A new look at neuroticism

A New Look at Neuroticism: Should We Worry So Much About Worrying?

Alexander Weiss[1] and Ian J. Deary

Department of Psychology, School of Philosophy, Psychology and Language Sciences, University of Edinburgh

Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh

[1]Address correspondence to Alexander Weiss, Department of Psychology, School of Philosophy, Psychology and Language Sciences, University of Edinburgh, 7 George Square, Edinburgh, EH8 9JZ, United Kingdom. Telephone: +44 131 650 3456; E-mail: alex.weiss@ed.ac.uk

Word count (Abstract + Main text + Acknowledgements): 2500

Number of tables: 5

Number of figures: 2

Number of references: 31

Abstract

People higher in neuroticism seem to have drawn the short straw of personality. However, there are multiple ways to score highly in neuroticism. Analyses of the short-scale Eysenck Personality Questionnaire-Revised in three large datasets revealed that higher neuroticism can mean having elevated scores on all items, elevated scores mainly on items related to anxiety and tension, or elevated scores mainly on items related to worry and vulnerability. Epidemiological and molecular genetic studies revealed that people in the first group are at greater risk for poorer mental and physical health, but that people in the latter two groups, and especially those people beset by worry and feelings of vulnerability, have better physical health. These findings suggest that future research on neuroticism and health should focus on different ways that people can exhibit high neuroticism.

A New Look at Neuroticism: Should We Worry So Much About Worrying?

Neuroticism features in nearly all personality models and questionnaires. For example, in the short-scale Eysenck Personality Questionnaire-Revised (EPQ-R; Eysenck, Eysenck, & Barrett, 1985), participants who answer "YES" to questions such as "Does your mood often go up and down?" obtain higher neuroticism scores. Other scales include items keyed to indicate low neuroticism, for example, "I seldom feel nervous." (McCrae, Costa, & Martin, 2005, p. 269).

Decades of research have provided evidence for neuroticism's reliability and construct validity. Neuroticism is substantially stable over time (Roberts & DelVecchio, 2000), self- and rater-reports agree quite strongly (Costa & McCrae, 1992a), and a third to half of its variation is attributable to genetic differences (Bouchard & Loehlin, 2001). People higher in neuroticism tend to be more stress prone, less happy, and less satisfied with their lives (Lahey, 2009; Strickhouser, Zell, & Krizan, 2017). They also tend to have poorer life outcomes: they are less well-educated, have lower incomes, and are more likely to divorce (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). Finally, people who are higher in neuroticism are more likely to be diagnosed with mental health disorders (Strickhouser, et al., 2017) and neurodegenerative diseases (e.g., Alzheimer's disease; Terracciano et al., 2014), and experience poorer physical health and die earlier (Deary, Weiss, & Batty, 2010; Roberts, et al., 2007; Strickhouser, et al., 2017).

Causal frameworks to explain relationships between neuroticism and health have been developed. One posits that neuroticism is related to mortality via its impact on health-related behaviors, thoughts, emotions, and physiological responses to stress. For example, the greater impulsiveness associated with neuroticism is said to lead to behaviors, such as cigarette smoking, which leads to increased mortality (Deary, et al., 2010). A second framework posits

that common causes, whether environmental stressors, genes, or other factors, lead to people being higher in neuroticism and having poorer health (Deary, et al., 2010).

Some findings on neuroticism and health are seemingly incompatible with these frameworks. Notably, some studies show that neuroticism is related to better health (e.g., type 2 diabetes; Čukić & Weiss, 2014) and reduced mortality (e.g., Korten et al., 1999). Other studies show that high neuroticism in the presence of high conscientiousness is especially protective (Turiano, Mroczek, Moynihan, & Chapman, 2013).

The studies that show that neuroticism can be protective are 'good studies': they have sufficient power and the neuroticism measures are reliable and well-validated. In response to these and other findings, some have urged researchers to revise or develop new causal frameworks that allow cultural, cohort, or period norms, or other influences, to modify how personality traits are expressed in health-relevant ways (H. S. Friedman, 2019; H. S. Friedman & Kern, 2014; H. S. Friedman, Kern, Hampson, & Duckworth, 2014; Shanahan, Hill, Roberts, Eccles, & Friedman, 2014). These models therefore allow that neuroticism may be related to health-promoting behaviors in some contexts, health-harming behaviors in other contexts, and health-neutral behaviors in still other contexts. These models also allow for the possibility that contextual factors may amplify or dampen associations between neuroticism and health.

We conducted a large epidemiological study that sought to determine why neuroticism was sometimes related to lower mortality. We found that, although the total neuroticism score was related to greater mortality, a component of neuroticism that captured worry and vulnerability was related to lower mortality (Gale et al., 2017). Follow-up studies involving other health outcomes (Weiss et al., 2019) and molecular genetic data (Hill et al., 2019) supported this possibility.

Factors and Facets of Personality

Personality traits are made up of lower-order facets that share variance in common, but also have what has been called unique, specific, or unshared variance (Costa & McCrae, 1995). For example, the Revised NEO Personality Inventory's (NEO-PI-R) neuroticism factor comprises anxiety, angry-hostility, depression, self-consciousness, impulsivity, and vulnerability. These facets correlate between .31 and .64 (median r = .47) with one another (Costa & McCrae, 1992, pp. 100-101).

