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Social-relational exposures and well-being: Using multivariate twin data to rule-out heritable and shared environmental confounds

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ABSTRACT

The aims of the present study were as follows: (1) Using a large sample of adults, estimate overlap between social-relational exposures measured at midlife and well-being measured at midlife and approximately 9-years later. (2) Using a subsample of twins, test for heritable variation in social-relational exposures, and (3) controlling for heritable and shared environmental variation, estimate overlap between social-relational exposures and well-being, both concurrently and approximately 9-years later. Results indicated small-to-moderate overlap between exposures and well-being (mean r = 0.29, range = 0.05–0.54). There was also evidence for heritable variation in exposures, and after accounting for these genetic factors, the degree of overlap between social-relational exposures and well-being decreased (mean r = 0.10, range = −0.07 to 0.33).

1. Introduction

Much attention has been dedicated to identifying features of the social environment that promote individual well-being, and a number of variables have been identified as risk and protective factors (Huppert, 2009). Cross-sectional studies indicate that well-being is correlated with parental affection (Flouri, 2004; Polcari, Rabi, Bolger, & Teicher, 2014), social support and strain (Chen & Feeley, 2014; Nguyen, Chatters, Taylor, & Mouzon, 2016), and work-family spillover (i.e. the transfer of behaviors, emotions, and values from one's occupational life to family life; Amstad, Meier, Fasel, Elfering, & Semmer, 2011; Grzywacz & Marks, 2000). Often included in studies as mediating variables, aspects of one's social-relational environment are often assumed, at least tacitly, to exert a causal influence on well-being and related psychosocial outcomes (Rijken & Groenewegen, 2008; Segrin & Rynes, 2009; Segrin & Taylor, 2007; Suresh & Sandhu, 2012). In turn, some investigators have made public policy recommendations based on correlational evidence, for example, suggesting that "Policy should pay specific attention to income support of the chronically ill and disabled in order to improve their opportunities for social participation" (Rijken & Groenewegen, 2008).

However, drawing such strong conclusions from correlational evidence may be dubious, in part, because individuals are not randomly assigned to social-relational environments. Rather, individuals select into and evoke responses from environments based on their heritable characteristics. This well-documented phenomenon, called genotype-environment correlation (Plomin, DeFries, & Loehlin, 1977) or niche-picking (Scarr & McCartney, 1983), has been pivotal in revising epidemiological and developmental models of causation, which now widely acknowledge reciprocal relations between persons and environments (LaFreniere & MacDonald, 2013; Leve & Cicchetti, 2016). Put differently, genotype-environment correlation refers to the non-random assortment of individuals into environments based on their genotypes. Consequently, the presence of heritable variation (h^2) in a measure of the environment *is* evidence for genotype-environment correlation, as this indicates that variation in the environment is partially accounted for by genetic differences between people.

Genetically informative research in humans has focused primarily on environments that are relevant to understanding the etiology of health-risk behaviors and psychiatric disorders, including substance-use disorders (Jaffee & Price, 2007; Kendler & Baker, 2007). Fewer studies have tested for heritable variation in social-relational exposures that are relevant to promoting positive psychological outcomes, including subjective or hedonic well-being, with a few noteworthy exceptions. These studies have shown that not only is subjective/hedonic well-being heritable (Bartels, 2015;

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Pluess, 2015), but also a number of social-relational constructs that are correlated with well-being, including financial status (Johnson & Krueger, 2006), social support (Wang, Davis, Wootton, Mottershaw, & Haworth, 2017), and positive life events (Wootton, Davis, Mottershaw, Wang, & Haworth, 2017).

For example, compared to those who lack social support, individuals with friends who are kind and supportive tend to report elevated levels of well-being (Chen & Feeley, 2014; Chu, Saucier, & Hafner, 2010). This commonly observed correlation *might* reflect a causal effect of friendship on well-being, whereby the social support provided by one's friends *causes* well-being to increase. Then again, individuals who are generally happy, satisfied, and easy-going may seek out or evoke more social support from their companions, compared to their depressed, dissatisfied, and worrisome counterparts. In this way, social support, whether it comes from a friend, spouse, or family member, may be influenced by the heritable characteristics of the recipient of support.

Unfortunately, the gold standard for assessing cause-effect relations, a randomized experimental design, is neither practical nor ethically permissible when studying the relations between social-relational exposures and many psychological outcomes. Researchers cannot randomly assign participants, for example, to spousal strain, poverty, or widowhood conditions. It may also be ethically dubious to experimentally manipulate life satisfaction. Given these methodological realities, quantitative genetic methods provide a means for testing hypotheses about the effects of social-relational exposures on psychological outcomes by ruling out non-causal explanations (Duncan et al., 2014; McGue, Osler, & Christensen, 2010; Schaefer et al., 2018), including overlapping genetic factors and potential sociodemographic confounds that contribute to the similarity of siblings raised in the same home.

Specifically, multivariate twin data can be used to estimate additive genetic, shared environmental, and non-shared environmental contributions to the associations between an exposure and a psychological outcome. The rationale behind the approach is simple. Identical twins are matched on genetic relatedness and early rearing conditions, including socioeconomic status, culture, neighborhood conditions, educational cohort, etc. Therefore, when identical twins differ with respect to an exposure and, furthermore, the difference is associated with an outcome of interest, that association cannot be accounted for by genetic factors or shared rearing conditions because the segregating genes and early rearing conditions of identical twins are, indeed, identical. In this sense, the classical twin design is a *quasi*-experimental design because it enables one to estimate the association between two constructs after ruling out heritable and shared-environmental factors that otherwise might provide a non-causal explanation for the association (Schaefer et al., 2018), such as overlapping genetic factors and shared rearing conditions that contribute to sibling similarity.

Multivariate twin data has been used to examine the relationship between various aspects of the social environment and well-being. Pertinent to the present study, in a large cross-sectional sample of 18-year-old twins, quality and quantity of social support were positively correlated with various aspects of well-being, including, but not limited to, positive and negative affect, life satisfaction, happiness, and gratitude (Wang et al., 2017). Importantly, both genetic factors and environmental factors not shared between twins contributed to these correlations, although genetic factors predominated (roughly 75% of the correlation between social support and well-being was explained by common genetic factors; Wang et al., 2017). Similarly, in a large cross-sectional sample of 16-year-old-twins, correlations between life events and well-being were largely accounted for by shared genetic factors, whereby heritable variation in life events overlapped with heritable variation in well-being (Wootton et al., 2017). In adulthood, a causal effect of widowhood on well-being cannot be ruled out, as bereaved twins who lost a spouse experience more depression and less life satisfaction, compared to their married co-twin (Liechtenstein, Gatz, Pedersen, Berg, & McClearn, 1996). We aim to contribute to this body of research by estimating concurrent and longitudinal correlations between twelve social-relational exposures and well-being, before and after accounting for genetic and environmental factors that contribute to the similarity of twins who were raised in the same home.

