Heritability and Genome-Wide Linkage Scan of Subjective Happiness

Meike Bartels,[1,2*] Viatcheslav Saviouk,[1] Marleen H. M. de Moor,[1] Gonneke Willemsen,[1] Toos C. E. M. van Beijsterveldt,[1] Jouke-Jan Hottenga,[1] Eco J. C. de Geus[1] and Dorret I. Boomsma[1]

[1] *Department of Biological Psychology, VU University, Amsterdam, the Netherlands*
[2] *EMGO+ Institute, VU University Medical Center, Amsterdam, the Netherlands*

Causes of individual differences in happiness, as assessed with the Subjective Happiness Scale, are investigated in a large of sample twins and siblings from the Netherlands Twin Register. Over 12,000 twins and siblings, average age 24.7 years (range 12 to 88), took part in the study. A genetic model with an age by sex design was fitted to the data with structural equation modeling in Mx. The heritability of happiness was estimated at 22% for males and 41% in females. No effect of age was observed. To identify the genomic regions contributing to this heritability, a genome-wide linkage study for happiness was conducted in sibling pairs. A subsample of 1157 offspring from 441 families was genotyped with an average of 371 micro-satellite markers per individual. Phenotype and genotype data were analyzed in MERLIN with multipoint variance component linkage analysis and age and sex as covariates. A linkage signal (logarithm of odds score 2.73, empirical *p* value 0.095) was obtained at the end of the long arm of chromosome 19 for marker D19S254 at 110 cM. A second suggestive linkage peak was found at the short arm of chromosome 1 (LOD of 2.37) at 153 cM, marker D1S534 (empirical *p* value of .209). These two regions of interest are not overlapping with the regions found for contrasting phenotypes (such as depression, which is negatively associated with happiness). Further linkage and future association studies are warranted.

Keywords: happiness, heritability, genome-wide linkage, twin-sibling

Pursuing happiness is a goal for nearly every human being and striving for happiness is one of the major life purposes. It is therefore not remarkable that research into the causes of individual differences of happiness and subjective wellbeing has become more and more part of the international research agenda (Layard, 2010). In a large sample of adolescent twins and their siblings subjective happiness (HAP) has been found to be part of the overall construct of subjective wellbeing (SWB) and the broad-sense heritability of HAP was estimated at 40% (Bartels & Boomsma, 2009). The remaining variance was accounted for by nonshared environmental factors. Previous research into the causes of variance in SWB is in line with this finding and heritability estimates in the range of 40 to 50% are reported (Lykken & Tellegen, 1996; Nes et al., 2006; Røysamb et al., 2002; 2003; Stubbe et al., 2005; Tellegen et al., 1988).

The significant heritability of SWB led to the formulation of the set point theory (Lykken, 1999), which hypothesizes that people adapt to major life events, both positive and negative, and that happiness more or less stays constant around an individuals set point throughout life, even if it is occasionally perturbed (see, for example, the often cited study by Brickman et al. (1978) on winning a lottery and paralysis victims). This hypothesis predicts that the heritability would remain stable throughout the life span, but formal testing of this idea is lacking.

In this article, causes of individual differences in HAP as a function of age and sex are estimated in a large data set with twins and siblings. Happiness was assessed with the subjective happiness scale (Lyubomirsky & Lepper, 1999), which is a widely used instrument in epidemiological studies on happiness and wellbeing. Based on the significant heritability found, we performed the first genome-wide linkage scan for happiness in 1157 individuals from 441 families.

Methods

Participants

This study brings together data collection from the Adult Netherlands Twin Register (ANTR, for details see Boomsma et al., 2006) and the Young Netherlands Twin Register (YNTR, for details see Bartels et al., 2007). The adult twin-family data collection is part of an ongoing study on health and lifestyle. Surveys are sent to the twin families every two to three years. Happiness data were collected in the sixth survey (2002). The response rate was 32% for twins and 40% for siblings (for details on the 2002 wave of

Received 10 February, 2010; accepted 3 March, 2010.

Address for correspondence: Meike Bartels, PhD, Department of Biological Psychology, room 2B-47, 1081 BT Amsterdam, The Netherlands. Email: m.bartels@psy.vu.nl

survey collection see Stubbe et al., 2005). The adolescent data form part of the longitudinal data collection of the YNTR. Young twins are registered at birth and when they reach the age of 13 their parents are asked for informed consent to send the twins and their non-twin siblings a survey. If their parents consented, 14-, 16-, and 18-year-old twins and their non-twin siblings received an online or paper and pencil self-report survey about the development of behavior, lifestyle, and wellbeing. When twins and siblings did not return the survey on time they were contacted by mail for a first reminder and next they were contacted by phone for a second reminder. The response rate was 43%.