The multifaceted nature of neuroticism has two consequences (see Table 1). First, although there are few ways that people can be very low or very high in neuroticism (they would have to be very high or very low on *all* facets), as one moves away from these extremes, there are *many* ways that people can get the same score. Second, for any outcome, two people with the same neuroticism score could be differently at risk and two people with different neuroticism scores could be equally at risk. This is because the size and direction of neuroticism-health associations depends on how a trait's facets are related to the outcome. Moreover, if an outcome is related to a particular facet and that facet is not captured by a particular scale, then by using that scale, researchers may not find an association between neuroticism and the outcome.

Testing whether different neuroticism facets have different associations with health sounds simple: just test the relationship between each facet and the health outcome. However, because facets are correlated, if one used this approach and found a significant association with one or more facets, it is not clear what these findings would mean. The associations could mean that the facet or facets were related to the outcome. However, because facet scores contain common neuroticism variance (they are all correlated), these findings could mean that the general neuroticism factor was related to the outcome. Also, because the strength of the association between each facet and the general neuroticism factor varies, if we find an association between health and one, but not another, neuroticism facet, this might be

Table 1

Illustration of How the Existence of Personality Facets Can Affect Causal Inference

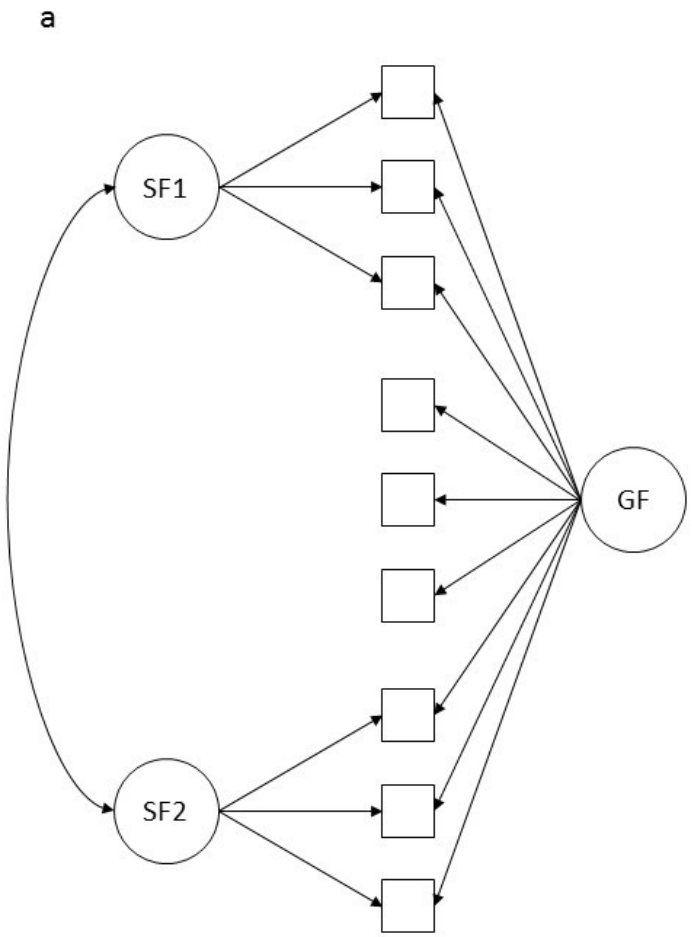
Participant	Anxiety	Angry Hostility	Depression	Self-Consciousness	Impulsiveness	Vulnerability	Neuroticism	Risk
1	15	20	10	21	25	10	101	Highest
2	25	13	12	12	22	17	101	High
3	17	16	18	14	15	21	101	Medium
4	24	15	17	11	9	25	101	Low
5	0	16	18	5	32	30	101	Lowest
6	11	11	19	11	0	30	82	Lowest
7	27	6	7	19	8	30	97	Lowest
8	14	21	4	10	0	30	79	Lowest
9	7	6	10	20	15	30	88	Lowest
10	13	14	27	22	28	30	134	Lowest

Note. This table presents raw scores on the six neuroticism facets and their raw neuroticism domain scores for 10 fictional participants who completed the NEO-PI-R (Costa & McCrae, 1992b). To compute the raw neuroticism domain score requires summing the scores for the six raw facet scores, each of which can ranges from 0 to 32 (Costa & McCrae, 1992b). A raw domain score of 100.3 on the self-report form of this instrument corresponds to one standard deviation above the mean, i.e., being high in neuroticism, based on combined gender norms in adults (Costa & McCrae, 1992b, p. 75). The first five rows show how different personality profiles at the facet level can all lead to the same high neuroticism score and how, if risk of a particular poor health outcome is associated with one facet (low scores on vulnerability in this instance), then the risk for each of these participants with the same neuroticism score will differ. The last five rows show that individuals with different neuroticism scores may have the same risk for poor health if they all have the same vulnerability score.

because the facet related to health is more closely related to the general neuroticism factor. In other words, to examine the role of facets, we must focus on the unique facet variance as it will not be drenched in general factor variance (Jang, McCrae, Angleitner, Riemann, & Livesley, 1998; Seltzer, Ones, & Tatar, 2017).

To examine the associations between personality facets and health or other outcomes, it is often best to use exploratory bifactor analyses (Wiernik, Wilmot, & Kostal, 2015). This statistical procedure distributes variance from a set of variables into a smaller number of variables (factors). Specifically, the variance for items is split into that which is related to a 'general factor' and that which is related to 'special factors' (see Figure 1a). The general factor, which is what one commonly refers to as 'neuroticism', consists of variation shared by all the items. The special factors are made up of the variation that are unique to a subset of items. As such, although special factors might be correlated with one another, as they are in the figure, they are statistically independent of the general neuroticism factor; they have a zero correlation with neuroticism as we commonly refer to it.

