# Mates and Marriage Matter: Genetic and Environmental Influences on Subjective Wellbeing Across Marital Status

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Specific environments and social relationships may alter the impact of genes. Previous studies have shown marriage to moderate heritability for depressive symptoms in females, suggesting that marriage provides protection or compensation against genetic risks. Similar mechanisms may be relevant for subjective wellbeing (SWB), which is considerably influenced by genes and almost universally associated with marital status. Questionnaire data on SWB from a population-based sample of 1250 monozygotic (MZ) and 981 dizygotic (DZ) male and female twin pairs ($n$ = 4462) were analyzed using structural equation modeling by means of Mx to investigate genetic and environmental influences on SWB across marital status. Resemblance for SWB in MZ twins exceeded that of DZ twins, but the magnitude of this difference varied across marital status. Genetic factors explained 51% and 54% of the variance in SWB among unmarried males and females, and 41% and 39% in married or cohabitating respondents. Remaining variance was attributable to the nonshared environment. The genetic influences were partly different ($r_g$ = 0.64) across marital status in females, but overlapping in married and single males. Our findings show that marriage moderates the magnitude of genetic influences on SWB in both males and females, with a smaller estimate of genetic influences for those with a marital or equivalent partner. The genetic influences on SWB are thus clearly contingent on the environmental context.

**Keywords:** subjective wellbeing, mental health, heritability, twin research, marital status

A number of twin and family studies have documented the importance of genetic differences for variation in happiness and wellbeing (e.g., Bartels & Boomsma, 2009; Lykken & Tellegen, 1996; Nes et al., 2006). Most of these studies report estimates of genetic and environmental influences based on the population at large, leaving gene–environment interplay virtually unexplored (Bartels & Boomsma, 2009; Lykken & Tellegen, 1996; Nes et al., 2006; Røysamb et al., 2002; 2003; Stubbe et al., 2005; Weiss et al., 2008). It is quite likely, however, that genetic and environmental influences on happiness and wellbeing vary systematically across subpopulations, gender and age cohorts, as well as with genotypes and environmental contexts.

The field of quantitative genetics has recently seen an explosion in studies of gene–environment interplay in mental health and behavior, moving far beyond simple reports of heritability and towards exploration of complex and developmentally plausible mechanisms including gene–environment (G × E) interactions. Broadly speaking, G × E interaction occurs either when genes alter the person's sensitivity to specific environmental features, or when environmental contexts differentially modify genetic effects (i.e., genetic dispositions are expressed differently in different environments). The latter effect can be explored by including measured environments in genetically informative designs. Previous research on such effects have shown genetic and environmental risk factors for depressive symptoms to vary across marital status in females (Heath et al., 1998) with genes accounting for 29% and 42% of the variance in the married and unmarried young respondents, and as much as 51% in the older unmarried respondents. Having a current marital or equivalent partner thus seems to reduce the impact of genetically inherited liability to depressive symptoms, suggesting that marriage or cohabitation may serve as protection or compensation against genetic risks. A marriage-like relationship is also shown to reduce the influence of genetic liability to alcohol consumption in females (Heath et al., 1989), with genetic liability accounting for only half as much

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of the variance in the young (< 30 years) and married (31%) as in the young and unmarried (60%).

Similar effects may very well be relevant for subjective wellbeing (SWB) which is regarded as the primary wellbeing index in psychological research (Ryan & Deci, 2001). SWB is negatively correlated with psychological distress and depressive symptoms, considerably influenced by genes (Lykken & Tellegen, 1996; Nes et al., 2006) and almost universally associated with marital status (Diener et al., 2000; Haring-Hidore et al., 1985; Wood et al., 1989). Quite consistently, married couples and unmarried people who live with a romantic partner report higher levels of SWB (Mastekaasa, 1992; 1994), whereas the widowed, the separated, and the divorced are more inclined to unhappiness (e.g., Lucas et al., 2003). The positive marriage effect even remains after additional demographic influences (e.g., income, age, education) and relationship quality are controlled for (Clark & Oswald, 1994; Dush & Amato, 2005; Kim & McKenry, 2002), and has been documented across various cultural contexts although minor cultural-specific factors have been reported (Diener et al., 2000). Minor variation in the magnitude of the effect is also reported across age groups and gender, with marriage constituting a greater source of happiness in younger than in older couples (Haring-Hidore et al., 1985) and the effect to be stronger for men than for women (Kiecolt-Glaser & Newton, 2001).

#### Genetic and Environmental Influences

Knowledge on causal factors explaining the marriage–happiness association is still fairly limited and the mechanisms involved probably complex. One relevant source of complexity could be G × E interaction. Different marital statuses are characterized, at least broadly, by different challenges and demands, and by partly different sources of satisfaction, wellbeing and distress (Kiecolt-Glaser & Newton, 2001). Genetic and environmental sources of wellbeing are therefore likely to differ in the married and the unmarried.