Phenotypic data on happiness were available for 12,279 subjects (9,604 twins and 2,675 siblings; 4,805 males and 7,474 females) from 5970 families. Ages range from 12 to 88, with a mean of 24.7 and a standard deviation of 11.7. Half of the sample was aged 12 to 19 and 10% of the sample was 40 years or older. Siblings were included in the analyses with a maximum of two siblings (one brother and one sister) per family. When more siblings were available, the sibling closest in age to the twin was selected for analyses. Zygosity of same-sex twins was determined by DNA typing for 37.5% of the same-sex twin pairs. For the other same-sex twins, zygosity was based questionnaire items. Agreement between zygosity based on these items and zygosity based on DNA was 97% (Rietveld et al., 2000; Willemsen et al., 2005). Zygosity information was missing for 349 families, since for these families only non-twin siblings participated in the study.

The final sample of 5621 families consisted of 844 families with MZM twins (627 complete pairs), 645 families with DZM twins (420 complete pairs), 1586 families with MZF twins (1181 complete pairs), 1027 families with DZF twins (690 complete pairs), 788 families with DOSmf twins (486 complete pairs), and 731 families with DOSfm twins (450 complete pairs). Data from all twin-sibling pairs with information on zygosity were used in the heritability analyses. A subsample was genotyped for linkage analyses (n = 1157 individuals from 441 families).

About 50% of the sample was between 12 and 19 years of age. For the estimation of the twin–sibling correlations and genetic and environmental components the sample was split into two groups. The first group consisted of twins aged 19 or younger with their non-twin siblings (mean age twins was 16.16 and mean age siblings was 17.91) and the second group consisted of twins aged 20 and older with their non-twin siblings (mean age twins was 32.42 and mean age siblings was 33.21). A detailed sample description for the total sample and the two age groups is provided in Tables 1 and 2.

Table 1

Distribution of the Sample Over Zygosity and Age

	Total sample	Younger sample	Older sample
MZM pairs	844[1] (627)[2]	433 (386)	411 (241)
DZM pairs	645 (420)	370 (299)	275 (121)
MZF pairs	1586 (1181)	616 (545)	970 (636)
DZF pairs	1027 (690)	448 (381)	579 (317)
DOS mf pairs	788 (493)	426 (335)	362 (158)
DOS fm pairs	731 (458)	386 (306)	345 (152)
Brothers	890	511	379
Sisters	1129	601	528

Note: [1] = number of families; [2] = total number of complete pairs.

Phenotype

Subjective Happiness (HAP) was assessed with the 4-item Subjective Happiness Scale (Lyubomirsky & Lepper, 1999). On a 7-point Likert scale, individuals had to indicate whether they agreed or disagreed with statements like: 'On the whole I'm a happy person' and 'On the whole, I'm not very happy'. Scores can range from 4 to 28 and are slightly skewed to the right. The four items showed good to excellent internal consistency (alpha's ranged from 0.79 to 0.94) and the test–retest reliability was .72 (Lyubomirsky & Lepper, 1999). The mean level in Dutch adolescents (twins and siblings) was 23.1 (Bartels & Boomsma, 2009).

Genotyping and Linkage Sample

DNA was extracted from either whole blood or buccal swabs using standard protocols. Samples were genotyped by the Mammalian Genotyping Service in Marshfield and the Molecular Epidemiology Section, Leiden University Medical Centre. Genotype data from these screens were combined. Pedigree relations were checked with Graphic Representation of Relationships. Errors of Mendelian inheritance were detected with Pedstats (Wigginton & Abecasis, 2005). Markers and samples were removed if the total error rate was >1%; in all of the other cases, the erroneous genotypes were set as unknown. Unlikely recombinants were detected using Merlin (Abecasis et al., 2002), and erroneous genotypes were removed with Pedwipe (Abecasis et al., 2002).

Families consisted of sibling pairs (DZ twin pairs, and/or twin pairs with one or more siblings). Individuals with >200 microsatellite markers genotyped were included in the linkage analysis.

The NTR linkage sample consisted of 711 families with 1438 founders and 1974 nonfounders (3412 subjects in total). 282 of those families had all founders genotyped, 138 families had one founder with missing genotype; 285 families had two founders with missing genotypes; five families had three founders with missing genotypes; and one complex family had four founders with missing genotypes. Data from the entire sample of 711 families were used for allele frequency estimation.

Out of 711 families in the NTR linkage sample, 122 families did not have a single subject phenotyped for HAP and another 129 families had only one

subject phenotyped and therefore did not contribute to the linkage signal (total 251). Of the remaining 460 families; 250 had two subjects phenotyped (19 of those families had just phenotyped MZ twins and did not contribute to the signal), 161 families had three subjects; 40 families had four subjects; six families had five subjects; and three families had respectively six, seven and nine subjects phenotyped.

Families that contributed to the linkage signal were those that had at least two non-cloned nonfounder subjects phenotyped. Since 129 families had only one subject phenotyped and therefore did not contribute to the linkage signal and 19 families had only a MZ pair phenotyped, the final sample for the linkage analysis consisted of 1157 subjects from 441 families (see Table 2 for a comparison of these individuals to the non-linkage and total sample). The average family size was 5.06 (4–12 siblings).

