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# Financial strain moderates genetic influences on self-rated health: support for diathesis–stress model of gene–environment interplay

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**ABSTRACT**

Data from the Interplay of Genes and Environment across Multiple Studies (IGEMS) consortium were used to examine predictions of different models of gene-by-environment interaction to understand how genetic variance in self-rated health (SRH) varies at different levels of financial strain. A total of 11,359 individuals from 10 twin studies in Australia, Sweden, and the United States contributed relevant data, including 2,074 monozygotic and 2,623 dizygotic twin pairs. Age ranged from 22 to 98 years, with a mean age of 61.05 (SD = 13.24). A factor model was used to create a harmonized measure of financial strain across studies and items. Twin analyses of genetic and environmental variance for SRH incorporating age, age$^2$, sex, and financial strain moderators indicated significant financial strain moderation of genetic influences on self-rated health. Moderation results did not differ across sex or country. Genetic variance for SRH increased as financial strain increased, matching the predictions of the diathesis–stress and social comparison models for components of variance. Under these models, environmental improvements would be expected to reduce genetically based health disparities.

Reducing socioeconomic inequalities in health by improving the health of the socioeconomically disadvantaged is a primary goal of health policy (WHO 1985). In the present study, we consider financial strain as an indicator of socio-economic inequality and describe its association with self-rated health (SRH), using a twin design. An extensive literature documents that socioeconomic status (SES) – encompassing occupational status, income,

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Supplemental data for this article can be accessed on the publisher's website.

and educational attainment – is one of the most robust predictors of health (Mirowsky and Ross 2003). It is associated with a broad array of outcomes including chronic disease (Mensah et al. 2005) and SRH (Moor, Spallek, and Richter 2017). The robustness and breadth of these associations has led to SES being identified as a "fundamental cause" of health disparities (Link and Phelan 1995). Nevertheless, the source of these SES-health associations continues to be heavily debated within social epidemiology and across disciplinary divides (Mackenbach 2012).

Social and health consequences of SES may depend on a person's own experience and perceptions of economic stressors as well as objective criteria (Glei, Goldman, and Weinstein 2018; Zavala 2014). For example, objective household income may not provide a complete picture of purchasing power or wealth, and the same level of income may reflect more or less financial strain in different cost-of-living contexts (Cundiff and Mathews 2017). Previous studies have demonstrated that subjective measures of financial strain predict mortality (Szanton et al. 2008), physical health (Singh-Manoux, Marmot, and Adler 2005), and subjective health (Arber, Fenn, and Meadows 2014), even after controlling for objective measures of SES such as income and education. Thus, our understanding of the etiology of associations between socioeconomic inequality and health can be expanded by considering measures that take into account the unique experiences of the individual within an environmental context.

In the last decade, several researchers have suggested that investigations of the associations between indicators of SES and health need to consider the role of genetic influences. It is generally accepted that there are genetic influences on both objective (Finkel et al. 2014) and subjective (Franz et al. 2017) measures of health. New theoretical models additionally posit gene-by-environment interplay (GxE). The most prominent models of GxE, diathesis–stress and social compensation models (Boardman, Daw, and Freese 2013; Reiss, Leve, and Neiderhiser 2013), predict that the importance of genetic factors (i.e., genetic variance) in health outcomes is maximized in adverse environments and minimized in favorable ones. The diathesis–stress model hypothesizes that high-risk environments (e.g., high financial strain) will exacerbate expression of genetic vulnerability for various health conditions. Social compensation is an extension of the diathesis–stress model predicting that a beneficial environment prevents the expression of an underlying genetic vulnerability (Shanahan and Hofer 2005). There is considerable evidence to support diathesis–stress/social compensation models of disease. For example, genetic influences on obesity and cardiometabolic risk are greater in adverse environments than in beneficial environments, such as higher education levels or higher perceived neighborhood cohesion (Johnson et al. 2011; Robinette, Boardman, and Crimmins 2018).

Other models of GxE, social enhancement (Reiss, Leve, and Neiderhiser 2013; Shanahan and Boardman 2009; Shanahan and Hofer 2005) and social distinction (Boardman, Daw, and Freese 2013), predict that the benefits of enriched environments are not distributed equally, but rather, accrue preferentially to a subset of individuals with genotypes that are responsive to the environment. As a consequence, social enhancement/distinction models predict that genetic variance is minimized in adverse environments and maximized in favorable ones. For example, in childhood, genetic variance and heritability for intelligence may be diminished in lower SES rearing environments and maximized in higher SES

rearing environments (Turkheimer and Horn 2014). To date, few studies have attempted to test how these models of GxE might apply to relationships between SES and subjective health.

