ORIGINAL RESEARCH

Exploring the Association Between Well-Being and Psychopathology in Adolescents

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Abstract Promotion of mental well-being and prevention of emotional and behavioral problems are suggested to go hand in hand. The present study examined the association between subjective well-being (SWB) and psychopathology and investigated the etiology of this association in a large population-based cohort study of adolescent twins ($n = 9,136$) and their non-twin siblings ($n = 1,474$) aged 12–20 years. Phenotypic, genetic, and environmental correlations between SWB and psychopathology were obtained from multivariate genetic modeling conditional on sex. An SWB factor score was used based on measures of subjective happiness, satisfaction with life, and quality of life. Psychopathology was obtained from all syndrome and broad-band scales of the Dutch version of the ASEBA Youth Self Report. Males reported significantly higher levels of SWB than females. Females reported significantly more internalizing problems while males report significantly higher levels of externalizing behavior. In both sexes, significant negative associations were found between SWB and psychopathology, with the strongest associations seen for SWB and the YSR syndrome scale anxious/depression behavior. The observed associations were primarily explained by genetic correlations while non-shared environmental influences were mainly domain specific. The genetic liability to lower levels of SWB are indicative of a genetic liability to higher levels of psychopathology, suggesting that it might be feasible to screen for emotional and behavioral problems before clear signs are present by screening on indices of subjective well-being.

There is a growing interest in positive aspects of psychological functioning, especially subjective well-being (SWB) and its possible preventive role for behavioral and emotional problems. Prevention of psychopathology is of increasing urgency given the wide-ranging individual, societal, and economic impact of these disorders (World Health Organization 2004, 2010). In addition, data on SWB may soon be used together with economic data to guide public policy (Stiglitz et al. 2009; Victor 2010). The value of promoting well-being in conjunction with prevention of psychopathology and the public policy implications depend in part on the sources of overlap between SWB and psychopathology.

Not surprisingly, SWB is negatively correlated with psychopathology, especially with depression and mood disorders (e.g. Greenspoon and Saklofske 2010; Proctor et al. 2009) but also with externalizing problems, such as violence, aggression, and substance (ab)use (e.g. Mac-Donald et al. 2005; Valois et al. 2001). The size of the

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association (correlations generally between −0.40 and −0.55) raises the possibility that SWB and psychopathology represent partly distinct dimensions of mental functioning, but also reveal that there is overlap. This has been described in the dual continuum model or the dual-factor mental health model (e.g. Greenspoon and Saklofske 2010; Russell and Carroll 1999; Ryff et al. 2006). Up until recently, only scant attention has been paid to the fact that associations between SWB and psychopathology can be a function of correlated genetic and/or correlated environmental factors.

Individual differences in both SWB and psychopathology have been shown to be partly due to genetic differences between individuals. Estimates of the heritability of SWB tend to hover around 40 %, with evidence for presence of additive and non-additive genetic influences (e.g. Bartels and Boomsma 2009; Lykken and Tellegen 1996; Nes et al. 2006; Røysamb et al. 2002, 2003; Stubbe et al. 2005). Combining the results of these studies indicate no difference in heritability estimates between adolescents and adults. In contrast causes of individual differences in psychopathology seem to vary with age. For most behavioral and emotional problems, except for Attention Problems, estimates of the influences of genetic factors vary across development (e.g. Bartels et al. 2007a; Hudziak et al. 2003; Legrand et al. 1999; Kendler et al. 2008; Kan et al. 2012). Changes in the magnitude of genetic influences seem inversely related to changes in the importance of environmental influences shared by children growing up in the same family. Non-shared environmental influences remain more or less constant. Furthermore, longitudinal studies with data from the Netherlands Twin Register indicate that stability of emotional and behavioral problems throughout childhood are mainly accounted for by genetic factors (Bartels et al. 2004, 2007b; Boomsma et al. 2005; Hoekstra et al. 2008; van Beijsterveldt et al. 2003; van Grootheest et al. 2007; Rietveld et al. 2004).

While the heritability estimates of previous studies with a focus on either SWB or psychopathology provide an indication that genetic factors could underlie the association among indices of wellbeing and psychopathology, a large scale study focusing on the underlying sources of covariance between SWB and psychopathology during adolescence is lacking. Two previous studies in adults (Kendler et al. 2011; Nes et al. 2008) explored the relationship between SWB and internalizing psychopathology, including major depression and generalized anxiety disorder. Both studies indicated that genetic factors underlie the phenotypic association between SWB and pscyhopathology of the internalizing subtype. The aim of the current study is to explore whether these observations hold in adolescents and whether they apply both internalizing and externalizing behavioral problems. To this end we conduct

a large scale genetically informative study into the sources of overlap between SWB and psychopathology. The large population based sample of adolescent twins and their non-twin siblings allows for estimation of sex-specific phenotypic, genetic, and environmental associations between SWB and psychopathology.

Methods

Participants

Participants were registered at birth with the Netherlands Twin Register (NTR) (van Beijsterveldt et al. 2013; Bartels et al. 2007a; Boomsma et al. 2006), established by the Department of Biological Psychology at the VU University in Amsterdam. For the current study, data from surveys collected in adolescent twins and their non-twin siblings were analyzed. The sample consisted of 10,610 individuals (9,136 twins and 1,474 non-twin siblings from 5,001 families, 43.7 % males). Mean age of the participants was 16.41 (1.56), with age ranging from 12 to 20 years (born in 1984–1999, 90 % between 13 and 19 years old). Data from non-twin siblings were included in the analyses with a maximum of 2 siblings (1 brother and 1 sister) per family. In families with data for more than one sibling ($n = 92$) and with two siblings of the same sex (49 of the 92 families), the data from sibling closest in age to the twin were selected for analyses. Zygosity was determined for 1210 same-sex twin pairs by DNA polymorphisms. For all other same-sex twin pairs, zygosity was determined by discriminant analysis, using longitudinally assessed questionnaire items from the previously collected parental reports. Agreement between zygosity assignment based on questionnaire information and zygosity determined by DNA markers was around 93 % (Rietveld et al. 2000; Willemsen et al. 2013).

