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The glass is half full *and* half empty: A population-representative twin study testing if optimism and pessimism are distinct systems

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Optimism and pessimism are associated with important outcomes including health and depression. Yet it is unclear if these apparent polar opposites form a single dimension or reflect two distinct systems. The extent to which personality accounts for differences in optimism/pessimism is also controversial. Here, we addressed these questions in a genetically informative sample of 852 pairs of twins. Distinct genetic influences on optimism and pessimism were found. Significant family-level environment effects also emerged, accounting for much of the negative relationship between optimism and pessimism, as well as a link to neuroticism. A general positive genetics factor exerted significant links among both personality and life-orientation traits. Both optimism bias and pessimism also showed genetic variance distinct from all effects of personality and from each other.

Keywords: optimism; pessimism; behavior genetics; twins; personality; Big Five

Introduction

> Prepare for the worst – and enjoy every moment of it. (Hadfield, 2013)

Optimism/pessimism impacts on accomplishment, health, and well-being (Carver, Scheier, & Segerstrom, 2010; Forgeard & Seligman, 2012), and an emerging literature has begun to describe possible neural substrates that might support these important life orientations (Sharot, Guitart-Masip, Korn, Chowdhury, & Dolan, 2012). Despite this, several important questions about the nature of optimism/pessimism are currently unresolved (Carver et al., 2010). Among these, it is unclear if optimism and pessimism are opposite ends of one dimension (Rauch, Schweizer, & Moosbrugger, 2007) or reflect two (or more) distinct systems (Herzberg, Glaesmer, & Hoyer, 2006). Second, the extent to which personality adequately explains optimism/pessimism is unclear (Sharpe, Martin, & Roth, 2011). Third, our understanding of the role of family environment, of genetics, and of unique life experiences in developing optimism and reducing pessimism is in its infancy. Here, we address these questions using a large representative sample of adult twins characterized on both personality and optimism and pessimism.

Dispositional optimism and pessimism (Scheier & Carver, 1987) are typically assessed by asking people whether they expect future outcomes to be beneficial or negative (Scheier & Carver, 1992). The most common measure is the revised Life Orientation Test (LOT-R: Scheier, Carver, & Bridges, 1994) consisting of 6 items each scored on a 5-point scale from 'Strongly disagree' to 'Strongly agree'. Example content includes 'I'm always optimistic about my future' and 'I hardly ever expect things to go my way'. The LOT returns separate optimism and pessimism scores for each individual. These overlap somewhat 'phenotypically'. This is simply the term used in genetics to describe manifested development: The observable characteristics of an individual resulting from the interaction of its genotype with the environment. In the present study, optimism and pessimism shared 22% of their phenotypic variance. Optimistic scores on this scale predict better outcomes in relationships (House, Landis, & Umberson, 1988), higher social status (Lorant et al., 2007), and reduced loss of well-being following adversity (Carver & Scheier, 1998). Health-preserving behaviors are associated with optimism, while health-damaging behaviors are associated with pessimism (Hooker, Monahan, Shifren, & Hutchinson, 1992).

At the genetic level, very little research has been reported to date. Plomin et al. (1992) reported the heritability of the LOT at .24 for optimism and .29 for pessimism, with a likely effect of shared environment on optimism (.13) but not pessimism. Mosing, Zietsch, Shekar, Wright, and Martin (2009) reported heritability for aggregate optimism/pessimism as .36 and also supported modest biologically mediated links between optimism and mental physical health, with 'genes predisposing to high optimism also predispose to good mental health and self-rated health'. Plomin et al. (1992) also found that optimism and pessimism contributed differently to the

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prediction of other outcomes: For instance, pessimism was genetically correlated with depression, whereas the genetic association of optimism with depression was nonsignificant. Currently, then, it is unknown whether one genetic trait underlies optimism and pessimism, or if these psychological processes reflect two or even more genetically distinct systems.

Behavioral studies of whether optimism and pessimism are best understood as a single factor or as weakly correlated systems with distinct etiology and correlates have yielded mixed results. Studies focused on predictive validity support retaining separate optimism and pessimism scales (Robinson-Whelen, Kim, MacCallum, & Kiecolt-Glaser, 1997). In addition, confirmatory modeling supports a two-dimensional measurement model (Herzberg et al., 2006). Yet others have suggested that this second dimension reflects only the social desirability of positively versus negatively worded items (Rauch et al., 2007). Pharmacological and imaging studies provide further insight into the complex origins of optimism and pessimism. This research implicates dopamine as creating a bias against pessimistic belief formation via blocking the formation of negative expectations about the future (Sharot, Guitart-Masip, et al., 2012). Anatomically, optimistic bias is associated with activation of the amygdala and rostral anterior cingulate cortex likely reflecting the integration of emotional information into autobiographical memory (Sharot, Riccardi, Raio, & Phelps, 2007). The finding that these areas are disregulated in depression provides further encouragement for understanding the biology of optimism/pessimism (Dayan & Huys, 2009). Finally, transcranial magnetic stimulation studies suggest that accurate belief formation incorporating bad news is normally blocked by interpretive functions implemented in the left inferior frontal gyrus (Sharot, Kanai, et al., 2012). The healthy ability to form accurate beliefs about risk emerges slowly over adolescence (Moutsiana et al., 2013), and a small study suggests that major depression may involve weakening of left inferior frontal gyrus activity which even in adulthood typically leaves belief formation biased toward good news (Korn, Sharot, Walter, Heekeren, & Dolan, 2014). While much work on belief formation and updating has focused on confirmation bias (Nickerson, 1998), recent studies of the accuracy and precision of processing and acquisition of valuable objective information indicate that updating is driven not by confirmation of expectations, but by whether information is 'good' or 'bad', with good news being preferentially encoded (Eil & Rao, 2011). These biological results, then, suggest genetic hypotheses about optimism and pessimism. They imply that optimism and pessimism reflect a complex, multi-componential system, likely to be reflected in complex genetic origins, which we test here.