Previous biometric studies have also indicated that the magnitude of the genetic effects, as well as the set of genes impacting on SWB, differ in males and females (Nes et al., 2006; Nes et al., 2009; Røysamb et al., 2002), and sex-related differences may be particularly relevant for marriage. Marriage commonly offers different roles to husbands and wives (Wood et al., 1989), and extensive research points to systematic sex differences in the experience and effect of marriage; for example, showing the protective effects of marriage on health to be stronger for men than for women.

The present study examines the impact of marriage on genetic and environmental influences for SWB. Previous estimates of genetic and environmental sources of SWB are mainly population-level estimates, which do not capture heterogeneity within population subgroups. Using a young adult Norwegian twin sample, we explore whether genetic dispositions for SWB are expressed differently in different social contexts or subgroups — in this case marriage, males and females.

## Method

### Sample

The Norwegian Institute of Public Health Twin Panel (NIPHTP) is an ongoing longitudinal study with a cohort sequential design (See Harris et al., 2002; 2006 for details). Twins were initially identified through information on plural births in the Medical Birth Registry of Norway (MBRN), which began in 1967 and requires mandatory notification of all pregnancies from 16 weeks gestation and registration of standardized information regarding all births in Norway. Questionnaire data were first collected in 1992 ($Q_1$) from twins born 1967–1974, and in 1998 ($Q_2$) from twins born 1967–1979. The present analyses are based on $Q_2$ data only. Responses to $Q_2$ were obtained from 8,045 twins, including 3,334 complete pairs and 1,377 singletons. The individual and pair-wise response rates were 63% and 53%, respectively. The analyses are based on data from same-sexed pairs only, and include responses from 742 and 604 monozygotic (MZ) and dizygotic (DZ) female twin pairs, and 508 and 377 male MZ and DZ pairs, altogether 4462 individuals aged 19 to 31 at the time of assessment.

### Zygosity

Initial zygosity assignment was based on discriminant analyses using seven questionnaire items regarding similarity and confusability in $Q_1$ and the same seven and two additional items in $Q_2$, previously shown to categorize correctly more than 97% (Harris et al., 1995). Twenty-four micro-satellite markers were then genotyped on a sub-sample of 676 like-sexed pairs in the sample. Results from these markers were used as dependent variable in a discriminant analysis with the questionnaire items as independent variables. Seventeen out of 676 pairs with DNA information were found to be misclassified by the questionnaire data and zygosity corrected. The total number of expected misclassified pairs could thus be estimated to be 1.38%.

### Measures

*SWB* was measured by a set of items originally suggested by Moum et al. (1990). The index was constructed using a mean score of 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?' (Six response categories, ranging from 1 = *Extremely satisfied* to 6 = *Extremely dissatisfied*. (2) 'Are you usually happy or dejected?' (Five response categories, ranging from 1 = *Dejected* to 5 = *Happy*. (3) 'Do you mostly feel strong and fit, or tired and worn out?' (Five response categories ranging from 1 = *Very strong and fit* to 5 = *Tired and worn out*. (4) 'Over the last month, have you suffered from nervousness (felt irritable, anxious, tense, or restless)?' (Four response categories ranging from 1 = *Almost all the time* to 4 = *Never*). Thus, the index comprises a cognitive aspect (life satisfaction), positive affect (happy, strong) and negative affect (worn out, nervous), thereby conforming to the generally accepted operationalization of SWB



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(Lucas et al., 1996). Differences in variances between the items due to different numbers of response categories were removed before the scores from each item were summed to make an index. Cronbach's α for the index was estimated to be 0.70 for the $Q_2$ data. Further description of the index can be found elsewhere (Røysamb et al., 2002).

A multi-sample confirmatory factor analysis including responses from 3429 males and 4587 females ($Q_2$) using Mplus (Muthén & Muthén, 2006) was conducted to further validate the scale and test for measurement invariance across males and females. A one factor model equating all four factor loadings across sex yielded excellent fit ($\chi^2_8 = 66.47$, RMSEA = .043, CFI = .99, TLI = .99) and not significantly worse fit than a model allowing the factor loadings to differ across males and females ($\Delta\chi^2_4 = 6.8$).

***Partnership status:*** The respondents were asked to report their current partnership status by the following single item 'Are you: (1) unmarried/not cohabiting, (2) married/cohabiting, (3) widow/widower, or (4) separated/divorced'. Thus our married data include both legally married individuals and cohabitating partners. Respondents who were widowed ($N = 6$) or separated/divorced ($N = 91$) were excluded from the analyses.

***Co-twin closeness:*** Different levels of social contact between MZ and DZ twin pairs may cause biased heritability estimates, and MZ co-twins in our sample have been shown to report more frequent contact than do DZ co-twins (Tambs et al., 1995). To test for the assumption of same degree of shared environment for MZ and DZ co-twins, we measured SWB co-twin closeness by a summed score index based on responses to 4 items measuring frequency of contact (personal or by telephone), quality of contact, and geographical distance.