The aims of the present study were as follows: (1) Using a large sample of adults, estimate concurrent and longitudinal correlations between a dozen social-relational exposures measured at midlife and well-being measured at midlife and approximately 9-years later in adulthood. (2) Using a subsample of twins, test for the presence of heritable variation in social-relational exposures. (3) Using multivariate twin data, estimate concurrent and longitudinal correlations between social-relational exposures and well-being, controlling for genetic and shared environmental factors.

2. Method

2.1. Sample

The sample included adults who participated in the National Survey of Midlife Development in the United States (MIDUS; Brim, Ryff, & Kessler, 2004). The first wave of data collection took place between 1995 and 1996 (N = 7109; Twin subsample = 1914). At the first wave, the average age of participants was approximately 46 years (range = 20–75 years). ~52% of the sample was female (~48% male), and ~92% self-reported white/European race/ethnicity, ~6% black/African American, and ~2% another race/ethnicity. At the second wave of data collection, between 2004 and 2006 (N = 4963; Twin N = 1484), the average age of participants was 55 years (range = 28–84 years). ~53% of the sample was female (~47% male), and ~90% self-reported white/European race/ethnicity, ~5% black/African American, and ~5% another race/ethnicity. Descriptive statistics for demographic variables can be found in the MIDUS codebook. Participants were paid $20 for each wave of data collection. Additional information regarding participant recruitment and data collection can be found elsewhere (Brim et al., 2004).

2.2. Analytic procedures & measurement

Data was obtained from the Inter-University Consortium for Political and Social Research (ICPSR; https://www.icpsr.umich.edu/icpsrweb) and prepared for analyses using R version 3.4.2, in combination with the 'MplusAutomation' package (Hallquist & Wiley, 2011). Inferential analyses consisted in two steps and were conducted using Mplus version 8 (Muthén & Muthén, 2012). First, a series of bivariate confirmatory factor analysis (CFA) models were used to operationalize social-relational exposures and well-being, while simultaneously estimating concurrent and longitudinal correlations between social-relational exposures and well-being. Second, a series of bivariate twin models were used to test for heritable, shared environmental, and non-shared environmental variation in social-relational exposures, and controlling for heritable and shared-environmental variation, estimate non-shared environmental contributions to concurrent and longitudinal correlations.

Siblings and twins were nested within the same family, and a subset of families had multiple sets of twins. Therefore, using the complex survey option in Mplus, a family identification number was included as a cluster variable to account for the non-independence of observations in both phenotypic CFA and bivariate twin models. Missing values were handled using

full-information maximum likelihood. The precision of effect sizes was evaluated using non-parametric bootstrapped standard errors and confidence intervals. Although the present study is not exploratory, given the sheer number of associations that were estimated (12 social-relational variables × 2 types of associations [concurrent and longitudinal] × 2 types of models [phenotypic and twin] = 48 correlations), a Bonferroni-corrected threshold was adopted when evaluating the statistical significance of correlations ($\alpha = 0.05/48 = 0.001$).

For all models, the values of observed indicators were coded so higher factor scores indicate higher levels of their respective constructs. For each construct, a single common factor was specified to account for covariation among observed indicators, scaled using unit variance identification (i.e. by fixing the variance of the common factor to one). The intercepts of scale scores and thresholds of item scores were freely estimated. Individual item scores were specified as ordinal indicators of a latent social exposure factor, and scales scores for positive affect (PA), negative affect (NA), and life satisfaction (LS) were specified as continuous indicators of a latent well-being factor. Therefore, models were estimated using weighted least squares with mean and variance adjustments (i.e. WLSMV), which is the default setting in Mplus when one or more observed indicator is binary or ordinal.

To control for potential confounds, social-relational exposure and well-being factors were regressed on a set of exogenous covariates, including mean-centered age, mean-centered age-squared, biological sex (male = −0.5, female = 0.5), and self-reported Black/African American race/ethinicity (1 = Yes, 0 = No). Suggested by a reviewer, sensitivity analyses were performed whereby the same models were fit to the data with additional covariates, including the highest level of education completed by mothers and fathers, and in twin models, the number of years that twins were raised in the same home ($M = 18.79$, $SD = 3.17$). Finally, the partial correlation between latent factors was estimated, which quantifies the magnitude of interdependence between the social-relational exposure and well-being factors after accounting for variance associated with study covariates. Note, an advantage to operationalizing focal study constructs as latent variables is that correlations are estimated free of unsystematic variation that is specific to individual indicators, including measurement error. Path diagrams of bivariate CFA and twin models can be found in supplemental materials.

2.2.1. Parental affection

Participants were asked seven questions about the relationship they had with their parents when they were children: (1) "How would you rate your relationship with your mother/father during the years you were growing up?" (2) "How much did [she/he] understand your problems and worries?" (3) "How much could you confide in [her/him] about things that were bothering you?" (4) "How much did [she/he] give you love and affection?" (5) "How much did [she/he] give you time and attention when you needed it?" (6) "How much effort did [she/he] put into watching over you and making sure you had a good upbringing?" (7) "How much did [she/he] teach you about life?" Maternal and paternal affection were measured separately. The first question was rated on a 5-point scale (5 – Excellent, 4 – Very good, 3 – Good, 2 – Fair, 1 – Poor). The remaining questions were rated on a 4-point scale (4 – A lot, 3 – Some, 2 – A little, 1 – Not at all). Factor loadings were high for items measuring maternal affection (range of $\lambda = 0.72$–0.92, $ps < 0.001$) and residual errors were small-to-moderate (range of $\sigma^2 = 0.16$–0.48). Similarly, factor loadings were high for items measuring paternal affection (range of $\lambda = 0.76$–0.92, $ps < 0.001$) and residual errors were small-to-moderate (range of $\sigma^2 = 0.16$–0.43).

2.2.2. Parental discipline

Participants were asked four questions about the nature of discipline received from each of their parents during childhood: (1) "How strict was [she/he] with [her/his] rules for you?" (2) "How consistent was [she/he] about the rules?" (3) "How harsh was [she/he] when [she/he] punished you?" and (4) "How much did [she/he] stop you from doing things that other kids your age were allowed to do?" Maternal and paternal discipline were measured separately, and questions were rated on a 4-point scale (4 – A lot, 3 – Some, 2 – A little, 1 – Not at all). Factor loadings were moderate-to-high for items measuring maternal discipline (range of $\lambda = 0.58$–0.90, $ps < 0.001$) and residual errors were small-to-moderate (range of $\sigma^2 = 0.18$–0.66). Similarly, factor loadings were high for items measuring paternal discipline (range of $\lambda = 0.70$–0.90, $ps < 0.001$) and residual errors were small-to-moderate (range of $\sigma^2 = 0.19$–0.51).

