sense in a framework involving both direct and indirect pathways. That is, a direct influence would imply a genetically based predisposition to hold a positive life view, corresponding to a top-down process (e.g. Brief et al., 1993; Feist et al., 1995). An indirect influence, however, refers to a genetically based tendency for characteristics that might influence SWB further down in the causal chain, possibly through a bottom up process. Given that SWB partly stems from an evaluation of the match between one's actual life and one's own standards and values (e.g. Pavot & Diener, 1993), and that to some extent different values and ideals are salient among women and men (e.g. Larsen & Cutler, 1996), the genotype of the individual might influence SWB through an interplay with the prevailing sub-cultural value systems. More specifically, if a certain culture posits that characteristic X is highly desirable for women, but not necessarily for men, those women genetically predisposed to have X might report higher levels of subjective well-being, partly because they hold an attribute that is congruent with what their gender culture proclaims to be valuable. Potential examples of X could be physical characteristics such as body shape, or different personality characteristics. Previous research on the sample of twins studied here indicated sex-specific genetic effects for body mass (Harris et al., 1995), thus possibly providing one specific example of a relevant characteristic.

The notion of a genetic dual mechanism process, as briefly outlined above, is proposed as a tentative explanation at this point. However, as a theoretical model it suggests that the genetics of subjective well-being can be conceived of as operating both by contributing to a general predisposition to hold a positive life-view, and by contributing to characteristics that are conducive to happiness given that they are valued by the acting culture or sub-culture. As such, it represents a possible theoretical account of the interplay between genetic factors and cultural value systems. Cantor and Sanderson (1999) have suggested that goal commitments are most conducive to happiness when the goals are valued by the culture or subculture to which the individual belongs, and Mallard et al. (1997) have found evidence of culture having a moderating effect upon the relationship between overall life satisfaction and satisfaction with specific life facets. We suggest that a number of human characteristics, such as accomplishments, traits, goals, attitudes, actions and physical attributes can be conducive to SWB, and will be particularly so when they are in accordance with the prevailing cultural and sub-cultural value systems. To the extent that these characteristics are genetically influenced, they represent variables for which the effect is partly moderated by the culture. This also implies that partly different genes might influence SWB in different cultures, and that as societies change — potentially involving changes in the value

systems shared by its members — the relative importance of different genes in affecting SWB might also change. Nevertheless, more conclusive evidence of the validity of the notion of a genetic dual mechanism process will only be provided by future research investigating the bi- and multivariate genetic relations between SWB and other characteristics. Another recommendation would be to conduct studies separating twins into discordant and concordant groups with regard to certain environmental exposures, thus enabling the study of gene-environment interaction.

So far, the discussion has focussed on interpretations and implications of the empirically best fitting model. Although this model did not include effects from genetic interaction and common environment, there are some potential caveats that require addressing. Whereas Lykken and Tellegen (1996) argued that virtually the entire effect of genes upon SWB is through a process of genetic interaction, our best fitting model did not support this. One interpretation of these apparently contrary findings is related to the SWB indices employed. Lykken and Tellegen (1996) used the Well Being scale of the MPQ, which primarily measures a facet of extraversion (Lykken et al., 1992). Thus, it is possible that this aspect of SWB is influenced by a non-additive genetic process to a higher extent than the aspect of SWB studied in the present project.

Another interpretation of our results can be provided by challenging the notion of additive genetic effects as primary to genetic interaction. That is, genetic interaction is generally modelled as a factor that operates in addition to, rather than instead of, additive genetic effects (Neale & Cardon, 1992). However, genetic interactions can theoretically operate regardless of the presence of additive effects, and it has been argued that additive genetic effects are negligible in SWB (Lykken & Tellegen, 1996). We tested models including a genetic interaction factor, while excluding the additive genetic factor, that is the ICE models. Under the sex-limitation conditions the ICE-models yielded an acceptable fit. In the ICE model only the C factor will contribute to DZ correlations, and in principle the model can account for the same data pattern as the AE model. Thus, with the present data set we cannot totally reject the notion of a genetic interaction factor, and only future studies with familial relationships beyond those of the classical twin design can yield more conclusive evidence regarding these issues in the SWB field.

Regarding other implications of the present findings in relation to the general literature on subjective well-being it should be noted that approximately 50% of the variance in SWB was explained by non-shared environment. Our study was not designed to identify the different environmental factors operating. Yet, a potentially important take-home implication would be that studies that report predictors of SWB which, in total, exceed this level of explained variance probably include variables that are also partly genetically based, and that the correlations between such predictors and SWB to some extent are due to a common genetic source. More importantly, future studies can test the contribution of genes and environment to the covariances between SWB and other constructs by incorporating such variables into multivariate models for twin and family data.

In summary, there were only marginal differences between women and men in mean level of SWB, and no difference in variance. However, we found sex differences with regard to the strength and character of the genetic factor. Genes explained more variation in subjective well-being among women than among men, or to put it the other way around, men were more influenced by their environment than were women. Moreover, there was evidence that, in part, different genes contribute to SWB for women and men. This finding can be interpreted as indicative of a dual mechanism process, in which genetic influence upon SWB operates both in a direct and

indirect manner. We suggest that the indirect path represents a causal chain which interacts with the values and ideals that are salient in the sociocultural context — for example gender culture — in which the individual is embodied.

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