Subjective Wellbeing and Sleep Problems: A Bivariate Twin Study

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The relationship between subjective wellbeing (SWB) and self-reported sleep problems was investigated in a cohort of Norwegian twins aged 18 to 31 years. Questionnaire data from 8045 same- and opposite-sex twins were analyzed using structural equation modeling to explore the relative effects of genetic and environmental influences on phenotypic variance and covariance. Special attention was paid to sex-specific effects. The correlation between the phenotypes was estimated to be –.43. Univariate analyses indicated considerable genetic influences for both SWB and sleep problems, for male and female twins alike. The best fitting bivariate model specified additive genetic and individual environmental factors for both phenotypes, and nonadditive genetic effects for sleep problems, with no sex-specific effects. Genetic and environmental effects accounted for 60% and 40% of the phenotypic correlation, respectively. Additive genetic factors affecting the two phenotypes were correlated (–.85), suggesting that part of the genetic effects that positively influence SWB also protect against sleep problems. In conclusion, the results indicate considerable overlap in genetic etiology for SWB and sleep problems, for males and females alike.

Experiencing good quality sleep is an important part of healthy functioning. However, as much as one third of the population commonly report problems sleeping (e.g., Heath, Eaves, et al., 1998). The prevalence is higher in females and older age groups (Ford & Kamerow, 1989), but even in young people 10% or more report problems sleeping (Levy et al., 1986). An estimated 60 to 70 million Americans suffer from some kind of sleep disturbance, costing about 150 billion dollars annually (National Sleep Foundation, 1993), and one third of Americans report having trouble staying awake during the day (National Sleep Foundation, 1997). In a Norwegian population sample (Statistics Norway, 2002), 13% of men and 22% of women report problems sleeping, representing a significant increase in sleep problems over the last 4 years.

Sleep problems are associated with quality of life (Kupfer, 1995), risk of accidents (Ohayon, 2002), work performance (e.g., Ohayon, 2002), and physical health (Kupfer, 1995). Evidence for a close relationship between sleep and mental illness is also well established (e.g., Ford & Kamerow, 1989; Kupfer, 1995; Pilcher et al., 1997). Sleep disturbance is strongly associated with most psychiatric disorders (Kupfer, 1995), and is considered the second most common symptom of mental distress (Heath, Eaves, et al., 1998). In mood disorders specifically, sleep disturbance plays a prominent role, and diagnostic criteria for depression have always included sleep complaints as a central characteristic (Perlis et al., 1997). About 80% of patients with major depression present sleep complaints (Reynolds & Kupfer, 1987), and individuals presenting sleep problems are more likely to report persistent or recurrent health problems or emotional distress (Heath, Eaves, et al., 1998). In general, good sleepers report better quality of life than mild or severe insomniacs (e.g., Léger et al., 2001). In the 1991 National Sleep Foundation Survey (US), 96% of respondents without insomnia complaints evaluated their life quality as excellent or good, as opposed to 81% and 70% with occasional and chronic insomnia.

Studies exploring the association between sleep disturbance and mental illness far outnumber studies investigating sleep and positive indicators of mental health. Since 1996, publications concerning sleep or sleep problems cited in Medline increased by more than 50% (Quan, 2003). Despite this increase, surprisingly few studies explored the relationship between sleep and mental health concepts such as subjective wellbeing (SWB), and the World Health Organization (WHO) consensus report on sleep and health (1998) strongly recommended more studies on the quality-of-life dimensions of sleeping problems.

Mental health is not merely characterized as the absence of illness, but is composed of various signs of wellbeing. Increasingly, the concepts of good mental

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health and SWB are used synonymously (Keyes & Lopez, 2002), and SWB is recognized as an important and additional source for evaluating or monitoring overall societal and economic development (Siegrist, 2003). SWB refers to people's subjective and multidimensional evaluations of their lives, and the construct embraces a cognitive component of life satisfaction as well as two affective components: the presence of positive affect and the absence of negative affect (e.g., Diener & Suh, 1997). These components capture distinct aspects of SWB, but are not entirely independent (Suh et al., 1996), and are assumed to reflect a single underlying dimension. The threefold structure of SWB is confirmed in a number of studies (Lucas et al., 1996) and appears to be moderately consistent across situations (Diener & Larsen, 1984) and the life span (Magnus & Diener, 1991). This stability partly reflects a general tendency to hold a positive outlook on life, and partly a cumulative effect of specific, positive life events, thus representing a top-down as well as a bottom-up process (e.g., Brief et al., 1993). Although life events influence SWB, most people eventually seem to adapt to changes and return to some kind of a biologically determined 'set-point'. This general stability of SWB has been partly attributed to substantial genetic influences (Lykken & Tellegen, 1996). To some degree, people seem to be born happy, not-so-happy or quite unhappy, or as Meehl (1975) points out, some people have more 'joy juice' than others, and seem to be 'born three drinks ahead'. Across studies, heritability estimates of SWB typically range from .25 to .55, and Lykken and Tellegen (1996) have calculated that genes explain approximately 80% of the variance in SWB over time, when considering the stable aspect of the construct. Thus, heritability seems to have a moderate influence on periodic fluctuations in SWB and considerable effect on the stable component of the construct.