2.2.3. Social support

Participants were asked four questions about how much social support they received from their family members, friends, and spouse/partner: (1) "How much do [the members of your family/your friends/your spouse or partner] really care about you?" (2) "How much do they understand the way you feel about things?" (3) How much can you rely on them for help if you have a serious problem?" and (4) "How much can you open up to them if you need to talk about your worries?" Family, friend, and spouse/partner support were all measured separately. Two additional questions were asked about spouse/partner support: (5) "How much does he or she appreciate you?" and (6) "How much can you relax and be yourself around him or her?" All questions were rated on a 4-point scale (4 – A lot, 3 – Some, 2 – A little, 1 – Not at all). Loadings onto common factors were high for items measuring family support (range of $\lambda = 0.83$–0.87, $ps < 0.001$), friend support (range of $\lambda = 0.87$–0.89, $ps < 0.001$) and spouse/partner support were high (range of $\lambda = 0.88$–0.92, $ps < 0.001$). After accounting for common variance, residual measurement errors were small-to-moderate for items measuring family support (range of $\sigma^2 = 0.24$–0.30), friend support (range of $\sigma^2 = 0.21$–0.25), and spouse/partner support (range of $\sigma^2 = 0.15$–0.22).

2.2.4. Social strain

Participants were asked four questions about how much strain they experience with their family, friends, and spouse/partner: (1) "How often do [the members of your family/friends/spouse or partner] make too many demands on you?" (2) "How often do they criticize you?" (3) "How often do they let you down when you are counting on them?" (4) "How often do they get on your nerves?" Family, friend, and spouse/partner strain were all measured separately. Two additional questions were asked about spouse/partner strain: (5) "How much does he or she argue with you?" and (6) "How often does he or she make you feel tense?" All questions were rated on a 4-point scale (4 – A lot, 3 – Some, 2 – A little, 1 – Not at all). The factor loadings for items measuring family strain (range of $\lambda = 0.65$–0.79, $ps < 0.001$), friend strain (range of $\lambda = 0.72$–0.80, $ps < 0.001$) and spouse/partner strain were moderate-to-high (range of $\lambda = 0.73$–0.86, $ps < 0.001$). After accounting for common variance, residual measurement errors were moderate for family strain (range of $\sigma^2 = 0.38$–0.58), friend strain (range of $\sigma^2 = 0.36$–0.48), and spouse/partner strain (range of $\sigma^2 = 0.29$–0.47).

2.2.5. Work-family spillover

Participants were asked to rate how often work has a negative influence on their life at home: (1) "Your job reduces the effort you can give to activities at home" (2) "Stress at work makes you irritable at home" (3) "Your job makes you feel too tired to do the things that need attention at home" and (4) "Job worries or

problems distract you when you are at home." Participants were also asked to rate how often their job has a positive impact on their life at home: (1) "The things you do at work help you deal with personal and practical issues at home" (2) "The things you do at work make you a more interesting person at home" (3) "Having a good day on your job makes you a better companion when you get home" and (4) "The skills you use on your job are useful for things you have to do at home". Positive and negative work-family spillover was measured separately, and all statements were rated on a 5-point scale (5 – All of the time, 4 – Most of the time, 3 – Sometimes, 2 – Rarely, 1 – Never). Factor loadings were moderate-to-high for both positive work-family spillover (range of $\lambda = 0.47–0.81$, $ps < 0.001$) and negative work-family spillover (range of $\lambda = 0.69–0.83$, $ps < 0.001$). After accounting for common variance, residual measurement errors were moderate-to-large for items measuring positive work-family spillover (range of $\sigma^2 = 0.35–0.78$) and negative work-family spillover (range of $\sigma^2 = 0.32–0.52$).

2.2.6. Well-being

Participants provided responses to sets of questions that were used to compute three scales: positive affect, negative affect, and life satisfaction. (1) The positive affect scale asked participants how often they feel a series of positive emotions (i.e. "cheerful", "in good spirits", "extremely happy", "calm and peaceful", "satisfied" and "full of life", $\alpha = 0.91$). (2) The negative affect scale asked participants how often they feel negative emotions (i.e. "so sad nothing could cheer you up", "nervous", "restless or fidgety", "hopeless", "that everything was an effort" and "worthless", $\alpha = 0.87$). Items measuring positive and negative affect were rated on a 5-point scale (1 = All of the time; 3 = Some of the time; 5 = None of the time). (3) The life satisfaction scale asked participants to rate their quality of life overall on a 11-point scale (0 = the worst possible; 10 = the best possible). It also includes domain satisfaction questions that ask participants to rate their satisfaction with work, health, and relationships with their partner and children ($\alpha = 0.67$). At the first and second measurement occasion, factor loadings were moderate-to-high for positive affect ($\lambda s = 0.83$ and 0.82, $ps < 0.001$), negative affect ($\lambda s = -0.76$ and -0.76, $ps < 0.001$), and life satisfaction ($\lambda s = 0.68$ and 0.70, $ps < 0.001$). After accounting for common variance among indicators of well-being, residual errors were moderate for positive affect ($\sigma^2 = 0.31$ & 0.33), negative affect ($\sigma^2 = 0.57$ & 0.58), and life satisfaction ($\sigma^2 = 0.46$ & 0.50).

3. Results

Model fit statistics for CFA models are reported in supplemental materials (mean RMSEA = 0.06, range of RMSEA = 0.03–0.08; mean CFI = 0.97, range of CFI = 0.92–0.99). Note that all social-relational exposures were significantly ($ps < 0.001$) correlated with well-being, both cross-sectionally and longitudinally, with three exceptions. The correlations between paternal discipline and well-being, measured concurrently ($r = 0.06$, CI.95% = 0.02–0.09, $p = .003$) and approximately 9-years later ($r = 0.05$, CI.95% = 0.01–0.10, $p = .014$), did not meet a Bonferroni-corrected threshold for statistical significance. The longitudinal correlation between maternal discipline and well-being also failed to meet a Bonferroni-corrected threshold for statistical significance ($r = 0.05$, CI.95% = 0.01–0.10, $p = .012$).

Correlations between social-relational exposures and well-being were generally small-to-moderate in magnitude (mean $r = 0.29$, range = .05 –0.54).[1] On average, concurrent correlations were larger (mean $r = 0.33$, range = 0.06–0.54) than longitudinal correlations (mean $r = 0.24$, range = 0.05–0.37). Of the 12 social variables included in the study, the strongest correlations were between well-being and negative work-family spillover, spouse/-partner support, and spouse/partner strain. Slightly weaker correlations were observed between well-being and positive work-family spillover, friend support, friend strain, family support, family strain, maternal affection, and paternal affection. Finally, the correlations between well-being and parental discipline, both maternal and paternal, approached zero. Although slightly attenuated, compared to concurrent correlations, the general pattern of effect sizes remained unchanged when well-being was measured approximately 9-years later.