Genetic links from personality to optimism/pessimism

Genetic analysis of the relationship of optimism–pessimism to the five-factor model of personality has not been undertaken previously. Behavior genetic studies using samples such as twins differing in zygosity or adoption designs gain the ability to fractionate apart normally confounded causes based in genes and environments. In a common design, used here, the ACE model (Neale & Maes, 1996) distinguishes additive genetic effects (termed A) from effects attributable to shared-environmental effects (termed C for common environmentality) and environmental effects which causes twins in a family to differ: termed E or unique environmental effects. Factors associated with a family, such as the home they live in, shared neighborhood factors, parental behaviors acting on all children in the family are among the kinds of factor typically linked to shared environment.

Given the biological complexity of optimism/pessimism, we might expect more than one personality dimension to be associated with optimism and/or pessimism. Behavioral studies attempting to incorporate optimism within the framework of personality have typically focused on the single dimension of neuroticism (Smith, Pope, Rhodewalt, & Poulton, 1989). These studies support the idea that optimism cannot be reduced to neuroticism or its facets, such as anxiety. For instance, Scheier et al. (1994) examined data in a large ($n = 4309$) student sample finding that optimism scores on the LOT had discriminant validity for the prediction of depression and at least some aspects of coping over and above measures of neuroticism, anxiety, self-mastery, and self-esteem. Incremental validity of optimism over neuroticism is not sufficient to distinguish optimism/pessimism from personality. Apart from the problem that incremental validity will arise for identical constructs whenever these constructs are measured with error (Aigner, Hsiao, Kapteyn, & Wansbeek, 1984), these analyses did not test the ability of the full five-factor model to account for optimism.

Studies that include a range of traits indicate relationships beyond neuroticism (Sharpe et al., 2011). For instance, optimism has been associated with higher subjective well-being (Diener, Oishi, & Lucas, 2003), suggesting that extraversion, neuroticism, and conscientiousness – personality traits which provide an affective buffer supporting higher well-being (Weiss, Bates, & Luciano, 2008) – may be related to optimism. Indeed, recent work has implicated not only neuroticism and extraversion but also conscientiousness and agreeableness in optimism (Sharpe et al., 2011). We therefore predicted genetic relationships from all personality domains barring openness to experience. We also tested if genetic influences from personality are sufficient to account completely for optimism/pessimism, and how personality inputs differ across optimism and pessimism.

Finally, genetic studies provide clear tests of the role of the environment. Leading theories of pessimism in particular attribute optimism/pessimism either to rearing or to repeated experience of uncontrollable negative events (Seligman, 2011). In the former case, we predict significant shared-environment effects. In the latter case, large unshared-environment effects are expected with negligible impact of genes. Supporting a role for rearing in optimism, adult optimism has been linked to greater parental warmth and financial security (Heinonen, Räikkönen, & Keltikangas-Järvinen, 2005). Such results, however, confound the roles of parenting and genetics, with both being transmitted. The present study, with genetic control, will be valuable in estimating the roles of shared and unshared environments, as well as of the genetic hypotheses outlined above.

Methods

Subjects

Subjects were all 939 female (mean age 54.13 years (SD = 11.95) and 763 male (mean age 54.02, SD = 11.4) twins who had completed the LOT-R and personality scales from among participants in Wave II of the MacArthur Foundation Survey for Midlife Development in the US, a nationally representative sample of households (Brim, Ryff, & Kessler, 2004). These comprised 153 male and 169 female MZ (identical) pairs, 115 male and 188 DZ female pairs, and 227 opposite sex pairs.

