Journal of Personality and Social Psychology
2014, Vol. 107, No. 4, 751–764

0022-3514/14/$12.00 http://dx.doi.org/10.1037/a0037009

Mutual Reinforcement Between Neuroticism and Life Experiences: A Five-Wave, 16-Year Study to Test Reciprocal Causation

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High neuroticism predicts psychopathology and physical health problems. Nongenetic factors, including major life events and experiences, explain approximately half of the variance in neuroticism. Conversely, neuroticism also predicts these life experiences. In this study, we aimed to quantify the reciprocal causation between neuroticism and life experiences and to gauge the magnitude and persistence of these associations. This longitudinal cohort study included 5 assessment waves over 16 years in a random sample of 296 Dutch participants (47% women) with a mean age of 34 years (SD = 12, range 16–63 years). Neuroticism was assessed with the Amsterdam Biographic Questionnaire. The experiences measured included positive and negative life events, long-term difficulties (LTDs), and change in life quality, all assessed by contextual rating procedures adapted from the Life Event and Difficulties Schedule. We fit structural equation models in Mplus. Results showed that neuroticism consistently predicted negative experiences, decreased life quality, and LTDs (β = 0.15 to 0.39), whereas effects on positive experiences were variable (β = 0.14). LTDs and deteriorated life quality each predicted small but persistent increases in neuroticism (β = 0.18), whereas improved life quality predicted small but persistent decreases (β = −0.13). This suggests set point change in neuroticism. Life event aggregates showed no persistent effects on the neuroticism set point. Neuroticism and life experiences showed persistent, bidirectional associations. Experience-driven changes in neuroticism lasted over a decade. Results support the corresponsive principle (reciprocal causation), suggesting a mixed model of change in neuroticism that distinguishes temporary changes in neuroticism from persistent changes in an individual's neuroticism set point.

Keywords: emotional stability, prospective, set point change, plasticity principle, positive life events

Supplemental materials: http://dx.doi.org/10.1037/a0037009.supp

Neuroticism is defined as the propensity to experience distress and negative emotions, including fear, sadness, anger, anxiety, irritability, loneliness, worry, self-consciousness, dissatisfaction, hostility, shyness, reduced self-confidence, and feelings of vulnerability (John, Robins, & Pervin, 2008). High neuroticism predicts most indices of psychopathology and physical health problems (Malouff, Thorsteinsson, & Schutte, 2005; Ormel et al., 2013; Saulsman & Page, 2004). Moreover, neuroticism predicts important life outcomes, such as occupational success, divorce, and mortality (Ozer & Benet-Martínez, 2006; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). The total economic costs associated with neuroticism are more than twice that of all of the common mental disorders combined (Cuijpers et al., 2010). Insights into change in neuroticism and the determinants of change are therefore important (Lahey, 2009) and may lead to new interventions to target vulnerability to mental disorders rather than the manifestations of those disorders.

Set Point Change in Neuroticism

Neuroticism was originally conceptualized as a dispositional behavioral trait (Eysenck, 1951) uninfluenced by the environment (McCrae et al., 2000). Some theorists acknowledged that life events might evoke subtle short-term state fluctuations in neuroticism but hypothesized that levels would gravitate back to their immutable set point (Costa, Herbst, McCrae, & Siegler, 2000; Fleeson, 2001). However, accumulating evidence suggests that many individuals change in neuroticism over their lifespan (Caspi, Roberts, & Shiner, 2005; Roberts & DelVecchio, 2000; Specht, Egloff, & Schmukle, 2011). For example, neuroticism shows a maturational mean-level change between ages 20 and 40 years of around a quarter of a standard deviation (d = 0.25) per decade (Bleidorn, Kandler, Riemann, Angleitner, & Spinath, 2009; Roberts, Walton, & Viechtbauer, 2006). Moreover, a 40-year follow-up study of neuroticism during midlife (20 to 60 years) showed that 16% of individuals increased and 13% decreased by more than 0.50 (Soto & John, 2012). Similarly, a study over 37 years, from midlife to old age (38 to 70 years), showed individual

This article was published Online First August 11, 2014.

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change in neuroticism of more than 1.00 for about a third of the women studied (Billstedt et al., 2014).

In sum, most individuals presumably remain relatively stable in their neuroticism set point, but reviewed evidence indicates developmental differences between individuals and the possibility for substantial change in neuroticism, provided that important changes in the environment also occur. There appears to be no point during the lifespan that neuroticism is immutable. This is known as the *plasticity principle* (Caspi & Shiner, 2011; Roberts & Wood, 2006). This suggests that the dispositional set point perspective is incomplete. We argue that experience-driven changes in neuroticism that last for years indicate a change in the set point rather than state fluctuations around a set point.

Experiential Effects on Neuroticism

Studies of genetically identical twins, reared together and apart, indicate that a substantial part of the variance in neuroticism is due to nongenetic and nonfamilial factors, mainly experiences that were not shared by both twins (Keller, Coventry, Heath, & Martin, 2005; Lake, Eaves, Maes, Heath, & Martin, 2000). The size of this nongenetic component of neuroticism increases with age (Laceulle, Ormel, Aggen, Neale, & Kendler, 2013; McCartney, Harris, & Bernieri, 1990). Moreover, even the temporal stability of neuroticism shows a substantial experiential component (Laceulle et al., 2013; Viken, Rose, Kaprio, & Koskenvuo, 1994).