We focus on subjective health because, similar to financial strain, it taps individual experience and perceptions of health. Researchers have suggested that SRH allows for the holistic integration of health, symptoms, and sensations that are not captured by objective health measures (Benyamini et al. 2003). As a result, SRH is broadly predictive of morbidity and mortality, often beyond objective assessments of physical health (Idler and Benyamini 1997). A recent twin analysis of measures of subjective health supported the idea that subjective health taps personal intuitions about health and that these personal intuitions reflect cultural definitions and personal concepts of health (Franz et al. 2017). Moreover, the heritability estimates for subjective health can vary widely from 0% to 46%, with significant gender and age differences indicated (Franz et al. 2017), and the ability to account for differences in heritability of subjective health is incomplete.

The goal of the current analysis was to examine the impact of financial strain on genetic and environmental influences on SRH using data from the Interplay of Genes and Environment across Multiple Studies (IGEMS) twin consortium (Pedersen et al. 2019). This study marks the first effort to test different models of GxE for subjective measures of SES (i.e., financial strain) and health (i.e., SRH). We compare the results of twin modeling to the predictions of GxE models. Following the diathesis–stress model, we predict that genetic variance in SRH will be higher among individuals reporting higher financial strain. Differences between models of GxE have implications beyond resolving theoretical viewpoints. Environmental improvements are expected to reduce or eliminate genetically based health disparities under some models (e.g., diathesis–stress) but expand them (e.g., social distinction) or have a mixed impact (e.g., differential susceptibility) under others.

## Method

### *Participants*

IGEMS is an international consortium of twin studies from Nordic countries, the USA, and Australia covering the adult lifespan (Pedersen et al. 2019). Ten IGEMS studies included measures of financial strain and subjective health. Australian studies represent 23.10% of the sample: The Australian Over 50′s study (AO50 (Mosing et al. 2009)) and the Older Australian Twins Study (OATS (Sachdev et al. 2009)). Overlap between the two Australian samples is accounted for in the data. Swedish studies, drawn from the population-based Swedish Twin Registry, represent 30.82% of the sample: the Swedish Adoption/Twin Study of Aging (SATSA (Finkel and Pedersen 2004)), Origins of Variance in the Old-Old (OCTO-Twin (McClearn et al. 1997)), Aging in Women and Men: A Longitudinal Study of Gender Differences in Health Behavior and Health among Elderly (GENDER (Gold et al. 2002)), and the Twin and Offspring Study in Sweden (TOSS (Neiderhiser and Lichtenstein 2008)). USA studies represent 46.08% of the sample: Vietnam Era Twin Study of Aging (VETSA (Kremen, Franz, and Lyons 2013)), Midlife in the United States (MIDUS (South and Krueger 2012)), the Carolina African-American Twin Study of Aging (CAATSA (Whitfield 2013)), and the Project Talent Twin and

**Table 1.** Sample characteristics.

| Study | N | MZ/DZ pairs | Age range | Median age | Mean age (SD) |
|---|---|---|---|---|---|
| *Australia* | | | | | |
| AO50 | 2,055 | 454/425 | 50–92 | 60.0 | 61.82 (8.63) |
| OATS | 569 | 138/131 | 65–90 | 69.7 | 71.28 (5.45) |
| *Sweden* | | | | | |
| GENDER | 498 | 0/248 | 69–89 | 72.7 | 73.35 (3.43) |
| OCTO-Twin | 646 | 131/165 | 80–98 | 82.5 | 83.41 (3.02) |
| SATSA | 1,721 | 215/364 | 26–92 | 62.8 | 60.51 (13.69) |
| TOSS | 636 | 142/169 | 32–54 | 44.0 | 43.87 (4.49) |
| *United States* | | | | | |
| CAATSA | 670 | 114/164 | 22–88 | 49.0 | 49.23 (14.40) |
| MIDUS | 1,158 | 169/132 | 25–74 | 44.0 | 45.21 (11.59) |
| PTTS | 2,183 | 370/561 | 67–74 | 70.0 | 70.01 (1.23) |
| VETSA | 1,223 | 341/264 | 51–60 | 54.0 | 55.42 (2.49) |
| *Total* | 11,359 | 2,074/2,623 | 22–98 | 63.0 | 61.05 (13.24) |

Note: MZ = monozygotic twin, DZ = dizygotic twin. AO50 = Australians Over 50, OATS = Older Australian Twin Study, SATSA = Swedish Adoption/Twin Study of Aging, OCTO-Twin = Origins of Variance in the Oldest Old, Gender = Aging in Women and Men: A Longitudinal Study of Gender Differences in Health Behavior and Health among Elderly, CAATSA = Carolina African American Twin Study of Aging, MIDUS = , PTTS = Project Talent Twin Study, VETSA = Vietnam Era Twin Study of Aging.

Sibling Study (PTTS (Flanagan 1962; Prescott et al. 2013)). The sample sizes and age ranges from the studies are presented in Table 1. A total of 11,359 individuals had data on both financial strain and SRH. Age ranged from 22 to 98 years, with a mean age of 61.05 (SD = 13.24); 56.15% of the sample was female.