Next, a series of bivariate twin models were fit to a subsample of same-sex monozygotic and dizygotic twins ($n = 643$ twin-pairs; 334 monozygotic; 309 dizygotic). These models were parameterized as bivariate Cholesky models (Loehlin, 1996), whereby a latent social-relational exposure factor was the primary variable and a latent well-being factor was the secondary variable. A path diagram of this model can be found in supplemental materials (bottom panel of Fig. S1). In these models the variances and covariance between an exposure and well-being are decomposed into two sets of latent genetic and environmental factors. The first set of latent factors (A1, C1, and E1) contain variance that is common to the exposure and well-being, as well as variance that is unique to the exposure. The second set of factors (A2 & E2) contain variance that is unique to well-being. In sensitivity anlyses, the number of years that twins were raised in the same home was introduced as an additional exogenous covariate of social-relational exposure and well-being factors, as well as the highest level of education completed by mothers and fathers.

The first additive genetic (a_1), shared environmental (c_1), and non-shared environmental (e_1) pathways capture latent genetic and environmental contributions to variation in the exposure. Because exposures are a measure of the social environment, additive genetic variation in an exposure provides evidence for gene-environment correlation. The additive genetic cross-path (a_{12}) and non-shared environmental cross-path (e_{12}) capture latent genetic and environmental contributions to covariation between the exposure and well-being. Specifically, a statistically significant (a_{12}) additive genetic cross-path indicates that genetic variation in the social-relational exposure is shared or overlaps with genetic variation in well-being. A statistically significant (e_{12}) non-shared environmental cross-path indicates that, within twin-pairs who are matched on genetic relatedness and early rearing conditions, the twin who reports higher levels of the exposure, on average, reports higher levels of well-being as well. Finally, the second set of additive genetic (a_2) and non-shared environmental (e_2) pathways capture residual variance in well-being that is unique of the exposure. Using these parameter estimates, path-tracing rules were followed to recast the total variance in latent exposures and well-being factors into additive genetic, shared-environmental, and non-shared environmental components:

$$h^2_{EXP} = a_1^2/[a_1^2 + c_1^2 + e_1^2]$$

$$c^2_{EXP} = c_1^2/[a_1^2 + c_1^2 + e_1^2]$$

$$e^2_{EXP} = e_1^2/[a_1^2 + c_1^2 + e_1^2]$$

$$h^2_{WB} = [a_2^2 + a_{12}^2]/[a_{12}^2 + a_2^2 + e_{12}^2 + e_2^2]$$

$$e^2_{WB} = [e_2^2 + e_{12}^2]/[a_{12}^2 + a_2^2 + e_{12}^2 + e_2^2]$$

Model fit statistics for bivariate twin models can be found in supplemental materials (mean RMSEA = 0.03, min. RMSEA = 0.01, max. RMSEA = 0.05; mean CFI = 0.98, min. CFI = 0.92, max.

[1] Results of sensitivity analysis: mean $r = 0.29$, range = 0.06–0.54.

CFI = 0.99). Parameter estimates are reported in Table 1. Notably, there was evidence for heritable variation in nearly all social-relational exposures (mean $h^2 = 0.32$, range = 0.03–0.57). There was also mixed evidence for shared environmental variation (mean $c^2 = 0.17$, range = 0.00–0.55), as well as evidence for non-shared environmental variation (mean $e^2 = 0.51$, range = 0.20–0.83).[2] Results are plotted in Fig. 1.

The same bivariate twin models were used to estimate concurrent and longitudinal correlations between social-relational exposures and well-being after accounting for heritable and shared environmental variation in both constructs- i.e. including only non-shared environmental sources of covariation. If an exposure is related to well-being for environmental reasons, that is, beyond the influence of heritable and sociodemographic factors that make siblings similar to each other, then the estimated correlation between the exposure and well-being should be significantly different than zero after accounting for additive genetic and shared environmental variation in both constructs. Put differently, the non-shared environmental contribution to the correlation should be greater than zero, which is equal to the square root of the non-shared environmentality of the exposure ($\sqrt{e^2_{EXP}}$), multiplied by the non-shared environmental correlation between the exposure and well-being ($rE_{12} = e_{12}/\sqrt{[e^2_{12} + e^2_2]}$), multiplied by the square root of the non-shared environmentality of the well-being factor ($\sqrt{e^2_{WB}}$). Alternatively, if the relationship between the exposure and well-being is not environmental in origin but is rather the result of shared genetic factors or socio-demographic confounding, then the non-shared environmental contribution to the correlation between the exposure and well-being factors should approach zero.

Results are depicted in Fig. 2, which compares partial correlations between exposures and well-being before ("Phenotypic Correlation Between Latent Factors") and after accounting for heritable and shared environmental variation ("Non-Shared Environmental Contribution"), with negative correlations reflected (i.e. multiplied by −1) to ease comparison of associations with different exposures. "Phenotypic Correlation Between Latent Factors" denotes partial correlations between the exposures and well-being estimated in the full sample (N > 6000), controlling for study covariates. "Non-Shared Environmental Contribution" denotes partial correlations between the exposures and well-being estimated in a subsample of same-sex monozygotic and dizygotic twins, including only non-shared environmental sources of covariation. Results indicate that, after accounting for heritable and shared environmental factors, the degree of overlap between social-relational exposures and well-being decreased considerably (mean $r = 0.09$, range = −0.07–0.32).[3] Nevertheless, for a number of social-relational exposures, specifically negative work-family spillover, spousal support and strain, and friend support and strain, non-shared environmental contributions to concurrent correlations were significantly different than zero ($ps < 0.001$), providing evidence that these associations are not merely the result of shared genetic factors or sociodemographic confounding. For positive work-family spillover, family support, and family strain, the non-shared environmental contributions to concurrent correlations were greater than zero and statistically significant by conventional standards ($ps < 0.05$) but were not significant after accounting for multiple comparisons ($ps > 0.001$). With respect to longitudinal phenotypic associations with well-being measured almost a decade after the exposure, after accounting for gene-environmental correlations, longitudinal associations were small and not significantly different than zero. Importantly, the size and precision of estimated effects remained largely unchanged in sensitivity analyses that included additional covariates.

4. Discussion

In the present study, we estimated the degree of concurrent and longitudinal overlap between a dozen social-relational exposures measured at midlife and well-being measured at midlife and almost a decade later in adulthood. Using a subsample of twins, we also tested for heritable variation in social-relational exposures. Finally, we estimated the degree of concurrent and longitudinal overlap between social-relational exposures and well-being, controlling for genetic and environmental factors that contribute to the similarity of twins who were raised in the same home. Results revealed small-to-moderate overlap between social-relational exposures and well-being. There was also evidence for gene-environment correlations, and after accounting for these genetic factors, the overlap between social-relational exposures and well-being decreased (mean change in $r = -0.19$, range = −0.03 to −0.35).[4] This suggests that the correlations between social-relational exposures and well-being that are commonly observed in cross-sectional and longitudinal studies are prone to overestimate the strength of environmental effects because they are partly the result of overlapping genetic factors that contribute to variation in both social-relational exposures and well-being.