Measures

Optimism and pessimism were each assessed using the Life Orientation Test-Revised (Scheier et al., 1994). Each item was responded to on Likert-response scales anchored from 1 ('A lot agree') to 5 ('A lot disagree'). Example optimism and pessimism items include 'In uncertain times, I usually expect the best' and 'I rarely count on good things happening to me', respectively. For items with a missing value, the mean value of completed items was imputed. Only one missing item was allowed per scale. Both scales showed acceptable reliability (Cronbach's $\alpha = .70$ and .81, respectively). Personality was assessed using the Midlife Development Inventory (MIDI), and a self-administered 25-item personality questionnaire (Lachman & Weaver, 1997) was mailed to each participant. Our measures of personality were scores on the five previously defined MIDI scales (Lachman & Weaver, 1997). Each score was calculated by obtaining the average of the ratings for items defining that dimension: Neuroticism was defined by moody, worrying, nervous, and calm (reverse-scored); extraversion was defined by outgoing, friendly, lively, active, and talkative; openness to experience was defined by creative, imaginative, intelligent, curious, broad-minded, sophisticated, and adventurous; agreeableness was defined by helpful, caring, warm, soft-hearted, and sympathetic; and conscientiousness was defined by organized, responsible, hardworking, and careless (reverse-scored). Respondents used 4-point Likert scales to indicate the degree to which each adjective on the questionnaire described them. To preserve power, effects of gender were not analyzed separately, based on previous research on the LOT-R indicating negligible sex-differences on these traits (Scheier et al., 1994).

Results

All analyses were conducted in R (R Core Team, 2014). Table 1 shows the phenotypic correlations among optimism and pessimism and the five-factor model domains. Average sum-scores (and SDs) for optimism and pessimism were 11.82 (2.42) and 6.85 (3.42), respectively.

All subsequent analyses were conducted using the R package 'OpenMx' (Boker et al., 2011, 2013) and the umx helper library (Bates, 2014). To minimize bias and maximize usage of data, all analyses used full information maximum likelihood estimation (FIML). The base or saturated model was a two-group Cholesky decomposition (see Figure 1). Models are formed for identical (MZ) twins, and for DZ twins to capture the facts that MZ twins share approximately 100% of genetic factors, while DZ twins share on average half this amount (50%). Within each group, covariation between the twins is modeled in terms of genetic (A), and environment effects which are common (C) or unique (E) to each twin, yielding the

Table 1. Phenotypic correlations among the variables. 1-twin from each pair only included, reliabilities (Cronbach's alpha) on the diagonal.

	Neuroticism	Extraversion	Openness	Agreeableness	Conscientiousness	Optimism	Pessimism
Neuroticism	.75						
Extraversion	−.232	.76					
Openness	−.234	.551	.76				
Agreeableness	−.129	.461	.359	.80			
Conscientiousness	−.258	.298	.316	.253	.65		
Optimism	−.433	.423	.386	.256	.219	.7	
Pessimism	.385	−.284	−.250	−.092	−.282	−.468	.81