#### Statistical Analyses

To estimate genetic and environmental effects conditional on environmental exposure (marital status), the pairs were categorized into three groups based on the relationship status of both twins: (1) concordant for both having a marriage-like relationship, (2) concordant for both having no partner, and (3) discordant. In discordant pairs, the single twin was always designated the 1st twin and the unmarried twin the 2nd twin, whereas the order of the twins were arbitrary in concordant pairs.

Presence of genotype–environment correlation (CorGE) may complicate the statistical procedures necessary for exploring gene–environment interaction (Purcell, 2002). CorGE refers to the fact that environmental risk and protection is not distributed randomly (i.e. the probability that an individual will experience a given environmental event depends on the genotype). Happy people are probably more fun to be with and may appear more attractive as marriage partners, resulting in happy people being more conducive to marriage and previous reports have shown the propensity to marry to be influenced by genes (Johnsen et al., 2004; Schnittker, 2008). Such gene–environment correlation will usually be reflected in higher cross-correlations between one twin's marital status and the co-twin's SWB in MZ than in DZ twin pairs. We therefore computed polyserial cross-correlations between one twin's marital status and the co-twin's SWB, separately for each zygosity group using LISREL 8.54 (Jøreskog & Sørbom, 1993). We also estimated the association between absolute intra-pair differences in SWB and social closeness (separately for each zygosity group) to test for the assumption of same degree of shared environment for MZ and DZ co-twins in our sample.

As an initial assessment of the importance of genetic and environmental influences, co-twin correlations were calculated. Biometric modeling was then used to further investigate genetic and environmental influences on SWB using the raw maximum likelihood (ML) estimation procedure in Mx (Neale et al. 1999), which allows for preliminary testing of basic assumptions concerning the homogeneity of means and variances within twin pairs and across zygosity and sex. Age was specified as a covariate in the preliminary analyses.

In classical twin modeling, genetic and environmental effects are modeled as the contribution of latent factors to the phenotypic variance of a given measure, in this case SWB. These latent factors represent the effects of many unidentified influences, including: additively (A) and nonadditively (D) acting genes and shared (C) and nonshared (E) environments. Additive genetic effects (*a*) comprise the effects from a large, but unknown number of individual alleles at loci influencing a particular phenotype additively. Nonadditive genetic effects (*d*) reflect interaction between alleles at the same locus (dominance) or between alleles across loci (epistasis), so that the expression of the genetic variant depends on the presence of other genetic variants.

Environmental variance is separated into either shared (*c*) or nonshared (*e*) effects, in which the former refers to all nongenetic influences causing resemblance between siblings, and the latter to all nongenetic influences causing differences.

The contributions of these latent genetic and environmental factors (*a, d, c, e*) are modeled as regression coefficients in the linear regression of the observed variables on the latent factors. This is possible because from genetic theory we know that MZ twins share all their genes, so that A and D are perfectly correlated in these pairs, whereas DZ twins share on average 50% of their segregating genes giving a genetic correlation of 0.5 (A) or 0.25 (D). As C includes all environmental influences causing similarity between co-twins regardless of zygosity, it is correlated 1.0 in all zygosity groups. E constitutes the residual variance after the effects of A, D, and C have been removed and also includes measurement error.

The genetic and environmental parameter estimates are derived by specifying a model according to



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the differential degree to which pairs of MZ and DZ twins are correlated for genetic and environmental effects (see Figure 1). However as the *d* and *c* parameters are fully negatively confounded, only one can appear in any given model. By convention, models specifying *d* are fit only when the ratio of MZ to DZ correlations exceeds 2.0 (Plomin et al., 1992).

In the present analyses we explore whether marital status modifies the effects of genes that influence variation in SWB. Data are analyzed separately for males and females using correlated factor (general G × E interaction) models (Figure 1) that allow us to distinguish between two types of effects of presence or absence of a marital-like relationship. The same genes and environmental influences may operate under both conditions (the presence or absence of a marital-like relationship) but the magnitude of the effects may differ (i.e., quantitative differences). Alternatively, some genes or environmental influences may be expressed only in married or in the unmarried (i.e., qualitative differences) people. More information on these models can be found elsewhere (Neale & Cardon, 1992).

The fit of the full models were compared to several nested submodels. ML analysis of raw data does not provide an overall measure of fit. However the difference in –2 log likelihood (–2LL) between the models is distributed as $\chi^2$, allowing the relative fit of submodels against the saturated model to be tested using the $\chi^2$ difference test ($\Delta\chi^2_{df}$). To select the best fitting model, we used the Akaike Information Criterion (AIC), which provides a summary index of both parsimony and fit ($\chi^2$ –2df; Akaike, 1987). Low AIC values (i.e., nonsignificant) indicates that the observed values do not deviate significantly from the expected values and the model yielding the lowest AIC value fits the observed data best.