Given the methodological barriers to studying the relationship between social-relational exposures and human individual differences, the present study provides evidence that social support and work-family spillover are related to hedonic well-being through genetic and environmental pathways. The near ubiquitous presence of heritable variation in social-relational exposures, in combination with non-shared environmental effects on well-being, is consistent with conceptualizing the relationship between exposures and well-being as transactional in nature. Results indicate that individuals seek out and evoke responses from their social-relational environment based on their heritable characteristics, including the tendency to experience positive emotions and general satisfaction with life. The social-relational contexts in which individuals are embedded are simultaneously associated with those characteristics because of environmental factors, which, in turn, further reinforces their interdependence.

The general pattern of correlations was similar when well-being was measured concurrently at midlife and longitudinally almost ten years later. However, longitudinal correlations between social-relational exposures and well-being were not significantly different than zero after accounting for genetic and shared environmental contributions to covariation. Moreover, after accounting for genetic and shared environmental factors, retrospective childhood assessments of parenting were not associated with well-being. This suggests that the environmental pathways between social-relational exposures and well-being are likely more proximate than distal. Put differently, social-relational exposures appear to be environmentally salient for well-being, and vice-versa, when individuals have recently been exposed, as opposed to nearly a decade later. This finding coincides with common sense intuition. What is happening now with your work, family, or friends likely matters more for your current levels of well-being, compared to what happened with work, your family, or friends almost ten years ago.

The present study did *not* find evidence that parental affection in childhood was related to well-being in middle adulthood after accounting for heritable and shared environmental factors. Mater-

[2] Results of sensitivity analysis: mean $h^2 = 0.32$, range = 0.01–0.61; mean $c^2 = 0.16$, range = 0.00–0.38; mean $e^2 = 0.52$, range = 0.20–0.89.

[3] Results of sensitivity analysis: mean $r = 0.07$, range = −0.11 to 0.27.

[4] Results of sensitivity analysis: mean change in r = −0.21, min. = −0.04, max. = −0.35.

Table 1
Parameter Estimates from Twin Models of the Association Between Social-Relational Exposures at Midlife and Well-Being at Midlife (Concurrent) and Later Adulthood (Longitudinal).

Predictor	Type of	Unstandardized Estimates													
Variable	Model	*a1*	*SE*	*c1*	*SE*	*e1*	*SE*	*a12*	*SE*	*e12*	*SE*	*a2*	*SE*	*e2*	*SE*
Maternal Affection	Concurrent	0.56	(0.06)	0.48	(0.07)	0.42	(0.02)	0.23	(0.04)	0.05	(0.02)	0.35	(0.05)	0.36	(0.03)
	Longitudinal	0.56	(0.07)	0.48	(0.06)	0.42	(0.02)	0.18	(0.04)	0.02	(0.04)	0.32	(0.05)	0.34	(0.03)
Maternal Discipline	Concurrent	0.57	(0.10)	0.36	(0.14)	0.37	(0.05)	0.03	(0.05)	0.07	(0.05)	0.42	(0.04)	0.36	(0.04)
	Longitudinal	0.57	(0.10)	0.36	(0.14)	0.37	(0.05)	0.12	(0.07)	−0.08	(0.05)	0.38	(0.07)	0.35	(0.05)
Paternal Affection	Concurrent	0.59	(0.08)	0.52	(0.10)	0.40	(0.03)	0.21	(0.06)	0.05	(0.04)	0.37	(0.06)	0.36	(0.04)
	Longitudinal	0.59	(0.09)	0.52	(0.10)	0.40	(0.02)	0.21	(0.06)	−0.01	(0.05)	0.33	(0.08)	0.36	(0.05)
Paternal Discipline	Concurrent	0.39	(0.15)	0.58	(0.08)	0.35	(0.05)	0.04	(0.13)	−0.04	(0.05)	0.42	(0.11)	0.36	(0.04)
	Longitudinal	0.39	(0.15)	0.58	(0.08)	0.35	(0.05)	0.02	(0.14)	−0.01	(0.06)	0.40	(0.12)	0.37	(0.05)
Family Support	Concurrent	0.20	(0.04)	0.09	(0.07)	0.26	(0.02)	0.28	(0.07)	0.10	(0.03)	0.27	(0.15)	0.32	(0.04)
	Longitudinal	0.20	(0.04)	0.09	(0.07)	0.25	(0.02)	0.27	(0.07)	0.02	(0.05)	0.23	(0.13)	0.33	(0.04)
Family Strain	Concurrent	0.23	(0.06)	0.20	(0.09)	0.30	(0.03)	−0.29	(0.07)	−0.11	(0.04)	0.25	(0.14)	0.32	(0.03)
	Longitudinal	0.24	(0.06)	0.20	(0.09)	0.31	(0.03)	−0.26	(0.09)	−0.08	(0.06)	0.27	(0.15)	0.34	(0.05)
Friend Support	Concurrent	0.21	(0.06)	0.12	(0.08)	0.42	(0.02)	0.26	(0.10)	0.11	(0.03)	0.32	(0.18)	0.34	(0.03)
	Longitudinal	0.21	(0.06)	0.12	(0.09)	0.42	(0.02)	0.22	(0.11)	0.07	(0.04)	0.32	(0.18)	0.35	(0.04)
Friend Strain	Concurrent	0.12	(0.04)	0.29	(0.03)	0.68	(0.03)	−0.39	(0.02)	−0.10	(0.02)	0.01	(0.00)	0.32	(0.02)
	Longitudinal	0.14	(0.05)	0.28	(0.05)	0.68	(0.03)	−0.37	(0.03)	−0.07	(0.02)	0.00	(0.00)	0.33	(0.03)
Spouse/Partner Support	Concurrent	0.46	(0.02)	0.00	(0.00)	0.78	(0.01)	0.35	(0.01)	0.11	(0.01)	0.00	(0.00)	0.31	(0.02)
	Longitudinal	0.47	(0.02)	0.00	(0.00)	0.76	(0.02)	0.24	(0.02)	0.05	(0.01)	0.24	(0.03)	0.31	(0.01)
Spouse/Partner Strain	Concurrent	0.43	(0.05)	0.00	(0.00)	0.61	(0.04)	−0.27	(0.05)	−0.14	(0.03)	0.23	(0.08)	0.28	(0.03)
	Longitudinal	0.44	(0.05)	0.00	(0.00)	0.62	(0.04)	−0.24	(0.06)	−0.05	(0.04)	0.24	(0.08)	0.32	(0.04)
Work-family Spillover(+)	Concurrent	0.30	(0.14)	0.32	(0.13)	0.54	(0.04)	0.11	(0.17)	0.10	(0.03)	0.40	(0.20)	0.35	(0.03)
	Longitudinal	0.31	(0.13)	0.31	(0.13)	0.54	(0.04)	0.13	(0.14)	0.02	(0.04)	0.38	(0.19)	0.36	(0.05)
Work-family Spillover(−)	Concurrent	0.28	(0.03)	0.00	(0.00)	0.41	(0.03)	−0.21	(0.04)	−0.21	(0.03)	0.34	(0.04)	0.28	(0.03)
	Longitudinal	0.30	(0.03)	0.00	(0.00)	0.46	(0.03)	−0.39	(0.05)	−0.03	(0.03)	0.07	(0.09)	0.36	(0.04)

Note. Bootstrapped standard errors (SE) are reported in parentheses to the right of parameter estimates. Well-being was the dependent variable in all models, whether measured concurrently or longitudinally, noted in the column titled "Type of Model". Social-relational exposures were measured when the average age of participants was approximately 46 years. In concurrent and longitudinal models, well-being was measured when the average age of participants was approximately 46 and 55 years, respectively.