Exploratory factor analysis differs from exploratory bifactor analysis in how it distributes the variance from the items. In exploratory factor analysis, the variance of the items is split into one or more factors, each representing the common variance shared by a subset of items (see Figure 1b). If multiple factors are extracted, none of these factors will be a general factor, and the degree to which there is a general factor will be reflected in correlations among these factors. That is, if one conducted a further exploratory factor analysis on these factors, as is shown in the figure, a general factor would emerge; however, this general factor would be correlated with the lower-order factors, which is not the case in bifactor analysis. It thus follows that, if a general factor is present in a set of personality items, choosing to use a bifactor analysis or factor analysis has consequences for the meaning of the results. This is because, once again, in bifactor analysis, the scores relating to



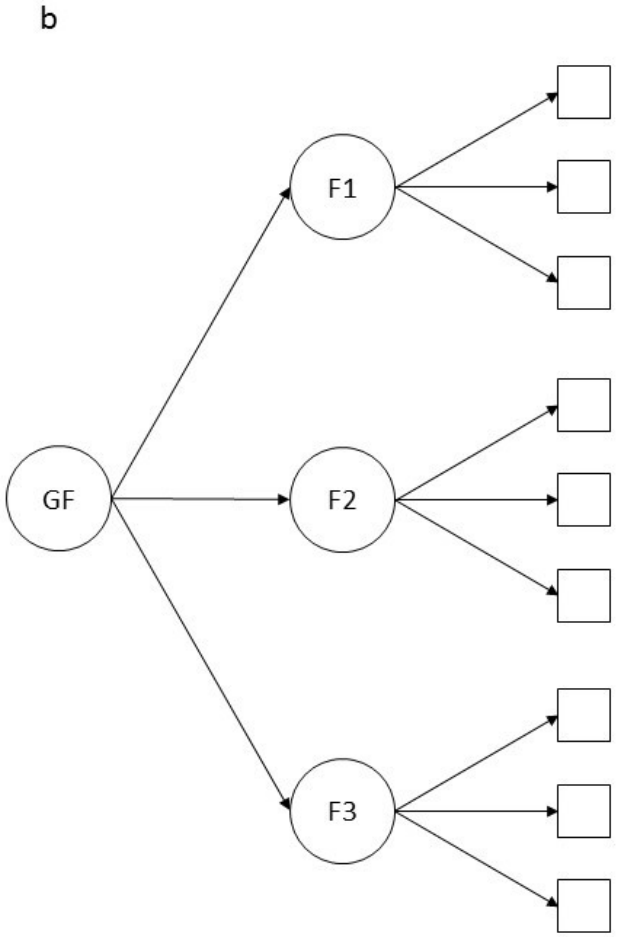


Fig 1. Rotation in exploratory bifactor analysis (a) enables one factor, the general factor (here, GF), to load on all items, and attempts to achieve a set of loadings (depicted by arrows from circles to boxes) for special factors (here, SF1 and SF2) in which each 'loads' on a discrete set of items (the boxes). Rotation in an exploratory factor analysis (b) attempts to achieve a set of loadings, in which each factor (here, F1, F2, and F3) loads on a discrete set of items (the boxes). Double-headed arrows indicate correlations

between factors. For simplicity, we did not depict residual variances. It is important to appreciate that, in the exploratory bifactor analysis, the general factor has a correlation of zero with the special factors. This is not the case in the exploratory factor analysis, in which the general factor is correlated with all the factors.

facets do not overlap with the general factor. On the other hand, in exploratory factor analysis, the facet scores would be correlated with the general factor.

Neuroticism, Special Factors of Neuroticism, and Health

Our first two studies tested whether facets of neuroticism had differential effects on health by analyzing data on 321,456 participants of UK Biobank (Sudlow et al., 2015). In our initial study (Gale, et al., 2017), we sought special factors, i.e., factors found in addition to, and uncorrelated with, a general neuroticism factor, in the 12-item neuroticism scale from the short-scale EPQ-R (Eysenck, et al., 1985). We found two (see Table 2). Based on the items that defined the first, e.g., "Would you call yourself a nervous person?", we named it "anxious/tense". Based on the items that defined the second, e.g., "Are you a worrier?" and "Are your feelings easily hurt?", we named it "worried/vulnerable". Although these special factors accounted for a small amount of variance, we later found them in two independent data sets (Weiss, et al., 2019). Next, we tested whether either or both special factors were related to death from all causes and death from cancer, cardiovascular disease, respiratory disease, and external (e.g., automobile accidents) causes (see Table 3). People with higher anxious/tense scores did not experience significantly greater or reduced mortality; people with higher worried/vulnerable scores were at reduced risk, and some associations were still significant when adjusting for health covariates and after correction for multiple tests.

In a second study, using the same participants, we investigated the relationships between the general neuroticism factor, the special neuroticism factors, and 16 health-related variables (Weiss, et al., 2019). Focusing on the analyses in which we examined associations of these covariates with neuroticism's general factor and the two special factors separately, one sees a striking pattern (see Table 4). For many of these variables, people who scored higher on the general factor of neuroticism showed a profile related to poorer health, whereas people who scored higher on worried/vulnerable showed a profile related to better health.

