The association between frequency of social media use, wellbeing, and depressive symptoms: disentangling genetic and environmental factors

S. Sametoglu[a, b], D. H. M. Pelt[a, b], M. Bartels[a ,b]

[a]Department of Biological Psychology, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands

[b]Amsterdam Public Health Research Institute, Amsterdam University Medical Centre, Amsterdam, The Netherlands.

Author Note

Data availability: The data from the Netherlands Twin Register can be requested from the Netherlands Twin Register (https://ntr-data-request.psy.vu.nl/) after completing the requirements of the data access committee.

Acknowledgements: Authors thank all the Netherlands Twin Register participants for their efforts.

Funding: This work and data collection were supported by a European Research Council Consolidator Grant (WELL-BEING 771057 PI Bartels). Meike Bartels is furthermore supported by an NWO VICI Grant (VI.C.211.054)

Correspondence concerning this article should be addressed to Selim Sametoğlu, Department of Biological Psychology, Faculty of Behavioural and Movement Sciences, Vrije Universiteit Amsterdam, Van der Boechorststraat 7-9, 1081 BT, Amsterdam, The Netherlands; e-mail: s.sametoglu@vu.nl

Conflict of interest: The authors have no relevant financial or non-financial interests to disclose.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The approval of the survey data collection was provided by the Medical Ethical Committee of the Amsterdam University Medical Centre (2018.389)

Informed consent: Informed consent was obtained from all individual participants included in the study.

Abstract

Meta-analyses consistently report small but significant associations (around r = -0.10) between wellbeing (WB) and social media use (SMU) and between anxious-depressive symptoms (ADS) and SMU (around r = 0.10). This study employs the classical twin design, utilizing data from 6492 individuals from the Netherlands Twin Register, including 3369 MZ twins (893 complete twin pairs, 1583 incomplete twin pairs) and 3123 DZ twins (445 complete, 2233 incomplete) to provide insights into the sources of overlap between WB/ADS and SMU. Both hedonic and eudaimonic WB scales were used. SMU was measured by (1) the time spent on different social media platforms (SMU_t), (2) the frequency of posting on social media (SMU_f), and (3) the number of social media accounts individuals have (SMU_n). Our results confirmed the low phenotypic correlations between WB/ADS and SMU (absolute correlations, between r = 0.03-0.10). For SMU, heritability estimates between 32 and 72% were obtained. The small but significant phenotypic correlations between WB/ADS and the SMU phenotypes were mainly determined by genetic factors (in the range of 80-90%). For WB and SMU, genetic correlations were between -0.10 and -0.0, and for ADS and SMU genetic correlations were between 0.10 and 0.23. Genetic correlations implied limited but statistically significant sets of genes that affect WB/ADS and SMU levels. Overall, the results indicate that there is evidence that the small associations between WB/ADS and SMU are partly driven by overlapping genetic influences. We encourage researchers and experts to consider more personalised approaches when considering the association between WB and SMU, as well as understanding the reasons for individuals' observed SMU levels.

Keywords: Classical twin design, Genetically informed designs, social media use, wellbeing, wellbeing

The association between frequency of social media use, wellbeing, and depressive symptoms:

disentangling genetic and environmental factors

As of April 2023, there are 4.80 billion social media users, equivalent to 59.9 % of the global population. Individuals daily spend on average around 2 and a half hours on social media platforms (Datareportal, 2023). Over the last decade, the number of people using social media platforms has been consistently growing. Public and governmental organizations are paying more attention to social media use, and its potential positive and negative effects on individuals, such as their wellbeing and mental health (Pereira et al., 2021; Izutsu et al., 2015).

Wellbeing can be broadly categorized as subjective (or hedonic) and psychological (or eudaimonic) wellbeing (Deci & Ryan, 2008; Ryff, 1989). Hedonic wellbeing consists of cognitive and affective evaluations of one's life, whereas eudaimonic wellbeing refers to positive functioning which entails multiple domains such as having positive relations, autonomy, environmental mastery, positive growth, purpose in life, and self-acceptance (Ryff, 1989). Higher levels of wellbeing are associated with a wide range of positive outcomes such as having better social relations, and finances, as well as higher school achievement, altruistic behaviour, and workplace functioning (Chapman & Guven, 2016; James et al., 2019; Maccagnan et al., 2019; Okabe-Miyamoto & Lyubomirsky, in press; Oswald et al., 2015; Steptoe, 2019; Walsh et al., 2018). At the nation level, higher wellbeing is associated with higher GDP, lower healthcare costs and sickness benefits (Hagerty & Veenhoven, 2003; Santini, Becher, et al., 2021; Santini, Nielsen, et al., 2021).

Similarly, depressive-anxious symptoms indicate a burden to society and individuals, given their association with increased healthcare costs in public health systems (Vasiliadis et

al., 2013), poverty (Ridley et al., 2020), suicidal ideation (Casey et al., 2008; Spijker et al., 2010), and loneliness (Lee et al., 2021) among many negative outcomes.

Association between WB/DS and SMU

In recent years, there has been a strong increase in the number of studies on the association between social media use with wellbeing and anxious-depressive symptoms (Valkenburg, 2022). For instance, multiple meta-review studies, combining results of the meta-analyses, indicated either small to moderate effect sizes or inconsistent results (Appel et al., 2020; Valkenburg, Beyens, et al., 2022; Valkenburg, Meier, et al., 2022). The most recent meta-review included 27 review studies on SMU and WB/ADS published (including 9 meta-analyses and 9 systematic reviews) between 2019 and 2021 (Valkenburg, Beyens, et al., 2022). This study reported that six out of seven meta-analyses found effect sizes that ranged from very small to moderate (absolute correlations, r = 0.05-0.17), while one meta-analysis reported mainly null results (Cunningham et al., 2021).

Overall, the lack of inconsistent results has been partly attributed to overreliance on cross-sectional research (e.g., Parry et al., 2022; Valkenburg, Beyens et al., 2022). As a potential solution to this, multiple studies used longitudinal data sets. For example, a study found small negative associations for WB with retrospectively reported and diary-logged digital screen engagement as a measure of SMU (r = - 0.08, and r = -0.02, respectively) (Orben & Przybylski, 2019). Similarly, another longitudinal study investigated ADS and SMU of a group of individuals from 10 to 16 years with 2-year lags. The results indicated null within-person level associations and a few small (between r = 0.04-0.07) associations at the between-person level (Steinsbekk et al., 2023). Other longitudinal studies applied a more person-centric approach by investigating whether the associations differed for each individual. Their results showed that the association between WB/ADS and SMU differs in

both direction and magnitude among among individuals over time. (Beyens et al., 2020; Pouwels et al., 2021).

It has also been considered whether different ways of assessing social media use could lead to different results (Hogue & Mills, 2019; Verduyn et al., 2017, 2022). Social media use can be categorised in active social media use (e.g., posting frequency, sending messages) or passive usage measures (e.g., scrolling), in which passive SMU is considered to have a negative influence on wellbeing, whereas active SMU's influence is considered to be less conclusive. For instance, a study using a population sample of Icelandic adolescents (*N* = 10,563) reported increased passive social media use to be associated with increased anxiety and depressive symptoms, and these effects persisted after controlling for time spent on social media. Another study, in a sample of adults between 18-49 age (N = 702), reported an association between increased passive SMU and negative wellbeing, but reported the opposite results for active SMU (Escobar-Viera et al., 2018). Further, although active and passive SMU distinctions in the assessment of SMU exist, there is no consensus on the usefulness of active versus passive social media usage measures in the field (Valkenburg, Van Driel, et al., 2022; Verduyn et al., 2017, 2022).

Overall, evidence indicated that small associations between WB/ADS and SMU are present. Furthermore, most studies assume the direction of the association to be from increased SMU to decreased WB/increased ADS, yet substantial evidence to claim the direction of causation is missing given the cross-sectional nature of most studies in this field.

Besides the size and the direction of the associations between WB/ADS and SMU variables, it is crucial to study the underlying sources of the associations between WB/ADS and SMU. In particular, by leveraging genetically informed designs, it can be investigated whether the phenotypic association between two variables is partly due to overlapping

genetic factors that influence both WB/ADS and SMU. As an example of a genetically informative design, the classical twin design can be used. Classical twin designs are based on data from monozygotic (MZ) and dizygotic (DZ) twins, which allow for disentangling the effects of genetic and environmental influences on the observed covariance between WB/ADS and SMU (i.e., De Vries et al., 2021). In addition to estimating the underlying sources of covariance, multivariate models provide the opportunity to calculate the genetic and environmental correlations that indicate the overlap in genetic and environmental factors between phenotypes.

For the relation between WB/ADS and SMU to be partly driven by genetics, it is a prerequisite that each of these traits is heritable. Evidence based on genetically informed designs (e.g., twin studies, molecular genetic studies) indicates individual differences in WB, ADS, and SMU to be partly accounted for by genetic factors. For instance, heritability estimates for WB were 40% to 50% (for a review and meta-analysis see, Bartels, 2015; Nes & Røysamb, 2017; Van De Weijer et al., 2022). Similarly, for ADS, twin-based heritability estimates ranging from 49% to 60% were found throughout childhood into adulthood in one study (Baselmans et al., 2018), supported by heritability estimates of 37% found in a meta-analysis (Sullivan et al., 2000) and a recent review study which has mentioned heritability estimates ranging between 30% to 50% (Kendall et al., 2021). Although based on a single study, heritability estimates of 67% and 40% have been reported for SMU, particularly for SMU for communicating with friends and using SMU for contacting family members living in other households, respectively (York, 2017).