From an evolutionary perspective, the association between sleep and negative emotionality is easily attributed to its obvious survival value, such as staying awake when threatened. The evolutionary aspects of positive emotions may be less obvious. A core hypothesis of positive health is that the experience of happiness and positive affect contributes to the effective functioning of biophysical systems, keeping the individual from disease, delaying morbidity, and helping the individual to maintain optimal functioning (Ryff et al., 2004). Thus, happiness may play an important adaptive role, for example, by signalling that one's environment is safe, and by facilitating availability of personal resources for activity coping with stressors, and for innovation (e.g., Fredrickson, 1998). The association may also partly reflect temperamental characteristics or personality factors associated with both sleep and SWB.

Twin surveys from Finland (Partinen et al., 1983) and Australia (Heath, Eaves, et al., 1998) have revealed more similar sleep patterns, sleep duration, and subjective sleep quality in monozygotic (MZ) than dizygotic (DZ) twins. Familial clustering is also documented in studies using electroencephalogram (EEG) recordings, polysomnography (PSG), investigating body movements, slow wave sleep and rapid eye movement (REM) density (Linkowski et al., 1991). Previous twin studies suggest that at least 33% of the variance in sleep quality and general sleep disturbance is accounted for by genetic factors (Heath et al., 1990; Hublin et al., 2001). To our knowledge, however, the way and the degree to which genetic and environmental factors explain the co-occurrence of sleep problems and SWB remains unexplored. Bivariate twin studies provide an excellent opportunity for exploring shared liability in co-occurring conditions (Neale & Kendler, 1995). The present study examines the correlated liabilities for SWB and self-reported sleep problems in a sample of young adult Norwegian twins, assuming co-occurrence to reflect pleiotropic effects of latent genetic and environmental risk factors. Sex-specific effects are investigated to address whether the same set of genes is associated with SWB and sleep disturbance, and to address whether there are sex differences in the magnitude of the genetic and environmental variance components.

Materials and Method

Sample

The Norwegian Institute of Public Health Twin Study is an ongoing longitudinal study with a cohort sequential design. The database includes data from all like- and unlike-sexed twins born in Norway between 1967 and 1979, where both twins survived to the age of 3 years and their current addresses could be obtained. Twins are identified through information contained in the Norwegian Medical Birth Registry. Questionnaire data were first collected in 1992 (Q1). In 1998, a second, greatly extended, questionnaire (Q2) was sent to all cohorts who received Q1 (twins born 1967 to 1974), plus to five new birth cohorts (twins born 1975 to 1979). Data for the present study come from Q2 and include responses from 8045 twins, comprising 3334 complete pairs and 1377 singletons, representing an individual response rate of 63% and a pairwise response rate of 53%. The mean age of the respondents was 25.52 (*SD* = 3.70). As zygosity information is not included in the Medical Birth Registry, zygosity assignment was based on questionnaire items, which have previously been shown to categorize more than 97% of the cases correctly (Magnus et al., 1983). Detailed description of the sample and zygosity determination is given elsewhere (Harris et al., 1995, 2002).

Measures

SWB was measured by a short version of the SWB scale developed by Moum et al. (1990). The index was constructed as a mean score (1–5) of the following four items: (1) 'When you think about your life at present, would you say that you are mostly satisfied with your life or mostly dissatisfied?' with six response categories, ranging from 1 = *extremely satisfied* to

6 = *very dissatisfied*; (2) 'Are you usually happy or dejected?' with five response categories, ranging from 1 = *dejected* to 5 = *happy*; (3) 'Do you mostly feel strong and fit, or tired and worn out?' with five response categories ranging from 1 = *very strong* and fit to 5 = *tired and worn out*; and (4) 'Over the last month, have you suffered from nervousness (felt irritable, anxious, tense or restless)?' with four response categories ranging from 1 = *almost all the time* to 4 = *never*. Thus, the index comprised a cognitive aspect (life satisfaction), positive affect (happy, strong) and negative affect (worn out, nervous), as such conforming to the generally accepted operationalization of global SWB. Item 1 and item 3 were reversed before the construction of the index, such that high scores reflect a high degree of SWB. Cronbach's α for the index was estimated to be .71. Further description of the index can be found elsewhere (Røysamb et al., 2002).