Table 2

Bifactor Structure of the 12 Items from the Short-Scale Eysenck Personality Questionnaire-Revised (N = 321,456)

		Special factors	
Item	General factor	Anxious/tense	Worried/vulnerable
Would you call yourself a nervous person?	**0.463**	**0.608**	0.026
Do you suffer from 'nerves'?	**0.439**	**0.490**	-0.028
Would you call yourself tense or 'highly strung'?	**0.481**	**0.352**	-0.020
Do you often feel 'fed-up'?	**0.708**	-0.158	-0.018
Do you ever feel 'just miserable' for no reason?	**0.673**	-0.154	0.027
Does your mood often go up and down?	**0.736**	-0.130	-0.047
Do you worry too long after an embarrassing experience?	**0.403**	0.009	**0.568**
Are your feelings easily hurt?	**0.458**	-0.024	**0.399**
Are you often troubled about feelings of guilt?	**0.450**	-0.014	**0.315**
Are you a worrier?	**0.481**	0.161	**0.309**
Do you often feel lonely?	**0.452**	-0.027	0.057
Are you an irritable person?	**0.492**	0.027	-0.043

Note. The bifactor structure was achieved by a bi-geomin rotation (Jennrich & Bentler, 2012) of three factors extracted from the 12 items. The first factor (the general factor) had substantial loadings on all of the items. The remaining two factors (the special factors) had substantial loadings on a subset of items. Note that the special factors are uncorrelated with the general factor. This rotation allowed the two special factors to be correlated, and revealed a medium-sized correlation between them, r =.312. Several indicators of how well this model fit the data were computed. The Root Mean Squared Error of Approximation (RMSEA) = 0.048, 90% confidence interval = [0.047 to 0.048], probability RMSEA ≤ .05 > .999; Comparative Fit Index = 0.975; Tucker-Lewis Fit Index =0.950; Standardized Root Mean Square Residual = 0.019. Factor loadings in boldface are salient, i.e., ≥ |0.3|. The values of these indicators show that the fit of the model to the data was excellent. Table adapted from Table S1 in Weiss, et al. (2019).

Table 3

Hazard Ratios for Mortality Risk and Each Standard Deviation Increase in Anxious/Tense and Worried/Vulnerable, Estimated Simultaneously (N = 321,456)

Cause of death and adjustments	Anxious/tense	Worried/vulnerable
All causes (n = 4497)		
Age, sex, and Neuroticism factor	1.00 [0.98, 1.03]	**0.88 [0.86, 0.91]**
All covariates	0.99 [0.96, 1.03]	**0.94 [0.90, 0.97]**
Cancer (n = 2912)		
Age, sex, and Neuroticism factor	0.96 [0.92, 1.00]	**0.93 [0.89, 0.97]**
All covariates	0.96 [0.92, 1.00]	0.97 [0.92, 1.01]
Cardiovascular disease (n = 925)		
Age, sex, and Neuroticism factor	1.00 [0.93, 1.07]	**0.84 [0.78, 0.91]**
All covariates	0.99 [0.92, 1.06]	0.93 [0.86, 1.01]
Respiratory disease (n = 688)		
Age, sex, and Neuroticism factor	1.02 [0.94, 1.11]	**0.84 [0.77, 0.91]**
All covariates	0.98 [0.90, 1.06]	0.93 [0.85, 1.01]
External causes (n = 422)		
Age, sex, and Neuroticism factor	1.03 [0.90, 1.13]	0.92 [0.82, 1.02]
All covariates	1.00 [0.90, 1.10]	0.97 [0.87, 1.08]

Note. The size and direction of relationships, as indicated by hazard ratios and their 95% confidence intervals (in square brackets), between each of the special factors of neuroticism and death from all causes and death from four major causes. Effects were derived using proportional hazards survival analysis and are described in Table 4 of Gale, et al. (2017). The hazard ratios anxious/tense and worried/vulnerable were adjusted for each other, and also for age, sex, and neuroticism in one model, and for these variables and additional covariates related to health behaviors (smoking status, alcohol consumption, exercise behavior, consuming fruits and vegetables), physical characteristics (body mass index, the lungs' forced expiratory volume in one second, systolic blood pressure, and grip strength), reaction time, number of reported diagnosis of a set of illnesses (vascular or heart problems, diabetes, cancer, asthma, chronic lung disease, deep vein thrombosis, or pulmonary embolism at baseline), and indicators of socioeconomic status, including the Townsend index of deprivation (Townsend, Phillimore, & Beattie, 1988), and educational achievement. Estimates in boldface and italics were associations that were significant at $p < .001$ and survived adjustment for the false discovery rate (Benjamini & Hochberg, 1995).

Table 4

Relationships Between Health Outcomes and Each Standard Deviation Increase in Anxious/Tense and Worried/Vulnerable, Estimated Separately (N = 321,456)