For the linkage analysis 757 autosomal and 22 X-linked microsatellite markers with an average spacing of 4.78 cM were used. The average number of markers genotyped was 371 (range 250–782) per individual. The Haldane mapping function was utilized for the variance components linkage analysis.

Genetic Analysis

Descriptive Statistics, Twin-Sib Correlations, and Genetic Modeling

Age and sex differences in means were tested with structural equation modeling in Mx (Neale et al., 2006). Twin and sibling correlations were estimated to summarize familial resemblance for HAP for the two age groups separately.

Data of twins and siblings provided the opportunity to decompose the variance of a trait into additive and non-additive genetic, shared environmental, and non-shared environmental components. Additive genetic factors (A) represent the sum of the effects of alleles over all loci that influence the trait. Non-additive genetic factors concern interactions between alleles, which can represent interaction between alleles at the same locus (D: dominance) or interaction between alleles at the different loci (epistasis). Common environmental factors (C) are shared by members of the same family and non-shared environmental factors (E) are unique to an individual. Non-additive genetic effects and shared environmental effects are confounded in the classical twin (sibling) design. Based on the literature (e.g. Bartels & Boomsma, 2009) and the twin-sibling correlation structure we considered models that include additive genetic, non-additive genetic, and non-shared environmental effects only.

Genetic Architecture

In order to test for age effects, sex-differences, and possible effects of age by sex interaction in the genetic architecture of HAP, genetic modeling was started with a 10 group design (five zygosity groups x two age groups). Model fitting was started with an ADE model for males and females and for younger and older twins with their siblings. The first significance of dominant genetic effects was tested by constraining the effect to zero in all groups. Next, it was tested whether influences of A and E could be constrained to be equal within sex over age groups (i.e., are genetic and environmental variances equal for younger and older males and equal for younger and older females (age effect)). Further, it was tested whether A and E of the older males group could be set equal to the A and E components of the younger males and females (age x sex interaction). Finally, it was tested whether the genetic architecture was similar for males and females by constraining the variance components to be equal in males and females (sex effect).

Raw-data Maximum Likelihood estimation of parameters in Mx (Neale et al., 2006) was used. Nested submodels were compared by hierarchic χ^2

Table 2

Subjective Happiness Observed Means Levels and Distribution of Sex and Age in the Total, Non-Linkage, and Linkage Sample

	Total sample			Non-linkage sample			Linkage sample		
	N	age	HAP	*N*	Age	HAP	*N*	age	HAP
Total	12279	24.69 (11.66)	22.71 (4.36)	11122	23.75 (11.14)	22.71 (4.38)	1157	33.75 (12.58)	22.76 (4.23)
Males	4805	23.52 (11.47)	23.02 (4.18)	4367	22.5 (10.77)	23.02 (4.19)	438	33.72 (13.19)	23.08 (4.15)
young	2734	16.4 (2)	23.25 (4.26)						
old	1967	32.96 (12.06)	22.78 (4.04)						
Females	7474	25.44 (11.71)	22.51 (4.46)	6755	24.55 (11.3)	22.52 (4.49)	719	33.78 (12.21)	22.55 (4.27)
young	3433	16.56 (2.07)	22.84 (4.43)						
old	3811	33.11 (11.28)	22.23 (4.48)						

tests. The χ^2 statistic is computed by subtracting –2LL (log-likelihood) for the full model from that for a reduced model ($\chi^2 = -2LL_1 - (-2LL_0)$). Given that the reduced model is correct, this statistic is χ^2 distributed with degrees of freedom (*df*) equal to the difference in the number of parameters estimated in the two models ($\Delta df = df_1 - df_0$).

Linkage Analysis and Empirical Significance

MERLIN 1.1.2 software (Abecasis et al., 2002) was used to estimate allele frequencies of the autosomal microsatellite markers and for the autosomal multipoint variance component linkage analysis with age and sex as covariates. The MINX modification of MERLIN was used for the chromosome X analysis. The genome scan was performed at 1 cM resolution with a total of 3546 autosomal and 189 X-linked positions analyzed for linkage. For the empirical evaluation of the results, 1000 nonlinked replicates of the autosomal genome were generated that preserved the allelic frequencies, marker distances and pattern of missing data using MERLIN. Each replicate was analyzed at 1 cM resolution and the largest LOD score from each analysis was extracted from the output into a LOD Table for the empirical evaluation.

Results

Descriptive statistics for HAP are presented in Table 2. The observed mean for the total sample of 12279 individuals is 22.7 (*SD* 4.4). A significant age by sex interaction effect is found for mean levels of HAP. For both males and females younger individuals are happier (M: $\chi^2_1 = 15.47$, $p = .00$; F: $\chi^2_1 = 19.38$, $p = .00$). Furthermore, within age groups males score higher on HAP than females (Older group: $\chi^2_1 = 14.83$, $p = .00$; Younger group: $\chi^2_1 = 18.32$, $p = .00$). Thus, younger males are most happy, followed by younger females, older males, and older females. No significant differences in HAP are observed between individuals who participated in the linkage analysis and the remaining of the sample ($p = .75$) (see Table 2). Mean age is higher for individuals who participated in the linkage analysis ($p = .00$).