Applying a multivariate approach genetic influence of 46% and 50% was found for the covariance between WB and ADS in two separate studies (Baselmans et al., 2018; Routledge et al., 2016). These results indicate that at least close to half of the (negative)

phenotypic correlation between WB and ADS was explained by genetic factors (and the remaining covariance was due to unique environmental factors). Furthermore, moderate to strong genetic correlations (r = -0.60/ r = -0.71) and environmental correlations (r = -0.48/ r = -0.43) were found. The negative genetic correlation between WB and ADS found in these studies indicated a moderate overlap between the underlying set of genetic factors that positively influence WB, negatively influence ADS, and vice versa. The same logic can be applied to the negative environmental correlations found between WB and ADS: a substantial part of the environmental factors positively associated with WB is the same for ADS but negatively associated with ADS levels.

The present study

In the present study, we apply a multivariate twin design to disentangle additive genetic and environmental effects (shared and non-shared) to investigate the underlying sources of the observed association between WB/ADS and SMU. Additionally, we calculate genetic and environmental correlations to assess the degree of overlap in the genetic and environmental factors that influence these phenotypes. We use both hedonic and eudaimonic WB measures to capture a complete picture of human well-functioning, and we use a separate ADS measure to assess illbeing (see the recommendations in, Valkenburg, Beyens, et al., 2022). To detect social media use from different angles we included the time spent on different social media platforms (SMU_t), the frequency of posting on social media (SMU_f), and the number of social media accounts individuals have (SMU_n)

The phenotypic association between WB and SMU, as well as ADS and SMU, may potentially differ for different sex and age groups, in that younger individuals and females might be more prone to lower wellbeing/higher illbeing associated with more social media

use (e.g., Orben et al., 2022). Therefore, we included both age and sex as a covariate in our analyses.

Method

The description of the dataset and the planned analyses were preregistered before analysing the dataset. The analysis plan and the (arguments for) deviations from this plan can be found on the Open Science Foundation (OSF) website (https://osf.io/jr4xc/)

Sample and Participants

The data used in this study were obtained from the Netherlands Twin Register (NTR; Ligthart et al., 2019) after meeting the requirements posited by the NTR Data Access Committee and obtaining their permission. In total, the sample included 6492 individuals, including 3369 MZ twins (893 complete twin pairs, 1583 incomplete twin pairs) and 3123 DZ twins (445 complete, 2233 incomplete). The mean age in the sample was 35.10 (*SD* = 14.93, range = 16 – 89). The sample consisted of 71% females (n = 4618).

Measures

Hedonic wellbeing - Hedonic wellbeing was assessed with the satisfaction with life scale (SWL, Diener et al., 1985), the subjective happiness scale (SHS, Lyubomirsky & Lepper, 1999), and a single-item measure of quality of life (QoL, Cantril, 1965). The SWL scale consisted of five items and the SHS consisted of four items (the fourth item was reverse-coded). Both measures were responded to on a 7-point Likert scale with answer options ranging from strongly disagree to strongly agree. An example item for satisfaction with life scale is: “I am satisfied with my life.” An example item for the subjective happiness scale is “Compared with most of my peers, I am less happy than they are.” The items were summed to obtain an overall SWL and SHS score.

The exact item used for QoL consisted of the following two sentences: “Where on the scale would you put your life in general? A score of 10 means the best life you can imagine, and zero means the worst life you can imagine”. The participants provided their responses by using a ten-step ladder. In the present sample, the reliability of the SWL and SH scales were 0.87 and 0.89, respectively. Single-item measures for wellbeing (such as Cantril Ladder in this case) are usually found reliable (Lucas & Brent Donnellan, 2012).

Eudaimonic wellbeing - Eudaimonic wellbeing was assessed by calculating a sum of responses on the Flourishing Scale (FL; Diener et al., 2010). The scale consists of eight items that could be responded to on a 7-point Likert scale with answer options ranging from strongly disagree to strongly agree. An example item was: “I am engaged and interested in my daily activities.” In the present sample, the reliability of the FL scale was 0.90.

Anxious-depressive symptoms - The anxious-depressive symptoms score (ADS) was assessed by calculating a sum score based on the 18 items from the Adults Self-Report Anxious-Depressed Syndrome Scale (Achenbach & Rescorla, 2003). The responses were given on a three-point Likert scale: (1) “Not at all”, (2) “somewhat or sometimes”, and (3) “very much or so often”. An example item was: “I worry about my future”. In the present sample, the reliability of the ADS scale was 0.92.

Time spent on different social media platforms (SMU_t) - was assessed by calculating the sum value of the responses obtained separately for five different social media platforms - Facebook, Instagram, Snapchat, Twitter, and LinkedIn. The exact item used in this study was: “How much time do you spend on ‘[PLATFORM NAME]’?”. The response options were on a 6-point Likert scale: 1-“never”, 2-“less than 30 minutes”, 3-“30 to 60 minutes”, 4-“1 to 2 hours”, 5- “2 to 3 hours”, 6- “more than 3 hours”.

The frequency of posting on social media (SMU_f) - was measured by a single item: "How often do you post on social media?" The response options were on a 6-point Likert scale: (1) "Never", (2) "once every couple of months", (3) "once every couple of weeks", (4) "weekly", (5) "daily", (6) "multiple times a day". We merged the categories (5) "daily" and (6) "multiple times a day" with each other as the responses provided for these categories (149 and 39) were considerably lower than the other categories (which were 1330, 1704, 1081, 396 from "1" to "4", respectively). The new category was labelled as (5) "daily".

The number of social media accounts (SMU_n) was measured by a single item. The exact item used in this study was: "On how many social media channels do you have an account (e.g., Facebook, Instagram, Twitter, LinkedIn, not Whatsapp)?". The participants were allowed to respond through one of the eight following options: "0", "1", "2", "3", "4", "5", "6", "7", "8 or more". We merged the last 2 answer categories ("7", and "8 or more") with the response category "6" given the number of individuals who selected these last two answer categories was noticeably more limited (25 and 87, respectively) than the responses to other categories (which were 361, 810, 1058, 748, 356, 119 from "0" to "6", respectively).

Analyses

Twin models leverage the differences in the degree of relatedness between monozygotic twin pairs (sharing 100% of the segregating genes) and dizygotic twin pairs (sharing on average 50%) to decompose the variance of a phenotype and the covariance between phenotypes in genetic and environmental variance/covariances. Additive genetic variance (A) indicates the proportion of variance that can be explained by the effect of independent alleles on the phenotype. Non-additive genetic variance (D) indicates the proportion of variance explained by interactions between alleles at the same locus (dominance) or between alleles from different loci (epistasis). Environmental variance

consists of environmental variance shared by members of the same family (C), and a unique environment component (E) including individual-specific environmental influences and measurement error.

In the classical twin model, models are not identified when both the C and D variance components are included, therefore a decision has to be made based on observed data and/or results from previous studies. In the observed data, if the cross-twin-cross-trait correlations for MZ twins are more than 2 times the correlations for DZ twins (i.e., rMZ > 2*rDZ), this is an indication of dominant genetic effects, whereas if the opposite is true (i.e., rMZ < 2*rDZ), shared environment effects are more plausible.

We first estimated phenotypic correlations, twin correlations, and cross-twin cross-trait with a saturated model using the OpenMx package (Boker et al., 2011) in R (R Core Team, 2000). We treated missing values in our data using the Full Information Maximum Likelihood (FIML) method. In the saturated model, we included all of the eight phenotypic variables, namely the four wellbeing scores, one anxious-depressive symptom score, and the three social media use scores. Age was included as a covariate on means given the presence of age differences in social media use (Datareportal, 2023). The age variable was divided by 100 to facilitate model estimation, which means that the regression coefficients are expressed in centuries. Sex is known to be a potential moderator for explaining the associations between WB/ADS and SMU (Kelly et al., 2018; Nesi & Prinstein, 2015). However, in the present study, the sample size was too small for testing sex differences; therefore, sex was only included as a covariate on means, similar to age.

Overall, our analyses were based on two zygosity groups (MZ and DZ twins). First, for each phenotype, we tested in a univariate model whether the assumption of equal means and variances for the oldest and youngest twins and for the MZ and the DZ twins held. All

versions of the saturated models, with a unique set of equality constraints on various parameters, were compared to the initial saturated model without any of the constraints. These comparisons were made using the chi-square ($\chi 2$) difference test. If the model fit changes significantly by introducing constraints/making the model more parsimonious (by finding significant $\chi 2$ difference test results at $p < 0.01$), this indicates the specifically tested assumption in the constrained model was successfully met. In addition, we utilised the AIC values between the models for comparison for both nested and non-nested models to assess model fit (smaller values indicated better fit). After checking the classical twin model assumptions, we estimated cross-twin cross-trait correlations in an 8-variate saturated model, with sex and age as the covariates on the means.

Cholesky model

Based on the cross-twin cross-trait correlations, we applied a Cholesky decomposition to decompose the phenotypic covariance matrix into genetic and environmental (co)variance components. Following the estimation of the Cholesky model, we proceeded with fitting a series of nested models by fixing the parameters for A and/or C (co)variance components to zero and comparing the log-likelihood values of the restricted models with the non-restricted models. Overall, we tested an ACE, AE, CE, and an E model.