## Results

### SWB Index Scores

The mean scores of the SWB index (0-10 scale) for the different zygosity groups are tabulated in Table 1.

Overall, both married males (mean = 7.63, *SD* = 1.43) and females (mean = 7.13, *SD* = 1.39) reported higher SWB than unmarried males (mean = 7.29, *SD* = 1.54) and females (mean = 6.90, *SD* = 1.65), but married males reported significantly higher SWB than married females ($p < .03$), and single males higher SWB than single females ($p < .04$).

### Heterogeneity of Means and Bariances

Means could not be constrained to be equal across all females regardless of zygosity and marital status ($\Delta\chi^2_{11} = 30.05$, $p = .00$, AIC = 8.05), and differed significantly across zygosity for married females ($\Delta\chi^2_5 = 14.78$, $p = .01$, AIC = 4.78) and for unmarried females ($\Delta\chi^2_5 = 13.27$, $p = .02$, AIC = 3.27).

In males, means could be set equal across zygosity for both married ($\Delta\chi^2_5 = 8.47$, $p = .13$, AIC = –1.53) and unmarried respondents ($\Delta\chi^2 = 2.75$, $p = .74$, AIC = –7.25), but could not be constrained to be equal across all males ($\Delta\chi^2_{11} = 40.99$, $p = .00$, AIC = 18.99).

Variances could be constrained to be equal across zygosity and marital status for males ($\Delta\chi^2_{11} = 13.61$, $p = .26$, AIC = –8.39) and for females ($\Delta\chi^2_{11} = 20.39$, $p = .04$, AIC = –1.61) without significantly compromising fit and the effect of age was not significant for males ($\Delta\chi^2_3 = 4.91$, $p = .18$, AIC = –1.10) nor females ($\Delta\chi^2_3 = 3.84$, $p = .28$, AIC = –2.16). There was, however, a

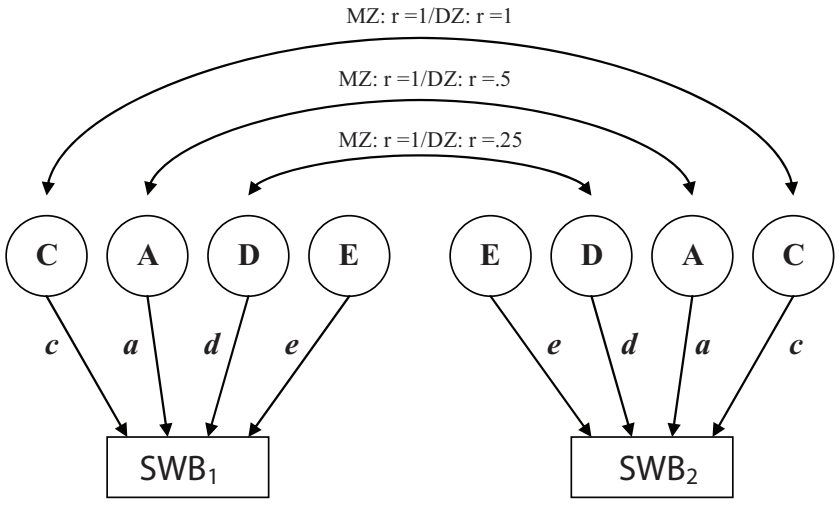


**Figure 1**

Basic univariate correlated factor model comprising additive (A) and non-additive (D) genetic factors and shared (C) and nonshared (E) environments.

Note: Capital letters A, D, C and E in circles denote the latent variables for additive genetic, non-additive genetic and shared and nonshared environmental effects.
MZ = monozygotic, DZ = dizygotic, SWB = subjective wellbeing, subscripts 1 and 2 denotes Twin 1 and Twin 2 in a given pair.
Parameters (small letters): a = additive genetic effect, d = nonadditive genetic effect, c = shared environmental effects e = nonshared environmental effect.



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**Table 1**

SWB Index Scores (Means, SD) for Male and Female Twin Pairs Conditional Upon Marital Status

| Marital status | Zygosity | SWB Mean (SD) | *N* |
|---|---|---|---|
| Single | | | |
| Females | | 6.90 (1.65) | 1240 |
| Males | | 7.29 (1.54) | 1063 |
| Concordant | | | |
| | $MZ_f$ | 7.08 (1.66) | 496 |
| | $DZ_f$ | 6.84 (1.57) | 318 |
| | $MZ_m$ | 7.41 (1.49) | 486 |
| | $DZ_m$ | 7.22 (1.59) | 322 |
| Discordant | | | |
| | $MZ_f$ | 6.87 (1.64) | 216 |
| | $DZ_f$ | 6.61 (1.68) | 210 |
| | $MZ_m$ | 7.15 (1.61) | 136 |
| | $DZ_m$ | 7.11 (1.50) | 119 |
| Married | | | |
| Females | | 7.13 (1.55) | 1452 |
| Males | | 7.63 (1.43) | 707 |
| Concordant | | | |
| | $MZ_f$ | 7.32 (1.39) | 556 |
| | $DZ_f$ | 6.98 (1.68) | 470 |
| | $MZ_m$ | 7.62 (1.43) | 258 |
| | $DZ_m$ | 7.56 (1.39) | 194 |
| Discordant | | | |
| | $MZ_f$ | 7.13 (1.52) | 216 |
| | $DZ_f$ | 6.78 (1.63) | 210 |
| | $MZ_m$ | 7.78 (1.43) | 136 |
| | $DZ_m$ | 7.61 (1.48) | 119 |

*Note*: MZ = monozygotic and DZ = dizygotic. Subscripts m and f indicates males and females, respectively.

nonsignificant trend for greater variances in both single males and single females.