The sleep index comprises three items as follows: (1) 'Have you, or have you ever had sleep problems?' with two response categories, *yes* and *no*; (2) 'How often have you used sleep medicine the last month?' with four response categories ranging from 1 = *never* to 4 = *almost every night*; and (3) 'Over the last month, have you suffered from problems falling asleep or other sleep problems?' with four response categories ranging from 1 = *never* to 4 = *almost every night*. The index was constructed as a mean score of items 2 and 3, plus the score (0 or 1) on item 1, thus ranging on a scale from 1 to 5. Cronbach's α for the index was calculated based on polychoric correlations due to the ordinal and dichotomous nature of the items, and estimated to be .86.

To further validate the scales and test for measurement invariance with respect to gender, a multisample confirmatory two-factor analysis (CFA) on all questionnaire items (four SWB items, three sleep items), was conducted. All item loadings exceeded .65 on the respective factors, and the model yielded a good overall fit, χ^2 (41, N = 5292) = 205.970, RMSEA = .039. Modeling analyses revealed no significant sex differences, ($\Delta\chi^2_8$ = 17.43). The correlation between the latent factors (SWB, sleep problems) was estimated to be –.65.

Statistical Analyses

The two indices, both constructed as 1- to 5-point scales, were each polytomized into four categories due to the ordinal character of the sleep index. The estimates were obtained under a liability threshold model positing continuous normal liability distributions, with distinct thresholds superimposed. Application of raw data methods allows for testing of homogeneity of thresholds within pairs, and across zygosity and sex, and between complete and incomplete twin pairs. Significant differences in liability-thresholds between complete and incomplete pairs suggest a cooperation bias correlated with the target variables, given that familial/genetic factors are of significant etiologic importance for the symptoms studied (Neale & Eaves, 1993). To further test for possible attrition biases, threshold differences between twins responding at both data collections (Q1 and Q2) and twins responding only at Q2 were explored.

Estimates of polychoric correlations, factor analyses and biometric modeling were conducted, using the software package Mx (Neale et al., 1999). We calculated polychoric correlations for the five zygosity groups as initial estimates of the importance of genetic and environmental influences in liability.

Basic biometric modeling for twin data was used to decompose phenotypic variance into the relative effects of three broad causes of variation — genetic (G), shared environmental (C), and nonshared or individual environmental factors (E). The effects (*g, c, e*) are modeled as regression coefficients in a linear regression of measured variables on unobserved, latent sources of variance (Boomsma et al., 2002). The biometric model derives estimates of these effects from comparing data from MZ twins who are perfectly correlated for genetic effects with DZ twins who share, on average, 50% of their genes. Greater similarity between MZ compared to DZ twins is considered evidence of genetic influences. Heritability refers to the total part of the phenotypic variance attributable to genetic influences (*g*), and comprises both additive effects (*a*) of individual alleles at loci influencing a particular phenotype, and nonadditive (*d*) effects, reflecting interactions between alleles at the same locus (dominance) or between alleles across loci (epistasis). Generally, models including the nonadditive genetic component (D) are fit only when the ratio of MZ to DZ correlations exceeds 2.0 (Plomin et al., 1992). The shared environment includes environmental factors contributing to similarity between twins, whereas the nonshared or individual environment (E) refers to environmental factors contributing to differences between twins, plus random error variance. Thus, E is not estimated directly and constitutes the residual variance after the effects of *a, d* and *c* have been removed. When analyzing data from twins reared together, C and D are negatively confounded, and alternate models including either C or D may be tested. The full model (ACE or ADE) is compared to several nested submodels. As maximum likelihood (ML) analyses of raw ordinal data do not provide an overall test of goodness-of-fit directly, the relative fit of the nested submodels against the full model may be obtained using the χ^2 difference test ($\Delta\chi^2_{df}$). Models including fewer parameters are preferable if not producing a significantly worse fit to the data. Alternatively, the Akaike Information Criterion (AIC), calculated as $\Delta\chi^2 - 2\Delta df$, combining parsimony and fit, may be utilized (Akaike, 1987). The latter method may, however, yield incorrect results if used alone (Sullivan & Eaves, 2002).