Adolescent twins, and their non-twin siblings (aged 12–25) received a self-report survey. Before inviting twins and their siblings to provide self-report data, parents of twins and siblings <16 years of age were contacted to ask for permission to send their children a self-report survey and to register non-twin siblings of the twins. Upon parental consent, questionnaires were sent to the twins and their siblings. Initially, the survey was presented in a paper and pencil version. In 2009 data collection continued with an online version of the questionnaire and the paper and pencil version was used as a second reminder. Response rate was 47 %. Non-response analyses showed that parents of twins who did not return the survey have in the past been also less willing to participate in survey research of the YNTR compared to parents of twins that participated. Other parental and familial characteristics, such as SES,

parental education, religion, and smoking and alcohol consumption during pregnancy were similar for families of non-responding twins compared to families of responding twins. Small, but significant differences were found for externalizing behavior at ages 7 and 12 with non-responders scoring higher (8.6 vs. 7.4 at age 7; 6.5 vs. 5.0 at age 12). In addition non-responders scored significantly lower on a general test of educational achievement at age 12, but again the difference is very small (536.4 vs. 538.3). No differences are found for internalizing problems at ages 3, 7, and 12 and externalizing problems at age 3. Furthermore, no differences were found for specific educational achievement tests for language and arithmetic. Total sample constitution and sample constitution as a function of zygosity is described in Table 1.

Measures

The Dutch Health Behavior Questionnaire (DHBQ) is a self-report instrument containing a broad range of measures on health, lifestyle, and behavior (Bartels et al. 2011). It includes the Youth Self Report (YSR) to assess adolescent psychopathology and three measures of Subjective Well-being. The YSR (Achenbach and Rescorla 2001) is a screening tool for behavioral and emotional problems in adolescents that comprises the Achenbach System of Empirically Based Assessments (ASEBA). Adolescents are asked to fill out 118 items (112 items of the 2001 version supplemented with 6 items from the previous version of the YSR) on a 3-point scale based on the occurrence of the behavior during the preceding 6 months: 0 if the problem item was *not true*, 1 if it was *somewhat or sometimes true,* and 2 if it was *very true or often true.* The eight syndrome scales (AD: anxious/depressed, SC: somatic complaints, WD: withdrawn/depressed, SP: social problems, TP:

thought problems, AP: attention problems, RB: rule-breaking behavior, AGG: aggressive behavior) and the broadband scales internalizing problems (INT: anxious/depressed, somatic complaints and withdrawn/depressed) and externalizing problems (EXT: rule-breaking and aggressive behavior) were based on the 2001 profile (Achenbach and Rescorla 2001). Dutch syndrome scales and comparability with the syndrome scales developed by Achenbach are reported in Verhulst et al. (1997).

Three measures of self-reported evaluations of SWB were used: (1) satisfaction with life (SAT) was assessed with the Satisfaction with Life Scale (Diener et al. 1985). The scale consists of 5 items which had to be answered on a 7-point scale ranging from 1 = '*strongly disagree*' to 7 = '*strongly agree*'. An example items is "My life is going more or less as I wished". Internal consistency of the scale was good with a Chronbach's Alpha of 0.86. (2) Subjective happiness (HAP) was assessed with the Subjective Happiness Scale (Lyubomirsky and Lepper 1999). The scale consists of 4 items which had to be answered on 7-point scale ranging from 1 = '*strongly disagree*' to 7 = '*strongly agree*'. An example items "Compared to most of my peers, I consider myself a happy person". Internal consistency of the scale was good with a Chronbach's Alpha of 0.84. (3) Quality of life in general (QLg) was assessed with the Cantril Ladder (Cantril 1965). The ladder has 10 steps: the top indicates the best possible life, and the bottom the worst possible life. Participants had to indicate the step of the ladder at which they place their lives in general.

Since the data were collected in a unselected population sample some of the measures showed a skewed distribution, which may cause small biases in parameter estimates. However, as transformation of the data does not remove these biases and the bias is known and small (unbiased estimate of the additive genetic effect, underestimation of

Table 1 Sample size and characteristics

Family constitution	Number of families	Zygosity	Number of families
One of the twin, no sibling	654	MZM	551
Twin pair, no sibling	2,876	DZM	476
One of the twin with a brother	38	MZF	792
One of the twin with a sister	71	DZF	571
One of the twin with a brother and a sister	1	DOSmf	576
Twin pair with a brother	603	DOSfm	545
Twin pair with a sister	707		
Brother only (no twin)	16	In total 5,001 families	
Sister only (no twin)	33	Cohort: 1984–1999	
Brother and sister only (no twin)	2	Mean age 16.41 (1.56)	

MZM monozygotic males, *DZM* dizygotic males, *MZF* monzygotic females, *DZF* dizygotic females, *DOSmf* male–female dizygotic twin pair, *DOSfm* female–male dizygotic twin pair

the shared environmental effect, and an overestimation of the unique environmental effect; see Derks et al. 2004) untransformed data were analyzed.

The SWB measures correlate substantially, with correlations in the range of 0.35–0.73 for males and 0.45–0.77 for females, indicating a moderate to large overlap between the measures. In our previous work (Bartels and Boomsma 2009) we showed that genetic factors are responsible for the moderate to high phenotypic associations between SAT, HAP, and QLg. Therefore a latent factor score for SWB was estimated for each participant in the dataset. This was done by specifying a factor model with the three observed measures of SWB loading on a single latent factor (SWB) in Mplus (Muthén and Muthén 2010). Given this factor solution and individuals' scores on the observed measures of SWB, a latent factor score can be estimated for each individual. This factor score has a mean of zero and a variance of 1, with a low score corresponding to low levels of SWB. To overcome problems of a low variance in this SWB factor score, we multiplied the factor score by 10.