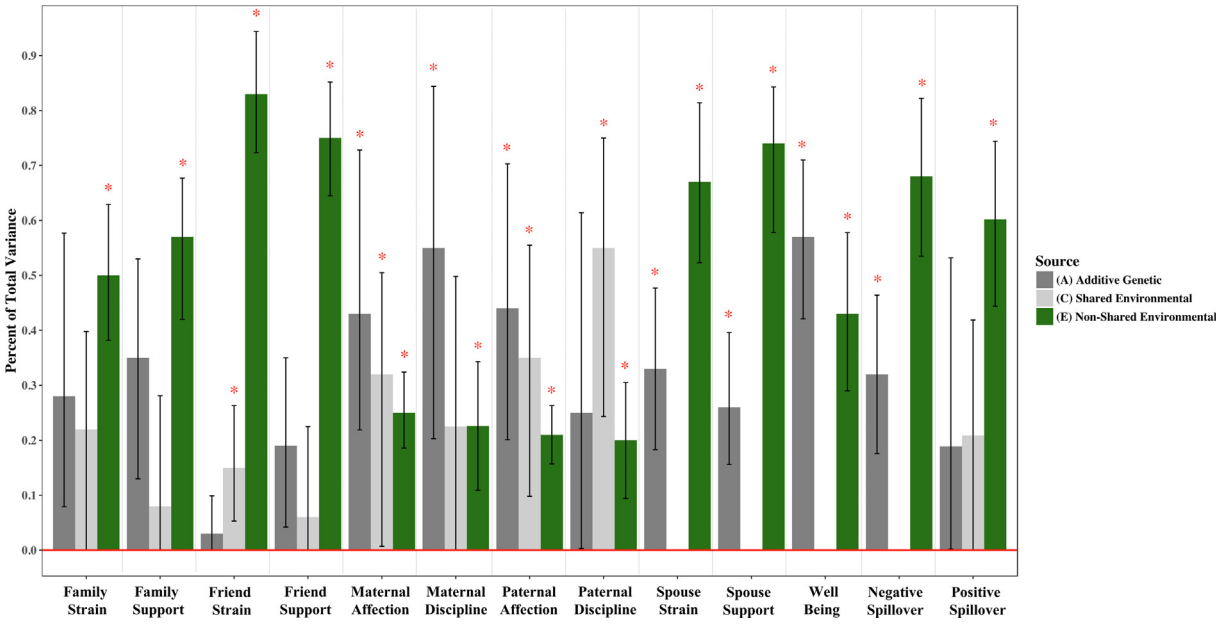

Fig. 1. Latent Genetic and Environmental Contributions to Variation in Social-Relational Exposures and Well-Being Measured at Midlife. Error bars depict 95% non-parametric bootstrapped confidence intervals. Asterisks denote portions of variance that were significantly different than zero at $p < .001$.

nal and paternal affection in childhood (reported retrospectively by adults) were modestly correlated with well-being at midlife, as well as a decade later in adulthood. However, results of the present study suggest that these associations are accounted for by common genetic factors that contribute to variation in both perceived parental affection and well-being. Similarly, there was little to no evidence that parental discipline in childhood was related to well-being through environmental pathways. Further, the phenotypic associations between parental discipline and well-being, both maternal and paternal, were trivial. The items used to measure discipline, however, did not include spanking, harsh corporal punishment, neglect, or abuse, which have been shown to be strongly associated with a number of deleterious outcomes (Cicchetti & Toth, 2005; Gershoff & Grogan-Kaylor, 2016; Vachon, Krueger, Rogosch, & Cicchetti, 2015). Indeed, cotwin-control studies have found that trauma, sexual abuse, and childhood maltreatment are associated with a number of detrimental outcomes through both heritable and environmental pathways (Brown