To explore sex differences, the models may be expanded to test for various sex-specific effects. A general sex-limitation model allows for evaluation of

Table 1

Descriptive Statistics

		Males		Females		*p*
Subjective wellbeing	Mean *(SD)*	3.72	(.59)	3.64	(.59)	< .01
Sleep Problems	Sleep problem?					
	Yes	6.4	(221)	9.6	(444)	< .01
	No	93.6	(3222)	90.4	(4158)	
% (*N*)	Sleep medication last month?					
	Every night	.2	(5)	.5	(19)	< .01
	Often	.2	(6)	.4	(13)	
	Sometimes	1.6	(48)	2.2	(80)	
	Never	98.1	(2935)	96.9	(3476)	
	Sleep problems last month?					
	Every night	1.9	(64)	2.0	(92)	*ns*
	Often	3.8	(130)	3.9	(181)	
	Sometimes	29.3	(1001)	32.1	(1477)	
	Never	65.1	(2227)	61.5	(2831)	

both quantitative and qualitative sex differences. The magnitude of genetic and environmental influences on the phenotypes for males and females are estimated separately, and the correlation between genetic factors in males and females (r_g) is not fixed, thus enabling us to estimate whether the same set of genes influence the trait in males and females. In a common sex-limitation model, only genetic effects that are common to both sexes may account for the phenotypic variance or covariance (r_g = 1), but the magnitude of the genetic and environmental parameters may vary. In the no sex–limitation model, sex-specific effects are removed and the variance components are set to be equal for males and females alike (Neale & Maes, 2003).

Univariate analyses were conducted to explore the genetic and environmental effects on the phenotypes separately. Bivariate models were then used to examine the extent to which genetic and environmental factors explained the correlation between the phenotypes. Multivariate models may be parameterized in different ways, and the Cholesky decomposition has been a popular approach (Loehlin, 1996). This parameterization may, however, prove problematic when modeling sex differences (Neale, Røysamb, & Jacobsen, 2005) and correlated factors models were therefore selected to analyze the bivariate data (Figure 1).

Results

Thresholds

There were no significant differences in thresholds within pairs, or between MZ and DZ twins when comparing same-sexed twins. However, constraining the thresholds to be equal across sex yielded a significant deterioration in fit for both SWB (Δ^2_6 = 43.67, p = .00) and sleep problems ($\Delta\chi^2_6$ = 35.47, p = .00), thus indicative of sex differences in the distribution for both measures. Specifically, the thresholds for SWB were higher for female than male responders, whereas the thresholds for sleep problems were lower for female than male responders. Tests of the equality of thresholds for complete versus incomplete twin pairs revealed no significant differences for either of the two measures, indicating that volunteering behavior was not associated with either phenotype. Further tests of attrition bias comparing twins responding to both data collections (Q1 and Q2) and twins responding only to Q2 revealed no differences in the threshold liability distributions for either measure.

Descriptive Statistics

The results of the descriptive analyses are displayed in Table 1. In accordance with the threshold test, significant sex differences were observed. Males scored slightly, though significantly, higher than females (p < .01) on the SWB index. In contrast, females scored significantly higher on the sleep index (p < .01). The SWB scores ranged from 1 to 5 for males, and 1.25 to 4.75 for females, whereas the scores on the sleep index ranged from 1 to 5 for both sexes. Sixty-five per cent of the males and 62% of the females did not report problems sleeping during the last month (not significant). A small percentage of the respondents reported using sleep medication. This was found to be significantly more prevalent among female than male responders.

Twin Correlations

The within-twin or phenotypic correlation between SWB and self-reported sleeping problems was negative and substantial (r = –.43). Table 2 displays the phenotypic correlations, the cross-twin correlations (i.e., correlations between twin 1 and twin 2 for each measure) and the cross-twin cross-trait correlations (i.e., correlations between twin1–trait1 and twin2–trait2) by zygosity with 95% confidence intervals (CI).

Table 2

Polychoric Twin Correlations for Sleep Problems and SWB: Within-Twin Correlations, Cross-Twin Correlations and Cross-Twin Cross-Trait Correlations by Zygosity

	n	Within-twin	Cross-twin: Sleep problems	Cross-twin: SWB	Cross-twin cross-trait
MZ_m	714	–.41 (–.47, –.34)	.47 (.36, .57)	.50 (.41, .57)	–.26 (–.33, –.18)
MZ_f	936	–.44 (–.48, –.38)	.43 (.35, .51)	.42 (.35, .49)	–.28 (–.34, –.21)
DZ_m	671	–.44 (–.50, –.37)	.05 (–.11, .20)	.26 (.15, .37)	–.13 (–.23, –.03)
DZ_f	862	–.43 (–.48, .37)	.16 (.05, .26)	.28 (.20, .36)	–.12 (–.19, –.05)
DZ_u	1528	–.42 (–.46, –.37)	.16 (.07, .24)	.16 (.09, .23)	–.08 (–.13, –.02)

Table 3

Univariate Model-Fitting Results and Correlation Estimates (r_g) for SWB

Sex effect	Model	A_m	C_m	E_m	A_f	C_f	E_f	r_g	–2LL	Δ_{df}	$\Delta\chi^2$	*p*	AIC
I	ACE	.43	.06	.51	.29	.14	.57	1.00	21577.04				
	AE	.50		.50	.45		.55	1.00	21579.24	2	2.20	.33	–1.80
II	ACE	.48	.01	.51	.30	.13	.57	1.00	21577.04	1	0.00	—	–2.00
	AE	.48		.52	.44		.56	1.00	21583.93	3	6.89	.08	0.89
III	ACE	.45	.00	.55	.45	.00	.55	1.00	21584.57	4	7.53	.11	–0.47
	AE	.45		.55	.45		.55	1.00	21584.57	5	7.53	.18	–2.47

Note: Sex effect I = general sex limitation.
Sex effect II = common sex limitation.
Sex effect III = no sex limitation.