Prospective evidence indicates that neuroticism is influenced by life experiences, such as those involving romantic relationships (e.g., new partner or divorce; see Mroczek & Spiro, 2003), work (e.g., being fired or laid off, hired, or promoted; see Sutin, Costa, Miech, & Eaton, 2009), and illness or severe financial hardship (Middeldorp, Cath, Beem, Willemsen, & Boomsma, 2008). However, it remains largely unknown which life events impact the neuroticism set point and how they affect it. Moreover, many changes in neuroticism may fade over time. In sum, recent work supports the plasticity principle and indicates that life experiences influence the neuroticism set point, although some experiences facilitate stability and others facilitate change (Fraley & Roberts, 2005; Laceulle et al., 2013). In the current study, we used five neuroticism assessments over 16 years to quantify the persistence of change in neuroticism driven by major life experiences.

Reciprocal Causation

The substantial nongenetic variance underlying neuroticism may reflect an individual's proactive, reactive, and evocative modification of the surrounding environment through selection of spouses, friends, vocations, or hobbies, for example (Caspi & Shiner, 2011; Kendler & Baker, 2007). It has long been believed that life events and experiences tend to shape personality development by intensifying the propensities that led individuals to those experiences in the first place. This snowballing effect, or reciprocal causation between experiences and personality (Caspi et al., 2005; Lüdtke, Roberts, Trautwein, & Nagy, 2011; Middeldorp et al., 2008), is called the *corresponsive principle*. The corresponsive principle suggests that mutually reinforcing feedback loops between the environment and personality shape personality development and stability.

It may even be that the neuroticism set point depends on the influence of genetically driven experiential effects (indirect genetic effects) rather than being directly determined by genetics (Nettle, 2007; Roberts & Jackson, 2008). The available evidence suggests that measures of the quality of an individual's life experiences can be as stable as personality (Bronfenbrenner, 2005; Kendler & Baker, 2007). For example, individual differences in life experiences have a substantial genetic basis (Kendler & Baker, 2007; Power et al., 2013). Such genetic effects are partially mediated by neuroticism, in line with the corresponsive principle (Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012; Kendler & Baker, 2007; Power et al., 2013). Many studies have shown that the incidence of negative (but not positive) major life events increases with increasing neuroticism (Lüdtke et al., 2011; Middeldorp et al., 2008; Specht et al., 2011), but, on average, individuals report more positive than negative life events (Berntsen, Rubin, & Siegler, 2011) and positive events associate with decreases in neuroticism (Jeronimus, Ormel, Aleman, Penninx, & Riese, 2013). The impact of negative experiences, however, seems more persistent than the impact of positive experiences (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001).

Hypotheses

We aimed to better define the rough contours of the corresponsive principle's feedback system. Five neuroticism assessments over 16 years enabled us to gauge the magnitude and course of reciprocal causation between neuroticism and life experiences (mutually reinforcing associations). Uneven intervals between each assessment wave may also illuminate the temporal dynamics of experiential effects on neuroticism. We hypothesized that neuroticism levels are more predictive of variance in life experiences than vice versa (Hypothesis 1). More specifically, we hypothesized, in line with the corresponsive principle, that major negative life events tend to increase neuroticism (Hypothesis 2A) and that higher neuroticism scores predict more negative life events (Hypothesis 2B). This may spark a malignant cycle that cements increases in neuroticism, resulting in a lasting set point increase. Similarly, we hypothesized, again in line with the corresponsive principle, that major positive life events predict decreases in neuroticism (Hypothesis 3A) and that decreases in neuroticism predict positive life events (Hypothesis 3B). This may spark a benign cycle of positive reinforcement, resulting in a lasting set point decrease. Finally, on the basis of empirical work discussed above (e.g., Baumeister et al., 2001; Jeronimus et al., 2013; Middeldorp et al., 2008), we hypothesized that increases in neuroticism driven by negative life events are less common than decreases in neuroticism driven by positive life events and that the positive effects are less persistent (Hypothesis 4). Thus, we argue that neuroticism is not immutable, that life experiences drive changes in neuroticism, and that experience-driven change in neuroticism that lasts for years indicates a change in the set point of neuroticism rather than state fluctuations around a set point.

Life Event and Difficulties Schedule (LEDS)

Earlier analyses of this data set focused on the stability of neuroticism (the autoregressive model in Ormel & Rijsdijk, 2000) and the etiology and course of psychological distress (Ormel & Wohlfarth, 1991; Ranchor & Sanderman, 1991). The use of life experience indices to predict neuroticism was not part of earlier

work. In this study, we invested in a life event assessment based on the LEDS, still the gold standard in life event research (Dohrenwend, 2006; Monroe, 2008). In the LEDS methodology, reviewers objectively rate the potential impact of specific events on individuals on the basis of the severity, duration, and ambiguity of the event; the context in which the event occurred; and the sociodemographic characteristics of the participant (Brown & Harris, 1978).

Interviews have limitations: They are costly and time consuming and may fail to capture embarrassing information (Grant, Compas, Thurm, McMahon, & Gipson, 2004; Monroe, 2008). Moreover, an individual's life situation may change in a way that is not captured by a life event interview but that affects neuroticism. This study combines the following methods to minimize the weaknesses of each: (a) survey data of life events, (b) interview-based panel ratings of life events and difficulties, and (c) change in the participant's environment or life situation.