Twin-twin and twin-sib correlations with the 95% confidence interval are presented in Table 3. DZ twin correlations could be set equal to twin-sib correlations. Since the MZ correlations are twice or even more than twice the DZ-sib correlations genetic influences on HAP are to be expected. MZF and DZF-sibling correlations are almost identical in the two age groups. However, MZM correlations are different in the two age groups. Note that the opposite-sex correlations are similar to the same-sex correlations, so there is no evidence for qualitative sex differences. Based on the twin-sibling correlations, model fitting was started with a model with both additive genetic and nonadditive genetics factors (ADE) and separate estimates for males and females and the young and old groups.

Table 3

Twin-Sibling Correlations (95% CI) for Happiness

	Young (95% CI)	Old (95% CI)
MZF	.42 (.35–.48)	.43 (.37–.48)
DZF/ F-F	.17 (.10–.23)	.17 (.10–.24)
MZM	.19 (.10–.28)	.29 (.17–.39)
DZM/ M-M	.08 (.01–.15)	.10 (.00–.21)
DOS/M-F	.18 (.13–.23)	.13 (.06–.19)

The dominant genetic component (D) could be dropped from the model for both sexes and age groups without a significant deterioration of model fit ($\chi^2_4 = 3.14$, $p = .54$). As expected from the MZF and DZF-sibling correlations, additive genetic and nonshared environmental effects could be constrained to be equal in the two age groups for women ($\chi^2_2 = .002$, $p = .99$). A and E of the older men could not be constrained to be equal to the genetic and environmental component in women ($\chi^2_4 = 27.95$, $p = .00$), so no sex by age interaction was observed. For men, genetic and environmental variance components could be constrained to be equal over age ($\chi^2_2 = 5.28$, $p = .07$). Significant sex differences in heritability were found ($\chi^2_4 = 37.06$, $p = .00$). In men individual differences in HAP were for 22% (CI: 16-28) accounted for by additive genetic effects, while in women 41% (CI: 37–45) of the variance in HAP was explained by additive genetic effects. The remaining variance was accounted for by nonshared environmental effects (78% (CI: 72–84) in men and 59% (CI: 55–63) in women.

Figure 1a shows the linkage results for the analysis of HAP. The highest LOD-score of 2.73 was found on chromosome 19 at 110 cM, the last marker on chromosome 19, D19S254 (empirical *p* value of .095; see Figure 1b). The previous marker, at 108.09 cM, was D19S210. In addition, a LOD score of 2.37 was found on chromosome 1, 153 cM, marker D1S534 (95% confidence interval: 151 cM, Marker D1S252 and 159 cM, marker D1S498; empirical *p* value of .209; see Figure 1c). To achieve a significance level of .05, a LOD score of >2.99 would be required for this dataset. To test if the LOD score for chromosome 19 was not inflated due to multipoint IBD information at the telomeric end of the chromosome, we conducted a two-point linkage analysis which resulted in a LOD-score of 2.49 for marker D19S254.

Discussion

This paper describes the largest genetic epidemiological study into the heritability of happiness and reports that individual differences in happiness are for 22% accounted for by genetic factors in men and for 41% in women. The remaining variance is accounted for by nonshared environmental influences. Heritability is equal for the younger (<20) and older (>19) cohort and no sex by age interaction is found. Additional analyses