	General factor	Anxious/tense	Worried/vulnerable	N vs. AT	N vs. WV	AT vs. WV
Logistic regression						
Current smoker	**1.27 [1.26, 1.29]**	**1.44 [1.41, 1.48]**	**0.77 [0.76, 0.79]**	S	D	D
Eats 5+ portions of fruit/vegetables daily	**0.90 [0.89, 0.91]**	1.00 [0.99, 1.01]	1.00 [0.99, 1.02]			
Drinks alcohol daily or almost daily	**0.98 [0.97, 0.99]**	**1.02 [1.01,1.03]**	**1.08 [1.07, 1.09]**	D	D	S
Vascular/heart problems	**1.22 [1.21, 1.23]**	**1.05 [1.04, 1.06]**	0.99 [0.98, 1.00]	S		
Diabetes	**1.21 [1.19, 1.23]**	**0.82 [0.81, 0.84]**	**0.81 [0.79, 0.83]**	D	D	S
Asthma	**1.14 [1.13, 1.15]**	1.02 [1.01, 1.04]	1.02 [1.01, 1.04]			
Chronic lung disease	**1.54 [1.49, 1.59]**	1.04 [0.99, 1.08]	**0.91 [0.87, 0.96]**		D	
Cancer	1.02 [1.01, 1.04]	0.99 [0.97, 1.01]	0.99 [0.97, 1.01]			
Deep vein thrombosis	**1.13 [1.10, 1.16]**	**0.91 [0.88, 0.94]**	**0.92 [0.88, 0.95]**	D	D	S
Pulmonary embolism	**1.14 [1.09, 1.19]**	**0.89 [0.84, 0.94]**	0.92 [0.87, 0.98]	D		
Linear regression						
Exercise taken	**-0.148 [-0.152, 0 .143]**	-0.004 [-0.005, 0.005]	**0.043 [0.042, 0.053]**		D	
Body mass index (kg/m^2)	**0.261 [0.243, 0.279]**	**-0.737 [-0.757, -0.712]**	**-0.560 [-0.582, -0.537]**	D	D	S
Systolic blood pressure (mm Hg)	**-0.421 [-0.487, -0.355]**	**0.422 [0.346, 0.498]**	**-0.500 [-0.584, -0.416]**	D	S	D
Grip strength (kg)	**-0.761 [-0.789, -0.734]**	**-0.411 [-0.443, -0.379]**	**-0.248 [-0.283, -0.213]**	S	S	S
Forced expiratory volume in 1s (l)	**-0.032 [-0.034, 0.030]**	-0.002 [-0.004, 0.001]	**0.023 [0.020, 0.026]**		D	
Reaction time (ms)	**4.72 [4.31, 5.13]**	**5.07 [4.59, 5.54]**	**0.937 [0.412, 1.470]**	S	S	S
Number of effects in different direction				6	8	2
Percent of effects in different direction				54.55%	72.73%	25.00%

Note. Adapted from Table S3 in Weiss, et al. (2019). Effect estimates for logistic regressions expressed in odds ratio per standard deviation of the general factor or special factors of neuroticism. Differences in the direction of significant effects between the general factor and anxious/tense (N vs. AT), between the general factor and

worried/vulnerable (N vs. WV), and between the two special factors (AT vs. WV) are noted in the last three columns. D = different direction, S = same direction. The last two columns representing the examination of the two special factors simultaneously are not depicted here.

In a third study (Hill, et al., 2019), a genome-wide association study of 270,059 UK Biobank participants, we determined the extent to which the general factor and two special factors were biologically and genetically distinct. We found that the general factor and the two special factors were all related to genetic variants in *different* regions of the genome. Moreover, although the genes underlying each factor were related to neurological and brain-based biology, and especially the brain's cortex, they were related to *different* parts and pathways of the brain. For example, genes underlying the general neuroticism factor were related to the creation of new neurons and the growth of dendrites, but genes underlying the worried/vulnerable special factor were related to high voltage gated calcium channel activity and the voltage gated calcium channel complex.

In this same study, we also examined, at the genetic level, the relationships between health outcomes, on the one hand, and, on the other hand, our neuroticism variables. To do so we computed genetic correlations between the neuroticism variables and the health variables. A genetic correlation indicates the degree to which the same genes are related to two measured variables (see Figure 2). We found four striking patterns of genetic correlations (see Table 5). First, as in our non-genetic studies (Gale, et al., 2017; Weiss, et al., 2019), the directions of the relationships between physical health and health behaviors, on the one hand, and the general neuroticism factor and the special factors, on the other, often were in opposite directions. The general and special factors were genetically related to poorer and better health outcomes, respectively. This finding is important because it is consistent with findings in our earlier, non-genetic studies. Second, with one exception, the relationships between the neuroticism variables and mental health were in the same direction, that is, greater risk. This finding is important because it is what one would expect to find if these findings were not some artifact. The sole exception, by the way, was for attention deficit hyperactivity disorder where the special factors were related to reduced risk and the general factor was related to