## Measures

### Financial Strain (FS)

Eleven IGEMS studies (listed in Supplemental Table S1) incorporated various items assessing financial strain or economic situation. Before proceeding to data analysis, we created a harmonized measure of financial strain. Four items were common across most of the studies: how well does your money cover your needs, do you have difficulty covering your monthly expenses, how does your economic situation compare to others of the same age, and do you usually have enough money for extra treats. We relied on data from the full sample and entire set of contributions for factor score generation. Factor analysis supported a single financial strain measure with factor loadings ranging from .681 to .897 (see Supplemental Table S2). Factors scores were generated on the available items for each individual using factor loadings calculated in full IGEMS sample. For any given individual, some items were missing and some items were present. Factor scores were then translated to T-score metric (mean of 50 and SD of 10 within each sample), with higher scores indicating more financial strain.

### Self-Rated Health (SRH)

The number of response options for the SRH item (how would you rate your overall health?) varied across studies ranging from 3 to 7 options. Based on an earlier analysis of various harmonization methods within IGEMS (Gatz et al. 2015), the most parsimonious and effective approach to harmonization was standardizing the variable within a study, transforming to a T-score, and then combining data across studies. Higher scores indicated worse subjective health.

### Statistical Method

The standard univariate twin method incorporates monozygotic (MZ) twins and dizygotic (DZ) twins to decompose the variance of any trait into the proportion attributed to additive genetic influences (A), common or shared environmental influences that contribute to similarity within families (C), and unique environmental influences that contribute to differences within families (E). MZ twins share all of their genetic material (A) and DZ twins share only half of their segregating genes. Shared environment (C) will contribute to the similarity of both MZ and DZ twins. Thus, we can estimate A and C by comparing the similarity of MZ and DZ twins. Unique environment (E) is the only component that can generate differences between MZ twins. In standard analysis of twin data, effects attributable to measurement error are typically included with E. Data from both complete and incomplete pairs can be included, with incomplete pairs contributing to the estimation of means. A moderation model was used incorporating three continuous moderator variables (age, age-squared, and financial strain) and one categorical moderator variable (sex) (van der Sluis et al. 2008).

All moderators were included in all models. The focus of the current analysis was financial strain; therefore, model comparison focused on financial strain moderation of A, C, and E in SRH while correcting for age, age-squared, and sex. The diathesis–stress model predicts that the importance of genetic factors in health outcomes is maximized in adverse environments and minimized in favorable ones; therefore, increasing A variance in SRH with increasing financial strain would support the diathesis–stress model. In contrast, the social enhancement model predicts, genetic variance is minimized in adverse environments and maximized in favorable ones; therefore, decreasing A variance in SRH with increasing financial strain would support social enhancement models. Model comparisons to investigate financial strain moderation of A, C, and E components of variance in SRH included three phases: model fitting in the full sample, dividing the sample by sex (two groups) and comparing model fit, and dividing the sample by country (three groups) and comparing model fit. Opposite sex DZ pairs could not be included when the sample was divided into male and female twin pairs; therefore, the sample size for that phase of model testing was reduced by 698 pairs. All statistical models were tested using the structural equation-modeling package Classic Mx 1.68 (Neale et al. 2004). Evaluation of relative fit of statistical models was performed using the likelihood-ratio test (LRT). Significant LRT values indicated that the reduction in parameters resulted in a significant reduction in model fit. Analyses were cross-sectional, using the baseline data for each study.

## Results

### Preliminary Analyses

There were no significant sex differences in mean FS or SRH and no significant country differences in mean SRH (see Supplemental Table S3). Analysis of variance indicated significant mean differences across country in FS ($F(2, 8596) = 10.84$, $p < .01$). ANOVA results were replicated using Welch's F statistic, which does not assume homogeneity of variance. Tukey's HSD post-hoc test indicated that mean FS was higher in Sweden than in Australia and the USA. Correlations between FS and SRH were significant at $p < .01$ in Sweden (.10) and the USA (.12), but not in Australia (.02). The correlation did not differ for

men and women (see Supplemental Table S3). Twin correlations in each study and country are reported in Supplemental Table S4. The heritability estimate for SRH in the full sample, corrected age, age-squared, and sex, was .12 (95% confidence interval = .05, .20) at the median financial strain. The heritability estimates for SRH were .32 (.07, .51) in Swedish sample, .11 (.03, .21) in the US samples, and .09 (.01, .22) in Australian samples.

## GxE Analyses

Five models were compared in the full sample and results of model comparisons presented in Table 2. First, the full model with all parameters was fit to the data as a baseline. In model 2, the significance of financial strain moderation of SRH was tested by dropping the relevant parameters from the model. In models 3 through 5, financial strain moderation of A, C, or E components of variance was tested individually. Dropping all financial strain moderation from the model resulted in a significant reduction in model fit (LRT = 15.03, df = 6, $p < .05$); however, models 3 through 5 indicate that the only significant moderation occurs for additive genetic variance. Estimated trends in components of variance in SRH across levels of financial strain are depicted in Figure 1, based on parameter estimates from the full moderation model (model 1). The figure shows that while environmental variance estimates (C and E) for SRH were fairly constant at different levels of financial strain, genetic variance (A) for SRH increased with increasing financial strain, as predicted by the diathesis–stress and social compensation models of GxE. Heritability for SRH increased from 6% at the 10th percentile of financial strain to 22% at the 90th percentile.