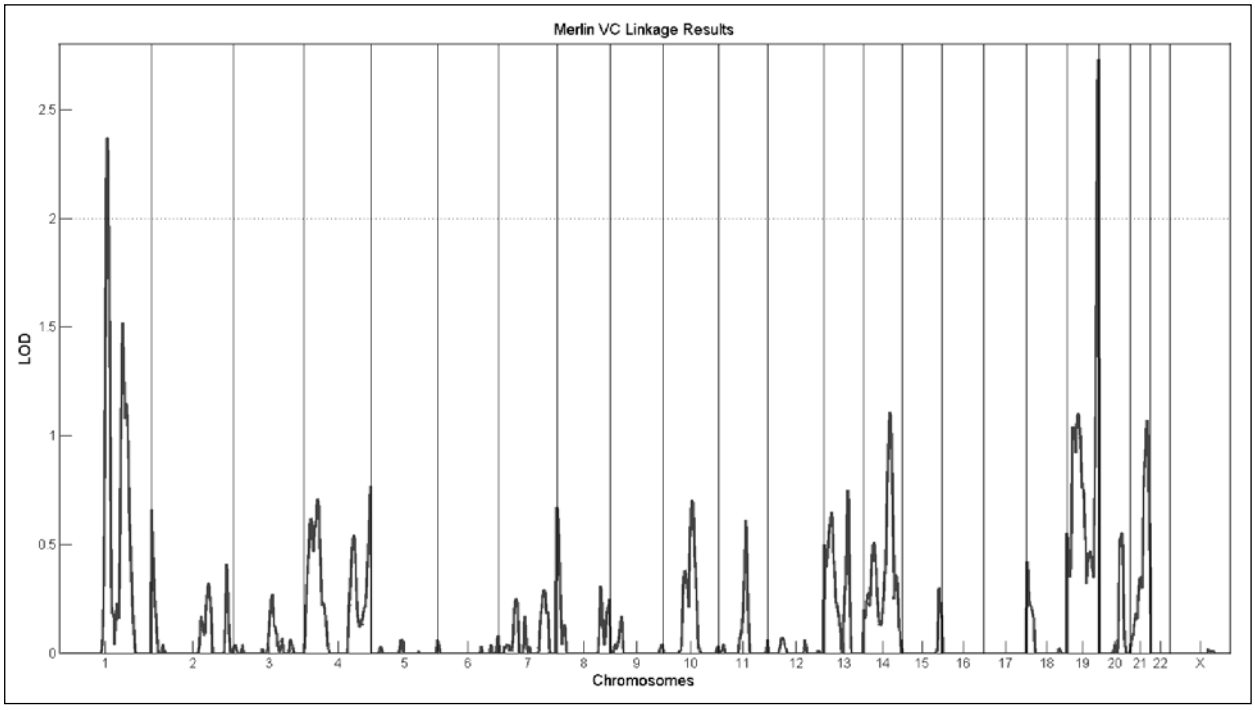

Figure 1a

Merlin VC linkage results across the whole genome.

(not reported but available upon request) with age as a continuous variable confirmed that the effect of age is absent. The heritability as reported for females is in line with a previous study using a partly overlapping sample of adolescent twins and siblings (Bartels & Boomsma, 2009). The latter study reported that happiness is influenced by an underlying set of genes that also influences satisfaction with life and quality of life, indicating the existence of an umbrella construct such as Subjective Wellbeing (SWB). Studies into the causes of individual differences in SWB also report heritabilities in the range of 40% (Lykken & Tellegen, 1996; Nes et al., 2006; Røysamb et al., 2002; 2003; Stubbe et al., 2005; Tellegen et al., 1988). The remaining variance is, in all studies, found to be accounted for by nonshared environmental influences. The heritability for males in this study is lower than reported in earlier studies. Sex differences in heritability estimates have been previously been found by Røysamb et al. (2002; 2003). They also reported larger heritabilities for women (54%) than for men (46%). The differences were, however, much smaller than found in the current study. Suggested 'environmental' correlates of SWB are income (Clark et al., 2008; Stutzer, 2004), education (Blanchflower & Oswald, 2004; Bukenya et al., 203), unemployment (Clark & Oswald, 1994; Lucas et al., 2004), religion (Ciarrochi & Deneke, 2005; Francis & Kaldor, 2002), exercise (Biddle & Ekkekakis, 2005; Stubbe et al., 2007), marriage (Brown, 2000), friendship (Lelkes, 2006; Pichler, 2006), and economic/political environment (Di Tella et al., 2003; Kahneman et al., 2004). Effect sizes, though, are rather small, taken together these effects account for about a quarter of the variance in SWB. The absence of an age effect on the heritability supports the set point theory (Lykken, 1999). It should be noted however, that more complex effects of age (e.g., nonlinear effect) cannot be ruled out.

This is the first study aiming to identify genomic regions of interest for happiness. Suggestive linkage was found for genomic regions on chromosome 1 and 19. The region on chromosome 19 (q13.43) harbors several protein coding genes (e.g., DUXA, AURKC, USP29, and several zinc finger protein genes), but none of these genes play a plausible role in explaining individual differences in HAP. The region on chromosome 1 (p12) also harbors several protein-coding genes (for example WARS2, PI4KB and SCA19), but these also do not play a plausible role in the variance of happiness. Although happiness is often seen as the counterpart of depression, the regions with suggestive linkage for happiness have not been reported for depression so far (e.g., Middeldorp et al., 2008). Furthermore the regions on chromosome 1 and 19 have also not been suggested in a linkage scan for borderline personality disorder features (Distel et al. 2008) and a linkage scan for Neuroticism (Wray et al., 2008). A next step would be to run a genome wide association analyses for happiness.