Method

Sample

We based our analyses on a five-wave data set collected over a 16-year period (1970–1986) by Ormel (1980; Ormel & Wohlfarth, 1991) and Sanderman (1988) from a random sample of 384 adults from the Dutch population. After baseline measurements (T_1) in 1970, 88 participants could not be followed up in the first two waves in 1975 (T_2) and 1976 (T_3; six died, five moved abroad, and 77 did not respond), leaving a final sample of 296 participants (77.1%; $n = 139$, 47% women). At the next wave (T_4) in 1984, another 49 participants could not be followed up (10 died, four moved abroad, and 35 did not respond). At the final wave (T_5) in 1986, 17 participants could not or did not complete follow-up measures (two died and 15 did not respond), whereas 230 participants responded. The total sample comprised 296 participants, of which 224 were interviewed five times over 16 years, whereas the other 72 participants missed T_4, T_5, or both. The maximum likelihood analyses used in our study are based on all available data from all participants. At T_1, the mean age of the participants was 34.3 years ($SD = 11.8$, range: 16–63 years); 26% were born between 1911 and 1925, 30% between 1926 and 1940, and 44% between 1941 and 1954. Comparison of the 224 responders and 72 nonresponders revealed no statistically significant differences in neuroticism, life events, or any of a broad range of sociodemographic-, distress-, and personality-related variables (Sanderman, 1988). However, attrition at all waves was related to age, because the rate of mortality was higher for older participants.

Measures

Education. At T_2, participants self-reported their level of education on a scale from 1 to 7: 1% of the participants reported no education (1); 26.7% reported only primary education (elementary school; 2); 25%, only lower secondary education (vocational school; 3); 19.9%, at least 3 years of secondary education (high school; 4); 13.2%, at least 5 years of secondary education (5); 6.1% reported bachelor-level higher education (6); and 6.8% reported graduate-level higher education (7). Data were missing for four participants (1.4%).

Neuroticism. All five waves assessed neuroticism (N_1–N_5) using the eight best discriminating items (see Table S1 in the supplemental materials) of the Amsterdamse Biografische Vragenlijst (ABV; Wilde, 1970). The ABV was the most popular Dutch neuroticism measure in the last quarter of the 20th century but lacks the breadth of modern neuroticism measures (e.g., NEO PI-R; Costa & McCrae, 1992). The content of the ABV is based on the Eysenck Personality Inventory (Eysenck & Eysenck, 1975). Items were scored on a 3-point scale (*no, don't know,* and *yes*). In our study, on average, 6.7% of our participants ($n = 20$) answered *don't know* on an item. For these, we followed the principles of Wilde's (1970) manual, in which he stated that undecided individuals (those answering *don't know*) were most likely to score high on neuroticism. Therefore, we calculated scale scores as the sum of the eight items (*no* = 1, *don't know* = 2, and *yes* = 2; theoretical scale range: 8–16). The average Cronbach's alpha over the five waves was .85 (range: .74–.98).

Life events, long-term difficulties, and life situation change. All five waves used investigator-based assessments of life experiences and a contextual rating procedure adapted from the LEDS (Brown & Harris, 1978). The specific characteristics of each measure, however, varied across waves due to the varying time intervals between assessment waves and available funding, as outlined below.

At T_2 (1975), we determined whether participants had experienced long-term difficulties (LTDs), irrespective of when the difficulties had arisen. The three inclusion criteria for LTDs were (a) sufficient stressfulness (impeding daily goals or plans), (b) duration of at least two months, and (c) presence of the difficulty in the 6 weeks prior to the T_2 assessment. Often, but not necessarily, a LTD (e.g., unemployment for at least two months) had evolved from a negative life event (fired because of factory closure). Other examples of LTDs are marital discord, having a handicapped child, or serious chronic illness of a family member. Three independent and well-trained reviewers assigned contextual ratings of LTDs and life events on the basis of written interview materials in accordance with the methodology of Brown and Harris (1978). They rated the impact of each event on the basis of the participant's biography and broader social context, such as gender, age, and sociocultural background (the participants' *microsituation*). Reviewers may thus have rated a particular life event differently for different participants. For example, giving birth to a wanted child while financially stable was rated differently than giving birth to an unplanned child in poverty.

Each reviewer also indicated whether LTDs were endogenous (brought about by the individual's own behavior) or exogenous (independent of the individual's own behavior). In cases of disagreement, we chose the position favored by the majority of reviewers. Reviewers rated the severity of each LTD ($\alpha = .92$) on a 4-point scale (Brown & Harris, 1978). We aggregated the mean ratings of severity into two indices, one for endogenous and one for exogenous LTDs.

At T_3 (1976), we measured positive and negative life events that had occurred over the 14 months preceding the interview (since T_2). Examples of negative life events include being fired or laid off, the end of a romantic relationship, divorce, fights with close friends, sickness, hospital admission, operations, accidents, or the death of a loved one. Examples of positive life events include starting a new job, starting a new romantic relationship, marriage,

pregnancy, giving birth, earning a degree, moving to a new home, or beginning cohabitation. Three independent reviewers rated the pleasantness of each positive life event ($\alpha = .93$) and long-term threat of each negative life event ($\alpha = .90$) on a 4-point scale (Brown & Harris, 1978), taking the participant's microsituation into account. This means that closely related life events were counted together (e.g., loss of job and financial loss arising from that loss of job). We then determined the aggregate of the pleasantness scores of all positive life events and threat scores of all negative life events at T_3.