**Table 2.** Model-fitting results for financial strain moderation of genetic and environmental variations in self-rated health.

| Model | Log likelihood | Degrees of freedom | Number of parameters | Likelihood ratio test (df) |
|---|---|---|---|---|
| *Full sample* | | | | |
| 1. Full model | 123640.85 | 16689 | 37 | |
| 2. Drop all FS moderation | 123655.88 | 16695 | 31 | 15.03 (6)* |
| 3. Drop FS moderation of A | 123651.79 | 16691 | 35 | 10.95 (2)** |
| 4. Drop FS moderation of C | 123641.18 | 16691 | 35 | 0.33 (2) |
| 5. Drop FS moderation of E | 123641.78 | 16691 | 35 | 0.94 (2) |
| *Sex comparison* | | | | |
| 6. Full model | 112019.34 | 15090 | 70 | |
| 7. Equate all FS moderation across sex | 112027.42 | 15096 | 64 | 8.09 (6) |
| 8. Equate FS moderation of A across sex | 112021.70 | 15092 | 68 | 2.36 (2) |
| 9. Equate FS moderation of C across sex | 112020.85 | 15092 | 68 | 1.51 (2) |
| 10. Equate FS moderation of E across sex | 112021.20 | 15092 | 68 | 1.86 (2) |
| *Country comparison* | | | | |
| 11. Full model | 123260.17 | 16621 | 111 | |
| 12. Equate all FS moderation across country | 123274.53 | 16633 | 99 | 14.39 (12) |
| 13. Equate FS moderation of A across country | 123262.22 | 16625 | 107 | 2.07 (4) |
| 14. Equate FS moderation of C across country | 123260.78 | 16625 | 107 | 0.63 (4) |
| 15. Equate FS moderation of E across country | 123269.13 | 16625 | 107 | 8.99 (4) |

Note: Likelihood ratio tests compared model fit to the full model within each section. A = additive genetic variance, C = shared rearing environmental variance, E = unique environmental variance. *$p < .05$; **$p < .01$

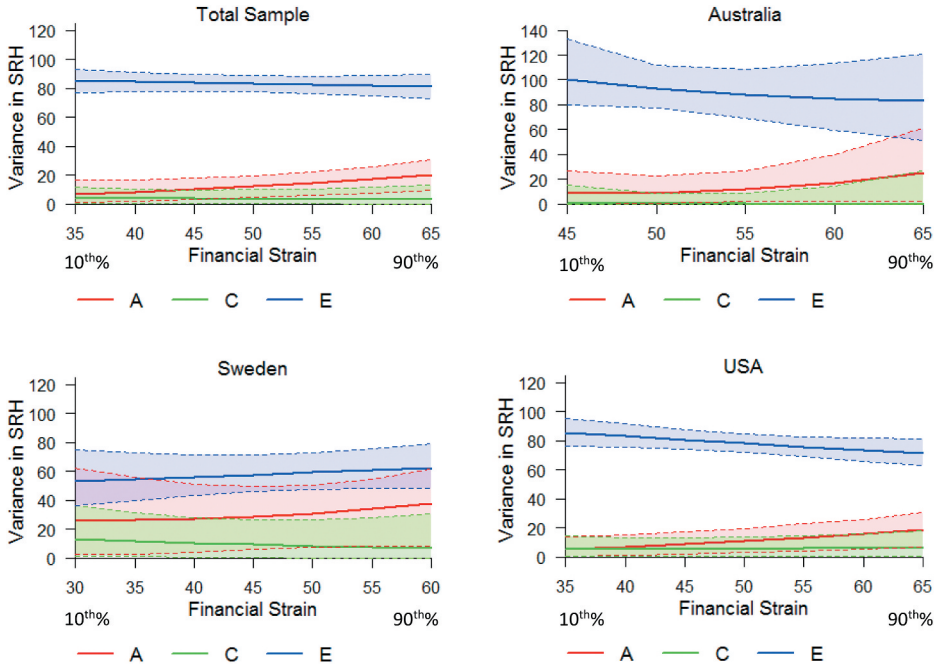


**Figure 1.** Financial strain moderation of additive genetic (A), shared environmental (C), and nonshared environmental (E) components of variance in self-rated health (SRH): total sample, Australia, Sweden, and United States. Dotted lines indicate the 95% confidence interval for the estimated variance components.