Common pathway model

In addition to the Cholesky model, the genetic and environmental effects can be modelled into common and specific factors in a common pathway (CP) model. More specifically, the CP model allows different phenotypes to be defined as indicators of a single or multiple latent factor(s), for which genetic and environmental variances can be estimated. In addition, variance components unique (i.e., independent of the common factor(s)) to each phenotype can be estimated (phenotype-specific/unique effects). In the CP model, a WB and

an SMU common factor were specified. The WB factor is comprised of our WB wellbeing indicators and the ADS indicator (negatively weighted). The SMU factor is comprised of the three SMU variables (see Figure 3). To make comparisons between the nested models, we used the chi-square difference tests.

< INSERT FIGURE 3 HERE >

Results

Descriptives and phenotypic correlations

Table 1 shows the descriptive values and Figure 1 shows the phenotypic correlations at alpha = 0.05 level. SMU_t and SMU_f were moderately correlated ($r > 0.6$), while SMU_n was weaker associated with SMU_t and SMUf (r = 0.3-0.4). Approximately half of the phenotypic associations between WB/ADS and SMU (7 out of 15) were statistically significant at alpha = 0.05 and ranged between r = 0.03 and r = 0.1 (absolute values).

< INSERT TABLE 1 HERE >

< INSERT FIGURE 1 HERE >

Covariates - age/sex and assumption tests

Age and sex had a significant effect on five and six of the eight variables, respectively. Higher age was positively associated with SHS (B = 0.035, SE = 0.004, $p <$ 0.0001), QoL (B = 0.011, SE = 0.001, $p < 0.0001$) and negatively associated with SMU_t (B = -0.088, SE = 0.003, $p < 0.0001$) and SMU_f (B = -0.048, SE = 0.001, $p < 0.0001$). Females had lower SWL (B = -0.585, SE = 0.163, $p < 0.001$), SHS (B = -0.389, SE = 0.142, $p < 0.010$), QoL (B = -0.129, SE = 0.035, $p < 0.001$), higher level of ADS (B = 1.761, SE = 0.209, $p <$

0.0001), higher SMU_t (B= 0.656, SE =0.092591125, $p < 0.0001$), and higher SMU_n (B = 0.220, SE =0.035, $p < 0.0001$).

The chi-square difference tests showed the assumptions of equal means and variance across co-twins and zygosities were met (see Supplementary Table S1 for the exact model fitting results).

Saturated model

Table 2 contains the twin correlations and the cross-twin cross-trait correlations estimated in the saturated model. The consistently higher MZ (cross-twin, cross-trait) correlations than the DZ (cross-twin, cross-trait) correlations indicated that additive genetic influences explained part of the (co)variance for all phenotypes. There was no evidence for a genetic dominance effect, therefore we proceeded with estimating a Cholesky ACE model.

< INSERT TABLE 2 HERE >

Cholesky model

The Cholesky ACE model provided a better fit compared to the saturated model based on the chi-square difference test (*p*-value > 0.01) and the AIC values between the two models (174123.86 versus 173994.19; see Table 3). Further comparing the fit of a Cholesky AE model revealed the shared environmental (C) (co)variance components could be constrained to zero (*p*-value > 0.01, AIC 173955.43).

< INSERT TABLE 3 HERE >

The unstandardized additive genetic (A) and unique environmental variances (E) based on the Cholesky AE model are shown in Supplementary Table S2. The unstandardized covariances for A and E are provided in Table S3. The standardized additive genetic (A) and

unique environmental variances (E) are shown in Table 4. The standardized covariances for A and E are provided in Table 5. In general, additive genetic factors (A) explained a moderate share of the variance in WB/ADS (between 38% and 47%; see Table 4). The additive genetic influences on SMU ranged between 32% and 72%. The remaining variance is accounted for by non-shared environmental factors (E), which ranged between 53% and 62% for WB/ADS, and between 28% and 68% for SMU.

< INSERT TABLE 4 HERE >

< INSERT TABLE 5 HERE >

Among the seven significant phenotypic correlations between WB/ADS and SMU variables, we only investigated the genetic and environmental influences on four of these associations[1]. The results indicated that phenotypic association between QoL with SMU_t and SMU_f (r = -0.07 and r = -.09 respectively) were largely determined by additive genetic factors (89% and 87%) in comparison to unique environmental factors (11% and 13%). Similarly, FL's association with SMU_n (r = 0.04), and the correlation of ADS with SMU_t (r = 0.07) were largely determined by genetic factors (99% and 80% respectively), where the influence of environmental factors was much more limited (1% and 20%).

Genetic and environmental correlations are shown in Figure 2. There were only small or non-significant genetic and environmental correlations between the WB/ADS and SMU variables. In total, only four statistically significant genetic correlations were observed, while none of the environmental correlations were statistically significant for the same variables. Both QoL and ADS had statistically significant genetic correlations with SMU_t and SMU_f (r

[1] This was because only in 4 of these 7 cases, additive genetic and environmental covariances had the same signs (e.g., either both values were positive or negative). When the same signs for both values are not observed in the data, genetic and environmental covariances cannot be interpreted in a meaningful way.

= -0.12 and -0.17; for QoL; r = 0.10 and 0.23). These results highlight a potentially small but significant overlap between the genetic factors that underly WB/ADS variables and SMU.

< INSERT FIGURE 2 HERE >

Common factor model

The common factor model with AE variance components provided a better fit than the model with all ACE components based on the chi-square differences test (p-value > 0.01) and the AIC values between the two models (174376.46 versus 174387.27, respectively). The Cholesky AE model provided a better fit than the common factor AE model (173955.43 versus 174376.46, respectively). The common factor model was used to provide a more fine-grained decomposition of the estimated variance components (i.e., through common and specific effects for each phenotype). The best-fitting common factor model is shown in Figure 3.

The phenotypic correlation between the common WB and SMU factors was -0.05 (-0.08, -0.01). The standardized additive genetic (A) and unique environmental variance (E) for the common WB factor were 47% and 53%, respectively. For the common SMU factor, the standardized additive genetic (A) and the unique environmental variances (E) for the SMU factor were 77% and 23%, also in their respective order. The genetic and environmental influences on the covariance between the common WB and SMU factors were 70% and 30%, respectively. A non-significant genetic correlation of r = -0.06 (-0.13, 0.02) and a non-significant environmental correlation of r = -0.04 (-0.14, 0.06) were found between common WB and SMU factors as well.

The total variance in the WB and ADS phenotypes was mostly due to common additive genetic effects (between 27% and 37% of the total variance) and not due to

phenotype-specific effects (between 3% and 16% of the total variance; see Figure 4). For the non-shared environmental effects, the common effects (between 30% and 41% of the total variance) also played a more central role and not the phenotype-specific effects (between 17% to 28%).

< INSERT FIGURE 4 HERE >

SMU_t was predominantly determined by common additive genetic effects (57% of the total variance), and not as much through the phenotype-specific effects (only 14% of the total variance). The common and phenotype-specific non-shared environmental effects were similar, and they explained a smaller portion of the total variance in SMU_t (17% and 12% of the total variance, respectively). Variance in SMU_f was largely determined by common (43%) and less by specific additive genetic factors (10%). The common non-shared environmental effects were smaller than the phenotype-specific counterparts (13% and 43% of the total variance, respectively).

Different from the other two SMU variables, the variance in SMU_n was largely determined by phenotype-specific environmental effects (60% of the total variance), while the remaining factors played a more limited role. These were common non-shared environmental, and common and specific additive genetic factors, explaining 4%, 14%, and 22% of the total variances, respectively.

Discussion

The current study investigated the underlying sources of overlap between wellbeing (WB), anxious-depressive symptoms (ADS), and social media use (SMU) leveraging data from a large population-based twin sample. Consistent with the literature, we found small or no associations between WB/ADS and SMU variables. Social media use was heritable with

estimates as high as 72% when measured through time spent daily on social media. The small but statistically significant associations between WB/ADS and SMU seemed to be due to overlapping genetic factors that influence both WB/ADS and SMU. The strong phenotypic relationship between time spent on social media and posting frequency also manifested in their genetic and environmental correlations.

The phenotypic correlation between SMU_t and SMU_f (r = 0.64) was primarily driven by additive genetic factors (80%), and the remaining part of the correlation can be explained by non-shared environmental influences. On the contrary, SMU_n's phenotypic correlations with SMU_t (r = 0.37) and SMU_f (r = 0.32) were less strongly influenced by additive genetic factors (63% and 55%, respectively) but more by non-shared environmental influences (about 37% and 45%, respectively).

We found strong genetic correlation between SMU_t and SMU_f (r = 0.82), which indicated strong overlap in genetic influences on these SMU variables. On the other hand, SMU_n had smaller genetic correlations with SMU_t and SMU_f (r = 0.48 and 0.42), indicating a smaller overlap with the other two SMU variables. We did not find strong environmental correlations among any of the SMU variables (r = ranging from 0.26 to 0.37) which indicated overlap in environmental influences were limited for these variables. These findings indicate that any intervention or prevention effort to limit the number of social media accounts will likely not generalize to time spent on social media and posting frequency. Alternatively, interventions to limit time spent on social media and posting frequency may have a greater chance of success due to their larger (genetic) overlap with each other, when the aim is to limit (problematic) amounts of social media use.

We found heritability estimates of 72% for SMU_t (CI 95% = between 68% - 75%), 32% for SMU_n (25% - 38%) and 54% for SMU_f (50% - 59%). Our results resembled the

results of the only study on the heritability of SMU, which found a heritability estimate of 40% (6-73%) for using social media to communicate with family members in other households and a heritability estimate of 67% (39-96%) for using social media to communicate with friends (York, 2017). The present study expands the evidence by finding similar heritability estimates for broader operationalizations of social media use not limited to using social media for specific motivations. We, for example, found individuals in the present study's sample report a wide range of motivations for using social media, such as "to follow the news," or "to be inspired", among others (see Supplementary Table S4).