The significant heritability, two nearly significant linkage peaks in a relative small sample, and the finding that these linkage peaks do not overlap with

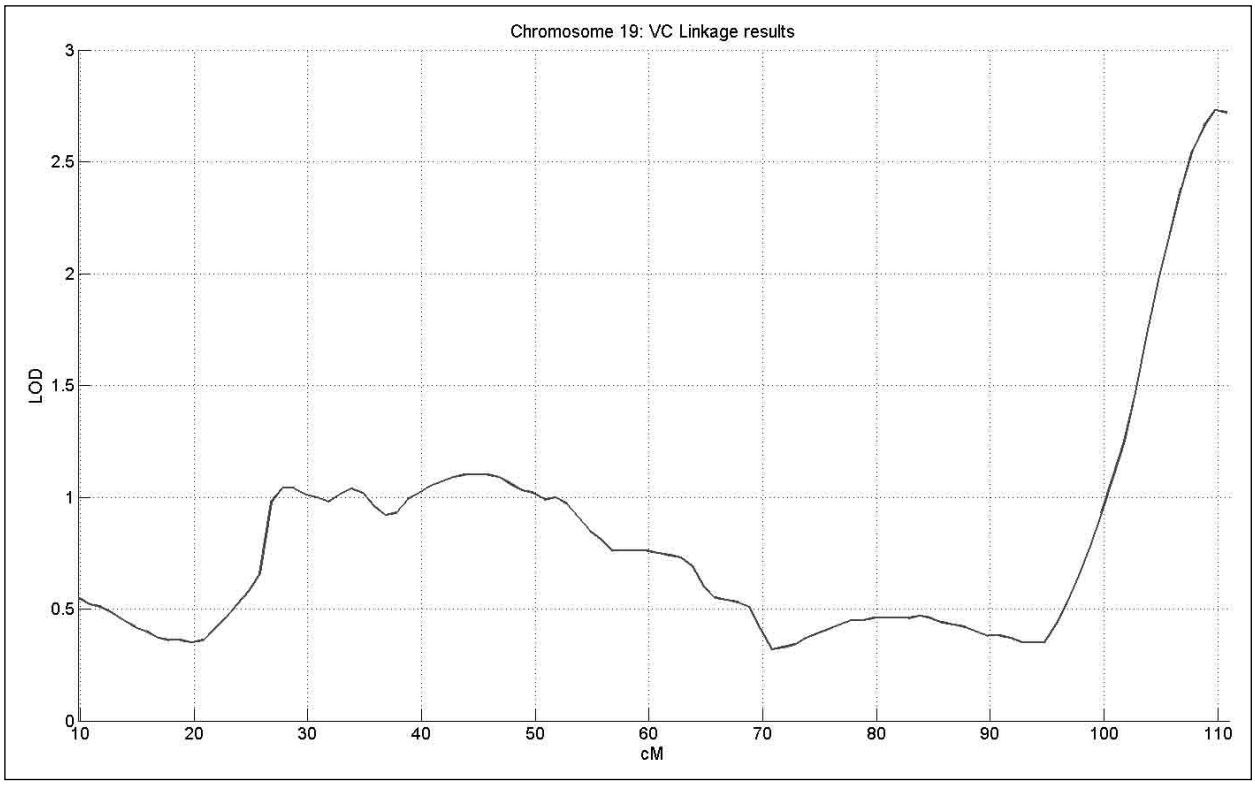

Figure 1b
Merlin VC linkage results for Chromosome 19.