Table 4

Univariate Model-Fitting Results and Correlation Estimates (r_g) for Sleep Problems

Sex effect	Model	A_m	D_m	E_m	A_f	D_f	E_f	r_g	–2LL	Δ_{df}	$\Delta\chi^2$	*p*	AIC
I	ADE	.08	.40	.52	.19	.24	.57	.34	15729.68				
	AE	.45		.55	.41		.59	.65	15734.08	2	4.40	.11	0.40
II	ADE	.08	.40	.52	.19	.24	.57	1.00	15729.68	1	0.00	—	–2.00
	AE	.43		.57	.40		.60	1.00	15735.49	3	5.81	.12	–0.19
III	ADE	.13	.32	.55	.13	.32	.55	1.00	15730.42	4	0.74	.95	–7.26
	AE	.41		.59	.41		.59	1.00	15735.72	5	6.03	.30	–3.97

Note: Sex effect I = general sex limitation.
Sex effect II = common sex limitation.
Sex effect III = no sex limitation.

Pair resemblance for SWB and self-reported sleep problems were considerably stronger in MZ than DZ twins, suggesting the etiological importance of genetic factors. For sleep problems, the correlations indicated the possible presence of nonadditive genetic effects ($r_{MZ}/r_{DZ} > 2.0$). For SWB, the correlational pattern neither strongly suggested nonadditive genetic or common environmental effects ($r_{MZ}\, r_{DZ} \sim 2.0$). Co-twin similarity for SWB was lower among opposite-sex DZ pairs than same-sexed DZ pairs. The confidence intervals were overlapping, but when testing the pooled correlation for the same-sexed DZ twins against the correlation for unlike-sexed DZ twins, a significant difference ($z = 2.2$, $p = .03$) was observed, thus suggesting sex-specific genetic effects. In contrast, the pattern of correlations for sleep problems was not indicative of sex–specific genetic influences. Cross-twin cross-trait correlations were greater in MZ than in DZ twins, indicative of genetic influences on the phenotypic association.

Univariate Modeling

Based on the results of the correlation analyses, we performed separate analyses for the two phenotypes. The results from the univariate modeling of SWB appear in Table 3.

An ACE model with general sex-limitation was utilized as the basic model. However, omitting the common environmental pathway did not produce any significant deterioration in fit ($\Delta\chi^2_2 = 2.25$, $p = .35$),

Table 5

Model-Fitting Results and Correlation Estimates for SWB and Self-Reported Sleep Problems From the Bivariate Analyses

Sex effect	model	A_m	E_m	A_f	E_f	A_m	D_m	E_m	A_f	D_f	E_f	–2LL	$\Delta\chi^2$	Δ_{df}	p	AIC	Males		Females	
																	r_g	r_e	r_g	r_e
I	1. AD_sE*	.50	.50	.45	.55	.14	.32	.54	.27	.16	.57	36421.75				–1.00	–.31	–.80	–.27	
	2. AE	.50	.50	.45	.55	.44		.56	.41		.59	36425.78	4.03	2	.13	0.03	–.58	–.30	–.61	–.27
II	3. AD_sE	.49	.51	.43	.57	.21	.25	.54	.20	.22	.58	36428.51	6.77	6	.34	–5.23	–.84	–.30	–.84	–.30
	4. AE	.49	.51	.43	.57	.42		.58	.40		.60	36432.22	10.47	8	.23	–5.53	–.59	–.29	–.59	–.29
III	**5. AD_sE**	**.45**	**.55**	**.45**	**.55**	**.20**	**.24**	**.56**	**.20**	**.24**	**.56**	**36430.00**	**8.24**	**11**	**.69**	**–13.76**	**–.85**	**–.30**	**–.85**	**–.30**
	6. AE	.45	.55	.45	.55	.41		.59	.41		.59	36433.45	11.70	12	.47	–12.30	–.59	.29	–.59	.29

Note: Sex effect I = general sex limitation
Sex effect II = common sex limitation
Sex effect III = no sex limitation.
* The *d* parameter is estimated for sleep problems only.

indicating that the familial aggregation of SWB may be accounted for most parsimoniously by genetic factors alone. The best model in terms of AIC values (ΔAIC = –2.47) was an AE model specifying equal parameters for males and females (no sex-limitation). This model did not result in a significant deterioration in fit ($\Delta\chi^2_5$= 7.50). In this model, parameter estimates for additive genetic effects was .45 (95% CI: .40–.50) and for individual environmental effects .55 (95% CI: .50–.60).