In general, our findings for the heritability of SMU showed that individuals may have different genetic liabilities which may result in very diverse social media use habits. This information can be utilized to explain differences in social media use both across different individuals but also across different families. Relatedly, the social media usage patterns of parents can be used to better understand the social media use of the children and adolescents. This is because parent and offspring's social media behaviour could be subject to shared genetic influences. Such information from the parents can be particularly useful for setting realistic goals in terms of determining the efficacy of interventions in which the aim is to decrease excessive amount of social media use, also referred to as "addictive social media use" (Andreassen et al., 2017). In addition to this, especially in adolescent target groups, interventions that primarily focus on leveraging within-family-based support to control addictive social media use might be less effective, as familial genetic predisposition might play a role in developing (problematic) patterns of social media use.

In the present study, 7 out of 15 phenotypic correlations between WB/ADS and SMU were statistically significant, yet very small (*r*s between 0.03 and 0.10). These results align with meta-review studies for wellbeing and illbeing variables (Orben, 2020; Parry et al.,

2022; Valkenburg, 2022) which have reported similar small or null associations between WB/ADS and SMU, underlining the potentially complex nature of the associations between these variables. Although strong claims are often made on the negative effects of social media use on wellbeing (e.g., Haidt, 2024), these are not substantiated in the current study.

Looking into these associations in more detail, we found that different WB measures manifested different correlations with SMU depending on the specific WB measure. Specifically, flourishing was positively associated with having more social media accounts (SMU_n) and spending more time on social media (SMU_t). The other WB measures were not related to having more social media accounts. Moreover, the signs of the associations between flourishing and the SMU variables were positive, whereas the other WB variables related to the SMU variables negatively. For instance, higher hedonic wellbeing levels negatively correlated with spending more time on social media (SMU_t) and more frequently posting on social media (SMU_f).

As the associations between WB/ADS and SMU variables were very small, they require caution when interpreting them. Nonetheless, it could be speculated that individuals with higher flourishing levels combine the use of more social media channels to utilize the unique advantages or the properties that each platform may offer (for instance, one platform can be better at following work related updates). The same individuals may also spend more time on social media, yet they do not post more frequently. This may indicate that higher flourishing might be associated with a more passive way of engaging with social media as opposed to more active ways such as posting. This is in line with the evidence that adolescents who flourish may spend more time on social media, but do not report themselves as ‘oversharing their activities’ compared to the general, ‘neither flourishing nor languishing’ group of users (Law et al., 2019). In contrast, lower hedonic wellbeing levels may be related

to spending more time on social media and posting more frequently without combining the use of multiple social media platforms. This finding is partly supported by previous evidence suggesting that oversharing on social media and dysfunctional amounts of social media use to be related to lower wellbeing levels in adolescents (Shabahang et al., 2024). Given the small overall correlation pattern, our interpretations of the present results have to be verified by future research.

The phenotypic correlations between WB/ADS and SMU (which were generally smaller than absolute $r = 0.1$) that were statistically significant were largely determined by additive genetic factors (between 80-90%). Given the (very) small phenotypic correlations between WB/ADS and SMU the covariance estimates for the genetic and environmental influences should be interpreted with care. We found that both quality of life and ADS had statistically significant genetic correlations with SMU_t and SMU_f (r = -0.12 and -0.17 for quality of life; r = 0.10 and 0.23 for ADS). No significant environmental correlations were found between WB/ADS and SMU variables. Therefore, although the evidence suggests increased social media use may be related to decreased wellbeing or mood (Appel et al., 2020; Valkenburg, Beyens, et al., 2022; Valkenburg, Meier, et al., 2022), these associations may not be causal but (partly) due to genetic confounding. Overall, any potential intervention aimed to increase wellbeing by the means of decreasing social media use will likely not be very successful due to their small phenotypic correlations. Nevertheless, some individuals may be genetically predisposed to use more social media and at the same predisposed to lower levels of wellbeing and lower levels of depressive symptoms. Therefore, interventions to increase wellbeing through limiting social media use may be more effective for some individuals compared to others due the genetic overlap between the two variables.

In addition to our Cholesky decomposition, we applied a common pathway model with two latent factors (WB and SMU) to obtain a more overarching view of common and unique influences. No statistically significant genetic correlation was found between the common WB and SMU factors. Potentially, this was due to the common factor model assuming a single genetic correlation estimate between all WB/ADS and SMU variables, muddling significant with non-significant and positive with negative signed genetic correlations at the indicator level.

In the common pathway model, it was also clear that SMU_t and SMU_f were more similar to each other than to SMU_n, as indicated by larger influences of the common genetic factor. Taken together with the high phenotypic and genetic correlations between SMU_t and SMU_f, these results may indicate that SMU_t cannot be considered a passive SMU measure given its considerable phenotypic and genetic correlation with SMU_f (posting frequency), which is clearly an active SMU measure. In addition, SMU_n seems to be more distinct from SMU_t and SMU_f. Since the SMU variables showed different (genetic) associations with each other and with the WB/ADS variables, future studies may want to include different SMU measures because they seem to capture different (genetic) aspects of mental health.

Limitations

In the present study, we had certain limitations. First, the present sample size did not allow for testing quantitative and qualitative sex differences. However, we included sex as a covariate to account for its influence on mean levels of WB/ADS and SMU. Second, the data from the present study were cross-sectional, making it impossible to make claims regarding the direction of causation between the variables. Fourth, our study employed the classical twin design, which has a series of assumptions (such as not modelling existing gene-environment correlations) (Derks et al., 2004; Kendler et al., 1993). In the present study, we

tested for equal mean and variances between twin siblings, and MZ and DZ twins for each of the phenotypes. However, we did not test for random mating, the equal environment assumption, or whether there were any gene-environment correlations, and gene-environment interactions present in the data. Among these unmodeled potential interplays, gene-environment interactions and gene-environment correlations can cause the overestimation of A and E variances, respectively (Little, 2013). Furthermore, the presence of assortative mating, if not modelled, causes underestimated A and overestimated C variances (Little, 2013). Although we did not test the equal environments assumption, it usually holds for many phenotypes (Willoughby et al., 2023). Fifth, our sample was from the Netherlands; therefore, the heritability estimates found for each phenotype may differ in other cultures, as it was found for other behavioural variables such as personality (Jang et al., 2006). Future research should replicate these findings in samples from other cultures and populations. Lastly, the distribution of the variables ADS and SMU_t showed signs of being positively skewed (the lowest scores were the most frequently present in the dataset). As a part of our pre-registered post-hoc analysis plan, we have repeated our main analyses, with the transformed values for ADS and SMU_t .The results revealed that our heritability estimates did not significantly change as the confidence intervals between the original and post-hoc heritability estimates overlapped with one another (see Supplementary Table S5 and S6, for the post-hoc estimates).

Concluding remarks

The present study employed a classical twin design based on a population-based sample to explore the underlying sources of overlap between wellbeing, anxious-depressive symptoms, and social media use. Our results confirmed the small associations between social media use and wellbeing/anxious-depressive symptoms in the literature (Appel et al., 2020;

Valkenburg, Beyens, et al., 2022; Valkenburg, Meier, et al., 2022), and suggested these associations to be partly driven by overlapping genetic factors. Further, we showed general social media use to be heritable as high as 72%. Given our findings, we encourage researchers and experts to consider more personalized approaches when considering the association between wellbeing and social media use intensity, as well as when understanding the reasons for individuals' use of social media use itself.

References

Achenbach, T. M., & Rescorla, L. A. (2003). *Manual for the ASEBA adult forms & profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth and Families.

Andreassen, C. S., Pallesen, S., & Griffiths, M. D. (2017). The relationship between addictive use of social media, narcissism, and self-esteem: Findings from a large national survey. *Addictive behaviors*, *64*, 287-293.

Appel, M., Marker, C., & Gnambs, T. (2020). Are social media ruining our lives? A review of meta-analytic evidence. *Review of General Psychology*, *24*(1), 60–74. https://doi.org/10.1177/1089268019880891

Bartels, M. (2015). Genetics of wellbeing and its components satisfaction with life, happiness, and quality of life: A review and meta-analysis of heritability studies. *Behavior Genetics, 45*, 137–156. https://doi.org/10.1007/s10519-015-9713-y

Baselmans, B. M. L., Willems, Y. E., van Beijsterveldt, C. E. M., Ligthart, L., Willemsen, G., Dolan, C. V., Boomsma, D. I., & Bartels, M. (2018). Unraveling the genetic and environmental relationship between well-being and depressive symptoms throughout the lifespan. *Frontiers in Psychiatry*, *9*, 261. https://doi.org/10.3389/fpsyt.2018.00261

Beyens, I., Pouwels, J. L., van Driel, I. I., Keijsers, L., & Valkenburg, P. M. (2020). The effect of social media on well-being differs from adolescent to adolescent. *Scientific Reports*, *10*(1), 10763. https://doi.org/10.1038/s41598-020-67727-7

Boker, S., Neale, M., Maes, H., Wilde, M., Spiegel, M., Brick, T., Spies, J., Estabrook, R., Kenny, S., Bates, T., Mehta, P., & Fox, J. (2011). OpenMx: An open source extended structural equation modeling framework. *Psychometrika*, *76*(2), 306–317. https://doi.org/10.1007/s11336-010-9200-6

Cantril, H. (1965). *The pattern of human concerns*. Rutgers University Press.