At T_4 (1984), less funding was available, which impeded the use of exactly the same methodology as at T_2. Additionally, the 8-year interval between T_3 and T_4 required a somewhat different approach than was applied at T_3, because life event assessments are less reliable over longer intervals. Also, the principal investigator at that time was focused on whether improvement versus deterioration of a person's overall life circumstances (the life situation) had a corresponding impact on well-being. At T_4, therefore, participants completed the Life Situation Schedule interview, which is used to collect information about a participant's current life situation and any major life changes since T_3. To collect this data, interviewers received information about the participant's life situation at T_3 but were kept ignorant of the participant's mental state and psychological characteristics. The interviewers collected factual information on the participant's microsituation (i.e., housing, neighborhood, financial situation, major interpersonal relationships, health status of significant others, stress and control in the work situation, amount of leisure time, and life events since T_3; Sanderman, 1988). Three reviewers independently rated this information, comparing participants' life situation at T_3 with that at T_4 using a 7-point scale to index change in life quality (1 = *very much deteriorated*; 4 = *neutral, neither improved nor deteriorated*; 7 = *very much improved*). These ratings were recoded into two variables, scaled to reflect their inverse relationship with neuroticism: deteriorated life quality (a rating of 0–4 = 0, 5 = 1, 6 = 2, and 7 = 3) and improved life quality (a rating of 1 = 3, 2 = 2, 3 = 1, and 4–7 = 0).

Finally, largely because of limited funding, T_5 (1986) used a simplified life event measurement that assessed life events since T_4 with a combination of a 95-item questionnaire with event ratings based on interviews that provided contextual and factual information about each event (Sanderman, 1988). The questions focused not only on the participant but also the participant's household members; interactions with others; and experiences with health, work, children, and loss. Contextual questions sought to determine when the event had happened, what had happened, what the outcome of the event was, what the consequences were, and whether the event had positive and/or negative effects. Interviewers subsequently gathered additional information during a short telephone interview, mostly concerning the life events. Reviewers rated the long-term threat or pleasantness of life events on a 6-point scale via an (investigator-based) procedure modeled after the Brown and Harris (1978) method. These scores were aggregated into positive and negative life event measures.

Statistics

Data cleaning and descriptives. We performed data cleaning steps and calculated descriptives in SPSS (Version 20, SPSS, Chicago, Illinois). All neuroticism and life experience measures were nonnormally distributed (Shapiro–Wilk tests, all ps $< .001$; visual inspections showed positive skews or floor effects). Neuroticism was most skewed ($z = 1.14$, standard error [*SE*] = 0.14) and kurtotic ($z = 0.52$, $SE = 0.28$) at T_3. However, even the distributions for LTDs at T_2 and positive and negative life events at T_3 deviated too much from zero to assume normality (skewness ranged from 1.73 to 2.12, all *SE*s = 0.14, and kurtosis ranged from 3.74 to 6.52, all *SE*s = 0.28; $z > 3.29$ is significant at $p < .001$). We observed floor effects for 20%–45% of the participants in most variables, rising as high as 77% for improved life quality at T_4. Therefore, we tested mean differences in neuroticism using the Wilcoxon signed rank tests (Zimmerman, 2011), which indicated differences in mean score location (two-tailed Monte Carlo, $k = 10{,}000$). We explored associations between variables using Spearman's rho (Bishara & Hittner, 2012). The multiple correlation squared of Spearman's rho indicates proportion of variance in the ranks that two variables share, usually approximating the multiple correlation squared. To reduce familywise alpha inflation, we only interpreted correlations that were significant at $p < .01$. Finally, we heuristically derived the temporal effects of neuroticism and life events based on follow-up times. Thus, if the time between two neuroticism assessments was 5 years (e.g., $N_1 - N_2$), the interposed LTDs occurred, on average, 2.5 years before T_2.

Effect sizes. We expressed our results with three effect size indices: correlations, Cohen's *d*, and partial regression coefficients (β). We classified correlations as small if between .10 and .29, moderate between .30 and .50, and large if above .50. Cohen's *d* expresses differences in standard deviation units, which we indexed as small from 0.20 to 0.49, medium from 0.50 to 0.80, and large if greater than 0.80, using conversion formulas derived from Borenstein, Hedges, Higgins, and Rothstein (2009). Finally, partial regression coefficients report the change in outcome per standard deviation change in a predictor. This enables comparison of predictors in a model.