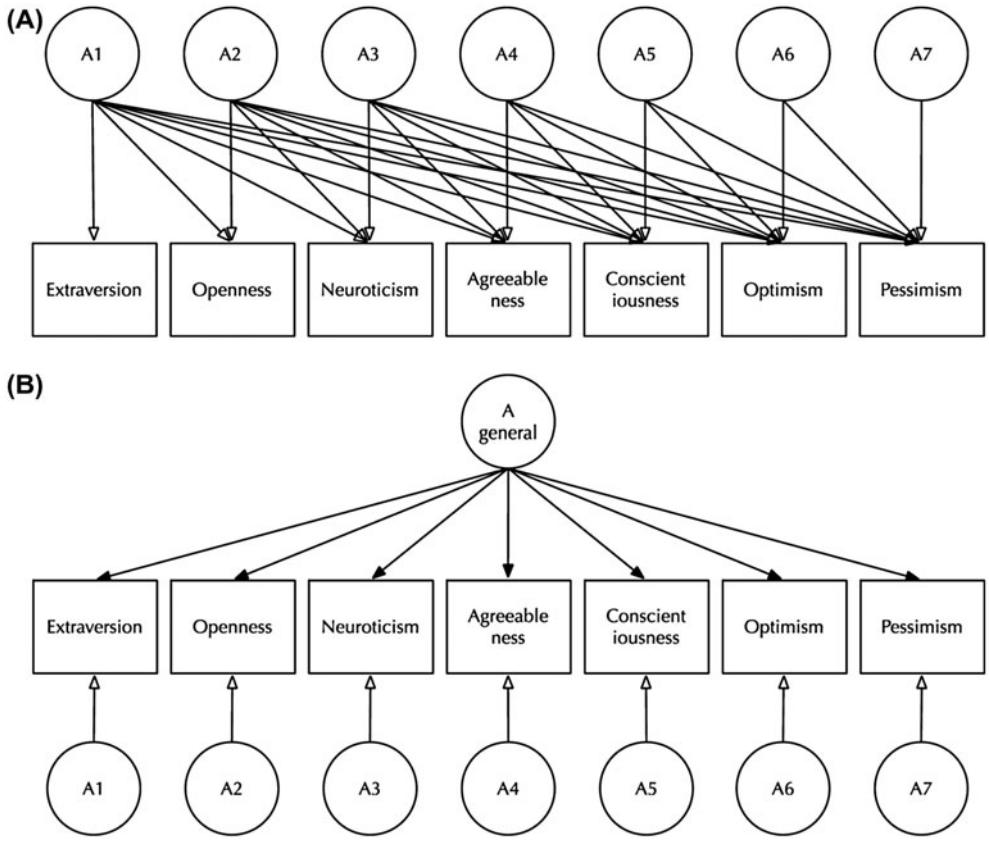

Figure 1. Cholesky (A) and independent pathway (B) models.
Notes: In the Cholesky model, each latent variable can load on all variables to beneath or to its right. This allows a saturated baseline model against which hypotheses can be tested. In the independent pathways model, general factors (1 in this case) loading on all variables are posited for each of A, C, and E. In addition, each measured variable is allowed to have unique variance components. For clarity, only the genetic paths are shown, but this model is duplicated for each of shared environment (C) and unique environment (E). The model is also duplicated for each twin, and in two groups: one for MZ and one for DZ twins.

classic 'ACE' model. Within the MZ group, twins share 100% of genetic effects, while in the DZ group, twin are modeled as sharing half this – or 50% of genetic effects. In both groups, shared environment correlates 1.0 and unique environment 0.0 by definition. Finally, in initial or saturated Cholesky decomposition, variance across the measured variables is broken down into as many A, C, and E latent effects as there are variables being modeled. This is done as 'lower triangle': with the first latent A, C, and E components able to load on all measured traits, while each subsequent latent A, C, or E variable picks up additional variance from each trait in sequence until the final variable has A, C, and E, components loading on it alone. This is a useful model in the present case, as it allows the researcher to ask, after taking into account the genetic and environmental influences on five personality traits, are their any additional effects required to account for optimism/pessimism? The answer to this question can be either affirmative, that is, optimism and/or pessimism require additional genetic factors to account for them, beyond those sufficient to account for personality, or in the negative, as, for instance, has been reported for subjective well-being where personality has been argued to provide a sufficient account of genetic variance in well-being (Weiss et al., 2008). All latent traits loading on personality in the base model, then, also loaded on optimism and pessimism: Thus, personality could (potentially) account for these two traits in part or in whole. Finally, six additional latent variables were added to allow for specific genetic, shared-environmental and unique environmental effects on optimism and pessimism: A total of seven latent variables for each of A, C, and E. To facilitate other researchers exploring these data, the covariance matrices for the MZ and DZ groups are provided in Tables 2 and 3. While the analyses presented here use FIML modeling on individual level data, the provided matrices (based on complete data) will yield very similar results.

To test the role of personality on optimism/pessimism, this saturated ACE model was first evaluated. The fit of this base model (model 1) is shown in Table 4.

Table 2. Covariances data for MZ twin pairs.

	N1	E1	O1	A1	C1	Opt1	Pess1	N2	E2	O2	A2	C2	Opt2	Pess2
N1	.473	−.094	−.113	−.063	−.056	−.997	.867	.223	−.048	−.066	−.022	−.009	−.551	.78
E1	−.094	.313	.136	.113	.083	.661	−.487	−.068	.119	.033	.060	.024	.243	−.39
O1	−.113	.136	.267	.066	.060	.535	−.515	−.042	.046	.097	.039	.011	.149	−.31
A1	−.063	.113	.066	.256	.043	.302	−.076	−.060	.084	.016	.106	.015	.300	−.21
C1	−.056	.083	.060	.043	.171	.241	−.385	−.017	.004	.008	.007	.049	−.009	−.19
Opt1	−.997	.661	.535	.302	.241	6.909	−4.164	−.432	.380	.308	.202	.074	2.409	−2.50
Pess1	.867	−.487	−.515	−.076	−.385	−4.164	10.769	.360	−.192	−.114	−.074	−.102	−1.867	4.30
N2	.223	−.068	−.042	−.060	−.017	−.432	.360	.427	−.085	−.077	−.038	−.028	−.857	.94
E2	−.048	.119	.046	.084	.004	.380	−.192	−.085	.281	.136	.122	.038	.453	−.40
O2	−.066	.033	.097	.016	.008	.308	−.114	−.077	.136	.283	.086	.063	.349	−.41
A2	−.022	.060	.039	.106	.007	.202	−.074	−.038	.122	.086	.231	.054	.320	−.28
C2	−.009	.024	.011	.015	.049	.074	−.102	−.028	.038	.063	.054	.134	.107	−.22
Opt2	−.551	.243	.149	.300	−.009	2.409	−1.867	−.857	.453	.349	.320	.107	5.633	−4.16
Pess2	.778	−.394	−.306	−.208	−.188	−2.497	4.302	.944	−.396	−.409	−.281	−.221	−4.162	9.55

Notes: N = Neuroticism; E = Extraversion; O = Openness; A = Agreeableness; C = Conscientiousness; Opt = LOT-R Optimism; Pess = LOT-R Pessimism. Suffixes '1' & '2' = twin 1 and twin 2.