Table 4 displays the results from the univariate modeling of sleep problems. An ADE model was used as the basic model due to the ratio of MZ to DZ correlations exceeding 2.0. The r_g was estimated to be unity, indicating no sex-specific genetic effects. The best fitting model in terms of AIC values was an ADE model with no sex-limitation. In this model, the parameters estimates for additive genetic effects were .13 (95% CI: .00–.38), nonadditive genetic effects .32 (95% CI: .05–.56) and individual environmental effects .55 (95% CI: .49–.62).

Bivariate Modeling

Based on the results of the univariate analyses, an ADE model, allowing for estimation of the *d* parameter for sleep problems only, as well as separate effects for males and females, was used as the basic model against which other nested reduced models were compared.

In the basic model, male parameter estimates for additive genetic variance were .50 (95% CI: .43–.57) and .14 (95% CI: .09–.47), for SWB and sleep problems respectively, and the corresponding estimates for female responders were .45 (95% CI: .39–.51) and .24 (95% CI: .11–.48). The parameter estimate for nonadditive genetic variance (sleep problems) for males was .32 (95% CI: .00–.41) whereas the respective estimate for females was .18 (95% CI: .00–.35). Estimates for the individual environmental factors for males were .50 (95% CI: .43–.57) and .54 (95% CI: .44–.64) for SWB and sleep problems, respectively. Corresponding estimates for females were .55 (95% CI: .49–.62) and .58 (95% CI: .50–.66). In total, six models were tested (Table 5). The best fit in terms of AIC values was observed for an ADE model, allowing for estimation of the *d* parameter for sleep problems only, and constraining the magnitude of parameters to be equal in males and females (model 5). The estimates for additive genetic effects for SWB and sleep problems in this model were .45 (95% CI: .41–.50) and .20 (95% CI: .11–.43) respectively, and the estimate for nonadditive effect for sleep problems was .24 (95% CI: .00–.35), whereas the individual environment was accounting for the remaining 55% (95% CI: .50–.59) and 56% (95% CI: .50–.63) of the variance. According to this model, the phenotypic correlation (–.43) could be decomposed into genetic (–.26) and environmental (–.17) components, explaining 60% and 40% respectively of the observed correlation. The correlation between additive genetic factors influencing SWB and sleep problems was estimated to be –.85 (95% CI: –1.00 — –.57), and the correlation between individual environmental factors was –.30 (95% CI: –.35 — –.23). Figure 1 depicts the results from the best fitting model with calculated path coefficients from the additive genetic factors, the nonadditive factor and the individual environmental factors, as well as estimated correlations between additive genetic factors and unique environment.

Discussion

Consistent with previous studies (Heath et al., 1990; Hublin et al., 2001; Røysamb et al., 2002), substantial heritability was found for both SWB and sleep problems. Further, and to our knowledge previously unexplored, 60% of the correlation between SWB and self-reported sleep problems was found to be attributable to common genetic factors. The additive genetic influences for SWB and sleep problems correlated substantially. Thus, we might consider the possibility of a primary and partly genetic process enhancing our ability for enjoyment and satisfaction and simultaneously facilitating sleep, consistent with the evolutionary hypothesis for positive health stating that SWB is genetically associated with the effective functioning of biophysical systems.

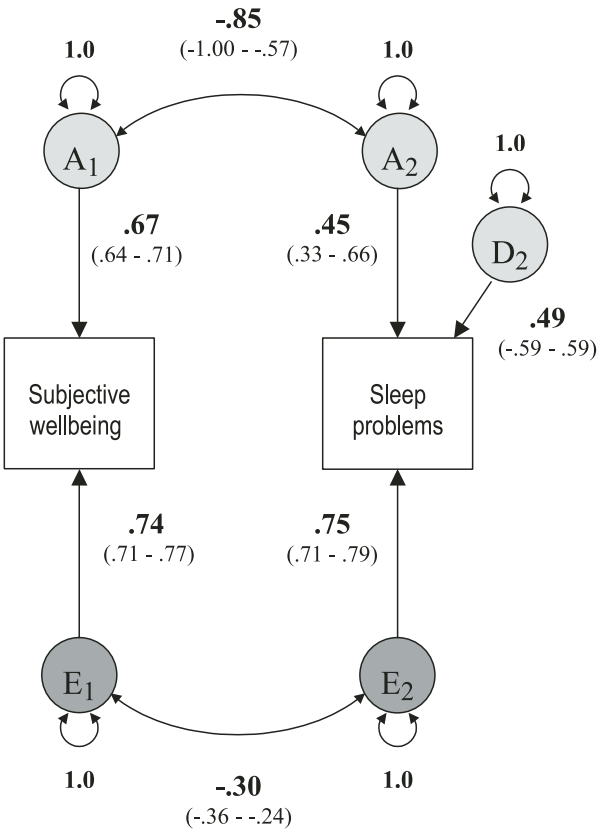

Figure 1

Results from the best fitting bivariate model (model 5).