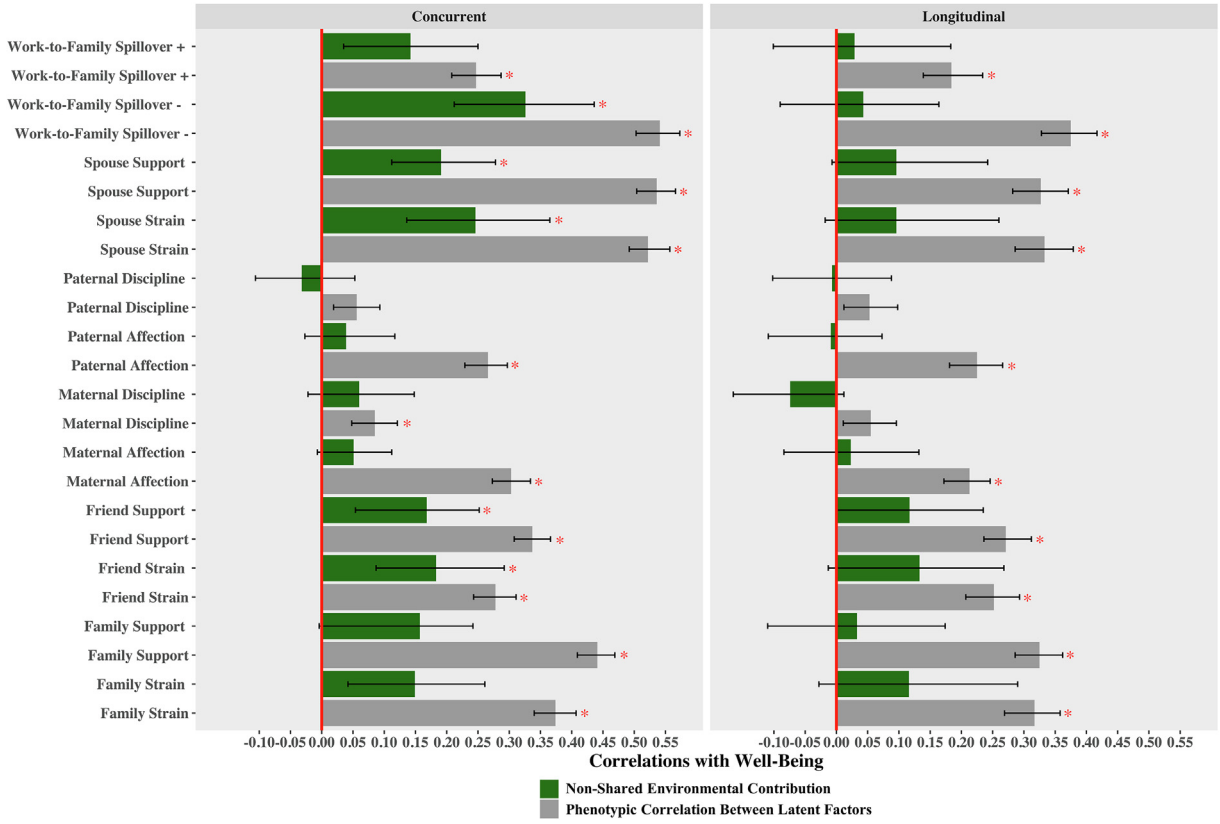

Fig. 2. Concurrent and Longitudinal Associations Between Social-Relational Exposures and Well-Being Before and After Accounting for Heritable and Shared Environmental Factors. Social-relational exposures were measured when the average age of participants was approximately 46 years. In concurrent and longitudinal models, well-being was measured when the average age of participants was approximately 46 and 55 years, respectively. Error bars depict 95% non-parametric bootstrapped confidence intervals. Asterisks mark correlations that were statistically significant at $p < .001$. "Phenotypic Correlation Between Latent Factors" denotes the correlations between social-relational exposures and well-being. "Non-shared environmental contribution" denotes the correlation after accounting for additive genetic and shared environmental variation- in other words, including only non-shared environmental contributions to covariation. Correlations between well-being and negative work-family spillover and social strain (family, friend, and spouse) were reflected (multiplied by −1) to ease visual comparison of effect sizes.

et al., 2014; Dinkler et al., 2017; Kendler et al., 2000; Nelson et al., 2002).

4.1. Limitations & future directions

The present study is not without limitations. One downside to analyzing data from a large population-representative sample is reliance on succinct measures that result in only modest measurement quality. Fittingly, in the present study social-relational exposures and well-being were measured using self-report questionnaires with a limited number of items. Although, the Cronbach's alpha of self-report scales met conventional standards for internal consistency and the factor loadings of individual items met conventional standards for inclusion, future studies would, nevertheless, benefit from incorporating additional sources of information to operationalize focal study constructs.

Although quasi-experimental in design, the present study falls short of a true experiment and, therefore, cannot provide direct evidence for a causal hypothesis or be used to draw definite conclusions about cause-effect relations. Rather, the present study provides estimates of covariation between social-relational exposures and well-being, after ruling out certain non-causal explanations. For many social-relational exposures covariation with well-being approached zero after accounting for these non-causal explanations, specifically overlapping genetic factors and shared environmental confounding. On the other hand, covariation between well-being and social support and work-family spillover remained significantly different than zero, after accounting for heritable and shared environmental factors. In addition, as the present study found that only contemporaneous associations are (at least partly) non-shared environmental in origin, reverse causality cannot be ruled out.

The latent well-being factor in the present study captured the tendency for individuals to endorse high positive emotions, low negative emotions, and high levels of satisfaction with one's family, friends, work, and life in general. However, despite the fact that positive affect, negative affect, and life satisfaction are themselves highly correlated, they often show differential correlations with other variables (Diener et al., 2017). Therefore, it remains an open question whether the latent environmental pathways documented in the present study will extend to more specific and fine-grained facets of well-being. The present study also focused on hedonic well-being, to the exclusion of alternative conceptualizations. For example, as opposed to balanced affect and life satisfaction, the eudaimonic tradition focuses more on the presence or absence of meaning in life and the fulfillment of one's potentials (Ryan & Deci, 2001). It remains unknown whether the results of the present study will extend to these alternative conceptualizations of well-being.

It is also important to remember the assumptions that underlie twin models and the consequences of violating those assumptions. For example, the assumption of no assortative mating, if violated, results in an inflation of shared-environmental variance. Thus, it is entirely possible that the magnitude of shared environmental variance in social-relational exposures was overestimated. On the other hand, if epistasis is present, then shared environmental vari-

ance will be underestimated. The bivariate twin models fit in the current study also assume no gene-environment interaction. Thus, it remains unknown whether the magnitude of genetic and environmental effects documented in the current study vary across levels of the measured exposure or other potential moderators. In addition, any inferences drawn from the present study should not be generalized to other cohorts and populations. It is unclear whether the genetic and environmental pathways between social-relational exposures and well-being documented in the present study will wax or wane in different countries, cultures, and times. Nevertheless, results of the present study suggest that social support and negative work-family spillover are related to well-being in adulthood, even after ruling out non-causal explanations related to heritable and shared environmental factors. Future studies stand to benefit from identifying the heritable characteristics that mediate genetic overlap between social-relational exposures and well-being, and, in turn, help to explain why different individuals encounter different kinds of environments that might lead to a better life.

Open practices statement

The current study was not formally preregistered because it conducted a secondary analysis of existing data, but data and study materials have been made available on a permanent third-party archive, the Inter-University Consortium for Political and Social Research (ICPSR). Requests to access the data should be directed to the ICPSR (https://www.icpsr.umich.edu/icpsrweb).

Author contributions

F.D.M. conducted analyses and drafted the manuscript in consultation with C.G.D. and R.F.K. All authors provided critical revisions and approved a final version of the manuscript prior to publication.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jrp.2019.103880.

References

Amstad, F. T., Meier, L. L., Fasel, U., Elfering, A., & Semmer, N. K. (2011). A meta-analysis of work–family conflict and various outcomes with a special emphasis on cross-domain versus matching-domain relations. *Journal of Occupational Health Psychology, 16*(2), 151.

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*(2), 137–156.

Brim, O. G., Ryff, C. D., & Kessler, R. C. (2004). *The MIDUS national survey: An overview*. University of Chicago Press.

Brown, R. C., Berenz, E. C., Aggen, S. H., Gardner, C. O., Knudsen, G. P., Reichborn-Kjennerud, T., ... Amstadter, A. B. (2014). Trauma exposure and Axis I psychopathology: A cotwin control analysis in Norwegian young adults. *Psychological Trauma: Theory, Research, Practice, and Policy, 6*(6), 652–660.

Chen, Y., & Feeley, T. H. (2014). Social support, social strain, loneliness, and well-being among older adults: An analysis of the Health and Retirement Study. *Journal of Social and Personal Relationships, 31*(2), 141–161.

Chu, P. S., Saucier, D. A., & Hafner, E. (2010). Meta-analysis of the relationships between social support and well-being in children and adolescents. *Journal of Social and Clinical Psychology, 29*(6), 624–645.