Statistical analyses

First, a tri-variate genetic model focusing on the overlap between SWB, INT, and EXT was evaluated. Second, to gain more insight into the overlap of SWB with the subscales of the YSR we ran bivariate analyses to disentangle the overlap between SWB and each subscale.

Descriptive statistics

Sex-differences in mean levels of SWB and psychopathology were tested by mixed-model analyses of variance in SPSS 18, with family as a random factor to account for the within-family dependence of the outcome variables. Significance thresholds for the descriptive was set at 0.05. A small but significant effect of age on mean levels has been reported for SWB and psychopathology (e.g. Bartels et al. 2011; Bartels and Boomsma 2009; Lamb et al. 2010), so age was included as a fixed effect on the means. Furthermore, in our previous work with an overlapping dataset, no differences between DZ twin and non-twins siblings were observed, so estimates were constrained to be equal.

Genetic modelling

The different degree of genetic relatedness between monozygotic (MZ), dizygotic (DZ) twin pairs and sibling pairs provides the opportunity to disentangle the phenotypic correlations in additive and non-additive genetic, shared environmental, and nonshared environmental components and to obtain estimates of genetic and environmental correlations. Additive genetic variance

(A) represents the additive effects of alleles over all loci that influence the trait. Non-additive genetic effects (D) comprise interactions between alleles at the same locus (dominance) and will also include effects of interaction among alleles at the different loci (epistasis). Shared environmental variance (C) is the part of the variance that is shared by members of a family which is not explained by genetic relatedness and non-shared environmental variance (E) is the part of the non-genetic variance that is unique to an individual. Phenotypic correlations, twin and twin-sibling correlations, and within person and cross-person cross-trait correlations were estimated using a saturated model in OpenMx (Boker et al. 2011). Next, an ACE model was fitted to the data in order to estimate the underlying sources of variance of SWB and psychopathology and their covariance. First, a tri-variate genetic model (SWB-INT-EXT) was fitted to the data with additive genetic, shared environmental and non-shared environmental influences (ACE model), including quantitative sex-differences. It was tested whether the influences of the genes and environment are of different magnitudes in males and females by constraining them to be equal. Next, the significance of the shared environmental component was tested.

For the bivariate genetic analyses, the ACE model was also fitted to the data. Tests for sex-differences in parameter estimates were carried out by restricting the estimates to be equal for males and females and testing if this led to a significant deterioration of fit. Next, we tested the significance of C separately in males and females.

After establishing the best fitting tri-variate and bivariate models, 95 % confidence intervals were estimated for all parameters. Genetic structural equation modeling in OPENMx was used with the raw-data ML procedure for estimation of parameters. Nested submodels were compared by hierarchic χ^2 tests. The χ^2 statistic is computed by subtracting –2LL (log-likelihood) for a reduced model from that for the full model ($\chi^2 = -2LL_0 - (-2LL_1)$). This χ^2 statistic is distributed with degrees of freedom (df) equal to the difference in the number of parameters estimated in the two models ($\Delta df = df_0 - df_1$). If the difference test is significant (threshold for the genetic models $p < 0.01$ due to multiple testing) the constraints on the nested model cause a significant deterioration of the model.

Results

Descriptive statistics

Means and standard deviations of SWB and psychopathology are presented in Table 2 for males and females separately. Males reported significantly higher levels of

Table 2 Number of individuals, means, and standard deviations for SWB and Psychopathology separately for males and females

	Males			Females		
	N	M	sd	N	M	sd
SWB	4318	0.072	0.88	5547	−0.056[*]	0.97
Withdrawn (WD)	4409	2.09	1.99	5723	2.62[*]	2.28
Somatic complaints (SC)	4386	1.76	2.02	5688	3.05[*]	2.67
Anxious/depressed (AD)	4390	3.06	3.43	5704	5.16[*]	4.71
Internalizing problems (INT)	4297	6.79	5.85	5590	10.56[*]	7.88
Rule-breaking behavior (RB)	4432	2.76	1.97	5746	2.46[*]	1.95
Aggressive behavior (AGG)	4407	6.02	4.41	5715	6.00	4.03
Externalizing problems (EXT)	4405	8.79	5.74	5713	8.46[*]	5.38
Social problems (SP)	4434	2.16	1.87	5757	2.10	1.89
Thought problems (TP)	4426	1.62	1.75	5748	1.92[*]	1.87
Attention problems (AP)	4419	4.63	2.77	5743	4.96[*]	2.85

SWB subjective well-being

* Females score significantly different than males ($p < 0.05$)

Table 3 Phenotypic correlations, twin correlations and cross-twin cross-trait correlations for SWB, INT and EXT

Males				Females			
Phenotypic				Phenotypic			
	SWB	INT	EXT		SWB	INT	EXT
SWB	1			SWB	1		
INT	−0.43 (−0.46, −0.41)	1		INT	−0.58 (−0.59, −0.56)	1	
EXT	−0.19 (−0.22, −0.16)	0.42 (0.39,0.44)	1	EXT	−0.30 (−0.32, −0.27)	0.47 (0.45, 0.49)	1
MZM				MZF			
	SWB	INT	EXT		SWB	INT	EXT
SWB	0.33 (0.26, 0.39)			SWB	0.45 (0.40, −0.49)		
INT	−0.26 (−0.30, −0.21)	0.47 (0.41, 0.52)		INT	−0.36 (−0.40, −0.32)	0.53 (0.48, 0.57)	
EXT	−0.15 (−0.20, −0.10)	0.23 (0.19, 0.28)	0.52 (0.46, 0.56)	EXT	−0.22 (−0.26, −0.18)	0.34 (0.30, 0.37)	0.61 (0.57, 0.64)
DZM/male–male				DZF/female–female			
	SWB	INT	EXT		SWB	INT	EXT
SWB	0.20 (0.14, 0.25)			SWB	0.29 (0.23, 0.34)		
INT	−0.14 (−0.18, −0.09)	0.24 (0.18, 0.30)		INT	−0.25 (−0.29, −0.20)	0.30 (0.24, 0.35)	
EXT	−0.10 (−0.14, −0.06)	0.14 (0.10, 0.18)	0.24 (0.20, 0.28)	EXT	−0.15 (−0.19, −0.11)	0.16 (0.12, 0.20)	0.25 (0.20, 0.29)
DOS/male–female							
	SWB	INT	EXT				
SWB	0.20 (0.16, 0.24)						
INT	−0.15 (−0.19, −0.12)	0.22 (0.18, 0.26)					
EXT	−0.08 (−0.10, −0.05)	0.09 (0.06, 0.12)	0.26 (0.21, 0.30)				