Casey, P., Dunn, G., Kelly, B. D., Lehtinen, V., Dalgard, O. S., Dowrick, C., & Ayuso-Mateos, J. L. (2008). The prevalence of suicidal ideation in the general population: Results from the

Outcome of Depression International Network (ODIN) study. *Social Psychiatry and Psychiatric Epidemiology*, *43*(4), 299–304. https://doi.org/10.1007/s00127-008-0313-5

Chapman, B., & Guven, C. (2016). Revisiting the relationship between marriage and wellbeing: Does marriage quality matter? *Journal of Happiness Studies*, *17*, 533–551. https://doi.org/10.1007/s10902-014-9607-3

Cunningham, S., Hudson, C. C., & Harkness, K. (2021). Social media and depression symptoms: A meta-analysis. *Research on Child and Adolescent Psychopathology*, *49*, 241–253. https://doi.org/10.1007/s10802-020-00715-7

Datareportal. (2023). *Global social media statistics*. https://datareportal.com/social-media-users

De Vries, L. P., Van Beijsterveldt, T. C. E. M., Maes, H., Colodro-Conde, L., & Bartels, M. (2021). Genetic Influences on the Covariance and Genetic Correlations in a Bivariate Twin Model: An Application to Well-Being. *Behavior Genetics*, *51*(3), 191–203. https://doi.org/10.1007/s10519-021-10046-y

Derks, E. M., Dolan, C. V., & Boomsma, D. I. (2006). A test of the equal environment assumption (EEA) in multivariate twin studies. *Twin Research and Human Genetics*, *9*(3), 403-411. https://doi.org/10.1375/twin.9.3.403

Deci, E. L., & Ryan, R. M. (2008). Hedonia, eudaimonia, and well-being: An introduction. *Journal of Happiness Studies*, *9*(1), 1–11. https://doi.org/10.1007/s10902-006-9018-1

Diener, E., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of Personality Assessment*, *49*(1), 71–75.

Diener, E., Wirtz, D., Tov, W., Kim-Prieto, C., Choi, D., Oishi, S., & Biswas-Diener, R. (2010). New well-being measures: Short scales to assess flourishing and positive and negative feelings. *Social Indicators Research*, *97*, 143–156. https://doi.org/10.1007/s11205-009-9493-y

Escobar-Viera, C. G., Shensa, A., Bowman, N. D., Sidani, J. E., Knight, J., James, A. E., & Primack, B. A. (2018). Passive and Active Social Media Use and Depressive Symptoms Among United States Adults. *Cyberpsychology, Behavior, and Social Networking*, *21*(7), 437–443. https://doi.org/10.1089/cyber.2017.0668

Hagerty, M. R., & Veenhoven, R. (2003). Wealth and happiness revisited–growing national income does go with greater happiness. *Social indicators research*, 64, 1-27. https://doi.org/10.1023/A:1024790530822

Haidt, J. (2024). *The anxious generation: How the great rewiring of childhood is causing an epidemic of mental illness*. Random House.

Hogue, J. V., & Mills, J. S. (2019). The effects of active social media engagement with peers on body image in young women. *Body Image*, *28*, 1–5. https://doi.org/10.1016/j.bodyim.2018.11.002

Izutsu, T., Tsutsumi, A., Minas, H., Thornicroft, G., Patel, V., & Ito, A. (2015). Mental health and wellbeing in the Sustainable Development Goals. *The Lancet Psychiatry*, *2*(12), 1052-1054.

James, P., Kim, E. S., Kubzansky, L. D., Zevon, E. S., Trudel-Fitzgerald, C., & Grodstein, F. (2019). Optimism and healthy aging in women. *American Journal of Preventive Medicine*, *56*(1), 116–124. https://doi.org/10.1016/j.amepre.2018.07.037

Jang, K. L., Livesley, W. J., Ando, J., Yamagata, S., Suzuki, A., Angleitner, A., Ostendorf, F., Riemann, R., & Spinath, F. (2006). Behavioral genetics of the higher-order factors of the Big Five. *Personality and Individual Differences*, *41*(2), 261–272. https://doi.org/10.1016/j.paid.2005.11.033

Kelly, Y., Zilanawala, A., Booker, C., & Sacker, A. (2018). Social media use and adolescent mental health: Findings from the UK Millennium Cohort Study. *EClinicalMedicine*, *6*, 59–68. https://doi.org/10.1016/j.eclinm.2018.12.005

Kendall, K. M., Van Assche, E., Andlauer, T. F. M., Choi, K. W., Luykx, J. J., Schulte, E. C., & Lu, Y. (2021). The genetic basis of major depression. *Psychological Medicine*, *51*(13), 2217–2230. https://doi.org/10.1017/S0033291721000441

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A test of the equal-environment assumption in twin studies of psychiatric illness. Behavior genetics, 23, 21-27. https://doi.org/10.1007/BF01067551

Lee, S. L., Pearce, E., Ajnakina, O., Johnson, S., Lewis, G., Mann, F., Pitman, A., Solmi, F., Sommerlad, A., Steptoe, A., Tymoszuk, U., & Lewis, G. (2021). The association between loneliness and depressive symptoms among adults aged 50 years and older: A 12-year population-based cohort study. *The Lancet Psychiatry*, *8*(1), 48–57. https://doi.org/10.1016/S2215-0366(20)30383-7

Ligthart, L., Beijsterveldt, C. E. M. van, Kevenaar, S. T., Zeeuw, E. de, Bergen, E. van, Bruins, S., Pool, R., Helmer, Q., Dongen, J. van, Hottenga, J.-J., Ent, D. van't, Dolan, C. V., Davies, G. E., Ehli, E. A., Bartels, M., Willemsen, G., Geus, E. J. C. de, & Boomsma, D. I. (2019). The Netherlands Twin Register: Longitudinal Research Based on Twin and Twin-Family Designs. *Twin Research and Human Genetics*, *22*(6), 623–636. https://doi.org/10.1017/thg.2019.93

Little, T. D. (Ed.). (2013). *The Oxford Handbook of Quantitative Methods in Psychology: Vol. 2* (1st ed.). Oxford University Press. https://doi.org/10.1093/oxfordhb/9780199934898.001.0001

Lucas, R. E., & Brent Donnellan, M. (2012). Estimating the Reliability of Single-Item Life Satisfaction Measures: Results from Four National Panel Studies. *Social Indicators Research*, *105*(3), 323–331. https://doi.org/10.1007/s11205-011-9783-z

Lyubomirsky, S., & Lepper, H. S. (1999). A measure of subjective happiness: Preliminary reliability and construct validation. *Social Indicators Research*, *46*(2), 137–155. https://doi.org/10.1023/A:1006824100041

Maccagnan, A., Wren-Lewis, S., Brown, H., & Taylor, T. (2019). Wellbeing and Society: Towards Quantification of the Co-benefits of Wellbeing. *Social Indicators Research*, *141*(1), 217–243. https://doi.org/10.1007/s11205-017-1826-7

Nes, R. B., & Røysamb, E. (2017). Happiness in Behaviour Genetics: An Update on Heritability and Changeability. *Journal of Happiness Studies*, *18*(5), 1533–1552. https://doi.org/10.1007/s10902-016-9781-6

Nesi, J., & Prinstein, M. J. (2015). Using social media for social comparison and feedback-seeking: Gender and popularity moderate associations with depressive symptoms. *Journal of Abnormal Child Psychology*, *43*, 1427–1438. https://doi.org/10.1007/s10802-015-0020-0

Okabe-Miyamoto, K., & Lyubomirsky, S. (in press). Happiness shapes and is shaped by social cognition and social connection. In D. Carlston, K. Johnson, & K. Hugenberg (Eds.), *The Oxford handbook of social cognition* (2nd ed.). Oxford University Press.

Orben, A. (2020). Teenagers, screens and social media: A narrative review of reviews and key studies. *Social Psychiatry and Psychiatric Epidemiology*, *55*(4), 407–414. https://doi.org/10.1007/s00127-019-01825-4

Orben, A., & Przybylski, A. K. (2019). Screens, Teens, and Psychological Well-Being: Evidence From Three Time-Use-Diary Studies. *Psychological Science*, *30*(5), 682–696. https://doi.org/10.1177/0956797619830329

Orben, A., Przybylski, A. K., Blakemore, S.-J., & Kievit, R. A. (2022). Windows of developmental sensitivity to social media. *Nature Communications*, *13*(1), 1649. https://doi.org/10.1038/s41467-022-29296-3

Oswald, A. J., Proto, E., & Sgroi, D. (2015). Happiness and productivity. *Journal of Labor Economics*, *33*(4), 789–822.

Parry, D. A., Fisher, J. T., Mieczkowski, H., Sewall, C. J., & Davidson, B. I. (2022). Social media and well-being: A methodological perspective. *Current Opinion in Psychology*, *45*, 101285. https://doi.org/10.1016/j.copsyc.2021.11.005

Pereira, M. A., Camanho, A. S., Marques, R. C., & Figueira, J. R. (2021). The convergence of the world health organization member states regarding the united nations' sustainable development goal 'good health and well-being'. Omega, 104, 102495. https://doi.org/10.1016/j.omega.2021.102495

Pouwels, J. L., Valkenburg, P. M., Beyens, I., van Driel, I. I., & Keijsers, L. (2021). Social media use and friendship closeness in adolescents' daily lives: An experience sampling study. *Developmental Psychology*, *57*(2), 309. https://doi.org/10.1037/dev0001148

R Core Team. (2000). R language definition. *Vienna, Austria: R Foundation for Statistical Computing*, *3*(1).