Cicchetti, D., & Toth, S. L. (2005). Child maltreatment. *Annual Review of Clinical Psychology, 1*, 409–438.

Diener, E., Heintzelman, S. J., Kushlev, K., Tay, L., Wirtz, D., Lutes, L. D., & Oishi, S. (2017). Findings all psychologists should know from the new science on subjective well-being. *Canadian Psychology/Psychologie Canadienne, 58*(2), 87–104.

Dinkler, L., Lundström, S., Gajwani, R., Lichtenstein, P., Gillberg, C., & Minnis, H. (2017). Maltreatment-associated neurodevelopmental disorders: A co-twin control analysis. *Journal of Child Psychology and Psychiatry, 58*(6), 691–701.

Duncan, G. E., Mills, B., Strachan, E., Hurvitz, P., Huang, R., Moudon, A. V., & Turkheimer, E. (2014). Stepping towards causation in studies of neighborhood and environmental effects: How twin research can overcome problems of selection and reverse causation. *Health & Place, 27*, 106–111.

Flouri, E. (2004). Subjective well-being in midlife: The role of involvement of and closeness to parents in childhood. *Journal of Happiness Studies, 5*, 335–358.

Gershoff, E. T., & Grogan-Kaylor, A. (2016). Spanking and child outcomes: Old controversies and new meta-analyses. *Journal of Family Psychology, 30*(4), 453–469.

Grzywacz, J. G., & Marks, N. F. (2000). Reconceptualizing the work–family interface: An ecological perspective on the correlates of positive and negative spillover between work and family. *Journal of Occupational Health Psychology, 5*(1), 111–126.

Hallquist, M., & Wiley, J. (2011). MplusAutomation: Automating Mplus model estimation and interpretation. R package version 0.6. Available online at: http://cran.R-project.org/web/packages/MplusAutomation/index.html.

Huppert, F. A. (2009). Psychological well-being: Evidence regarding its causes and consequences. *Applied Psychology: Health and Well-Being, 1*(2), 137–164.

Jaffee, S. R., & Price, T. S. (2007). Gene–environment correlations: A review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry, 12*(5), 432–442.

Johnson, W., & Krueger, R. F. (2006). How money buys happiness: Genetic and environmental processes linking finances and life satisfaction. *Journal of Personality and Social Psychology, 90*(4), 680.

Kendler, K. S., & Baker, J. H. (2007). Genetic influences on measures of the environment: A systematic review. *Psychological Medicine, 37*(5), 615–626.

Kendler, K. S., Bulik, C. M., Silberg, J., Hettema, J. M., Myers, J., & Prescott, C. A. (2000). Childhood sexual abuse and adult psychiatric and substance use disorders in women: An epidemiological and cotwin control analysis. *Archives of General Psychiatry, 57*(10), 953–959.

LaFreniere, P., & MacDonald, K. (2013). A post-genomic view of behavioral development and adaptation to the environment. *Developmental Review, 33*(2), 89–109.

Leve, L. D., & Cicchetti, D. (2016). Longitudinal transactional models of development and psychopathology. *Development and Psychopathology, 28*, 621–622.

Liechtenstein, P., Gatz, M., Pedersen, N. L., Berg, S., & McClearn, G. E. (1996). A co-twin—control study of response to widowhood. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 51*(5), P279–P289.

Loehlin, J. C. (1996). The Cholesky approach: A cautionary note. *Behavior Genetics, 26*(1), 65–69.

McGue, M., Osler, M., & Christensen, K. (2010). Causal inference and observational research: The utility of twins. *Perspectives on Psychological Science, 5*(5), 546–556.

Muthén, L. K., & Muthén, B. O. (2012). *Mplus version 8 user's guide*. Los Angeles, CA: Muthén & Muthén.

Nelson, E. C., Heath, A. C., Madden, P. A., Cooper, M. L., Dinwiddie, S. H., Bucholz, K. K., ... Martin, N. G. (2002). Association between self-reported childhood sexual abuse and adverse psychosocial outcomes: Results from a twin study. *Archives of General Psychiatry, 59*(2), 139–145.

Nguyen, A. W., Chatters, L. M., Taylor, R. J., & Mouzon, D. M. (2016). Social support from family and friends and subjective well-being of older African Americans. *Journal of Happiness Studies, 17*(3), 959–979.

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin, 84*(2), 309–322.

Pluess, M. (Ed.). (2015). Genetics of psychological well-being: the role of heritability and genetics in positive psychology. Series in Positive Psychology.

Polcari, A., Rabi, K., Bolger, E., & Teicher, M. H. (2014). Parental verbal affection and verbal aggression in childhood differentially influence psychiatric symptoms and wellbeing in young adulthood. *Child Abuse & Neglect, 38*(1), 91–102.

Rijken, M., & Groenewegen, P. P. (2008). Money does not bring well-being, but it does help! The relationship between financial resources and life satisfaction of the chronically ill mediated by social deprivation and loneliness. *Journal of Community & Applied Social Psychology, 18*(1), 39–53.

Ryan, R. M., & Deci, E. L. (2001). On happiness and human potentials: A review of research on hedonic and eudaimonic well-being. *Annual Review of Psychology, 52*(1), 141–166.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype→ environment effects. *Child Development, 54*(2), 424–435.

Schaefer, J. D., Moffitt, T. E., Arseneault, L., Danese, A., Fisher, H. L., Houts, R., ... Caspi, A. (2018). Adolescent victimization and early-adult psychopathology: Approaching causal inference using a longitudinal twin study to rule out noncausal explanations. *Clinical Psychological Science, 6*(3), 352–371.

Segrin, C., & Rynes, K. N. (2009). The mediating role of positive relations with others in associations between depressive symptoms, social skills, and perceived stress. *Journal of Research in Personality, 43*(6), 962–971.

Segrin, C., & Taylor, M. (2007). Positive interpersonal relationships mediate the association between social skills and psychological well-being. *Personality and individual differences, 43*(4), 637–646.

Suresh, S., & Sandhu, D. (2012). Social skills and well-being: The mediating role of positive relations with others. *Indian Journal of Positive Psychology, 3*(1), 71–74.

Vachon, D. D., Krueger, R. F., Rogosch, F. A., & Cicchetti, D. (2015). Assessment of the harmful psychiatric and behavioral effects of different forms of child maltreatment. *JAMA Psychiatry, 72*(11), 1135–1142.

Wang, R. A. H., Davis, O. S., Wootton, R. E., Mottershaw, A., & Haworth, C. M. (2017). Social support and mental health in late adolescence are correlated for genetic, as well as environmental, reasons. *Nature: Scientific Reports, 7*(1), 13088.

Wootton, R. E., Davis, O. S., Mottershaw, A. L., Wang, R. A. H., & Haworth, C. M. (2017). Genetic and environmental correlations between subjective wellbeing and experience of life events in adolescence. *European Child & Adolescent Psychiatry, 26*(9), 1119–1127.