Table 3. DZ covariances.

	N1	E1	O1	A1	C1	Opt1	Pess1	N2	E2	O2	A2	C2	Opt2	Pess2
N1	.338	−.074	−.071	−.039	−.054	−.523	.777	.036	.002	.030	.008	.004	−.179	.171
E1	−.074	.351	.171	.154	.072	.479	−.529	−.041	.041	.026	.022	−.007	.075	−.153
O1	−.071	.171	.276	.114	.072	.382	−.408	−.012	.030	.040	.022	.008	.094	−.217
A1	−.039	.154	.114	.274	.070	.282	−.233	−.040	.023	.002	.045	−.012	.008	−.106
C1	−.054	.072	.072	.070	.176	.234	−.454	−.004	.029	.036	.030	.029	.078	−.148
Opt1	−.523	.479	.382	.282	.234	4.931	−3.027	−.137	−.005	−.111	.077	.007	.646	−.684
Pess1	.777	−.529	−.408	−.233	−.454	−3.027	10.215	.336	−.117	.029	−.007	−.010	−1.080	2.756
N2	.036	−.041	−.012	−.040	−.004	−.137	.336	.339	−.028	−.045	−.012	−.029	−.680	.749
E2	.002	.041	.030	.023	.029	−.005	−.117	−.028	.308	.130	.107	.041	.418	−.295
O2	.030	.026	.040	.002	.036	−.111	.029	−.045	.130	.299	.051	.062	.491	−.381
A2	.008	.022	.022	.045	.030	.077	−.007	−.012	.107	.051	.206	.037	.204	−.093
C2	.004	−.007	.008	−.012	.029	.007	−.010	−.029	.041	.062	.037	.160	.120	−.398
Opt2	−.179	.075	.094	.008	.078	.646	−1.080	−.680	.418	.491	.204	.120	6.331	−3.511
Pess2	.171	−.153	−.217	−.106	−.148	−.684	2.756	.749	−.295	−.381	−.093	−.398	−3.511	9.812

The role of shared or common environment ('C') was estimated at very low levels and could be dropped with negligible effect on fit ($\chi^2(28) = 6.32$, $p = 1.0$: see model 2 Table 4). The effect of genetic similarity was tested by dropping additive genetic effects ('A') from the saturated model: This caused a significant reduction in fit ($\chi^2(28) = 47.19$, $p = .013$). All further testing was therefore conducted with reference to the AE model as a comparison model, which decomposed subject's responding into components due to additive genetic differences, and to the environmental effects making twins unique from each other.

The hypothesis that personality has no genetic effects on optimism/pessimism was tested by setting to zero all genetic paths into optimism and pessimism emanating from each of the five latent traits underlying the five personality domains. This model in which personality had no impact on optimism/pessimism could be rejected ($\chi^2(13) = 79.3$, $p < .001$: see model 3 Table 4). The contrary hypothesis – that genetic effects on optimism and on pessimism are entirely accounted for by genetic effects from the five personality domains – was tested next. Dropping the unique genetic paths of optimism and for pessimism, however, lead to significant loss of fit ($\chi^2(3) = 18.69$, $p < .001$).

The two tests above confirm both that personality traits are highly significant influences on optimism and personality, and that, at least at the domain-level, personality is not a complete account of the genetics of optimism and pessimism.

We next moved to focus on the specific personality traits influencing optimism and pessimism, and to determine whether optimism and pessimism themselves are genetically distinct from each other. First, nonsignificant paths from personality to optimism and pessimism were removed, along with nonsignificant paths among the personality traits (which had to this point been allowed to genetically correlate to maximize power to reject the null

Table 4. Genetic model-fitting analyses for 5FM and life orientation test scores for the best-fitting AE model.

	Model	EP	Δ−2LL	Δdf	*P*-value (see note below)	AIC	Model to compare with
1	Saturated ACE	91				2188	
2	AE	63	6.31	28	1.000	2139	1
3	CE	63	47.19	28	.013	2179	1
4	AE, no paths from 5FM to O/P	50	79.3	13	<.001	2192	2
5	AE, no specific genetics for O/P	60	18.7	3	<.001	2151	2
6	Best reduced ACE model	51	13.9	12	.309	2129	2
7	IP model	49	118	42	<.001	2222	1
8	IP, allowing for correlated E	63	16	28	.970	2148	1
9	IP, no specific C	56	.008	7	1.000	2134	8
10	m9 + reducing shared environment	52	2.434	11	.996	2128	8
11	m9 + dropping all C from N, optimism, and pessimism	49	12	3	.007	2135	10
12	Final model: M9 and reduced E (Figure 2)	48	1.3	4	.864	2122	10

Abbreviations: EP = estimated parameters; Δ−2LL = change in $-2 \times \log(\text{likelihood})$; Δdf = change in degrees of freedom from the comparison model. *P*-value = χ^2 test of significance of the change in likelihood; AIC = Akaike information criterion (smaller is better); Boldface type indicates the best-fitting model by AIC (see Figures 1 and 2).
Notes: For models with parameters fixed at zero, the χ^2 test of significance can be biased: Readers should therefore focus on AIC as a guide to model fit. In this case, both were in agreement.