#### Gene–Environment Correlation

All cross-correlations between one twin's marital status and the co-twin's SWB score were insignificant and the absolute value less than .05. In addition, the pattern of cross-correlations was not systematically higher in MZ than DZ co-twins, indicating that a genetic predisposition to SWB does not lead to an increased probability of getting married (i.e., no indication of gene-environment correlation) in our sample.

#### Social Closeness

Absolute difference in co-twin's SWB score was not significantly related to current co-twin contact. Thus there was no evidence for increased resemblance in siblings with frequent contact compared to sisters and brothers with less frequent social contact.

#### Twin Correlations

Co-twin correlations for SWB varied significantly across zygosity and marital status in both males and females. Table 2 displays the different co-twin correlations by zygosity and marital status with 95% confidence intervals (CI).

Correlations for SWB were generally higher among MZ than DZ co-twins, indicating genetic effects on variation in SWB. However, the magnitude of the correlations, and the difference between MZF and DZF co-twin correlations, varied across marital status, suggesting different magnitude of genetic and environmental influences in married and unmarried males and females. In both males and females, the difference between the MZ and DZ correlation was greater in single as opposed to married twins, suggesting that heritability for SWB is higher in single than in married respondents.

#### Model Fitting

The results of genetic model fitting are summarized separately for females (Table 3) and males (Table 4).

#### Females

The full model (model 1) against which the nested submodels were compared, was an ACE model specifying both quantitative and qualitative genetic effects. Pathways from A and C were in turn fixed to zero to test for significant contribution of additive genetic and shared environmental effects.

The CE and E models (model 2 and model 4) were firmly rejected by the $\chi^2$-test ($20.25_3$; $176.57_4$), indicating significant influences from additive genetic effects on SWB. An AE model (model 3) specifying both quantitative and qualitative genetic differences across marital status fitted the data well, suggesting that familiar resemblance for SWB could be explained solely by additive genetic influences which partly differ in single and married respondents ($r_g < 1$). This model also turned out to be the best-fitting model in terms of AIC values (AIC = –2.82) when compared to further reduced models (models 5–8). The best-fitting model to the female data was thus an AE model which incorporated

**Table 2**

Twin Correlations With 95% Confidence Intervals

| | MZ | Pairs (*N*) | DZ | Pairs (*N*) |
|---|---|---|---|---|
| Females | | | | |
| Concordant single | 0.55 (0.46–0.63) | 248 | 0.27 (0.12–0.41) | 159 |
| Concordant 'married' | 0.33 (0.22–0.43) | 278 | 0.22 (0.10–0.34) | 235 |
| Discordant | 0.24 (0.11–0.23) | 216 | 0.23 (0.10-0.35) | 210 |
| Males | | | | |
| Concordant single | 0.49 (0.39–0.58) | 243 | 0.20 (0.04–0.34) | 161 |
| Concordant 'married' | 0.41 (0.25–0.54) | 129 | 0.17 (0.00–0.36) | 97 |
| Discordant | 0.49 (0.35–0.61) | 136 | 0.26 (0.08-0.42) | 119 |



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**Table 3**

Fit Measures: Females

| Effect | Model | $a^2_s$ | $c^2_s$ | $e^2_s$ | $a^2_m$ | $c^2_m$ | $e^2_m$ | $r_g$ | –2LL | $\Delta\chi^2$ | $\Delta$df | $p$ | AIC |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| I | 1. ACE | 0.44 | 0.1 | 0.46 | 0.3 | 0.08 | 0.62 | 0.54 | 10043.07 | | | | |
| | 2. CE | — | 0.43 | 0.57 | — | 0.23 | 0.77 | — | 10063.32 | 20.25 | 3 | 0 | 14.25 |
| | 3. AE | 0.54 | — | 0.46 | 0.39 | — | 0.51 | 0.64 | 10044.25 | 1.18 | 2 | 0.56 | –2.82 |
| | 4. E | — | — | 1 | — | — | 1 | — | 10219.64 | 176.57 | 4 | 0 | 166.57 |
| II | 5. ACE | 0.54 | 0 | 0.46 | 0.16 | 0.17 | 0.66 | 1 | 10045.82 | 2.75 | 1 | 0.1 | 0.75 |
| | 6. AE | 0.52 | — | 0.48 | 0.31 | — | 0.69 | 1 | 10051.73 | 8.66 | 3 | 0.03 | 2.66 |
| III | 7. ACE | 0.34 | 0.07 | 0.59 | 0.34 | 0.07 | 0.59 | 1 | 10060.9 | 17.83 | 4 | 0 | 9.83 |
| | 8. AE | 0.42 | — | 0.58 | 0.42 | — | 0.58 | 1 | 10061.44 | 18.37 | 5 | 0 | 8.37 |