In subsequent phases of model comparison, sex and country differences in these results were tested. The same five models were tested across sexes and countries: full model as a baseline, equate all financial strain moderation parameters across groups, and then equate financial strain moderation of A, C, or E components of variance across groups. As shown in Table 3, none of these reduced models resulted in a significant change in model fit, indicating that the pattern of financial strain moderation of A, C, and E in SRH was similar across sexes and countries. Replication of the general pattern of the financial strain moderation of A for SRH in the three countries is indicated in Figure 1.

## Discussion

The goal of the current analysis was to examine the extent to which financial strain moderates genetic and environmental influences on SRH, as a means to test models of gene-by-environment interplay. Results indicated that an underlying genetic vulnerability to poor subjective health was maximized in the unfavorable environment indicated by high financial strain, supporting the diathesis–stress and social compensation models of gene–environment interplay. Moreover, this result was replicated across three countries with very different systems of government support of health and welfare.

The heritability estimates for SRH reported here were generally lower than the heritability estimates reported by other researchers, which tend to range from 25% to 50% (Franz et al. 2017; Mosing et al. 2010; Svedberg et al. 2005). Differences in samples, measurements, and models may explain some of the differences in the heritability estimates. For example,

some samples included only male twins (Romeis et al. 2000), insufficient twin pairs to find significant sex differences (Mosing et al. 2010), or only younger twins (Silventoinen et al. 2007), although in a large consortium, sample age and sex differences in genetic influences on SRH were significant (Franz et al. 2017). Studies have incorporated multi-item measures of SRH (Svedberg et al. 2005) and investigated genetic influences on SRH in the context of other variables, such as optimism (Mosing et al. 2010). To our knowledge, the current analysis represents the first direct comparison of the heritability estimates for SRH from different countries, even though evidence suggests possible country differences in mean SRH (Hardy, Acciai, and Reyes 2014). Country differences in mean SRH do not necessarily translate to country differences in components of variance, and in the current analysis, it did not translate to country differences in the financial strain moderation of genetic influences on SRH.

It is likely that there are various pathways through which increased financial strain can trigger the underlying diathesis for poor SRH. Social-psychological explanations focus on the impact of relative deprivations associated with economic hardship or financial strain (i.e., poor housing, poor access to health care and education, crowding, food deserts) and the subsequent psychological and physiological outcomes (i.e., inadequate diet, poor health care) which in turn affect health (Hoebel and Lampert 2020). In contrast, neurobiological explanations posit that individuals' perceptions and resulting physiological reactivity to financial strain act as a stressor may lead to consequences in health outcomes via endogenous stress pathways (Hoebel and Lampert 2020). For example, lower subjective SES is associated with higher cortisol reactivity and higher abdominal fat distribution, as well as higher self-reported chronic stress (Adler et al. 2000). Associations also exist for biomarkers that relate to functions of immune system and inflammatory processes in the body (Derry et al. 2013; Steptoe 2012). Ultimately, a multidisciplinary framework incorporating both psychological and neurobiological mechanisms may provide insight into the cascade of effects linking financial strain and SRH (Hoebel and Lampert 2020).

It is important to consider these results in the context of possible confounds, including common-method variance, the scope of subjective measures of health and SES, "third" variable issues, or reverse causation (Boardman, Domingue, and Daw 2015). The relationship between financial strain and SRH may arise from common-method variance: how people rate themselves (Li, Zhang, and Muennig 2018; Singh-Manoux, Marmot, and Adler 2005). Individuals who perceive themselves as unable to meet their financial needs may also be more likely to perceive themselves as having poor health, independent of actual SES and health status. However, it is possible to consider the common methodology as a strength. Researchers have suggested that SRH contributes to the prediction of mortality and morbidity over and above measures of physical health because the subjective nature of SRH allows for the holistic integration of health, symptoms, and sensations that are not captured by objective health measures (Benyamini et al. 2003). Similarly, financial strain likely allows individuals to globally assess SES as a stressor incorporating all salient aspects (Cundiff and Mathews 2017; Hoebel and Lampert 2020; Singh-Manoux, Marmot, and Adler 2005).

It is also possible that the shared variance between financial strain and SRH arises from unmeasured confounds or "third" variables, including objective SES, personality traits, or psychosocial factors that contribute to both financial strain and subjective health (Hoebel and Lampert 2020). In the context of models of GxE, a third variable such as neuroticism

could underlie the shared genetic variance for these subjective measures of SES and health. However, phenotypic analyses that have controlled for third variables such as self-esteem, perceived control, trust, cynicism, mastery, or neuroticism report that associations between SRH and subjective SES remained significant (Lundberg and Kristenson 2008). In the IGEMS studies, we find only modest phenotypic correlations of SRH and financial strain with possible third variables such as neuroticism and extraversion.