hypotheses of personality impact on optimism/pessimism. This final model did not fit significantly worse than the AE model ($\chi^2(12) = 13.88$, $p = .309$) and was best according to AIC (dropping any further paths increased Akaike Information Criterion [AIC]). In this model, both optimism and pessimism retained specific heritable influences, that is, genetic effects not explicable simply in terms of personality, nor reflecting a single factor influencing both optimism and pessimism. This Cholesky model, then, indicated that optimism and pessimism could not be treated as simple opposite ends of a single bipolar dimension: The equal-but-opposite loadings on the latent traits which this implies did not appear.

Theory-based modeling

While the results from the Cholesky model presented above established that optimism and pessimism could not be reduced to personality effects, the model has several limitations for constraining how we think about optimism and pessimism. The ordering of variables impacts on the model: Whichever variable is placed first must do dual duty as representing both specific effects on that initial variable, and any general effects shared by the first and subsequent variables. While this does not alter fit, it does alter the substantive meaning of the model (Loehlin, 1996). A better model would abstract this general factor. In the next steps of modeling, therefore, the independent pathway (IP) model (Neale & Maes, 1996) was fitted. This is an order-independent model which imposes considerable structure on the data and which explicitly allows for general effects. The structure of this model is shown in Figure 1 panel B. In addition to allowing for general effects of genes and environments, this model specifies the genetics of the five-factor model domains as they are theoretically predicted to occur: with genetic origins for each independent of the other four domains.

As is common, an IP model allowing only specific unique environment effects (i.e. no covariation among traits other than via the general pathways) fit less well than did the baseline ACE model ($p < .001$, see model 7, Table 4). This reflected un-modeled covariation distributed into many small but cumulatively significant correlations among individual scales: Allowing the unique environment component of the model to take a Cholesky form lead to a model that fitted the data well (see Table 4, model 8).

This IP model could be reduced considerably without significant loss of fit. With regard to shared (family-level) environment, while all measure-specific shared-environment effects (i.e. family-level environment effects specific to one a single dimension) could be dropped without substantive loss of fit ($\chi^2(7) = .01$, $p = 1.000$; see model 9, Table 4), some significant general-factor shared-environment effects were highly significant. Specifically, the effects of shared environment on N, optimism, and pessimism were substantial, and dropping these caused a significant loss of fit ($\chi^2(3) = 12.03$, $p = .007$; see model 11, Table 4). Effects of the general shared environment factor could be dropped without significant loss of fit for the personality dimensions other than N ($\chi^2(11) = 2.43$, $p = .996$; model 10, Table 4). Finally, four pathways in the unique environment matrix were small and all could be dropped without significant loss of fit ($\chi^2(4) = 1.28$, $p = .864$, model). This yielded the best-fitting model of the data by AIC, see model 12, Table 4.

Finally, we tested whether allowing personality-specific genetic factors effects on optimism and pessimism improved model fit. Allowing for such connections did not improve fit significantly ($\chi^2(10) = 11.61$, $p = .312$). In

addition, adding just paths from neuroticism (the trait most often implicated in optimism/pessimism) to optimism and to pessimism also did not significantly improve fit ($\chi^2(6) = 2.96$, $p = .814$). These results, then, suggest no shared genetic relations between specific personality effects and optimism or pessimism (over and above the significant general genetic influence). The final model, then, was one with a general genetic influence, specific genes for optimism, specific genes for pessimism a shared (family-level) environmental influence on neuroticism, optimism, and pessimism, and large but unstructured unique environmental influences (see model 12, Table 4), This is shown graphically in Figure 2 (the environmental matrix is shown separately in Table 5).

Discussion

Several findings of interest for understanding optimism/pessimism emerged from the analyses. First, in addition to clear support for heritable effects on optimism and pessimism, these two traits were clearly distinguishable from the five factors of personality, both genetically and environmentally. Second, support was found for significant and substantial effects of family-level environment and of personal or unique environmental influences. Third, optimism and pessimism were themselves differentiated by loadings on separate genetic influences impacting on one dimension but not the other. Support was thus found for multiple genetic and environmental influences serving to differentiate, and to shape the two life orientations. Fourth, the IP model indicated that the observed associations of optimism, pessimism, and personality reflect two distinct effects. The first was a genetic association, in particular of optimism, but also (negatively) of pessimism with a general genetic influence across the domains (see general genetic factor, Figure 2). The second was a significant effect of shared environmental influence. This family environment factor acted to increase optimism and to lower neuroticism and pessimism (or, adversely, to raise neuroticism, and pessimism, and lower optimism). These results are discussed below, including a speculative attempt to integrate the genetic findings with recent neurocognitive research.