Modeling Procedure

Structural equation models. We used a system of regression equations specified in path analysis or structural equation models (SEMs) in Mplus Version 6.12 software (Muthén & Muthén, 1999). SEM enables simultaneous modeling of several related regression relationships (e.g., neuroticism at T_3 can be both dependent and independent in the same model) and estimation of measurement error as well. Approximately 20%–25% of the variance in our neuroticism measure was due to measurement error (Ormel & Rijsdijk, 2000). Modeling neuroticism as a latent variable would adjust for this. A latent variable approach, however, is not possible for the experiential measures because many stressful life experiences are stochastically independent. We therefore applied a conservative observed-variable approach, which enables comparison of the magnitude and persistence of both experiential (life stress) and neuroticism effects. Figure 1 shows our a priori baseline model. In this figure, horizontal paths quantify the strength of temporal associations in the model as β coefficients, while vertical paths reflect correlated change. Creating this baseline model allows us to test our hypotheses, and adjustments based on these tests enable us to construct a final model that best

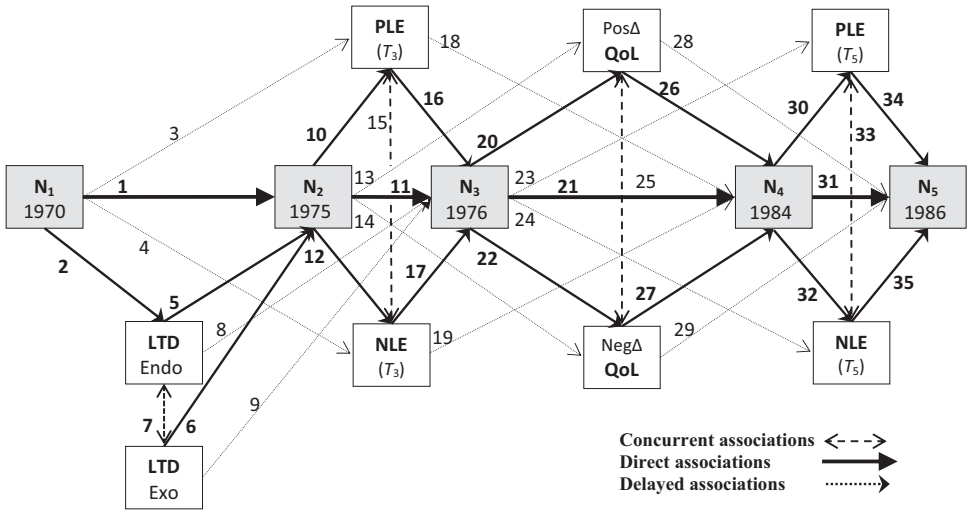

Figure 1. Baseline model: Mutual reinforcement between neuroticism and experiential effects over five waves and 16 years. N_1 = neuroticism at baseline measured in 1970; LTD = long-term difficulties; Endo = endogenous; Exo = exogenous; PLE = positive life events; NLE = negative life events; PosΔQoL = improved life quality; NegΔQoL = diminished life quality. All details about all variables are reported in the Method section. The depicted paths are indicated with labels 1–35, but all paths between experiential measures were estimated freely (even those not shown in the figure). Effects of N_1 on exogenous LTDs (Path 36) and delayed neuroticism effects (T_{x+2-4}) on experiences (Paths 37–48) are neither depicted nor estimated in the baseline model (but are given in Table S3 of the supplemental materials). Paths 36–48 are fully described in the Method section and tested in secondary models, and all results are given in the Result section (see also Table S4 of the supplemental materials).

quantifies the mutually reinforcing relationship between neuroticism and life experiences.

Estimators and tests. We calculated our models using robust maximum-likelihood estimation (MLR) because MLR deals with missing data and provides robust standard errors that account for the nonnormality in our data. Multivariate kurtosis distorts the distribution of the chi-square test statistics and thus inhibits a comparison of nested models via chi-square difference tests with specified degrees of freedom, henceforth denoted as $\Delta\chi^2(\Delta df)$. MLR provides a correction factor that enables the calculation of Santorra–Bentler-corrected $\Delta\chi^2(df)_{SB}$. When the asymptotic nature of this correction led to negative $\Delta\chi^2(df)_{SB}$, we calculated only strictly positive $\Delta\chi^2(df)_{SB}$-tests via a clone model. For details, see Bryant & Satorra, 2012). Nested model modifications that improved on baseline fit converged in the final model, in which all insignificant paths were fixed at zero and those that significantly improved fit were freely estimated.

Fit indices. After checking the assumptions underlying the fit indices (Bentler & Chou, 1987), we evaluated the baseline and final models on the basis of six fit indices (Geiser, 2013): (a) the chi-square, $\Delta\chi^2(df)$; (b) the comparative fit index (CFI $\geq$ .90 for an acceptable model); (c) the root-mean-square error of approximation (RMSEA $<$ .05 in a good model); (d) the Tucker–Lewis index (TLI $\geq$ .90 is desirable); (e) the Akaike information criterion (AIC); and (f) the Bayesian information criterion (BIC). We considered the model that best fit the data to be the one that performed significantly better in $\Delta\chi^2(df)_{SB}$-tests and produced the largest value on CFI and the lowest on AIC, BIC, and RMSEA. Finally, we checked the robustness of the modeling results (see Table S4 in the supplemental materials).

Model preparation. We began to examine the reciprocal relationship between neuroticism and life experiences and its persistence by fitting a saturated baseline model, freely estimating all paths between life experience measures and modeling paths between these life experiences and neuroticism. Figure 1 shows most of the paths, while details on all the paths appear below. This baseline model fit served as a reference for a subsequent series of nested models that diverged from the baseline via either constrained or freed paths. Note that in the case of freed paths, the baseline model was actually nested. We quantified significant ($p <$ .05) improvement or deterioration in comparison to baseline fit using $\Delta\chi^2(\Delta df)$ tests and created a final model that included all of the improvements to best fit the data. We used this final model to test our hypothesis regarding the magnitude and persistence of the reciprocal associations between neuroticism and life experiences. Finally, we used indirect effect models to study the presence, persistence, and magnitude of indirect effects. Figure 1 displays Paths 1–35 and Table 1 categorizes every path (1–48; a detailed specification for each path appears in Table S3 of the supplemental materials).