Although the current investigation focused on financial strain moderation of SRH, it is possible that health may impact socio-economic factors and resulting perceptions of financial strain. Health expenses associated with major illnesses could be a substantial source of financial strain in countries without universal health care. Results from longitudinal studies, sensitivity analyses, and experimental designs provide support for a direction of effect from subjective SES to health outcomes (Cohen et al. 2008; Hoebel and Lampert 2020; Li, Zhang, and Muennig 2018). For example, lower subjective SES predicts subsequent declines in health, even when controlling for objective SES and baseline health (Singh-Manoux, Marmot, and Adler 2005). In the current sample, we also investigated the possibility that SRH moderates genetic variance for financial strain, but modeling produced no consistent evidence for that association. Moreover, regardless of differences in health-care systems in Australia, Sweden, and the USA, financial strain moderation of genetic variance in SRH did not differ significantly across countries.

Finally, the current statistical model estimated genetic and environmental influences on both financial strain and SRH, providing an estimate of shared genetic variance (rG) and shared environmental variance (rC). Estimates for both rG (.95) and rC (.99) were quite high in the full sample, although estimates should be interpreted with caution in the context of modest phenotypic correlations and heritability estimates. The results suggest the possibility of forms of pleiotropy, in which genetic variance for SRH arises from genetic influences on financial strain (mediational pleiotropy) or the same set of genes may give rise to variance in both SRH and financial (biological pleitropy) (Boardman, Domingue, and Daw 2015). In combination with the results of the moderation model, these estimates of shared genetic variance emphasize the role of genetic influences in the linkage between financial strain and SRH.

In conclusion, results indicated financial strain moderated genetic variance for SRH, as predicted by the diathesis–stress and social compensation models of gene-by-environment interplay. Results were consistent across Australia, Sweden, and the United States, although they may not generalize to non-Western countries. Under the diathesis–stress model, environmental improvements would be expected to reduce genetically based health disparities. Improvements could focus on objective factors, such as access to education, access to health care, higher minimum wage, or universal basic income, which should in turn lead to lower financial strain. It is also possible that subjective SES, per se, may be malleable, providing a means for impacting health disparities. These results are a powerful demonstration of why GxE processes should be taken into account in trying to understand social determinants of health. The fundamental cause has a different impact, dependent on genetic endowment.

## Disclosure Statement

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## References

Adler, N. E., E. S. Epel, G. Castellazzo, and J. R. Ickovics. 2000. Relationship of subjective and objective social status with psychological and physiological functioning: Preliminary data in healthy, White women. *Health Psychology* 19 (6):586. doi:10.1037/0278-6133.19.6.586.

Arber, S., K. Fenn, and R. Meadows. 2014. Subjective financial well-being, income and health inequalities in mid and later life in Britain. *Social Science & Medicine* 100:12–20. doi:10.1016/j.socscimed.2013.10.016.

Benyamini, Y., T. Blumstein, A. Lusky, and B. Modan. 2003. Gender differences in the self-rated health–mortality association: Is it poor self-rated health that predicts mortality or excellent self-rated health that predicts survival? *The Gerontologist* 43:396–405. doi:10.1093/geront/43.3.396.

Boardman, J. D., J. Daw, and J. Freese. 2013. Defining the environment in gene-environment research: lessons from social epidemiology. *American Journal of Public Health* 103 (S1):S64–S72. doi:10.2105/AJPH.2013.301355.

Boardman, J. D., B. W. Domingue, and J. Daw. 2015. What can genes tell us about the relationship between education and health? *Social Science & Medicine* 127:171–80. doi:10.1016/j.socscimed.2014.08.001.

Cohen, S., C. M. Alper, W. J. Doyle, N. Adler, J. J. Treanor, and R. B. Turner. 2008. Objective and subjective socioeconomic status and susceptibility to the common cold. *Health Psychology* 27 (2):268. doi:10.1037/0278-6133.27.2.268.

Cundiff, J. M., and K. A. Mathews. 2017. Is subjective social status a unique correlate of physical health? A meta-analysis. *Health Psychology* 36:1109–25. doi:10.1037/hea0000534.

Derry, H. M., C. P. Fagundes, R. Andridge, R. Glaser, W. B. Malarkey, and J. K. Kiecolt-Glaser. 2013. Lower subjective social status exaggerates interleukin-6 responses to a laboratory stressor. *Psychoneuroendocrinology* 38 (11):2676–85. doi:10.1016/j.psyneuen.2013.06.026.

Finkel, D., L. Gerritsen, C. A. Reynolds, A. K. Dahl, and N. L. Pedersen. 2014. Etiology of individual differences in human health and longevity. In *Annual review of gerontology and geriatrics – genetics*, ed. R. L. Sprott, 189–227. New York: Springer.

Finkel, D., and N. L. Pedersen. 2004. Processing speed and longitudinal trajectories of change for cognitive abilities: The Swedish adoption/twin study of aging. *Aging, Neuropsychology, and Cognition* 11:325–45. doi:10.1080/13825580490511152.

Flanagan, J. C. 1962. Project Talent. *Applied Psychology* 11:3–14. doi:10.1111/j.1464-0597.1962.tb00441.x.