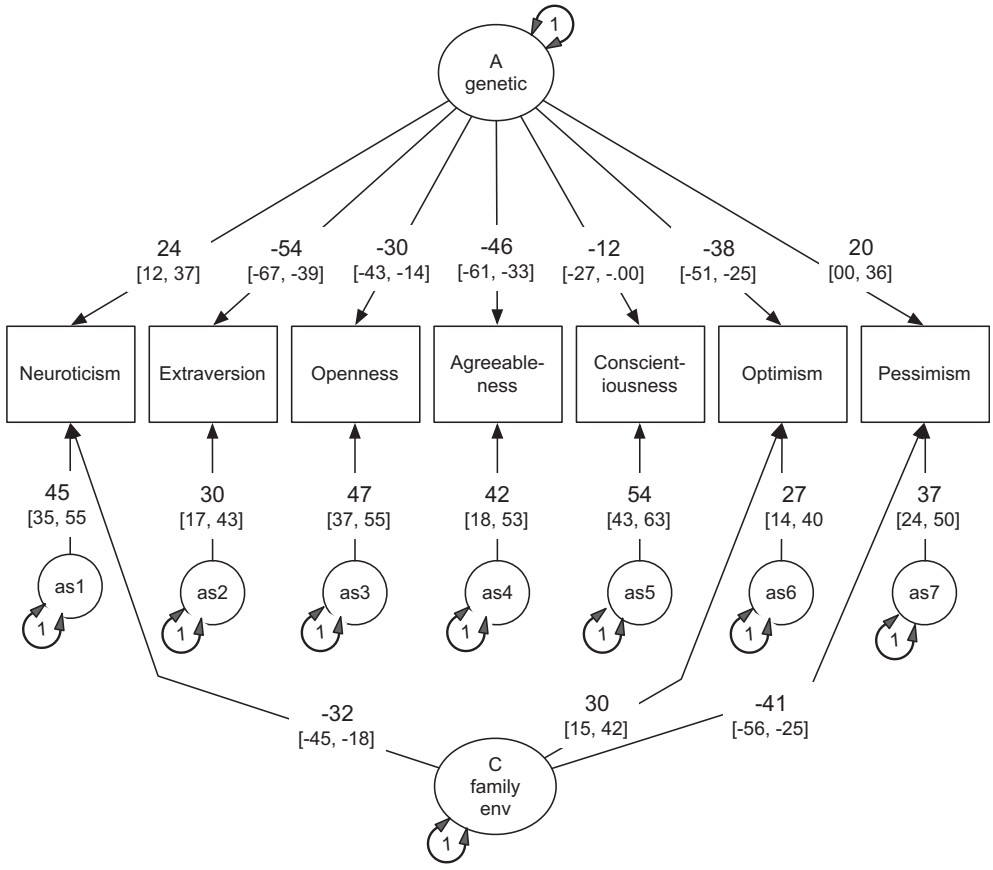

Figure 2. Independent pathway model of personality, optimism, and pessimism.
Notes: Path estimates followed by 95% CIs in square brackets (decimal places omitted for clarity). 'A genetic' is a general genetic factor. 'as' latent variables are traits-specific additive genetic effects. 'C family env' represents a latent shared-environment effect on neuroticism, optimism, and pessimism.

Table 5. Significant effects of specific environment on personality and on optimism and pessimism (note, 95% confidence intervals in brackets).

Domain	E1	E2	E3	E4	E5	E6	E7
Neuroticism	.80 [.79, .85]						
Extraversion	–.08 [−.16, .003]	.78 [.71, .86]					
Openness	–.18 [−.25, −.10]	.43 [.33, .52]	.68 [.62, .75]				
Agreeableness	.	.27 [.18, .37]	.11 [.04, .19]	.72 [.66, .78]			
Conscientiousness	–.21 [−.27, −.13]	.22 [.13, .31]	.19 [.12, .25]	.16 [.09, .23]	.73 [.67, .80]		
Optimism	–.34 [–42, –25]	.19 [.10, .28]	.13 [.06, .19]	.	.	.72 [.66, .79]	
Pessimism	.28 [.19, .35]	–.13 [−.22, −.04]	–.11 [−.17, −.05]	.	–.17 [−.22, −.11]	–.2 [−.27, −.13]	.69 [.63, .76]

The initial ACE model, in addition to confirming numerous previous reports showing heritable influences on personality (Lewis & Bates, 2014; Tellegen et al., 1988), indicated that optimism and pessimism were unlikely to be reducible to personality or to mirror images of each other. To characterize these findings in more detail, we moved to a more theoretically potent model in which the substantive meaning and interpretability of the model is not confounded with order of entry into the model (Loehlin, 1996). The IP model (Neale & Maes, 1996) imposes considerable structure on the model and, therefore, is both more readily falsified and is more informative: This model is discussed below with respect to the hypotheses and findings regarding genetic and environmental effects associated with optimism and with pessimism.