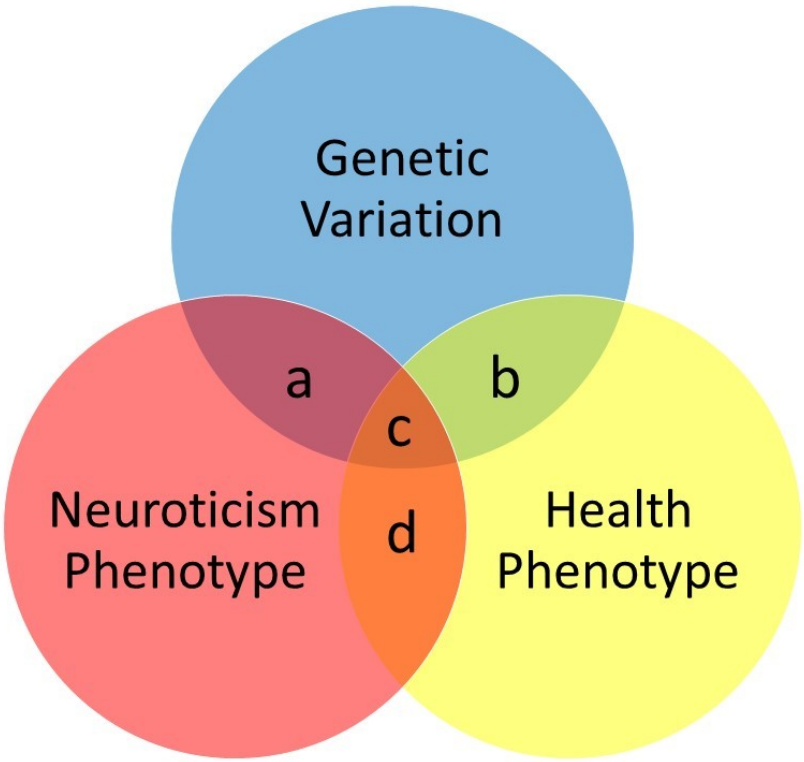


Fig 2. A genetic correlation indicates the degree to which the same genes are related to two measured variables. For any pair of heritable traits, that is, traits in which some proportion of the variation can be explained by genes, it is possible to estimate a genetic correlation. In the example above, some variation in the neuroticism phenotype (areas a and c) and the health phenotype (areas b and c), is heritable. The phenotypic correlation between these traits, that is, what one would obtain by computing a Pearson correlation coefficient (r), is represented by areas c and d. The genetic correlation (r_g) is the proportion of the phenotypic correlation attributable to genetic effects, and is represented by area c. There are multiple ways to estimate genetic correlations. For example, one can do so in a twin study by comparing the size of correlations between variables A and B in monozygotic twins to cross-twin correlations between these variables in dizygotic twins. A cross-twin correlation is simply the correlation between variable A in one twin and variable B in his or her co-twin, and vice versa. If the two variables are genetically correlated, we would find that the cross-twin correlations are greater in monozygotic twins, who share all of their genes in common, than in dizygotic twins, who share, on average, half of their genes in common. If molecular genetic data are available, one can estimate genetic correlations directly by determining the proportion of genes for one trait that are genes for the other trait.

Table 5

Genetic Correlations Between Health Outcomes and the General Factor or Neuroticism, the Anxious/Tense Facet, and the Worried/Vulnerable Facet (N = 270,059)

	General factor	Anxious/tense	Worried/vulnerable	N vs. AT	N vs. WV	AT vs. WV
Longevity						
Parents age at death	*-0.16*	***0.16***	***0.21***			S
Longevity	***-0.18***	0.06	0.08			
Mental health						
Bipolar disorder	0.07	***0.17***	***0.10***			S
Schizophrenia	***0.16***	***0.28***	***0.17***	S	S	S
Major depressive disorder	***0.68***	***0.21***	***0.19***	S	S	S
Anorexia nervosa	***0.14***	***0.20***	***0.22***	S	S	S
Attention-deficit hyperactivity disorder	***0.30***	***-0.26***	***-0.22***	D	D	S
Autism spectrum disorder	0.12	0.09	***0.16***			
Cardiovascular						
Coronary artery disease	***0.10***	***-0.15***	***-0.11***	D	D	S
Diastolic blood pressure	0.01	***0.19***	-0.02			
Systolic blood pressure	-0.04	***0.22***	-0.01			
Metabolic						
Type 2 diabetes	-0.04	***-0.15***	*-0.09*			
Obesity	0.02	***-0.41***	***-0.22***			S
Health and well-being						
Alzheimer's disease[a]	*0.11*	0.03	-0.03			

Self-rated health	***-0.48***	***0.15***	***0.15***	D	D	S
Smoking (ever vs. never)	***0.16***	***-0.26***	***-0.21***	D	D	S
Alcohol intake	***-0.11***	0.06	***0.09***		D	
Tiredness	***0.67***	-0.04	0.08			
Forced expiratory volume in one second	-0.03	0.07	0.05			
Subjective well-being	***-0.68***	***-0.16***	***-0.27***	S	S	S
Anthropometric						
Body mass index	0.04	***-0.38***	***-0.19***			S
Chronotype	-0.05	-0.03	-0.04			
Sleep duration	***-0.15***	0.05	-0.03			
Reproductive						
Age at first birth	***-0.26***	***0.28***	***0.25***	D	D	S
Number of children	***0.11***	***-0.26***	***-0.12***	D	D	S
Number of effects in different direction				6	7	0
Percent of effects in different direction				60.00%	63.64%	0.00%

Note. Table adapted from Hill, et al. (2019). Correlations indicate the overlap between the genetic variance relating to the neuroticism variables and the other variables. Estimate in italics were significant at $p < .001$ and estimates in boldface and italics survived adjusting for the false discovery rate (Benjamini & Hochberg, 1995). Differences in the direction of significant effects between the general factor and anxious/tense (N vs. AT), between the general factor and worried/vulnerable (N vs. WV), and between the two special factors (AT vs. WV) are noted in the last three columns. D = different direction, S = same direction. [a] In the original paper, Hill, et al. (2019) included two sets of genetic variants for Alzheimer's disease. The genetic correlations between the three neuroticism variables and the two sets of genetic variants for Alzheimer's disease were close to identical.

increased risk. Third, higher blood pressure was solely genetically related to the anxious/tense factor. This was important because it was evidence for convergent and discriminant validity. Fourth, the genetic correlations between the general factor and major depressive disorder, subjective well-being, self-rated health, and tiredness, were much larger than the comparable correlations of these health variables and the special factors.