Neuroticism effects. We examined associations between neuroticism measurements at the various assessment waves and associations between neuroticism and life experiences. Associations between subsequent neuroticism measures are henceforth called *stability effects*. We distinguished four temporal dimensions for neuroticism effects on life experiences: *direct neuroticism effects* on life experiences during a single interval (T_{x+1}) and *delayed neuroticism effects* on life experiences over two (T_{x+2}), three (T_{x+3}), or four (T_{x+4}) measurement intervals. Notably, our model treated delayed neuroticism effects as unmediated by interposed

Table 1
Characteristics of the Paths in Figure 1 and Model Estimates for Each Block

Description of the paths in Figure 1				Estimates		
Type	Span	No.	Path labels	*df*	$\Delta\chi^2(\Delta df)_{SB}$	Result
Stability		4	1, 11, 21, 31	47	523.41***	Fit
Neuroticism						
Direct		7	2, 10, 12, 20, 22, 30, 32	50	38.66***	Fit
Delayed	T_{x+2}	6	3, 4, 13, 14, 23, 24	49	2.74	@0
Delayed	T_{x+3}	6	37–42, not depicted in Figure 1	37	4.13	@0
Concurrent		4	7, 15, 25, 33	47	71.19***	Fit
Experiential						
Direct		8	5, 6, 16, 17, 26, 27, 34, 35	51	35.93***	Fit
Delayed	T_{x+2}	6	8, 9, 18, 19, 28, 29	49	30.77***	Fit
Delayed	T_{x+3}	6	43–48, not depicted in Figure 1	37	7.79	@0

Note. $\Delta\chi^2(\Delta df)_{SB}$ = change in χ^2 compared with baseline; Fit = fitted in the baseline model (Figure 1); @0 = constraint at zero; T_{x+1} = subsequent measurement wave; T_{x+2} = two waves further in time; No. = total number of paths in a category; *df* = degrees of freedom of the estimated model, where Δdf from baseline is similar to the total number of paths in a category. Details about all types of effects, their persistence, all variables, and model estimations for neuroticism and experiential effects are reported in the Method section. Table S4 in the supplemental materials shows results for all individual paths and each type of effect. Paths 1–35 are pictured in Figure 1 (see the Method section or Table S3 in the supplemental materials for Paths 36–48).
*** $p < .001$, two-tailed.

neuroticism or life experiences measures: For example, paths calculating the delayed effect of neuroticism at T_1 on change in life quality between T_3 and T_4 do not include T_2 measurements as a mediating factor (Paths 37–38). However, as stated in the introduction, we do not expect delayed effects across two or more intervals and presume such long-term influences to be mediated (carried forward) by interposed neuroticism and life experience measures. Accordingly, we fixed delayed effects for three or four intervals at zero in our baseline model and, therefore, removed them from Figure 1 (Path 37–48).

We used secondary models to test these assumptions. If freely estimated delayed paths (T_{x+3} and T_{x+4}) significantly improved model fit compared with baseline, it would discredit our assumption and we would include these delayed paths in the final model. However, our assumptions would be supported if fixing delayed neuroticism effects (T_{x+2-4}) at zero in the model caused no significant change in model fit. We also tested our assumption that exogenous LTDs between T_1 and T_2 are independent of neuroticism at T_1. Deviance in model fit from the baseline model would indicate the importance of these neuroticism effects.

Experiential effects. We also examined the relationship between experiential measures and neuroticism, as well as concurrent effects of various life experience measures. Concurrent effects are associations between different life experience measures from the same assessment wave. Additionally, we distinguished four temporal dimensions for experiential effects on neuroticism similar to the dimensions for neuroticism's effects on experiences: *direct experiential effects* on immediate neuroticism assessments (T_{x+1}) and *delayed experiential effects*, or spillover, on later neuroticism assessments (T_{x+2}, T_{x+3}, T_{x+4}). Note that although we defined *delayed experiential effects* as unmediated by interposed neuroticism or life event measures in the baseline model, we actually expect long-term life event influences to be mediated (carried forward) by interposed neuroticism measures. Consequently, we fixed delayed paths of three or four intervals at zero in the baseline model and removed them from Figure 1. Delayed experiential effects would, in theory, arise from LTDs to affect neuroticism at T_3 (T_{x+1}), T_4 (T_{x+2}), or T_5 (T_{x+3}, Paths 43–46, respectively) and from positive and negative life events at T_3 to affect neuroticism at T_5 (T_{x+3}, Paths 47 and 48). We tested this with secondary models that either fixed delayed experiential effects at zero (T_{x+2}) or estimated them freely (T_{x+3}, T_{x+4}), comparing the results against baseline fit. The models in which concurrent and direct experiential effects were fixed at zero quantified impact in terms of deviance from baseline fit.

Results

Mean Level

Table 2 provides descriptives. Neuroticism remained stable over the five waves (10.00 at T_1; 9.98 at T_5). Nevertheless, we observed a small but significant mean-level decrease between 1975 and 1976 (T_2–T_3, $\Delta = -0.24$, $z = -3.164$, $p < .005$), stability between 1976 and 1984 (T_3–T_4, $\Delta = -0.09$, $z = 0.530$, $p = .60$), and a small increase between 1984 and 1986 (T_4–T_5, $\Delta = 0.25$, $z = 2.031$, $p < .05$).