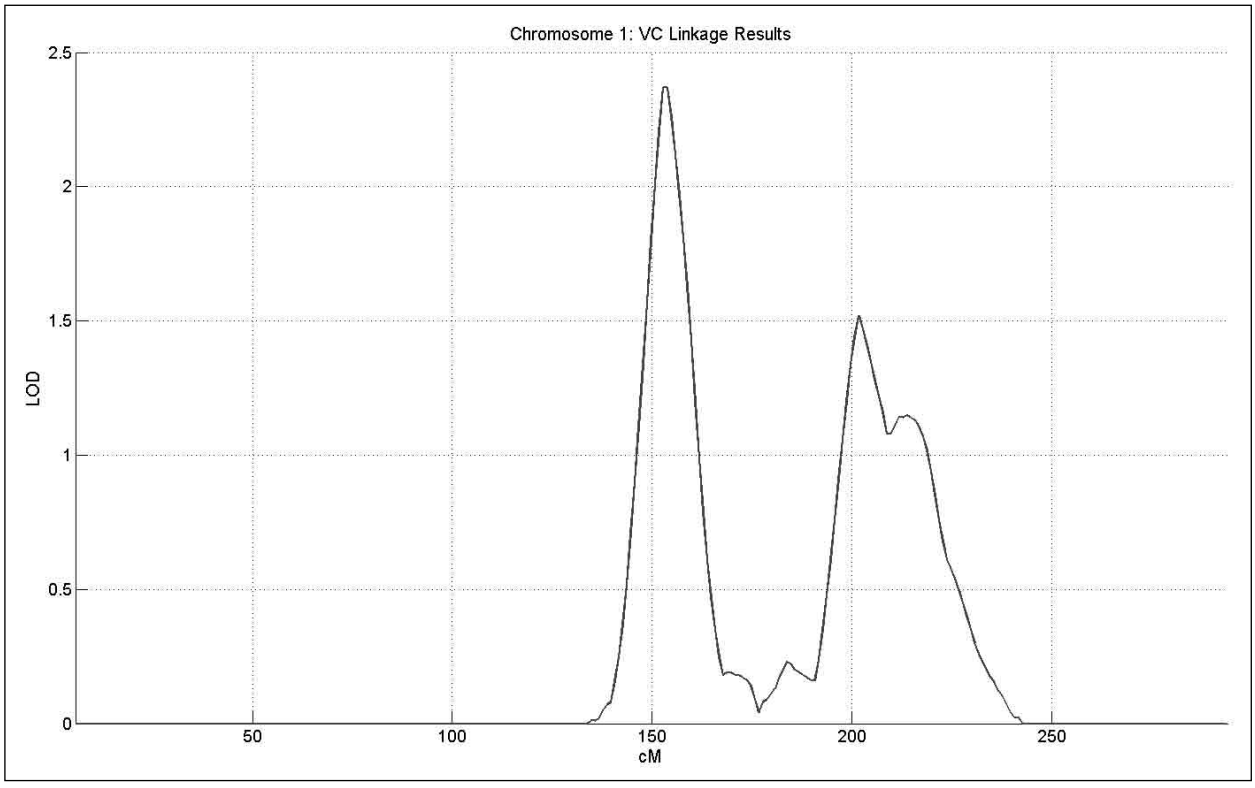

Figure 1c
Merlin VC linkage results for Chromosome 1.

finding for obvious counterparts such as depression, makes happiness an interesting phenotype to study for gaining new insight into the causes of individual differences in overall human wellbeing.

Acknowledgments

This study was supported by Spinozapremie (NWO/SPI 56-464-14192) and Twin-Family Database for Behavior Genetics and Genomics (NWO 480-04-004). Data collection in the adolescent twin families was supported by NWO/VENI and NWO/Bilateral grants awarded to M.B.

M.B. is financially supported by a senior fellowship of the EMGO+ institution.

S. S. is supported by an EU Marie Curie Research Training Networks grant (EUTwinS, PI Igor Nenadic).

We thank Drs J Weber (Mammalian Genotyping Service, Marshfield) and PE Slagboom (Molecular Epidemiology Section, Leiden University Medical Centre, The Netherlands) for microsatellite genotyping. Analyses were carried out on the Genetic Cluster Computer (http://www.geneticcluster.org) which is financially supported by the Netherlands Scientific Organization (NWO480-05-003) and the VU University Faculty of Psychology and Education.

References

Abecasis, G. R., Cherny, S. S., Cookson, W. O., & Cardon, L. R. (2002). Merlin—rapid analysis of dense genetic maps using sparse gene flow trees. *Nature Genetics, 30*, 97–101.

Bartels, M., Van Beijsterveldt, C. E. M., Derks, E. M., Stroet, T. M., Polderman, J. C., Hudziak, J. J., et al. (2007). Young Netherlands Twin Register (Y-NTR): A longitudinal multiple informant study of problem behavior. *Twin Research and Human Genetics*, *10*, 3–11.

Bartels, M., & Boomsma, D. I. (2009). Born to be happy? The etiology of Subjective Wellbeing. *Behavior Genetics*, *39*, 605–615.

Biddle, S. J. H., & Ekkekakis, P. (2005). Physical active lifestyles and well-being. In F. A. Huppert, N. Baylis, & B. Keverne (Eds.), *The science of well-being* (pp. 141–168). Oxford: Oxford University Press.

Blanchflower, D. G., & Oswald, A. J. (2004). Well-being over time in Britain and the USA. *Journal of Public Economics*, *88*, 1359–1386.

Boomsma, D. I., Geus, de E. J. C., Vink, J. M., Stubbe, J. H., Distel, M. A., Hottenga, J. J., et al. (2006). Netherlands Twin Register: From twins to twin families. Twin *Research and Human Genetics*, *9*, 849–857.

Brickman, P., Coates, D., & Janoff-Bulman, R. (1978). Lottery winners and accident victims: Is happiness relative? *Journal of Personality and Social Psychology*, *36*, 917–27.

Brown, S. L. (2000). The effect of union type on psychological well-being: Depression among cohabitors versus marrieds. *Journal of Health and Social Behaviour*, *41*, 241–255.

Bukenya, J. O., Gebremedhin, T. G., & Schaeffer, P. V. (2003). Analysis of rural quality of life and health: A spatial approach. *Economic Development Quarterly*, *17*, 280–293.

Ciarrochi, J. W., & Deneke, E. (2005). Happiness and the varieties of religious experience: Religious support, practices, and spirituality as predictors of well-being. *Research in the Social Scientific Study of Religion*, *15*, 209–223.

Clark, A., Frijters, P., & Shields, M. A. (2008). A survey of the income happiness gradient. *Journal of Economic Literature*, *46*, 95–144.

Clark, A., & Oswald, A. (1994). Unhappiness and unemployment. *The Economic Journal*, *104*, 648–659.

Distel, M. A., Hottenga, J. J., Trull, T. J., & Boomsma, D. I. (2008). Chromosome 9: Linkage for borderline personality disorder features. *Psychiatric Genetics, 18,* 302–307.