SWB ($p < 0.05$) than females. Females reported significantly more internalizing problems while males report significantly higher levels of externalizing behavior ($p < 0.05$). The difference in externalizing behavior is based on a significant higher score on rule-breaking behavior for males, while no differences in mean levels were found for aggressive behavior between males and females.

Tri-variate phenotypic, twin, and cross-twin cross-trait correlations

Phenotypic correlations, twin correlations and cross-twin cross-trait correlations for SWB, INT and EXT are provided in Table 3. For males phenotypic correlations between SWB and INT and SWB and EXT were −.43 and −0.19 respectively. The phenotypic correlation between INT and EXT is 0.42. For females a similar picture emerges, but the size of all correlation is larger (SWB–INT: −0.58; SWB–EXT: −0.30; INT–EXT: 0.47). Twin correlations indicate that familial resemblance is mainly accounted for by additive genetic influences and that non-shared environmental influences account for a large proportion of variance. Cross-twin cross-trait correlations are higher for MZ than for DZ twins and twin-sibling relatives, providing evidence for the influence of genetic effects on the association between SWB and psychopathology.

Tri-variate genetic model fitting and variance/covariance decomposition

Model fitting results for the tri-variate analyses are presented in Table 4 and show that constraining the variance/covariance components to be equal for males and females gave a significant deterioration in model fit ($\chi^2_{18} = 520.77$, $p = 0.00$). Shared environmental influences could be dropped from the model without a significant deterioration of fit ($\chi^2_{12} = 16.6$, $p = 0.17$). Since the twin-sibling and the cross-twin/sibling correlations provided some evidence for the significance of shared environmental influences for females an $AE_{males}-ACE_{females}$ models was applied to the data, but this model gave a less parsimonious solution to the data than the AE model.

Table 5 provides the standardized variance/covariance components and their 95 % confidence intervals for SWB, INT, EXT based on the best fitting AE model. Heritability is estimated to be around 34 % for the SWB factor score in males and 47 % in females. For males genetic influences account for 45 and 49 % of the variance in INT and EXT, respectively, and for 53 and 58 % in females. For both males and females the remaining variance is accounted for by non-shared environmental influences.

The off-diagonal estimates provide the percentages of the covariance between SWB and INT and EXT that is accounted for by genetic and non-shared environmental components. For males the main source of covariance constitutes of genetic factors. Notably, the genetic influences on the covariance between SWB and psychopathology (SWB-INT; 58 %; SBW-EXT: 86 %) are larger than the influences of genetic factors on the covariance between INT and EXT (56 %). The remaining influence on the covariance is accounted for by non-shared environmental factors. For females a similar picture emerges. In females

Table 4 Tri-variate model fitting results

Model	−2LL	df	χ^2	df	p
1, ACE with sex-differences	194771.2	29825			
2, ACE no sex-differences	195291.96	29843	520.77	18	0.00
3. AE sex differences	**194787.8**	**29837**	**16.6**	**12**	**0.17**
4. AE$_{males}$_ACE$_{females}$	194807.49	29831	7.41	6	0.28

Note The best fitting model is printed in bold

Table 5 Standardized estimates (95 % CI) for additive genetic and nonshared environmental influences on SWB, INT, EXT and their covariance based on the best fitting model

	A			E		
	SWB	INT	EXT	SWB	INT	EXT
Males						
SWB	0.34 (0.28, 0.39)			0.66 (0.61, 0.72)		
INT	0.58 (0.49, 0.67)	0.45 (0.40, 0.50)		0.42 (0.33, 0.51)	0.55 (0.50, 0.60)	
EXT	0.86 (0.68, 1.06)	0.56 (0.47, 0.65)	0.49 (0.44, 0.54)	0.14 (−0.06, 0.32)	0.44 (0.35, 0.53)	0.51 (0.46, 0.56)
Females						
SWB	0.47 (0.42, 0.51)			0.53 (0.49, 0.58)		
INT	0.66 (0.60, 0.71)	0.53 (0.49, 0.57)		0.34 (0.29, 0.40)	0.47 (0.43, 0.51)	
EXT	0.78 (0.68, 0.87)	0.72 (0.66, 0.78)	0.58 (0.54, 0.62)	0.22 (0.13, 0.32)	0.28 (0.22, 0.34)	0.42 (0.38, 0.46)

however, the influences of genetic factors is stronger for the association between INT and EXT (72 %) than between SWB and INT (66 %). Remarkably, the estimate of genetic influences on the covariance is highest for the overlap between SWB and EXT (78 %). Confidence intervals for these genetic influences on the covariances are overlapping though in females. Genetic correlations between SWB and INT (♂: −0.64; ♀: −0.76) and EXT (♂: −0.40; ♀: −0.45) are in the same range for males and females and are stronger for overlap between SWB and INT than SWB and EXT (Table 6). The non-shared environmental correlations are moderate for SWB and INT (♂: −0.30; ♀: −0.40), but lower for SWB and EXT (♂: −0.04; ♀: −0.14). As expected the correlations are negative for the overlap between SWB and psychopathology but positive for the overlap between INT and EXT (♂: 0.35; ♀: 0.30), indicating that for SWB and psychopathology partly overlapping genes and environmental influences play a role, but with effects in the opposite directions.