With respect to the debate regarding whether optimism and pessimism are distinct constructs or opposite ends of a single bipolar construct, the present evidence supported viewing optimism and pessimism as distinct constructs (see model 12 Table 4 and Figure 2). The observed moderate inverse covariation among optimism/pessimism was seen as partly reflecting a moderate shared loading on a general pro-social or desirable behavior genetic factor, especially for optimism. In part, the covariation of optimism and pessimism also reflected significant shared family environmental influences. In respect of models in which the two constructs are viewed as simply more or less complex outcomes of personality, the final models suggested no direct genetic links from personality (no paths from any specific genetic influences on the big 5 to optimism or to pessimism). The link with neuroticism was not genetic but rather environmental. The genetic correlation of these two traits was −.66, and the unique environment correlation, while weaker, was also significant: −.38. This result, then, indicates that, at both a biological and environmental level, factors exist which exert opposite effects on optimism and pessimism.

Importantly, the analyses also revealed significant specific genetic influences on both optimism and pessimism. That is genetic factors affecting each of these traits individually. This is strong evidence for an ultimate irreducibility of optimism and pessimism into a single trait at the biological level. Similar, larger, effects from the unique environment specific for each trait buttressed this separability. Future studies with multiple measures of each construct would be valuable, or even item-level heritability analyses, but within these data, and *based on modeled genetic and familial covariance (rather observed or phenotypic covariance),* the idea of optimism and pessimism as bi-polar opposites or as reducible to personality was not supported.

Linking to neurobiological findings

The complexity of the genetic and environmental origins of optimism and pessimism examined here mirrors that of the findings from contemporary biological research on optimism/pessimism noted in the introduction. Given that optimism and pessimism are linked to clinical outcomes (Korn et al., 2014) and optimal maturation (Moutsiana et al., 2013), the present findings suggest that it will be important for future research to establish connections between the neuroscientific literature, the positive psychological literature, and behavior genetic approaches. In particular, the dimensions of the genetic architecture articulated in the present study should be able to be mapped coherently onto dimensions revealed by neuroimaging. As noted above, neuroimaging suggests that optimism bias is related to a psychological function for processing ‘good news’ and involving left inferior frontal gyrus activation/deactivation (Korn et al., 2014; Sharot, Kanai, et al., 2012). Future work combining imaging and behavioral or molecular genetic data may, then, seek to test the hypothesis that genetic effects specific to

optimism reflect volume or activity differences in this region (Lewis, Kanai, Rees, & Bates, 2014). Similarly, the antagonistic effect of dopamine on pessimistic belief formation or the processing of 'bad news' (Sharot, Guitart-Masip, et al., 2012) suggests that genetic effects specific to pessimism may reflect variation in dopaminergic function, and specific genetic polymorphisms within dopamine pathways and this, while speculative, can be tested. Finally, the general genetic effect present across multiple personality traits including optimism/pessimism suggests a need to test whether areas linked to optimism–pessimism may reflect this factor. For instance, anatomical volumes and connectivity of the amygdala, the rostral anterior cingulate cortex, and the connectivity of these systems implicated in optimism (Sharot et al., 2007) may reflect variation in neuroticism, extraversion, agreeableness, and/or conscientiousness.

Finally, but importantly, we turn to the significant effect of shared environmental influence. Shared environment consists of non-genetic factors which are shared by siblings and which serve to make them more similar to each other. Example mechanisms that act at this level include parental education and behavior, family socioeconomic status, influences of the neighborhood such as school, security, etc. Family environment has been suggested to have nonsignificant effects on adult personality (Harris, 1995). Here, a significant family environment factor was found. This acted on optimism, pessimism, and neuroticism, increasing the former, and lowering the latter two (or, adversely, family-level effects acting to raise neuroticism, and pessimism, and lower optimism).

The genetic findings in this paper indicate that we may have intrinsic systems for our processing and orientating to positive and negative events forming a system for resilience – a notion intrinsic to positive psychology (Seligman, 2002). The significant family environment effects are evidence that resilience may be nurtured by family environment. Identifying these specific events within the family environment and, indeed, and the significant environments beyond the family (the significant 'E' or unique environments consist of effects impacting on optimism–pessimism, but not shared by siblings) will be important to identify targets for growth and enhanced resilience.

Limitations and conclusion

While strengths of the research included the breadth of personality domains studied and of maintaining as optimism and pessimism separate measures, other traits warrant attention also. Future research including cognate traits such as failure avoidance and motivation would be valuable (Atkinson, 1967). It will also be important to address the possibility of gene × environment interactions amplifying the development of optimism and pessimism (Bates, Lewis, & Weiss, 2013)

In conclusion, the research indicated that optimism and pessimism are at least partially biologically distinct, resulting in two distinct psychological tendencies: One affecting optimism bias and the tendency to see promise in the future – 'the glass half full', and a second factor linked to genetic and environmental factors leading to processing of negative events as being more likely and less avoidable. Evidence was found also for significant influences from multiple levels of the environment including family environments affecting stability, optimism, and pessimism, which, if malleable, might be targeted for improving well-being and achievement.

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