Note: I = qualitative and quantitative genetic differences across marital status, II = quantitative genetic differences across marital status, III = no differences across marital status. Subscripts m and s indicates married and single, respectively. Parameters: a = additive genetic effect, c = shared environmental effect, e = environmental effect. The $r_g$ indicates the correlation between genetic factors for married and unmarried respondents.

both quantitative and qualitative genetic effects, indicating that (1) the genetic and environmental influences on SWB differ across marital status in females, and that (2) the set of genes influencing SWB are partly different in the married and the unmarried. Heritability in this best-fitting model was estimated to be 0.39 (95%CI: 0.29-0.48) and 0.54 (95%CI: 0.46–0.61) in married and unmarried females, respectively, and thus lower in married than in single respondents (Figure 2). The correlation between genetic factors for SWB in married and unmarried female twins was estimated to be 0.64 (95%CI: 0.40–0.90). Variance not attributable to additive genetic factors was accounted for by the nonshared environment and explained 61% and 46% in married and single respondents, respectively.

### Males

The full male model was an ADE model due to the differences in correlations between MZ and DZ twins exceeding 2.0 for both the concordant groups. This is also supported by previous findings indicating considerable non-additive genetic influences on SWB (e.g. Bartels & Boomsma, 2009; Lykken & Tellegen, 1996; Stubbe et al., 2005).

Moderate additive (0.34–0.37) and minor nonadditive (0.08–0.15) genetic effects were estimated for single and married males in this full model. However, deleting either the non-additive genetic effect (model 2) or the additive genetic effect (model 3) resulted in somewhat better fit in terms of AIC values (AIC = –1.06 versus AIC = –3.52 respectively). The E model (model 4) was firmly rejected by the $\chi^2$-test ($142.86_4$), indicating significant influences from genetic factors on SWB. Dropping the qualitative genetic effect did not significantly worsen the fit, indicating that the same genetic sources are influencing SWB in single and married males. A model specifying only quantitative genetic differences across marital status, and dropping the non-additive genetic effect (model 7), fitted the data best in terms of AIC (AIC = –5.52). This model fitted the data better than further reduced models (model 8–10), which equated the parameter estimates in married and unmarried respondents. In this best-fitting model, heritability was estimated to be

**Table 4**

Fit Measures: Males

| Effect | Model | $a^2_s$ | $c^2_s$ | $e^2_s$ | $a^2_m$ | $c^2_m$ | $e^2_m$ | $r_g$ | –2LL | $\Delta\chi^2$ | $\Delta$df | $p$ | AIC |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| I | 1. ADE | 0.37 | 0.15 | 0.48 | 0.34 | 0.08 | 0.58 | 1 | 6351.91 | | | | |
| | 2. DE | — | 0.52 | 0.48 | — | 0.42 | 0.58 | 1 | 6354.85 | 2.94 | 2 | 0.23 | –1.06 |
| | 3. AE | 0.51 | — | 0.49 | 0.41 | — | 0.59 | 1 | 6352.39 | 0.48 | 2 | 0.79 | –3.52 |
| | 4. E | — | — | 1 | — | — | 1 | 1 | 6494.77 | 142. 86 | 4 | 0 | 134.86 |
| II | 5. ADE | 0.37 | 0.15 | 0.48 | 0.34 | 0.08 | 0.58 | 1 | 6351.91 | 0 | 1 | — | –2 |
| | 6. DE | — | 0.52 | 0.48 | — | 0.42 | 0.58 | 1 | 6354.85 | 2.94 | 3 | 0.4 | –3.06 |
| | 7. AE | 0.51 | — | 0.49 | 0.41 | — | 0.59 | 1 | 6352.39 | 0.48 | 3 | 0.92 | –5.52 |
| III | 8. ADE | 0.34 | 0.13 | 0.53 | 0.34 | 0.13 | 0.53 | 1 | 6357.8 | 5.89 | 4 | 0.21 | –2.11 |
| | 9. DE | | 0.52 | 0.48 | | 0.52 | 0.48 | 1 | 6360.85 | 8.95 | 5 | 0.11 | –1.05 |
| | 10. AE | 0.47 | — | 0.53 | 0.47 | — | 0.53 | 1 | 6358.16 | 6.25 | 5 | 0.28 | –3.75 |

*Note:* I = qualitative and quantitative genetic differences across marital status, II = quantitative genetic differences across marital status, III = no differences across marital status. Subscripts m and s indicates married and single, respectively. Parameters: a = additive genetic effect, d = nonadditive genetic effect, e = environmental effect. The $r_g$ indicates the correlation between genetic factors for married and unmarried respondents.