Correlations

Table 3 gives correlations. Women scored higher on neuroticism ($d = 0.57$, $R^2 = 7\%$). Younger participants were better educated ($d = 0.52$), reported more positive life events at T_3 ($d = 0.58$) and T_5 ($d = 0.41$), and improved life quality at T_4 ($d = 0.43$). Both younger and better educated participants reported fewer exogenous LTDs (ds = 0.49 and 0.41, respectively) and more positive and negative life events at T_5 (both ds = 0.40). The stability of neuroticism decreased over time from a correlation of .72 over 1 year ($T_2 \rightarrow T_3$) to .65 over 5 years ($T_1 \rightarrow T_2$), .60 over 8 years ($T_3 \rightarrow T_4$), .51 over 11 years ($T_2 \rightarrow T_5$), and .41 over 16 years ($T_1 \rightarrow T_5$, $R^2 = 17\%$).

We observed moderate direct neuroticism effects on life events including endogenous LTDs at T_2 ($d = 0.70$, $R^2 = 11\%$) and negative life events at T_3 ($d = 0.32$). Neuroticism also showed a

Table 2
Descriptives Variables

Wave	Year	Variable	Abbreviation	*n*	Range	*M*	*SD*
T_1	1970	Gender	Gender	296	0–1	0.53	0.49
T_1	1970	Age (years)	Age	296	11–58	29.27	11.82
T_2	1975	Education level	Education	292	1–7	3.63	1.51
T_1	1970	Neuroticism	N1	296	8–16	10.00	2.03
T_2	1975	Neuroticism	N2	296	8–16	10.06	1.98
T_3	1976	Neuroticism	N3	296	8–16	9.82	2.01
T_4	1984	Neuroticism	N4	256	8–16	9.73	1.88
T_5	1986	Neuroticism	N5	224	8–16	9.98	2.14
T_1–T_2	1975	Endogenous long-term difficulties[a,b]	LTD Endo	291	0–27	3.49	4.32
T_1–T_2	1975	Exogenous long-term difficulties[a,b]	LTD Exo	291	0–29	3.26	4.22
T_2–T_3	1976	Negative life events	NLEs	296	0–22	4.94	5.14
T_2–T_3	1976	Positive life events	PLEs	296	0–23	3.64	4.12
T_3–T_4	1984	Diminished life quality[a,b,c]	NegΔQoL	247	0–2	0.31	0.62
T_3–T_4	1984	Improved life quality[a,b,c]	PosΔQoL	247	0–2	0.54	0.65
T_4–T_5	1986	Negative life events	NLEs	296	0–22	3.92	4.81
T_4–T_5	1986	Positive life events	PLEs	296	0–19	2.85	3.58

Note. $N = 157$ men and 139 women. T_1 = baseline; T_2 = follow-up; T_3 = third wave of measurement; T_4 = fourth wave of measurement; T_5 = fifth wave of measurement. All details on all variables are reported in the Method section.
[a] Majority rating among three raters. [b] Endogenous or brought about by the individual's own behavior versus exogenous or not brought about by the individual's own behavior. [c] With reference to the baseline at T_3.

weak delayed association with negative life events at T_3 over 5.5 years (T_{x+2}, $d = 0.35$, $R^2 = 2\%$, Path 4). We also observed five direct experiential effects on neuroticism, associating (a) endogenous long-term difficulties with neuroticism at T_2 ($d = 0.70$, $R^2 = 11\%$), (b) negative life events with neuroticism at T_3 ($d = 0.30$), (c) deteriorated life quality ($d = 0.54$) and (d) improved life quality with neuroticism at T_4 ($d = -0.43$, $R^2 = 4\%$), and (e) negative life events with neuroticism at T_5 ($d = 0.54$). In addition, endogenous LTDs and negative life events at T_3 showed delayed associations with neuroticism (T_{x+2}, *d*s = 0.72 and 0.41, respectively; T_{x+3}, both *d*s = 0.61, $R^2 = 8\%$). Notably, the delayed association of endogenous LTDs with neuroticism at T_5 persisted at least 13.5 years (T_{x+4}, $d = 0.56$, $R^2 = 7\%$, Path 45).

Taken together, these correlations indicate that although improved life quality is associated with decreases in neuroticism, no positive life events do. We also found no delayed effects on neuroticism. Decreases in neuroticism associated neither with subsequent positive life events nor with improved life quality. However, all negative life events, LTDs, and deteriorated life quality were associated with increases in neuroticism, even showing delayed effects in later waves, with some associations persisting through four intervals ($r = .27$ over 13.5 years). Increases in neuroticism were associated with subsequent negative life events, even at later waves, and more endogenous LTDs and decreased quality of life.

Modeling

The baseline model, depicted in Figure 1, guided our subsequent nested modeling. In Table 1, we report on all of the paths in the baseline model, our model manipulations, and the $\Delta\chi^2(\Delta df)$ test results for each block. We first present results for the tested baseline assumptions, followed by neuroticism effects and experiential effects. Together, these shaped the final model.

A priori assumptions. Tests of our a priori assumptions showed that, indeed, there were no neuroticism effects on exogenous LTDs at T_2, $\chi^2(1)_{SB} = 0.71$, $p = .40$. Thus, we kept them fixed at zero in the final model. Constraining the concurrent effects between life event measures at zero resulted in a void model ($p < .001$).