Franz, C., D. Finkel, M. Panizzon, K. Spoon, K. Christensen, M. Gatz, N. Pedersen, R. Krueger, J. Neiderhiser, and C. Reynolds. 2017. Facets of subjective health from early adulthood to old age. *Journal of Aging and Health* 29 (1):149–71. doi:10.1177/0898264315625488.

Gatz, M., C. A. Reynolds, D. Finkel, C. Hahn, Y. Zhou, and C. Zavala. 2015. Data harmonization in aging research: Not so fast. *Experimental Aging Research* 41 (5):475–95. doi:10.1080/0361073X.2015.1085748.

Glei, D. A., N. Goldman, and M. Weinstein. 2018. Perception has its own reality: Subjective versus objective measures of economic distress. *Population and Development Review* 44 (4):695–722. doi:10.1111/padr.12183.

Gold, C. H., B. Malmberg, G. E. McClearn, N. L. Pedersen, and S. Berg. 2002. Gender and health: A study of older unlike-sex twins. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences* 57B:S168–S176. doi:10.1093/geronb/57.3.S168.

Hardy, M. A., F. Acciai, and A. M. Reyes. 2014. How health conditions translate into self-ratings: A comparative study of older adults across Europe. *Journal of Health and Social Behavior* 55 (3):320–41. doi:10.1177/0022146514541446.

Hoebel, J., and T. Lampert. 2020. Subjective social status and health: Multidisciplinary explanations and methodological challenges. *Journal of Health Psychology* 25:173–85. doi:10.1177/1359105318808804.

Idler, E. L., and Y. Benyamini. 1997. Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior* 38:21–37. doi:10.2307/2955359.

Johnson, W., K. O. Kyvik, A. Skytthe, I. J. Deary, and T. I. A. Sorenson. 2011. Education Modifies Genetic and Environmental Influences on BMI. *PLoS One* 6 (1):e16290. doi:10.1371/journal.pone.0016290.

Kremen, W. S., C. E. Franz, and M. J. Lyons. 2013. VETSA: The Vietnam Era Twin Study of Aging. *Twin Research and Human Genetics: The Official Journal of the International Society for Twin Studies* 16:399–402. doi:10.1017/thg.2012.86.

Li, S., Q. Zhang, and P. Muennig. 2018. Subjective assessments of income and social class on health and survival: An enigma. *SSM-population Health* 6:295–300. doi:10.1016/j.ssmph.2017.10.005.

Link, B. G., and J. Phelan. 1995. Social conditions as fundamental causes of disease. *Journal of Health and Social Behavior* 80–94. doi:10.2307/2626958.

Lundberg, J., and M. Kristenson. 2008. Is subjective status influenced by psychosocial factors? *Social Indicators Research* 89 (3):375. doi:10.1007/s11205-008-9238-3.

Mackenbach, J. P. 2012. The persistence of health inequalities in modern welfare states: The explanation of a paradox. *Social Science & Medicine* 75 (4):761–69. doi:10.1016/j.socscimed.2012.02.031.

McClearn, G. E., B. Johansson, S. Berg, N. L. Pedersen, F. Ahern, S. A. Petrill, and R. Plomin. 1997. Substantial genetic influence on cognitive abilities in twins 80 or more years old. *Science* 276:1560–63. doi:10.1126/science.276.5318.1560.

Mensah, G. A., A. H. Mokdad, E. S. Ford, K. J. Greenlund, and J. B. Croft. 2005. State of disparities in cardiovascular health in the United States. *Circulation* 111 (10):1233–41. doi:10.1161/01.cir.0000158136.76824.04.

Mirowsky, J., and C. E. Ross. 2003. *Education, social class, and health*. New York, New York: Aldine deGruyter.

Moor, I., J. Spallek, and M. Richter. 2017. Explaining socioeconomic inequalities in self-rated health: A systematic review of the relative contribution of material, psychosocial and behavioural factors. *Journal of Epidemiology and Community Health* 71 (6):565–75. doi:10.1136/jech-2016-207589.

Mosing, M. A., N. L. Pedersen, N. G. Martin, and M. J. Wright. 2010. Sex differences in the genetic architecture of optimism and health and their interrelation: A study of Australian and Swedish twins. *Twin Research and Human Genetics* 13 (4):322–29. doi:10.1375/twin.13.4.322.

Mosing, M. A., B. P. Zietsch, S. N. Shekar, M. J. Wright, and N. G. Martin. 2009. Genetic and Environmental Influences on optimism and its relationship to mental and self-rated health: A study of aging twins. *Behavior Genetics* 39:597–604. doi:10.1007/s10519-009-9287-7.

Neale, M. C., S. M. Boker, G. Xie, and H. H. Maes. 2004. *Mx: Statistical modeling*. 6th ed., 126 MCV. Richmond VA 23298: Department of Psychiatry. Box.