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0.51 (95%CI: 0.42–0.59) and 0.41 (95%CI: 0.30–0.52) in single and married males, respectively. The remaining variance was attributable to the nonshared environment, including error (Figure 2).

## Discussion

An extensive literature points to the importance of interpersonal relationships for mental health and well-being, and marital relationships have been shown to be particularly important (Kiecolt-Glaser & Newton, 2001). In this study, we conducted a series of analyses to further explore the relationship between marriage and mental health, focussing on marriage as a potential moderator of genetic influences on SWB.

### Quantitative Differences

Consistent with most previous findings on marriage and wellbeing, we found higher levels of SWB in the married as opposed to the unmarried. We also note that males report higher levels of SWB than females. In line with previous biometric studies of SWB (Bartels & Boomsma, 2009; Lykken & Tellegen, 1996; Røysamb et al., 2002; 2003; Schnittker et al., 2008; Weiss et al., 2008), our results reveal considerable genetic influences on SWB within the range of estimates obtained from previous studies ($h^2$ = 0.35–0.50). However, the magnitude of these genetic effects varies according to marital status. In males as well as females, genetic influences on variation in SWB were shown to be significantly greater in unmarried ($h^2_m$ = 0.51, $h^2_f$ = 0. 54) than in married ($h^2_m$ = 0.41, $h^2_f$ = 0.39) respondents, indicating that different environmental settings, such as living within or outside a marital-like relationship, provide different opportunities for the genetic potential for SWB to be actualized. The effects of genes on SWB are thus clearly contingent on co-action with the environment.

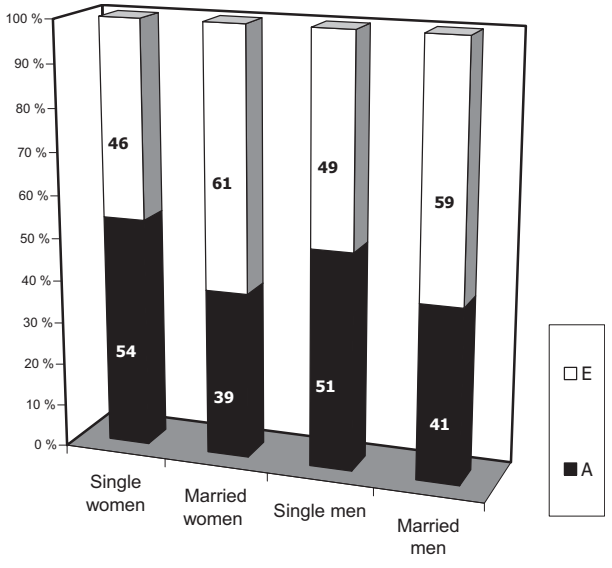


**Figure 2**

Proportion of the phenotypic correlations accounted for by genetic and environmental factors.

Note: A = additive genetic influences, E = nonshared environmental influences.

What is it about the context of marriage that matters for gene expression? A number of studies have indicated that heritability tend to attenuate in settings that are characterized by higher levels of social control, and to increase in settings with lower levels of social constraints (Boomsma et al., 1999; Heath et al., 1998; Heath et al., 1989; Koopmans et al., 1999; Rose et al., 2001). This pattern is predicted by the social control model (Shanahan & Hofer, 2005) which hypothesizes that social norms and habits constrain the activities and choices of individuals within social contexts, consequently preventing the genetic expression. The causes of behavior in more structured or less varied environments are therefore likely to be less dispositional and more situational, whilst genetic differences are more likely to explain behavior in less structured environments. Previous studies have shown marriage and cohabitation to reduce the impact of genetically inherited liability to both depressive symptoms and alcohol consumption in women (Heath et al., 1998; Heath et al., 1989). Religiosity (Boomsma et al., 1999; Koopmans et al., 1999) and regional residency (Rose et al., 2001) have likewise been shown to moderate genetic influences on behavior problems such as disinhibition and patterns of alcohol use and smoking. Along with these previous reports, our results may suggest that environments that provide a more limited range of opportunities to express individual differences (i.e., are more 'controlling'), constrain the expression of dispositional genes. Marriage is a small and intimate form of social network, a well-defined social arena that tends to be both specialized and compartmentalized, usually providing relatively unambiguous clues about behavior in a wide range of situations. In contrast, single living commonly provides fewer salient behavioral cues, causing the individual to rely more heavily on innate dispositions.