A indicates the latent additive genetic factors, D nonadditive genetic factor, and E individual environmental factors. The subscripts 1 and 2 refer to SWB and sleep problems, respectively. The magnitude of each path is shown in the figure, and must be squared to equal the proportion of variance in the observed variable accounted for by the latent factors.

An alternative interpretation suggests that our findings may result from a genetically influenced top-down process simultaneously affecting the perception, and thus the self-report, of SWB, sleep quality and sleep quantity. Within such a framework, the observed genetic overlap may reflect a genetic disposition or propensity for a positive outlook on life, tending to produce positive evaluations of various subdomains of life. Previous studies have demonstrated that characteristically happy individuals tend to construe their encounters and life events more favorably, and remember pleasant aspects of life better than individuals located at the low end of the hedonic capacity continuum (Lyubomirsky & Tucker, 1998).

It is widely accepted that subjective recollections of sleep may represent inaccurate portrayals of objective sleep problems. When 'objective' ratings (e.g., PSG, EEG) are examined, associations between sleep and self-reported quality of life are sometimes found to be weak (e.g., Watten et al., 1997). The relationship between quality of sleep and measures of health, well-being and sleepiness has been found to be stronger than sleep quantity and the respective measures (e.g., Pilcher et al., 1997), suggesting that the association between sleep and mood may result from either positive or negative affective recall bias. Accordingly, reported sleep problems may primarily represent the individual's current affect state, expressing the influence of affective valence on perceptions of sleep and health.

Our data, however, suggest that SWB and sleep disturbance are not synonymous constructs showing divergent validity, thus indicating that reported sleep problems do not merely mirror the emotional state of the respondents. Rather, the discrepancies between objective and subjective measures of sleep may arise from, for example, subtle alterations of sleep phenomenology (Hall et al., 2000) or biochemical effects. Such alternative explanations address the commonly reported discrepancy between subjective and objective measures of sleep, and argue for a more complex understanding of the phenotypic relationship than is suggested by the affect-bias hypothesis. At the neurophysiological or biochemical level, the results may reflect common genetic mechanisms influencing neurophysiological processes or transmitter systems involved in both regulation of sleep and mood (Adrien, 2002). Hitherto, dysregulation of the serotonergic system, which is concurrently involved in the control of sleep and affectivity, seems to represent the most probable candidate. A genetic liability to dysfunctional or decreased serotonergic activity (Meltzer & Lowy, 1987) or abnormalities in the ratio of cholinergic to aminergic neurotransmission systems (e.g., Janowsky et al., 1983) is suggested to be related to the association between mood and sleep.

Our results indicate that individual environmental factors account for a moderate part of the phenotypic association (40%). The study was, however, not designed to identify which specific environmental influences may have an effect on both phenotypes. A twin study by Heath, Eaves, et al. (1998) investigating the effects of lifestyle on subjective sleep disturbance, found smoking, educational level, marital status, number of children and consumption of tea, coffee and alcohol to explain only a small proportion of the variance in subjective sleep disturbance. When ignoring the effects of genotype and personality, lifestyle factors accounted for only 4% of the variance in sleep disturbance, and interaction between genotype and risk factors explained only a relatively small proportion of the variance. However, diverse stress factors (e.g., job stress, commuting, chronic financial strain, family conflicts) and life events (e.g., significant loss) are commonly found to play important roles in the pathogenetical process of sleep problems (e.g., Hall et al., 2000) and global sleep dissatisfaction (Ohayon & Zulley, 2001), as well as influencing individual evaluations of SWB.