Conclusions

The findings described here represent key results from our recent work on the special factors of neuroticism (additional results can be found in Gale, et al., 2017; Hill, et al., 2019; Weiss, et al., 2019). These findings, we would argue, indicate that there may be a need to refine how we think about the relationships between neuroticism and health. Specifically, the importance of examining variance specific to neuroticism facets, especially related to worry and vulnerability, adds a new wrinkle to causal models. The special neuroticism factor, whose items relate to being worried and feeling vulnerable, for example, may be related to better health because it promotes health vigilance. This explanation is consistent with a recent study in which the authors found that the neuroticism variation that overlapped with "bodily vigilance", which was measured by questions such as "How much do you wonder about why your body feels the way it does?", was associated with better health, and that the other neuroticism variance was related to poorer health (Weston & Jackson, 2018). It is also consistent with earlier reports that people high in neuroticism tend to report symptoms more frequently even if there is no underlying disease (Costa & McCrae, 1987) and seek medical attention more often (B. Friedman, Veazie, Chapman, Manning, & Duberstein, 2013), and so these individuals may be more likely to be diagnosed and treated at earlier stages of a disease process (H. S. Friedman, 2019). Further work in this direction will bring us closer to understanding the causal framework undergirding personality and health, and neuroticism itself.

As a medical or psychological professional, or a friend, loved one, or acquaintance, we often tell others not to worry so much. Health-wise, we have found phenotypic and genetic evidence for an upside to worry.

Acknowledgements

The work was done within the Centre for Cognitive Ageing and Cognitive Epidemiology, which is funded by the UK's Medical Research Council and Biotechnology and Biological Sciences Research Council (MR/K026992/1).

References

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society: Series B (Methodological), 57*, 289-300. http://dx.doi.org/10.2307/2346101

Bouchard, T. J., Jr., & Loehlin, J. C. (2001). Genes, evolution, and personality. *Behavior Genetics, 31*, 243-273. http://dx.doi.org/10.1023/A:1012294324713

Costa, P. T., Jr., & McCrae, R. R. (1987). Neuroticism, somatic complaints, and disease: Is the bark worse than the bite? *Journal of Personality, 55*, 299-316. http://dx.doi.org/10.1111/j.1467-6494.1987.tb00438.x

Costa, P. T., Jr., & McCrae, R. R. (1992a). Four ways five factors are basic. *Personality and Individual Differences, 13*, 653-665. http://dx.doi.org/10.1016/0191-8869(92)90236-I

Costa, P. T., Jr., & McCrae, R. R. (1992b). *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO-FFI) professional manual*. Odessa, FL: Psychological Assessment Resources.

Costa, P. T., Jr., & McCrae, R. R. (1995). Domains and facets: Hierarchical personality assessment using the Revised NEO Personality Inventory. *Journal of Personality Assessment, 64*, 21-50. http://dx.doi.org/10.1207/s15327752jpa6401_2

Čukić, I., & Weiss, A. (2014). Personality and diabetes mellitus incidence in a national sample. *Journal of Psychosomatic Research, 77*, 163-168. http://dx.doi.org/10.1016/j.jpsychores.2014.07.004

Deary, I. J., Weiss, A., & Batty, G. D. (2010). Intelligence and personality as predictors of illness and death: How researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities.

Psychological Science in the Public Interest, 11, 53-79. http://dx.doi.org/10.1177/1529100610387081

Eysenck, S. B. G., Eysenck, H. J., & Barrett, P. (1985). A revised version of the psychoticism scale. *Personality and Individual Differences, 6*, 21-29. http://dx.doi.org/10.1016/0191-8869(85)90026-1

Friedman, B., Veazie, P. J., Chapman, B. P., Manning, W. G., & Duberstein, P. R. (2013). Is personality associated with health care use by older adults? *Milbank Memorial Fund Quarterly, 91*, 491-527. http://dx.doi.org/10.1111/1468-0009.12024

Friedman, H. S. (2019). Neuroticism and health as individuals age. *Personality Disorders: Theory, Research, and Treatment, 10*, 25-32. http://dx.doi.org/10.1037/per0000274

Friedman, H. S., & Kern, M. L. (2014). Personality, well-being, and health. *Annual Review of Psychology, 65*, 719-742. http://dx.doi.org/10.1146/annurev-psych-010213-115123

Friedman, H. S., Kern, M. L., Hampson, S. E., & Duckworth, A. L. (2014). A new life-span approach to conscientiousness and health: combining the pieces of the causal puzzle. *Developmental Psychology, 50*, 1377-1389. http://dx.doi.org/10.1037/a0030373

Gale, C. R., Čukić, I., Batty, G. D., McIntosh, A. M., Weiss, A., & Deary, I. J. (2017). When is higher neuroticism protective against death? Findings from UK Biobank. *Psychological Science, 28*, 1345-1357. http://dx.doi.org/10.1177/0956797617709813

Hill, W. D., Weiss, A., Liewald, D., Davies, G., Porteous, D. J., Hayward, C., . . . Deary, I. J. (2019). Genetic contributions to two special factors of neuroticism are associated with affluence, higher intelligence, better health, and longer life. *Molecular Psychiatry*. http://dx.doi.org/10.1038/s41380-019-0387-3

Jang, K. L., McCrae, R. R., Angleitner, A., Riemann, R., & Livesley, W. J. (1998). Heritability of facet-level traits in a cross-cultural twin sample: Support for a

hierarchical model of personality. *Journal of Personality and Social Psychology, 74*, 1556-1565. http://dx.doi.org/10.1037/0022-3514.74.6.1556

Jennrich, R. I., & Bentler, P. M. (2012). Exploratory bi-factor analysis: The oblique case. *Psychometrika, 77*, 442-454. http://dx.doi.org/10.1007/s11336-012-9269-1

Korten, A. E., Jorm, A. F., Jiao, Z., Letenneur, L., Jacomb, P. A., Henderson, A. S., . . . Rodgers, B. (1999). Health, cognitive, and psychosocial factors as predictors of mortality in an elderly community sample. *Journal of Epidemiology and Community Health, 53*, 83-88. http://dx.doi.org/10.1136/jech.53.2.83