The environment may also 'get under the skin' through other pathways. Ryff & Singer (2005) have emphasized that the emotional experience in social relationships is likely to be the key element in mapping pathways to positive health. Differentiating between the different pathways through which the putative social forces of marriage operate is beyond the scope of this study, however, as we cannot identify whether the institutional aspects (e.g., laws, regulations) of marital unions, the informal habits and norms, the emotional experiences, or the psychological characteristics of the partner, constitute the main influence. Our findings do attest, however, that marital relationships moderate the expression of genetic dispositions for SWB.

### Qualitative Differences

Besides showing that the magnitude of the genetic influences varies across marital status, the results indicate that partly different sets of genes are influencing



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SWB across marital status in females ($r_g$ = 0.64 (95%CI: 0.40–0.90)).

Does marriage trigger new (different) genetic effects in females? Partly different genetic sources of SWB in married and unmarried females may indicate biological changes at the molecular level, but could also reflect alterations in psychosocial circumstances of a sufficient magnitude to elicit new genetic sources for SWB. Such genetic effects illustrate that despite the DNA not undergoing change, different life-situations, circumstances, or developmental stages, may make different genetic factors salient for SWB. Biological influences do not operate independently of socio-contextual features. A voluminous literature has linked characteristics of the social environment to physical and mental health (e.g., Ryff & Singer, 2005). A recent study has also evidenced that social-environmental risk factors are linked to global alterations in human gene transcription, providing a functional genomic explanation for increased levels of inflammatory disease in chronically lonely individuals (Cole et al., 2007).

The different social, practical, and economic circumstances characterising life within and outside marital unions are also not likely to operate independently of biological or genetic features. Circumstances generate needs, challenges and sources of satisfaction and distress, that are related to different genetic factors. Marriage and cohabitation commonly provide emotional, social and practical support, activate new sources of identity and self-esteem, and offer new roles such as that of a spouse and a parent (e.g., Wood et al., 1989). Institutionalization and joint investments (e.g. financial or relationship-specific) provide security, community recognition, and a shared history, which in itself may become a source of meaning, identity, and happiness (Misick & Bumpass, 2006). These important factors may trigger new sources of happiness and well-being reflecting different genetic sources. Marriage may thus work as a contextual trigger (Shanahan & Hofer, 2005) of new genetic variance in young adult females. Genes contributing to happiness in young unmarried women may therefore be different from genes contributing to happiness among their married sisters.

#### Limitations

Our results should be interpreted in the context of a number of limitations. In twin studies, the individual environmental effect also subsumes measurement error, and estimates of familial resemblance are therefore proportionally deflated by decreasing reliability. Cronbach's α for the SWB index used here was estimated to be 0.70. The heritability estimates may therefore be moderately underestimated, and the true effect from the non-shared environment moderately overestimated in the current study.

Attrition may similarly lead to biased estimates of the genetic and environmental parameters (Heath, Madden et al., 1998). The response rate was lower than optimal (53%), and no definite conclusion can be drawn regarding selection bias at the entry of our twin study. Health information and demography from the first assessment ($Q_1$) have been tested as predictors of participation in the data wave used here ($Q_2$) (Tambs et al., 2008), and show a moderate selection towards good mental health, but do not seem to seriously threaten the representativeness of our sample. However, our conclusions may not be fully accurate reflections of the entire population.

As the analyses are based entirely on young adult Norwegian twins, the results may also not fully extrapolate to other ethnic groups or age groups. The effect of marriage may vary considerably with age and differential selection into or out of marital-like relationships may have different implications in different age groups. The normative biological, psychological, and social changes occurring during young adulthood are larger and involve more life-changing roles, demographic diversity, instability, and identity decisions than any other adult life stage (Arnett, 2000). The social and emotional support, as well as social constraints associated with marriage or marital-like unions, may therefore be particularly important during this life phase.

Contrary to some previous reports we have not made systematic distinctions between married and cohabitating partners in these analyses due to our data not containing the necessary information to make this distinction. This lack of differentiation would clearly be problematic should there be important qualitative differences between the selection into, or the effects of married and unmarried unions, with regard to SWB. Some studies have shown that cohabitating individuals report poorer mental health and wellbeing than the married (e.g., Lamb et al., 2003), whereas other studies have not documented significant differences (e.g. Horwitz & White, 1998). Differences between married and unmarried unions seem to decline, however, when cohabitation becomes an institutionalized alternative to formal marriage (Horwitz & White, 1998; Mastekaasa, 2006). This is clearly the case in Norway, and research based on Norwegian samples has reported only small differences in wellbeing between the married and cohabitating unions (e.g., Mastekaasa, 1995).

#### Conclusion

The present study shows that mates and marriage matter for the genetic expression on SWB. The magnitude of the genetic effects is smaller in both males and females with a marital or equivalent partner, indicating that marriage or marital-like unions are sufficiently important to moderate genetic influences on SWB. Results also indicate that the set of genes impacting on SWB partly differs across marital status in females, suggesting that marital unions may elicit new genetic sources for SWB in females. The findings clearly show that the genetic and environmental influences on SWB transact and interplay through changing circumstances and environmental settings, thus being contingent on co-action with the environment.



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