Bivariate phenotypic, twin, and cross-twin-cross-trait correlations

To gain more insight in the overlap between SWB and psychopathology we ran bivariate analyses for SWB with each YSR subscale. Phenotypic correlations are depicted in Fig. 1. For males phenotypic correlations ranged from −0.18 to −0.44. Correlations were stronger (more negative) for internalizing related subscales withdrawn and anxious/depressed. For females phenotypic correlations ranged from −0.25 to −0.58, indicating that the overlap between SWB and psychopathology is stronger in females than in males. Twin and cross-twin cross-trait correlations are presented in Table 8 and indicate that the genetic architecture of the overlap between SWB and the psychopathology subscales is largely similar for each scale and for males and females.

Model fitting results are presented in Table 9. The order of model fitting is based on the twin correlations and cross-twin cross-trait correlations. The best fitting model is chosen on the basis of parsimony. In Fig. 1 the underlying sources of the phenotypic correlations are presented. For males the phenotypic correlations are accounted for by genetic and non-shared environmental factors. No significant influences of shared environment on the covariance between SWB and psychopathology were found for males. A similar picture emerged for the females, with the largest part of the phenotypic correlation accounted for by genetic factors.

Genetic and environmental correlations between SWB and psychopathology are reported in Table 7. In males the genetic correlations were much larger than the non-shared environmental correlations, indicating that to a significant extent the same genes influence both constructs. The lower non-shared environmental correlations indicate that environmental influences are more distinct. For females genetic correlations were also strong and non-shared environmental correlations were weaker.

Discussion

This study obtained several results that bear on the relationship between SWB and psychopathology and are essential to value the role of SWB in psychopathology prevention. First, there were substantial shared genetic influences on SWB and psychopathology, with large correlations for the overlap of SWB with INT than for SWB with EXT. Second, the nonshared environmental influences on SWB and psychopathology were largely unique to each domain. Previous research by Kendler and colleagues (Kendler et al, 2011) focused on internalizing psychopathology and mental well-being in adults and reported a phenotypic correlation of −0.54 of which 86 % was mediated by genetic factors and 14 % by nonshared environmental factors. No quantitative sex-differences were found. Important, the overlap between genetic factors was large while the overlap between environmental factors was

Table 6 Genetic and environmental correlations (95 % CI) for the overlap between SWB, INT, and EXT

r_g				r_e			
	SWB	INT	EXT		SWB	INT	EXT
Males							
SWB	1				1		
INT	−0.64 (−0.72, −0.56)	1			−0.30 (−0.36, −0.24)	1	
EXT	−0.40 (−0.49, −0.31)	0.50 (0.43, 0.57)	1		−0.04 (−0.11, 0.02)	0.35 (0.29, 0.41)	1
Females							
SWB	1				1		
INT	−0.76 (−0.81, −0.71)	1			−0.40 (−0.44, −0.35)	1	
EXT	−0.45 (−0.51, −0.38)	0.60 (0.55, 0.65)	1		−0.14 (−0.20, −0.08)	0.30 (0.25, 0.35)	1

Note r_g genetic correlation, r_e nonshared environmental correlation

Fig. 1 Phenotypic correlations between SWB and psychopathology and the proportions that are accounted for by genetic and nonshared environmental influences
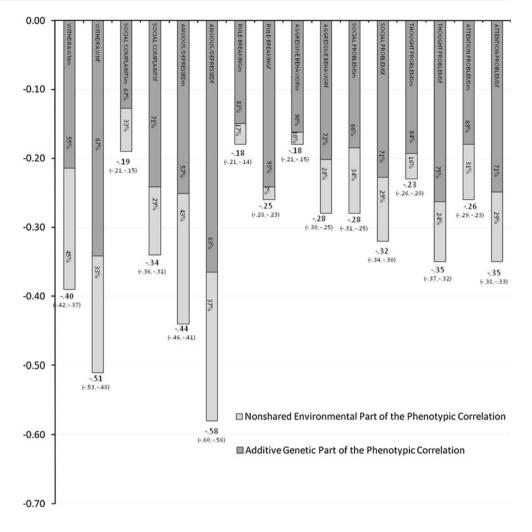

Table 7 Genetic and Environmental Correlations between SWB and psychopathology, with their 95 % confidence intervals

	SWB			
	Males		Females	
	r_g	r_e	r_g	r_e
Withdrawn/depressed	−0.63 (−0.72, −0.53)	−0.27 (−0.32, −0.21)	−0.74 (−0.80, −0.68)	−0.30 (−0.36, −0.26)
Somatic complaints	−0.36 (−0.48, −0.25)	−0.10 (−0.16, −0.04)	−0.53 (−0.61, −0.46)	−0.18 (−0.23, −0.12)
Anxious/depressed	−0.67 (−0.75, −0.58)	−0.30 (−0.35, −0.25)	−0.76 (−0.81, −0.72)	−0.42 (−0.46, −0.37)
Rule-breaking behavior	−0.36 (−0.47, −0.26)	−0.05 (−0.11, 0.01)	−0.47 (−0.54, −0.41)	−0.04 (−0.09, 0.02)
Aggressive behavior	−0.38 (−0.49, −0.29)	−0.03 (−0.09, 0.03)	−0.39 (−0.46, −0.33)	−0.15 (−0.21, −0.10)
Social problem	−0.52 (−0.62, −0.41)	−0.15 (−0.20, −0.09)	−0.50 (−0.58, −0.43)	−0.17 (−0.22, −0.11)
Thought problems	−0.50 (−0.60, −0.40)	−0.06 (−0.13, 0.00)	−0.53 (−0.60, −0.47)	−0.17 (−0.22, −0.11)
Attention problems	−0.47 (−0.59, −0.37)	−0.13 (−0.19, −0.06)	−0.54 (−0.60, −0.47)	−0.20 (−0.25, −0.14)

Note r_g genetic correlation, r_e nonshared environmental correlation

small. We reported a phenotypic correlation of −0.43 for males (58 % A and 42 % E) and of −0.58 for females (66 % A, 34 % E). We also found that the overlap between genetic factors is substantial (♂: r_g = −0.64; ♀: −0.76) in contrast to the overlap in environmental factors (♂: r_e = −0.30; ♀: −0.40).