Ridley, M., Rao, G., Schilbach, F., & Patel, V. (2020). Poverty, depression, and anxiety: Causal evidence and mechanisms. *Science*, *370*(6522), eaay0214. https://doi.org/10.1126/science.aay0214

Routledge, K. M., Burton, K. L. O., Williams, L. M., Harris, A., Schofield, P. R., Clark, C. R., & Gatt, J. M. (2016). Shared versus distinct genetic contributions of mental wellbeing with depression and anxiety symptoms in healthy twins. *Psychiatry Research*, *244*, 65–70. https://doi.org/10.1016/j.psychres.2016.07.016

Ryff, C. D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology*, *57*(6), 1069.

Santini, Z. I., Becher, H., Jørgensen, M. B., Davidsen, M., Nielsen, L., Hinrichsen, C., Madsen, K. R., Meilstrup, C., Koyanagi, A., Stewart-Brown, S., McDaid, D., & Koushede, V. (2021). Economics of mental well-being: A prospective study estimating associated health care costs and sickness benefit transfers in Denmark. *The European Journal of Health Economics*, *22*(7), 1053–1065. https://doi.org/10.1007/s10198-021-01305-0

Santini, Z. I., Nielsen, L., Hinrichsen, C., Nelausen, M. K., Meilstrup, C., Koyanagi, A., McDaid, D., Lyubomirsky, S., VanderWeele, T. J., & Koushede, V. (2021). Mental health economics: A prospective study on psychological flourishing and associations with healthcare costs and sickness benefit transfers in Denmark. *Mental Health & Prevention*, *24*, 200222. https://doi.org/10.1016/j.mhp.2021.200222

Shabahang, R., Shim, H., Aruguete, M. S., & Zsila, A. (2024). Oversharing on social media: Anxiety, attention-seeking, and social media addiction predict the breadth and depth of sharing. *Psychological reports*, *127*(2), 513-530. https://doi.org/10.1177/00332941221122861

Spijker, J., De Graaf, R., Ten Have, M., Nolen, W. A., & Speckens, A. (2010). Predictors of suicidality in depressive spectrum disorders in the general population: Results of the Netherlands Mental Health Survey and Incidence Study. *Social Psychiatry and Psychiatric Epidemiology*, *45*(5), 513–521. https://doi.org/10.1007/s00127-009-0093-6

Steinsbekk, S., Nesi, J., & Wichstrøm, L. (2023). Social media behaviors and symptoms of anxiety and depression. A four-wave cohort study from age 10–16 years. *Computers in Human Behavior*, 107859. https://doi.org/10.1016/j.chb.2023.107859

Steptoe, A. (2019). Happiness and health. *Annual Review of Public Health*, *40*, 339–359.

Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: Review and meta-analysis. *American Journal of Psychiatry*, *157*(10), 1552–1562. https://doi.org/10.1176/appi.ajp.157.10.1552

Valkenburg, P. M. (2022). Social media use and well-being: What we know and what we need to know. *Current Opinion in Psychology*, *45*, 101294. https://doi.org/10.1016/j.copsyc.2021.12.006

Valkenburg, P. M., Beyens, I., Meier, A., & Vanden Abeele, M. M. P. (2022). Advancing our understanding of the associations between social media use and well-being. *Current Opinion in Psychology*, *47*, 101357. https://doi.org/10.1016/j.copsyc.2022.101357

Valkenburg, P. M., Meier, A., & Beyens, I. (2022). Social media use and its impact on adolescent mental health: An umbrella review of the evidence. *Current Opinion in Psychology*, *44*, 58–68. https://doi.org/10.1016/j.copsyc.2021.08.017

Valkenburg, P. M., Van Driel, I. I., & Beyens, I. (2022). The associations of active and passive social media use with well-being: A critical scoping review. *New Media & Society*, *24*(2), 530–549. https://doi.org/10.1177/14614448211065425

Van De Weijer, M. P., De Vries, L. P., & Bartels, M. (2022). Happiness and well-being: The value and findings from genetic studies. In *Twin Research for Everyone* (pp. 295–322). Elsevier. https://doi.org/10.1016/B978-0-12-821514-2.00016-7

Vasiliadis, H.-M., Dionne, P.-A., Préville, M., Gentil, L., Berbiche, D., & Latimer, E. (2013). The Excess Healthcare Costs Associated With Depression and Anxiety in Elderly Living in the Community. *The American Journal of Geriatric Psychiatry*, *21*(6), 536–548. https://doi.org/10.1016/j.jagp.2012.12.016

Verduyn, P., Gugushvili, N., & Kross, E. (2022). Do Social Networking Sites Influence Well-Being? The Extended Active-Passive Model. *Current Directions in Psychological Science*, *31*(1), 62–68. https://doi.org/10.1177/09637214211053637

Verduyn, P., Ybarra, O., Résibois, M., Jonides, J., & Kross, E. (2017). Do Social Network Sites Enhance or Undermine Subjective Well-Being? A Critical Review. *Social Issues and Policy Review*, *11*(1), 274–302. https://doi.org/10.1111/sipr.12033

Walsh, L. C., Boehm, J. K., & Lyubomirsky, S. (2018). Does happiness promote career success? Revisiting the evidence. *Journal of Career Assessment*, *26*(2), 199–219. https://doi.org/10.1177/1069072717751441

Willoughby, E. A., Polderman, T. J. C., & Boutwell, B. B. (2023). Behavioural genetics methods. *Nature Reviews Methods Primers*, *3*(1), 10. https://doi.org/10.1038/s43586-022-00191-x

York, C. (2017). A regression approach to testing genetic influence on communication behavior: Social media use as an example. *Computers in Human Behavior*, *73*, 100–109. https://doi.org/10.1016/j.chb.2017.03.029

Tables

Table 1. Descriptives for the 8 phenotypic variables (N, M, SD, Range)

	N	**M**	**SD**	**Range**
SWL	5554	26.99	5.49	5-35
SHS	5541	22.07	4.77	4-28
QoL	5605	7.59	1.17	0-10
FL	5542	45.86	6.08	8-56
ADS	4207	5.68	6.06	0-33
SMU_t	4295	8.6	2.74	5-26
SMU_n	4732	2.66	1.54	0-6
SMU_f	4699	2.24	1.07	1-5

Note SWL = Satisfaction with life, SHS = Subjective happiness, QoL= quality of life, FL = Flourishing, ADS = Anxious-Depressive symptoms, SMU_t = Time spent (daily) on social media, SMU_n = Number of social media accounts, SMU_f = Frequency of posting on social media.

Table 2. Twin correlations and cross-twin cross-trait correlations.

	SWL	SHS	QoL	FL	ADS	SMUt	SMUn	SMUf
SWL	0.42/ 0.26	0.20	0.23	0.20	-0.15	0.00	-0.02	-0.03
SHS	0.35	0.40/ 0.19	0.22	0.19	-0.16	-0.08	-0.06	-0.15
QoL	0.34	0.33	0.36/ 0.28	0.19	-0.18	-0.09	-0.08	-0.14
FL	0.34	0.33	0.31	0.36/ 0.19	-0.15	-0.01	-0.04	-0.05
ADS	-0.34	-0.40	-0.33	-0.32	0.49/ 0.25	0.10	0.04	0.15
SMU_t	0.05	-0.01	-0.01	0.07	0.06	0.68/ 0.36	0.17	0.35
SMU_n	0.01	0.00	0.03	0.06	-0.03	0.22	0.30/ 0.14	0.04
SMU_f	0.03	-0.04	-0.01	0.06	0.05	0.48	0.18	0.53/ 0.45

Note: MZ correlations below the diagonal and before the dash, DZ correlations above the diagonal and after the dash. SWL = satisfaction with life, SHS = subjective happiness, QoL = quality of life, FL = flourishing, ADS = anxious-depressive symptoms, SMU_t = time spent (daily) on social media, SMU_n = number of social media accounts, SMU_f = frequency of posting on social media.

Table 3. Model fitting results for the multivariate models of wellbeing and social media use

	Baseline	Comparison	ep	-2LL	df	AIC	ΔLL	Δdf	*p*
1	Saturated model	-	304	173515.86	3909	174123.86	-	-	-
2	Saturated model	**Cholesky ACE**	**140**	**173714.19**	**3926 3**	**173994.19**	**198.33**	**164**	**0.035 (n.s.)**
3	Cholesky ACE	-	140	173714.19	3926 3	173994.19			
4	Cholesky ACE	**Cholesky AE**	**104**	**173747.43**	**3929 9**	**173955.43**	**33.24**	**36**	**0.60 (n.s.)**
5	Cholesky ACE	Cholesky CE	104	173803.19	3929 9	174011.19	89.01	36	0.000002
6	Cholesky ACE	Cholesky E	68	174742.04	3933 5	174878.04	1027.85	72	0.000000
7	CP - ACE	-	73	174241.27	3933 2	174387.27	-	-	-
8	CP - ACE	**CP - AE**	**62**	**174252.46**	**3934 3**	**174376.46**	**11.19**	**11**	**0.43 (n.s.)**

Note. ep = number estimated parameters, − 2LL = minus two times the log-likelihood, df = degrees of freedom, AIC = Akaike information criterion; best-fitting model in bold letters, CP = Common pathway model, n.s. = non-significant chi-square difference test result at alpha = 0.1.