Neiderhiser, J. M., and P. Lichtenstein. 2008. The Twin and Offspring Study in Sweden: Advancing our understanding of genotype-environment interplay by studying twins and their families. *Acta Psychologica Sinica* 40 (10):1116–23.

Pedersen, N. L., M. Gatz, B. K. Finch, D. Finkel, D. A. Butler, A. D. Aslan, … M. McGue. 2019. IGEMS: The consortium on interplay of genes and environment across multiple studies—an update. *Twin Research and Human Genetics* 1–8. doi:10.1017/thg.2018.75.

Prescott, C. A., D. L. Achorn, A. Kaiser, L. Mitchell, J. J. McArdle, and S. J. Lapham. 2013. The project TALENT twin and sibling study. *Twin Research and Human Genetics* 18:437–48. doi:10.1017/thg.2012.71.

Reiss, D., L. D. Leve, and J. Neiderhiser. 2013. How genes and the social environment moderate each other. *American Journal of Public Health* 103 (S1):S111–S121. doi:10.2105/AJPH.2013.301408.

Robinette, J. W., J. D. Boardman, and E. Crimmins. 2018. Perceived neighborhood social cohesion and cardiometabolic risk: A gene× environment study. *Biodemography and Social Biology* 64 (3–4):173–86. doi:10.1080/19485565.2019.1579084.

Romeis, J. C., J. F. Scherrer, H. Xian, S. A. Eisen, K. Bucholz, A. C. Heath, … W. R. True. 2000. Heritability of self-reported health. *Health Services Research* 35 (5 Pt 1):995.

Sachdev, P. S., A. Lammel, J. N. Trollor, T. Lee, M. J. Wright, D. Ames, … P. R. Schofield. 2009. A comprehensive neuropsychiatric study of elderly twins: The older Australian twins study. *Twin Research and Human Genetics* 12(573–582. doi:10.1375/twin.12.6.573.

Shanahan, M. J., and J. D. Boardman. 2009. Genetics and Behavior in the Life Course: A Promising Frontier. In *The craft of life course research*, ed. G. H. E. A. J. Z. Giele, 215–35. New York: Guilford.

Shanahan, M. J., and S. M. Hofer. 2005. Social context in gene–environment interactions: retrospect and prospect. *Journals of Gerontology B 60*65–76. doi:10.1093/geronb/60.Special_Issue_1.65.

Silventoinen, K., D. Posthuma, E. Lahelma, R. J. Rose, and J. Kaprio. 2007. Genetic and environmental factors affecting self-rated health from age 16–25: A longitudinal study of Finnish twins. *Behavior Genetics* 37 (2):326–33. doi:10.1007/s10519-006-9096-1.

Singh-Manoux, A., M. G. Marmot, and N. E. Adler. 2005. Does subjective social status predict health and change in health status better than objective status? *Psychosomatic Medicine* 67 (6):855–61. doi:10.1097/01.psy.0000188434.52941.a0.

South, S. C., and R. F. Krueger. 2012. Genetic strategies for probing conscientiousness and its relationship to aging. *Developmental Psychology* 50:1362–76. doi:10.1037/a0030725.

Steptoe, A. 2012. Socioeconomic status, inflammation, and immune function. *The Oxford Handbook of Psychoneuroimmunology*, ed. S. C. Segerstrom, 234–252. New York: Oxford University Press.

Svedberg, P., M. Gatz, P. Lichtenstein, S. Sandin, and N. L. Pedersen. 2005. Self-rated health in a longitudinal perspective: A 9-year follow-up twin study. *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 60 (6):S331–S340. doi:10.1093/geronb/60.6.S331.

Szanton, S. L., J. K. Allen, R. J. Thorpe Jr, T. Seeman, K. Bandeen-Roche, and L. P. Fried. 2008. Effect of financial strain on mortality in community-dwelling older women. *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 63 (6):S369–S374. doi:10.1093/geronb/63.6.S369.

Turkheimer, E., and E. E. Horn. 2014. Interactions between socioeconomic status and components of variation in cognitive ability. In *Behavior genetics of cognition across the lifespan*, ed. D. Finkel and C. Reynolds, 41–68. New York: Springer.

van der Sluis, S., C. V. Dolan, M. C. Neale, and D. Posthuma. 2008. A general test for gene-environment interaction in sib pair-based association analysis of quantitative traits. *Behavior Genetics* 38 (4):372–89. doi:10.1007/s10519-008-9201-8.

Whitfield, K. E. 2013. A registry of adult African American twins: The Carolina African American twin study of aging. *Twin Research and Human Genetics* 16 (1):476–80. doi:10.1017/thg.2012.79.

WHO. 1985. *Targets for health for all*. Copenhagen: World Health Organization.

Zavala, C. 2014. *Subjective socioeconomic status in daily cognitive functioning and cognitive aging*. (Publication No. 3644052) [Doctoral dissertation, University of California, Riverside]. ProQuest Dissertations and Theses Database.