Nes et al. (Nes et al. 2008) also reported sex difference, with genetic factors being the major source of overlap

between internalizing problems and satisfaction with life in males and shared environment also accounting for overlap in females. The power concern, raised by Nes et al., is covered by our larger sample of twins and additional siblings (Posthuma and Boomsma, 2000).

The unique broad focus of our study with measures of both internalizing as well as externalizing problems and the ability of estimating sex-specific effects provides large added value to field of research on the etiology of the overlap between SWB and psychopathology. The focus on both internalizing as well as externalizing problems reveals that there is also a significant overlap between SWB and externalizing problems, which in both males and females are mainly accounted for by genetic factors.

The finding of the high genetic overlap between SWB and psychopathology is of value in the design and development of new prevention. It indicates that a genetic liability to lower SWB is indicative of a genetic liability to higher psychopathology. The commonality of heritable influences on SWB and psychopathology may lead to the identification of the vulnerable at risk groups prior to any manifestation of psychopathology (Lewinsohn et al. 1991). It could, for example, be suggested that SWB screening can be used as an innovative psychopathology prevention strategy, with the possibility of capturing individuals at risk for the development of psychopathology, before onset of early psychopathological symptoms, based on a low score on SWB.

Since the overlap between psychopathology and SWB is mainly genetic, prevention has to deal with individual differences. This would suggest that population wide launched initiatives to promote SWB need to be reconsidered and probably redesigned to become more tailored for distinct groups of (genetically distinct) individuals to reach its optimal goal. New studies into gene-intervention interaction are highly needed to investigate the effects of genetic differences on intervention effects.

Finally, founded in the field of epidemiology and somatic medicine, it has been proposed that larger benefits to overall public health and mental capital are to be expected when the bell curve of mental health in the human population is shifted a little to the healthy site, the so-called population strategy (Rose, 2008). A relative slight increase in the level of SWB of the bulk of the population, possibly induced by a SWB promotion program, may have a larger preventive effect than targeting the much smaller group of people at high risk. The genetic overlap between SWB and psychopathology, and thus the overlap in vulnerability, makes this theory ground for novel approaches to reduce psychopathology.

While interpreting the results, the following limitation should be kept in mind. We used the Youth Self Report in a normal population sample, resulting in skewed data. Based on the paper by Derks et al. (2004), we decided not to transform the data to approach normality. Use of these skewed data could have resulted in an underestimation of the shared environmental effects and an overestimation of the nonshared environmental effects. We do, however, not expect a large influence on the reported genetic architecture for the overlap between SWB and psychopathology, since SWB is normally distributed. We, furthermore, relied on adolescent self-report data. A multiple rater approach, with for example parental or teacher ratings would have been more reliable (Bartels et al., 2007b), but approaching both the adolescent themselves and their parent would have resulted in a lower response rate and consequently lower power for the current study. Furthermore, in the Dutch school system adolescent do have many teachers at the same time, resulting in the absence of a specific teacher with enough knowledge about the pupil to fulfill the requirement to fill out a teacher report form.

Working with data of a voluntary survey study has the implicit risk of sample selection. In order to check for possible selection on level of psychopathology, we compared our means on the YSR broadband and subscales to those reported in the Dutch manual of the YSR (Verhulst et al. 1997). For some subscale our means are higher (TP and AP), while for the remaining they are slightly lower. It should be noted however that our sample is about eighteen times larger than the community sample.

In conclusion, the relationship between SWB and psychopathology has been identified as being sex-specific and complex, with a large role for shared genetic influences on SWB and psychopathology and more disparate nonshared environmental influences on SWB and psychopathology. As a result of these various forces, SWB and psychopathology cannot be considered as opposing ends of a single mental health continuum. The genetic overlap between SWB and psychopathology justifies the integration of prevention and promotion in the field of Mental Health, as has been suggested by the World Health Organization, however this strategies should be selected and indicated, which could for example be achieved by initial screening on SWB to assess risk for psychopathology.

Appendix

See Tables 8 and 9.

Table 8 Twin and Cross-twin cross trait correlations for SWB and psychopathology with their 95 % confidence intervals