Lahey, B. B. (2009). Public health significance of neuroticism. *The American psychologist, 64*, 241-256. http://dx.doi.org/10.1037/a0015309

McCrae, R. R., Costa, P. T., Jr., & Martin, T. A. (2005). The NEO-PI-3: A more readable Revised NEO Personality Inventory. *Journal of Personality Assessment, 84*, 261-270. http://dx.doi.org/10.1207/s15327752jpa8403_05

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin, 126*, 3-25. http://dx.doi.org/10.1037/0033-2909.126.1.3

Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science, 2*, 313-345. http://dx.doi.org/10.1111/j.1745-6916.2007.00047.x

Seltzer, B. K., Ones, D. S., & Tatar, A. (2017). Using personality facets to understand the nature of personality-satisfaction relationships: Findings from meta-analytic bifactor latent modeling. *Career Development International, 22*, 477-506. http://dx.doi.org/10.1108/CDI-08-2017-0141

Shanahan, M. J., Hill, P. L., Roberts, B. W., Eccles, J., & Friedman, H. S. (2014). Conscientiousness, health, and aging: the life course of personality model. *Developmental Psychology, 50*, 1407-1425. http://dx.doi.org/10.1037/a0031130

Strickhouser, J. E., Zell, E., & Krizan, Z. (2017). Does personality predict health and well-being? A metasynthesis. *Health Psychology, 36*, 797-810. http://dx.doi.org/10.1037/hea0000475

Sudlow, C., Gallacher, J., Allen, N., Beral, V., Burton, P., Danesh, J., . . . Collins, R. (2015). UK Biobank: An open access resource for identifying the causes of a wide range of complex diseases of middle and old age. *PLoS Medicine, 12*, e1001779. http://dx.doi.org/10.1371/journal.pmed.1001779

Terracciano, A., Sutin, A. R., An, Y., O'Brien, R. J., Ferrucci, L., Zonderman, A. B., & Resnick, S. M. (2014). Personality and risk of Alzheimer's disease: New data and meta-analysis. *Alzheimer's & Dementia, 10*, 179-186. http://dx.doi.org/10.1016/j.jalz.2013.03.002

Townsend, P., Phillimore, P., & Beattie, A. (1988). *Health and deprivation: Inequality and the North*. Beckenham, England: Croom Helm.

Turiano, N. A., Mroczek, D. K., Moynihan, J., & Chapman, B. P. (2013). Big 5 personality traits and interleukin-6: Evidence for “healthy Neuroticism” in a US population sample. *Brain, Behavior, and Immunity, 28*, 83-89. http://dx.doi.org/10.1016/j.bbi.2012.10.020

Weiss, A., Gale, C. R., Čukić, I., Batty, G. D., McIntosh, A. M., & Deary, I. J. (2019). Conditioning on a collider may or may not explain the relationship between lower neuroticism and premature mortality in the study by Gale et al. (2017): a reply to Richardson, Davey Smith, and Munafò (2019). *Psychological Science, 30*, 633-638. http://dx.doi.org/10.1177/0956797619833325

Weston, S. J., & Jackson, J. J. (2018). The role of vigilance in the relationship between neuroticism and health: a registered report. *Journal of Research in Personality, 73*, 27-34. http://dx.doi.org/10.1016/j.jrp.2017.10.005

Wiernik, B. M., Wilmot, M. P., & Kostal, J. W. (2015). How data analysis can dominate interpretations of dominant general factors. *Industrial and Organizational Psychology, 8*, 438-445. http://dx.doi.org/10.1017/iop.2015.60

Recommended Readings

Costa, P. T., Jr., & McCrae, R. R. (1995). Domains and facets: Hierarchical personality assessment using the Revised NEO Personality Inventory. *Journal of Personality Assessment, 64*, 21-50. doi:10.1207/s15327752jpa6401_2

- An overview and discussion of personality facets, including their measurement, their relationships to domains and one another, and of their common and unique variance.

Deary, I. J., Harris, S. E., & Hill, W. D. (2019). What genome-wide association studies reveal about the association between intelligence and physical health, illness, and mortality. *Current Opinion in Psychology, 27*, 6-12. doi:10.1016/j.copsyc.2018.07.005

- A useful review of work, including a discussion of the methods used, in the burgeoning literature on the genetic correlations between individual differences variables (cognitive ability in this case) and health outcomes.

Deary, I. J., Weiss, A., & Batty, G. D. (2010). Intelligence and personality as predictors of illness and death: How researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities. *Psychological Science in the Public Interest, 11*, 53-79. doi:10.1177/1529100610387081

- An in-depth, comprhensive, and clear review of the literature on the associations between intelligence and personality and health.

Reise, S. P., Moore, T. M., & Haviland, M. G. (2010). Bifactor models and rotations: exploring the extent to which multidimensional data yield univocal scale scores. *Journal of Personality Assessment, 92*, 544-559. doi:10.1080/00223891.2010.496477

- A detailed discussion of and tutorial on bifactor analysis. This paper focuses mainly on confirmatory bifactor analyses, but exploratory methods are discussed, if briefly.

Wiernik, B. M., Wilmot, M. P., & Kostal, J. W. (2015). How data analysis can dominate interpretations of dominant general factors. *Industrial and Organizational Psychology, 8*, 438-445. doi:10.1017/iop.2015.60

- An easy to follow discussion and demonstration of the problem of using sum scores to investigate lower-order constructs when there is a large general factor, and how bifactor analyses is a solution to this problem.