Di Tella, R., MacCulloch, R., & Oswald, A. (2003). The macroeconomics of happiness. The *Review of Economics and Statistics*, *85*, 809–827.

Francis, L. J. & Kaldor, P. (2002). The relationship between psychological well-being and Christian faith and practice in an Australian population sample. *Journal for the Scientific Study of Religion*, *49*, 179–184.

Kahneman, D., Krueger, A. B., Schkade, D. A., Schwarz, N., & Stone, A. A. (2004). Toward national well-being accounts. *American Economic Review*, *94*, 429–434.

Layard, R. (2010). Measuring Subjective Well-Being. *Science*, *327*, 534–535.

Lelkes, O. (2006). Knowing what is good for you. Empirical analysis of personal preferences and the 'objective good'. *The Journal of Socio-Economics*, *35*, 285–307.

Lucas, R. E., Clark, A. E., Georgellis, Y., & Diener, E. (2004). Unemployment alters the set point for life satisfaction. *Psychological Science*, *15*, 8–13.

Lyubomirsky, S., & Lepper, H. S. (1999). A measure of subjective happiness: Preliminary reliability and construct validation. *Social Indicators Research*, *46*, 137–155.

Lykken, D. & Tellegen, A. (1996). Happiness is a stochastic phenomenon. *Psychological Science*, *7*, 186–189.

Lykken, D. (1999). *Happiness: What studies of twins show us about nature, nurture, and the happiness set-point*. New York: Golden Books.

Middeldorp, C. M., Hottenga, J. J., Slagboom, P. E., Sullivan, P. F., Geus de, E. J. C., Posthuma, D., Willemsen, G.,& Boomsma, D. I. (2008). Linkage on chromosome 14 in a genome-wide linkage study of a broad anxiety phenotype. *Molecular Psychiatry*, *13*, 84–89.

Neale, M. C., Boker, S. M., Xie, G., & Maes, H. H. (2006). *Mx: Statistical modeling* (7th ed.) Richmond: VCU, Department of Psychiatry.

Nes, R. B., Røysamb, E., Tambs, K., Harris, J. R., & Reichborn-Kjennerud, T.(2006). Subjective well-being: genetic and environmental contributions to stability and change. *Psychological Medicine*, *6*, 1033–42.

Pichler, F. (2006). Subjective quality of life of young Europeans. Feeling happy but who knows why? *Social Indicators Research*, *75*, 419–444.

Rietveld, M. J., van Der Valk, J. C., Bongers, I. L., Stroet, T. M., Slagboom, P. E., & Boomsma, D. I. (2000). Zygosity diagnosis in young twins by parental report. *Twin Research, 3*, 134–141.

Røysamb, E., Harris, J. R., Magnus, P., Vitterso, J. & Tambs, K. (2002). Subjective well-being. Sex-specific effects of genetic and environmental factors. *Personality and Individual Differences*, *32*, 211–223.

Røysamb, E., Tambs, K., Reichborn-Kjennerud, T., Neale, M. C. & Harris, J. R. (2003). Happiness and health: Environmental and genetic contributions to the relationship between subjective well-being, perceived health, and somatic illness. *Journal of Personality and Social Psychology*, *85*, 1136–1146.

Stubbe, J. H., Moor de, M. H. M., Boomsma, D. I., & de Geus, E. J. C. (2007). The association between exercise participation and well-being: A co-twin study. *Preventive Medicine*, *44*, 148–152.

Stubbe, J. H., Posthuma, D., Boomsma, D. I., & de Geus, E. J. C. (2005). Heritability of life satisfaction in adults: A twin-family study. *Psychological Medicine*, *35*, 1581–1588.

Stutzer, A. (2004). The role of income aspirations in individual happiness. *Journal of Economic Behaviour and Organisation*, *54*, 89–109.

Tellegen, A., Lykken, D. T., Bouchard, T. J., Wilcox, K. J., Rich, S. & Segal, N. L. (1988). Personality similarity in twins reared apart and together. *Journal of Personality and Social Psychology*, *54*, 1031–1039.

Wigginton, J. E., & Abecasis, G. R. (2005). PEDSTATS: Descriptive statistics, graphics and quality assessment for gene mapping data. *Bioinformatics, 21*, 3445–3447.

Willemsen, G., Posthuma, D., & Boomsma, D. I. (2005). Environmental factors determine where the Dutch live: Results from the Netherlands Twin Register. *Twin Research and Human Genetics*, *8*, 312–317.

Wray, N. R., Middeldorp, C. M., Birley, A. J., Gordon, S. D., Sullivan, P. F., Visscher, P. M., Nyholt, D. R., Willemsen, G., de Geus, E. J. C., Slagboom, P. E., Montgomery, G. W., Martin, N. G., & Boomsma, D. I. (2008). Genome-wide linkage analysis of multiple measures of neuroticism of 2 large cohorts from Australia and the Netherlands. *Archives of General Psychiatry, 65,* 649–658.