	MZM	DZM/twin brother	MZF	DZF/twin sister	DOS/brother–sister
Subjective well-being	0.33 (0.26, 0.40)	0.19 (0.13, 0.25)	0.45 (0.40, 0.49)	0.29 (0.24, 0.34)	0.20 (0.16, 0.25)
Withdrawn/depressed	0.40 (0.33, 0.45)	0.15 (0.09, 0.21)	0.47 (0.42, 0.52)	0.20 (0.14, 0.25)	0.16 (0.12, 0.19)
SWB-WD	−0.24 (−0.29, −0.19)	−0.09 (−0.13, −0.04)	−0.34 (−0.37, −0.30)	−0.19 (−0.23, −0.15)	−0.11 (−0.14, −0.09)
Somatic complaints	0.35 (0.28, 0.42)	0.18 (0.12, 0.25)	0.45 (0.40, 0.50)	0.22 (0.17, 0.28)	0.18 (0.14, 0.22)
SWB-SC	−0.12 (−0.17, −0.07)	−0.06 (−0.10, −0.01)	−0.22 (−0.26, −0.18)	−0.18 (−0.22, −0.14)	−0.08 (−0.10, −0.05)
Anxious/depressed	0.42 (0.35, 0.47)	0.18 (0.12, 0.25)	0.48 (0.44, 0.53)	0.26 (0.20, 0.31)	0.22 (0.18, 0.26)
SWB-AD	−0.25 (−0.30, −0.19)	−0.14 (−0.19, −0.09)	−0.35 (−0.39, −0.31)	−0.22 (−0.26, −0.18)	−0.13 (−0.16, −0.10)
Rule-breaking behavior	0.45 (0.39, 0.51)	0.23 (0.18, 0.29)	0.55 (0.50, 0.59)	0.24 (0.18, 0.29)	0.18 (0.14, 0.22)
SWB-RB	−0.14 (−0.19, −0.09)	−0.09 (−0.13, −0.05)	−0.24 (−0.27, −0.20)	−0.13 (−0.17, −0.09)	−0.07 (−0.10, −0.04)
Aggressive behavior	0.51 (0.45, 0.51)	0.19 (0.13, 0.25)	0.57 (0.52, 0.61)	0.22 (0.16, 0.27)	0.20 (0.16, 0.24)
SWB-AGG	−0.15 (−0.19, −0.10)	−0.08 (−0.12, −0.04)	−0.19 (0.22, −0.15)	−0.14 (−0.18, −0.10)	−0.08 (−0.12, −0.05)
Social problems	0.41 (0.34, 0.47)	0.15 (0.09, 0.21)	0.48 (0.42, 0.52)	0.19 (0.13, 0.24)	0.17 (0.13, 0.20)
SWB-SP	−0.20 (−0.25, −0.15)	−0.08 (−0.13, −0.04)	−0.21 (−0.25, −0.17)	−0.17 (−0.21, −0.13)	−0.09 (−0.12, −0.06)
Thought problems	0.48 (0.42, 0.54)	0.20 (0.13, 0.26)	0.54 (0.49, 0.58)	0.25 (0.19, 0.30)	0.19 (0.15, 0.23)
SWB-TP	−0.18 (−0.22, −0.12)	−0.09 (−0.14, −0.05)	−0.25 (−0.29, −0.21)	−0.18 (−0.21, −0.13)	−0.12 (−0.15, −0.08)
Attention problems	0.46 (0.40, 0.52)	0.15 (0.09, 0.21)	0.49 (0.44, 0.53)	0.23 (0.17, 0.28)	0.17 (0.13, 0.21)
SWB-AP	−0.16 (−0.21, −0.11)	−0.10 (−0.15, −0.06)	−0.23 (−0.27, −0.19)	−0.18 (−0.22, −0.14)	−0.10 (−0.14, −0.07)

MZM monozygotic males correlation, *DZM/twin brother* dizygotic males and twin sibling correlation, *MZF* monzygotic females correlation, *DZF/twin sister* dizygotic females and twin sister correlation, *DOS/brother–sister* male–female and female–male correlation for both twins and siblings

Table 9 Model fitting results and parameter estimates (95 % CI) based on the best fitting (bold-faced) model

Withdrawn/Depressed	−2LL	df	χ²	df	p
ACE	113043.5	19973			
ACE, no sex differences	113172.8	19982	129.3	9	0.00
AE	**113052.68**	**19979**	**9.17**	**6**	**0.16**

Variance–covariance

	Males A	Males E	Females A	Females E
SWB	0.34 (0.29, 0.40)	0.66 (0.60, 0.71)	0.47 (0.43, 0.51)	0.53 (0.49, 0.57)
With	0.55 (0.45, 0.65)	0.45 (0.35, 0.55)	0.67 (0.61, 0.73)	0.33 (0.27, 0.39)
With	0.36 (0.30, 0.41)	0.64 (0.59, 0.70)	0.44 (0.40, 0.49)	0.56 (0.51, 0.60)

Correlations

	Males A	Females A	Males E	Females E
r_g	−0.63 (−0.72, −0.53)	−0.74 (−0.80, −0.68)		
r_e			−0.27 (−0.32, −0.21)	−0.30 (−0.36, −0.26)

Somatic complaints	−2LL	df	χ²	df	p
ACE	116251.1	19915			
ACE, no sex differences	116662.2	19924	411.09	9	0.00
AE	**116260.41**	**19921**	**9.32**	**6**	**0.16**

Variance–covariance

	Males A	Males E	Females A	Females E
SWB	0.34 (0.29, 0.40)	0.66 (0.60, 0.71)	0.47 (0.43, 0.51)	0.53 (0.49, 0.57)
SC	0.67 (0.46, 0.87)	0.33 (0.13, 0.54)	0.71 (0.62, 0.81)	0.29 (0.19, 0.38)
SC	0.35 (0.29, 0.41)	0.65 (0.59, 0.71)	0.43 (0.38, 0.48)	0.57 (0.52, 0.62)

Correlations

	Males A	Females A	Males E	Females E
r_g	−0.36 (−0.48, −0.25)	−0.53 (−0.61, −0.46)		
r_e			−0.09 (−0.16, −0.04)	−0.18 (−0.23, −0.12)

Anxious/depressed	−2LL	df	χ²	df	p
ACE	125051.1	19935			
ACE, no sex differences	125532.44	19944	481.38	9	0.00
AE	**125060.07**	**19941**	**9.01**	**6**	**0.17**

Variance–covariance

	Males A	Males E	Females A	Females E
SWB	0.34 (0.29, 0.40)	0.66 (0.60, 0.71)	0.46 (0.42, 0.50)	0.54 (0.50, 0.58)
AD	0.57 (0.47, 0.66)	0.43 (0.34, 0.53)	0.63 (0.57, 0.68)	0.37 (0.32, 0.43)
AD	0.40 (0.34, 0.45)	0.60 (0.55, 0.66)	0.49 (0.45, 0.53)	0.51 (0.47, 0.55)

Correlations

	Males A	Females A	Males E	Females E
r_g	−0.67 (−0.75, −0.58)	−0.76 (−0.81, −0.72)		
r_e			−0.30 (−0.35, −0.25)	−0.42 (−0.46, −0.37)

Rule-breaking behavior	−2LL	df	χ²	df	p
ACE	112963.5	20019			
ACE, no sex differences	113036.4	20028	72.87	9	0.00
AE	**112975.4**	**20025**	**11.94**	**6**	**0.06**

Table 9 continued

Note: This is a rotated, multi-section table. It is reconstructed below by behavior. Each behavior shows a model-fitting section (ACE / ACE, no sex differences / AE) and variance–covariance / correlation estimates for Males and Females. Bold values indicate the best-fitting (AE) model.