Table 4. Standardized estimates for additive genetic effects (A) and unique environmental effects (E) from the best-fitting model (Cholesky AE).

	A (%)	**E (%)**
SWL	42 (37, 47)	58 (53, 63)
SH	39 (33, 44)	61 (56, 67)
QoL	39 (33, 44)	61 (56, 67)
FL	38 (32, 43)	62 (57, 68)
ADS	47 (41, 52)	53 (48, 59)
SMU_t	72 (68, 75)	28 (25, 32)
SMU_n	32 (25, 38)	68 (62, 75)
SMU_f	54 (50, 59)	46 (41, 50)

Note. SWL = satisfaction with life, SH = subjective happiness, QoL = quality of life, FL = flourishing, ADS = anxious-depressive symptoms, SMU_t = time spent (daily) on social media, SMU_n = number of social media accounts, SMU_f = frequency of posting on social media.

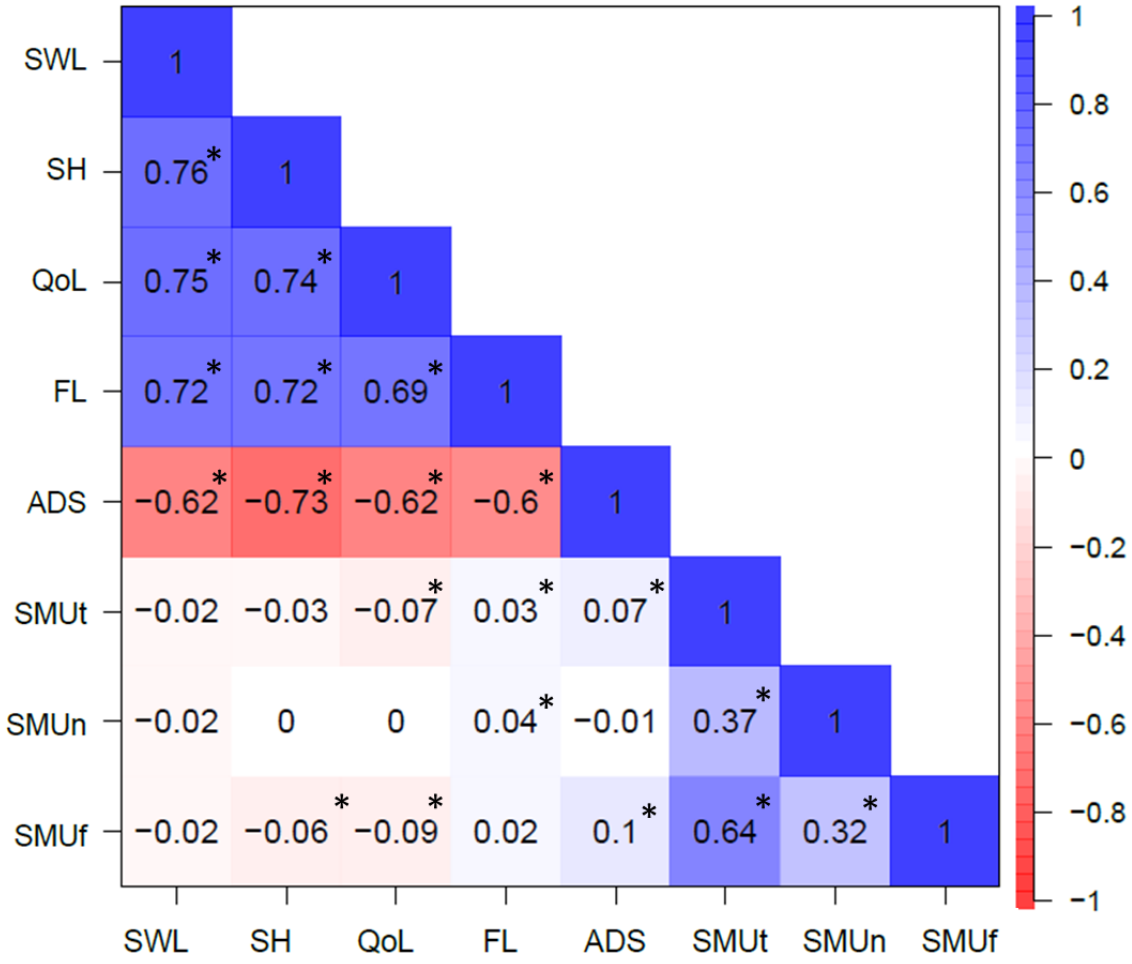
Table 5. Standardized covariances in percentages for additive genetic effects (A) (below diagonal) and unique environmental effects (E) (above diagonal) from the best fitting model (Cholesky AE).

	SWL	SH	QoL	FL	ADS	SMU_t	SMU_n	SMU_f
SWL	-	55 (49,61)	54 (48,61)	53 (47,59)	51 (44,58)	156 (NA,NA)	164 (NA,NA)	83 (0,241.50)
SH	45 (39,51)	-	54 (48,61)	52 (46,59)	51 (44,57)	-2 (NA,NA)	61 (NA,NA)	-11 (-91,NA)
QoL	46 (39,52)	46 (39,52)	-	52 (46,59)	48 (41,55)	11 (NA,NA)	-315 (NA,NA)	13 (-31, NA)
FL	47 (41,54)	48 (41,54)	48 (41,54)	-	47 (40,55)	-18 (NA,NA)	1 (NA,NA)	26 (NA,NA)
ADS	49 (42,56)	50 (43,56)	52 (45,59)	53 (45,60)	-	20 (NA,NA)	-372 (NA,NA)	-15 (NA,NA)
SMU_t	-56 (NA,NA)	102 (NA,NA)	89 (NA,NA)	118 (NA,NA)	80 (NA,NA)	-	37 (28,48)	20 (16,25)
SMU_n	-64 (NA,NA)	39 (NA,NA)	415 (NA,NA)	99 (NA,NA)	472 (NA,NA)	63 (53,73)	-	45 (34,58)
SMU_f	17 (-2449, NA)	111 (NA,191)	87 (NA,1.32)	74 (NA,NA)	115 (NA,NA)	80 (75,84)	55 (43,66)	-

Note. SWL = satisfaction with life, SH = subjective happiness, QoL = quality of life, FL = flourishing, ADS = anxious-depressive symptoms, SMU_t = time spent (daily) on social media, SMU_n = number of social media accounts, SMU_f = frequency of posting on social media. NA = CIs were unable to be estimated given small phenotypic correlations. In some cases, unstandardized values for additive genetic and unique environmental covariances had the same signs (e.g., either both values were positive or negative). When the same signs for both values are not observed in the data, standardized genetic and environmental covariances results with percentages higher than 100 which cannot be interpreted meaningfully.

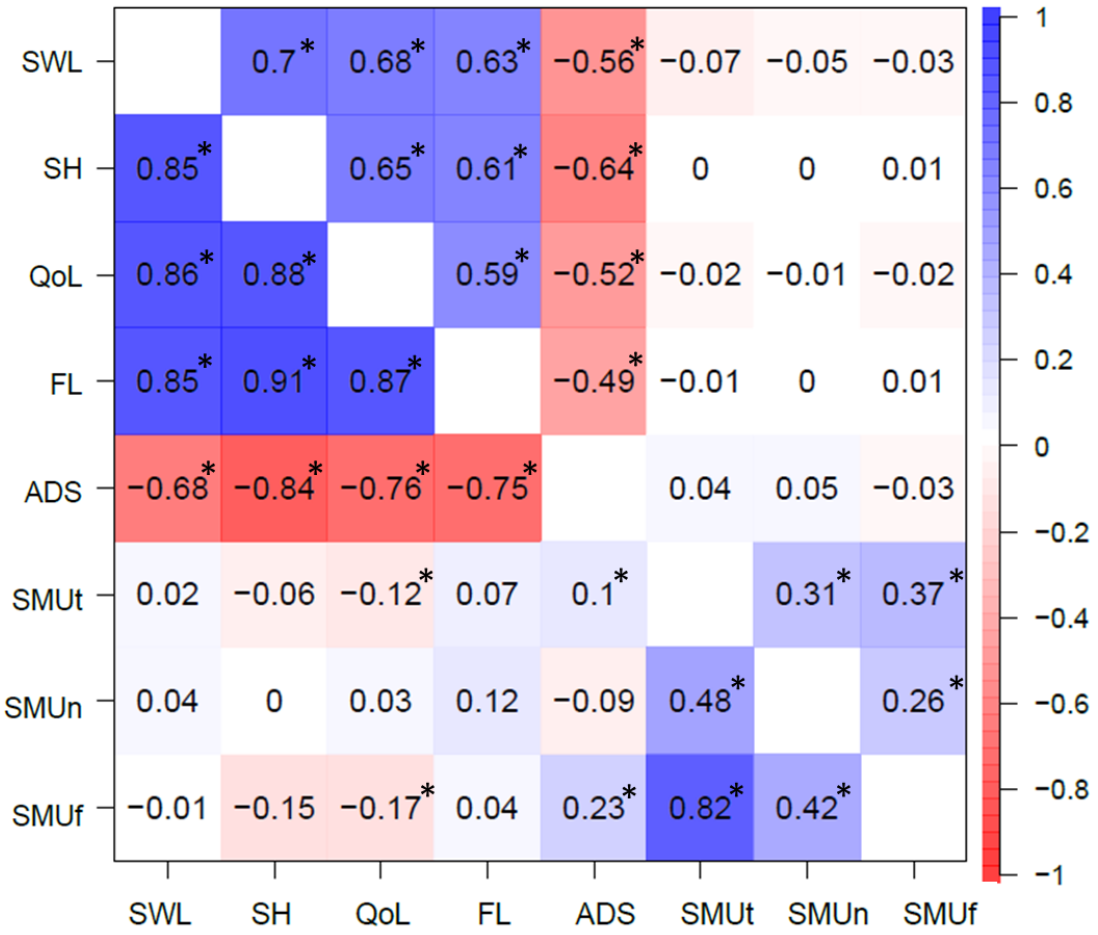
Figures

Figure 1. Phenotypic correlations among study variables.