Significant but modest sex differences in the degree of SWB and the prevalence of sleep problems were observed. Specifically, male responders were

found to be happier and reported a lower prevalence of sleep problems than their female counterparts. These findings are in accordance with previous findings. Heritability for the SWB index based on the current sample was reported in a multivariate twin study by Røysamb et al. (2003) to be .44 for both males and females. Our bivariate model estimate was .45. This slight difference is probably due to the former study using an asymptotic covariance matrix approach, whereas the present study used the raw data method, and different phenotypes were included in the models. However, Røysamb et al. (2003) found a significant qualitatitive sex difference in genetic effects on SWB. A former univariate twin study, also based on the Norwegian Twin Panel (Q1), found evidence for both qualitative and quantitative sex differences in the heritability of SWB (Røysamb et al., 2002). In our study, cross-twin polychoric correlations for SWB suggested sex differences, but the best fitting models in terms of AIC did not include sex-specific genetic effects. In twin studies, very large samples are generally required to have sufficient statistical power to detect differences in genetic effects below .1 to .2 (Neale et al., 1994). Therefore, the possibility of sex differences in the heritability of SWB cannot be ruled out completely. However, the bivariate analyses indicated no evidence for sex-specific genetic processes contributing to the association between SWB and sleep problems.

Limitations

These results should be interpreted in the context of the following methodological limitations.

Correlated liability models were used to investigate the association between SWB and self-reported sleep problems. Our results reflect the limitations specified in these particular models, but do not rule out the possibility of other causal relationships. Co-occurrence in the same individual of symptoms or disorders may arise from various mechanisms, such as direct phenotypic causation (e.g., reduced SWB causing sleep problems), or reciprocally interacting phenotypes (Neale & Kendler, 1995). Alternative models of co-occurring phenotypes are possible in a cross-sectional data set (Gillespie et al., 2003; Heath et al., 1993; Neale & Kendler, 1995), but the statistical power to detect differences in these models are usually very low with samples of our size (Neale & Kendler, 1995). Longitudinal data are better suited to explore causal mechanisms, and should be used in future studies.

Although self-report data are widely accepted as reflecting stable aspects of functioning, they may not correspond perfectly to more 'objective' assessment. The traits examined were also based on a small number of items (four and three items). However, confirmatory multisample factor analysis yielded high factor loadings, good overall fit, and noninvariance across sexes, indicating good psychometric properties. Nevertheless, the correlation between factors was higher (–.65) than the corresponding correlation between sum score indices (–.43), suggesting substantial contribution from uncorrelated measurement errors, and a possible overestimation of the nonshared environment. Our heritability estimates from the univariate analysis are, however, in accordance with previous findings for sleep problems (Heath et al. 1990; Hublin et al., 2001) and SWB (e.g., Lykken & Tellegen, 1996).

Differential attrition and nonresponse may potentially lead to biased estimates of genetic and environmental parameters (Heath, Madden, et al., 1998). The individual and pair response rate for the present study was 63% and 53%, respectively, thus lower than optimal. The response rate was particularly low among the youngest cohorts, born 1975 to 1979. The modest response rate may partly reflect Norwegian law prohibiting more than one reminder as well as the increased length of the Q2 questionnaire. However, tests of the homogeneity of thresholds did not differ between pair responders and singletons, or between twins responding at both data collections (Q1 and Q2) and twins responding only at Q2. Also, comparing the twin–co-twin correlations for SWB at Q1 for nonresponders and responders at Q2, no significant differences were observed. Corresponding analyses for the sleep index were not possible due to different sleep items in the Q1 data. The standard problems of small and self-selected samples are also usually ruled out when the sample is being derived from a population-based twin register.

Sex differences in specific sleep symptoms were not investigated due to a rather broadly defined phenotype. Recently, Khan et al. (2002) observed significantly more hypersomnia and fatigue in female twins whereas male respondents reported more insomnia and agitation.

Another limitation concerns the age of our sample. This study included young adults, aged between 18 and 31 years. Generally, epidemiological studies show a higher prevalence of sleeping problems with increasing age, rising up to 50% in older cohorts (greater than 65 years; Ohayon, 2002). A sex difference in prevalence is also found to be greater in older cohorts (greater than 45 years), the ratio of women to men being approximately 1.7 compared to a ratio of 1.4 in younger individuals (Ohayon, 2002). Also, Heath, Eaves, et al. (1998) observed increased effects of genetic liability on sleep disturbance in older women. Thus, our results may not be generalized to older cohorts.

Conclusion

Analyzing SWB and self-reported sleep problems in young adult Norwegian twins, we found that the additive genetic factors affecting the two phenotypes correlated (–.85), and explained 60% of the phentotypic association. Our results suggest that genetic factors positively influencing SWB also protect against problems sleeping. We observed sex differences in the distribution of both phenotypes. However, the best fitting bivariate model specified no sex-specific genetic effects. The correlation

between individual environmental effects was –.30, and the individual environment explained 40% of the phenotypic correlation. Future studies may attempt to disentangle this complex interplay between genes and environment, to further clarify the specific etiological contributions of each effect; for example, by exploring possible causal pathways, or by controlling for genetic effects and investigating specific environmental influences, protective factors or suboptimal environments underlying the association between happiness and sleep.

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