Rule-breaking behavior

Model fitting

	−2LL	df	χ²	df	p
ACE	128009.5	19963			
ACE, no sex differences	128139.5	19972	130.06	9	0.00
AE	**128018.06**	**19969**	**8.57**	**6**	**0.20**

Variance–covariance and correlations

	Males			Females		
	A: SWB	RB		A: SWB	RB	
SWB	0.34 (0.28, 0.39)			0.47 (0.43, 0.51)		
RB	0.83 (0.61, 1.05)	0.44 (0.38, 0.49)		0.93 (0.81, 1.04)	0.52 (0.47, 0.56)	
	E: SWB	RB		E: SWB	RB	
SWB	0.66 (0.61, 0.72)			0.53 (0.49, 0.57)		
RB	0.17 (−0.06, 0.40)	0.56 (0.51, 0.62)		0.07 (−0.04, 0.19)	0.48 (0.44, 0.53)	
r_g	−0.36 (−0.47, −0.26)			−0.47 (−0.54, −0.41)		
r_e	−0.05 (−0.11, 0.0)			−0.04 (−0.09, 0.02)		

Aggressive behavior

Variance–covariance and correlations

	Males			Females		
	A: SWB	AGG		A: SWB	AGG	
SWB	0.34 (0.28, 0.39)			0.47 (0.42, 0.51)		
AGG	0.90 (0.69, 1.12)	0.47 (0.41, 0.52)		0.72 (0.61, 0.82)	0.54 (0.49, 0.58)	
	E: SWB	AGG		E: SWB	AGG	
SWB	0.66 (0.61, 0.72)			0.53 (0.49, 0.58)		
AGG	0.10 (−0.12, 0.31)	0.37 (0.31, 0.43)		0.28 (0.18, 0.39)	0.46 (0.42, 0.51)	
r_g	−0.38 (−0.49, −0.29)			−0.39 (−0.46, −0.33)		
r_e	−0.03 (−0.09, 0.03)			−0.15 (−0.21, −0.10)		

Social problems

Model fitting

	−2LL	df	χ²	df	p
ACE	111946.69	20032			
ACE, no sex differences	111999.56	20041	52.87	9	0.00
AE	**111956.21**	**20038**	**9.53**	**6**	**0.15**

Variance–covariance and correlations

	Males			Females		
	A: SWB	SP		A: SWB	SP	
SWB	0.34 (0.29, 0.40)			0.46 (0.42, 0.50)		
SP	0.66 (0.52, 0.79)	0.37 (0.31, 0.43)		0.71 (0.61, 0.81)	0.44 (0.39, 0.49)	
	E: SWB	SP		E: SWB	SP	
SWB	0.65 (0.60, 0.71)			0.54 (0.50, 0.58)		
SP	0.34 (0.21, 0.48)	0.63 (0.57, 0.69)		0.29 (0.19, 0.39)	0.56 (0.51, 0.61)	
r_g	−0.52 (−0.62, −0.41)			−0.50 (−0.58, −0.43)		
r_e	−0.15 (−0.20, −0.09)			−0.17 (−0.22, −0.11)		

Thought problems

Model fitting

	−2LL	df	χ²	df	p
ACE	111040.12	20015			
ACE, no sex differences	111122.22	20024	82.1	9	0.00
AE	**111049.71**	**20021**	**9.58**	**6**	**0.14**

Variance–covariance (SWB, TP) — Males and Females (E, SWB …) [table continues]

Table 9 continued

Thought problems

	Males		Females	
	SWB	TP	SWB	TP
A				
SWB	0.34 (0.29, 0.40)		0.47 (0.43, 0.51)	
TP	0.84 (0.68, 1.0)	0.44 (0.39, 0.50)	0.75 (0.67, 0.84)	0.56 (0.50, 0.61)
Correlations r_g	−0.50 (−0.60, −0.40)		−0.53 (−0.60, −0.47)	
E				
SWB	0.66 (0.60, 0.71)		0.53 (0.49, 0.57)	
TP	0.16 (0.00, −0.32)	0.51 (0.46, 0.55)	0.25 (0.16, 0.33)	0.49 (0.45, 0.54)
Correlations r_e	−0.06 (−0.13, 0.00)		−0.17 (−0.22, −0.11)	

Attention problems

	−2LL	df	χ^2	df	p
ACE	119862.79	20003			
ACE, no sex differences	119934.98	20012	72.18	9	0.00
AE	**119878.01**	**20009**	**15.21**	**6**	**0.02**

	Males		Females	
	SWB	AP	SWB	AP
Variance–covariance **A**				
SWB	0.34 (0.29, 0.40)		0.54 (0.50, 0.58)	
AP	0.67 (0.52, 0.82)	0.40 (0.34, 0.45)	0.71 (0.62, 0.79)	0.47 (0.43, 0.52)
Correlations r_g	−0.47 (−0.58, −0.36)		−0.54 (−0.60, −0.47)	
E				
SWB	0.66 (0.60, 0.71)		0.46 (0.42, 0.50)	
AP	0.33 (0.18, 0.48)	0.53 (0.49, 0.57)	0.29 (0.21, 0.38)	0.53 (0.48, 0.57)
Correlations r_e	−0.14 (−0.20, −0.07)		−0.20 (−0.25, −0.14)	

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