Note. *significant correlation at alpha = 0.05. SWL = satisfaction with life, SH = subjective happiness, QoL = quality of life, FL = flourishing, ADS = anxious-depressive symptoms, SMU_t = time spent (daily) on social media, SMU_n = number of social media accounts, SMU_f = frequency of posting on social media.

Figure 2. Genetic and environmental correlations (shown at the lower and upper diagonal of the table, respectively) among study variables.



Note. *significant correlation at alpha = 0.05. SWL = satisfaction with life, SH = subjective happiness, QoL = quality of life, FL = flourishing, ADS = anxious-depressive symptoms, SMU_t = time spent (daily) on social media, SMU_n = number of social media accounts, SMU_f = frequency of posting on social media.

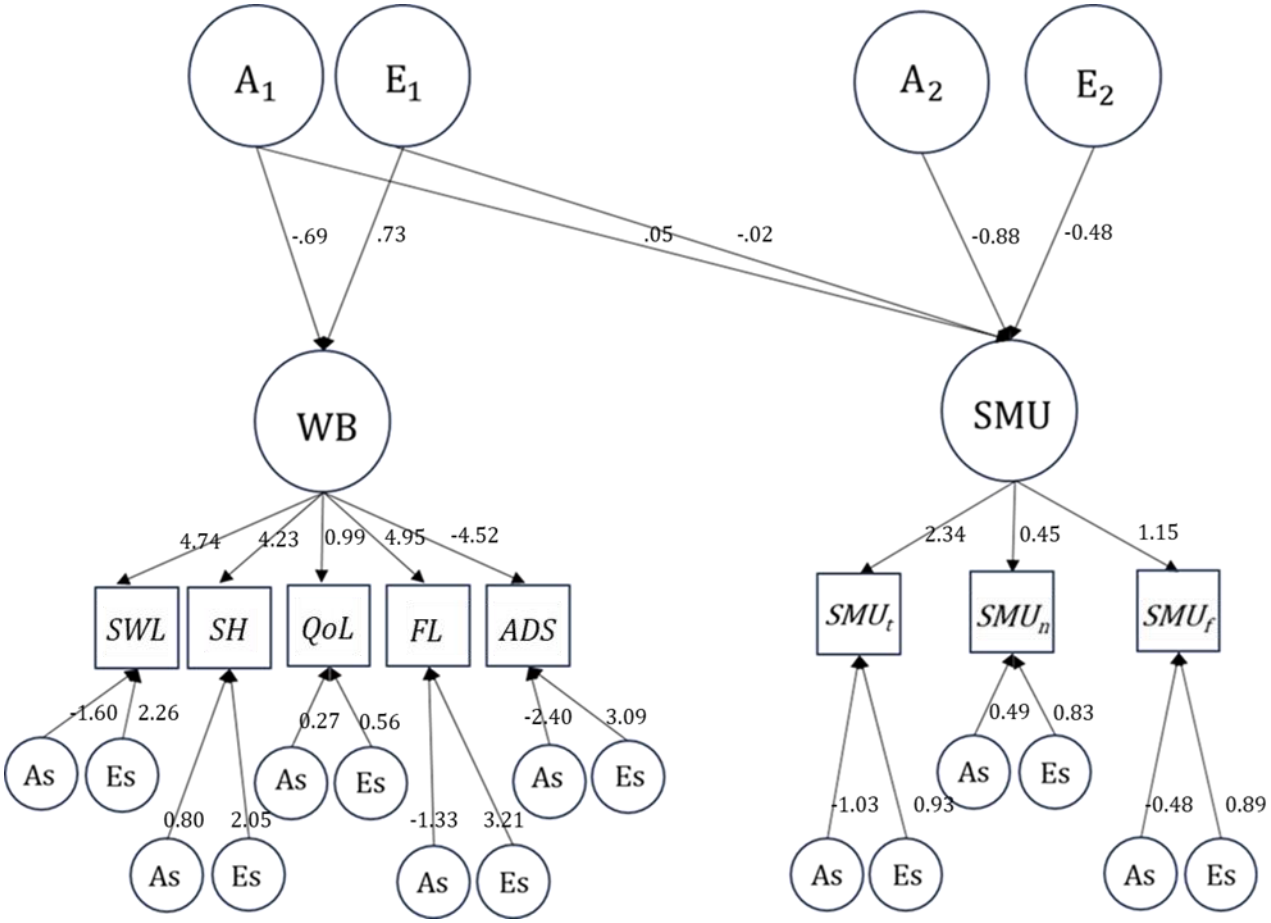
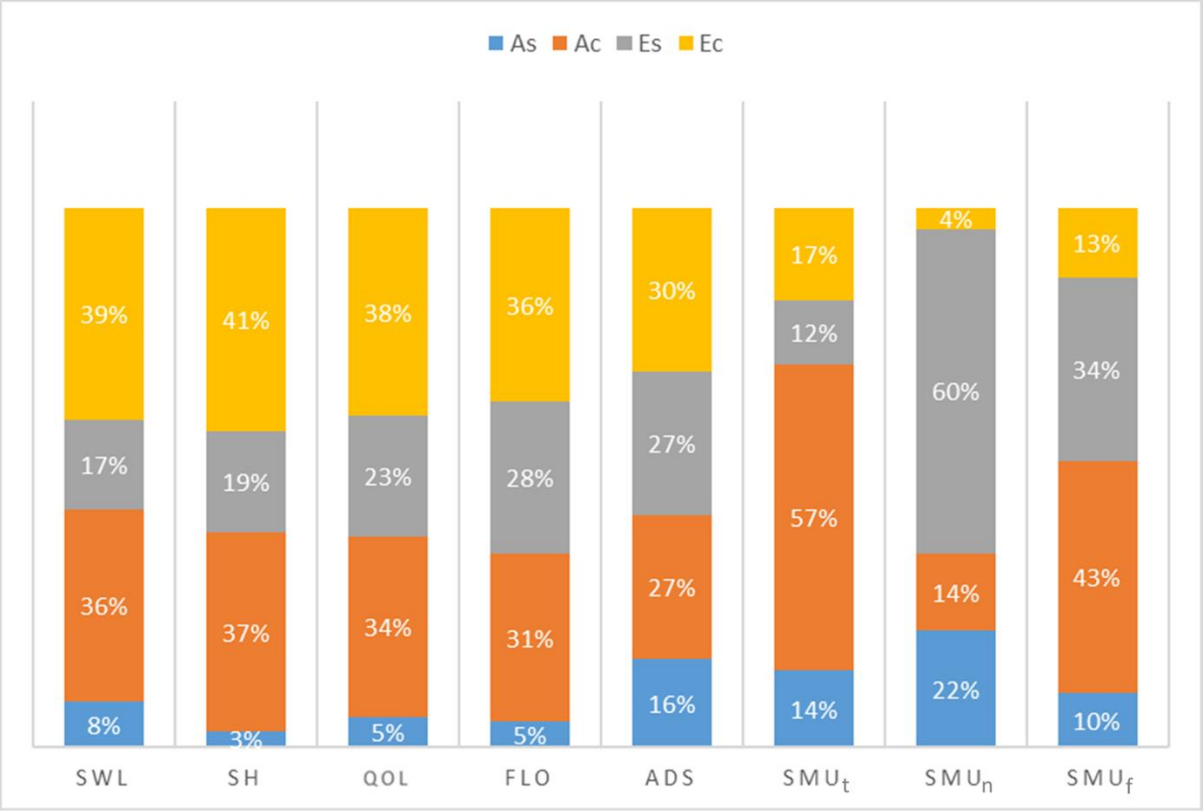


Figure 3. Unstandardized two-factor common pathway model for a single twin (effects from covariates are not included for simplifying purposes)

Note. WB = wellbeing factor SMU, = social media use factor, A = additive genetic variance, E = environmental variance. As and Es refer to the phenotype-specific residual variances for A and E variances. SWL = satisfaction with life, SH = subjective happiness, QoL = quality of life, FL = flourishing, ADS = anxious-depressive symptoms, SMU_t = time spent (daily) on social media, SMU_n = number of social media accounts, SMU_f = frequency of posting on social media.

Figure 4. Percentage of variance explained through common additive genetic and environmental factors or phenotype-specific effects based on the common pathway model



Note. Ac = percentage of total additive genetic variance explained by common genetic effects, As = percentage of total variance explained by additive genetic effects specific for a given phenotype, Ec = percentage of the total variance explained by common unique environmental effects, Es = percentage of total variance explained by unique environmental effects specific for a given phenotype. SWL = satisfaction with life, SH = subjective happiness, QoL = quality of life, FL = flourishing, ADS = anxious-depressive symptoms, SMU_t = time spent (daily) on social media, SMU_n = number of social media accounts owned, SMU_f = posting on social media.