Tests of neuroticism effects on experiences showed that removing direct neuroticism effects significantly decreased the model fit ($p < .001$). Closer inspection revealed this to be mostly due to the effect of neuroticism at T_1 on endogenous LTDs, $\chi^2_{SB}(1) = 29.92$, $p < .001$. Freely estimated delayed neuroticism effects did not improve the fit of the model significantly (T_{x+2}, $p = .84$; T_{x+3}, $p = .66$). Therefore, we included only stability and direct neuroticism effects in the final model.

Tests of experiential effects on neuroticism showed that fixing direct effects at zero diminished the model fit significantly ($p < .001$). Freely estimated delayed experiential effects over two intervals improved the model fit compared with baseline ($p < .001$) but had no effect over three or more intervals ($p = .25$). Thus, we included direct and delayed experiential effects over one and two intervals in the final model. Table 1 contains all of the details on modeling, and results for all individual paths appear in Table S4 of the supplemental materials.

Final Model

The final model, depicted in Figure 2, allowed us to test our hypotheses. Table 4 shows the magnitude and persistence of each path. The final model showed higher CFI/TLI (.93/.90) and lower RMSEA (.06), AIC (14,832), and BIC (14,987) values than the baseline model (see the Method section) but did not have a significantly improved fit, $\chi^2(17) = 12.04$, $p = .80$. The model fit was acceptable and the sample-size-to-free-parameter ratio was above 7. Details can be found in the supplemental materials. The relatively low TLI may be due to the mainly small correlations between variables.

Neuroticism effects. The final model in Figure 2 illustrates the high stability of neuroticism. We found five direct neuroticism

Table 3
Associations Between Variables as Spearman's Rhos

Wave	Year	Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. T_1		Gender	—															
2. T_1	1970	Age	.03	—														
3. T_2	1975	Education	.12*	**−.23*** **	—													
4. T_1	1970	N1	**−.25*** **	−.07	.09	—												
5. T_2	1975	N2	**−.31*** **	.00	.05	**.65*** **	—											
6. T_3	1976	N3	**−.24*** **	.10	−.06	**.61*** **	**.72*** **	—										
7. T_4	1984	N4	**−.29*** **	.03	.01	**.49*** **	**.61*** **	**.60*** **	—									
8. T_5	1986	N5	**−.27*** **	.04	−.04	**.41*** **	**.51*** **	**.52*** **	**.65*** **	—								
9. T_1–T_2	1975	LTD Endo	−.11	.10	−.14*	**.33*** **	**.33*** **	**.34*** **	**.29*** **	**.27*** **	—							
10. T_1–T_2	1975	LTD Exo	−.07	**.24*** **	**−.20*** **	.04	.01	.14*	.05	.10	.01	—						
11. T_2–T_3	1976	NLEs (T_3)	−.06	−.06	−.04	**.17*** **	**.16*** **	**.15*** **	**.20*** **	**.29*** **	.12*	.06	—					
12. T_2–T_3	1976	PLEs (T_3)	.04	**−.28*** **	.13*	.13*	.08	.02	.02	−.00	−.05	−.05^x	**.36*** **	—				
13. T_3–T_4	1984	PosΔQoL	−.07	**.17*** **	−.16*	.06	.10	.11	**.26*** **	.12	**.19*** **	.07	−.08	**−.17*** **	—			
14. T_3–T_4	1984	NegΔQoL	−.00	**−.21*** **	.06	.03	−.03	.01	**−.21*** **	−.01	−.06	−.06	.13*	.09	**−.49*** **	—		
15. T_4–T_5	1986	NLEs (T_5)	−.11	**−.15*** **	**.17*** **	.02	.11*	.03	.14*	**.26*** **	.11	−.03	.08	−.03	−.02	.03	—	
16. T_4–T_5	1986	PLEs (T_5)	−.08	**−.20*** **	**.23*** **	.04	.13*	.09	.10	.14*	.02	−.06	.11	.13*	−.10	.04	**.47*** **	—

Note. N = 157 men, 139 women (47%; score = 0). PosΔQoL = improved life quality; LTD = long-term difficulties; Endo = endogenous; Exo = exogenous; NegΔQoL = diminished life quality; NLEs = negative life events; PLEs = positive life events; T_1 = baseline wave; T_2 = follow-up; T_3 = third wave of measurement; T_4 = fourth wave of measurement; T_5 = fifth wave of measurement. Correlations significant at $p < .01$ were interpreted and are reported in bold.
* $p < .05$, two-tailed. ** $p < .01$. *** $p < .001$.

effects, the largest for endogenous LTDs on neuroticism at T_2 (d = 0.96). It is interesting that the other direct neuroticism effects for the three negative life event measures (all ds = 0.40) and one positive life event measure (d = 0.38 at T_5) all showed similar magnitudes to each other. Neuroticism predicted diminished life quality over 8 years, which was the most persistent direct neuroticism effect we measured. We observed no delayed neuroticism effects. Most variance was explained for neuroticism at T_3 (R^2 = .60, SE = 0.05, $p < .001$; all residuals, intercepts, and R^2s are reported in Table S6 of the supplemental materials).

Experiential effects. We also observed direct experiential effects, such as an increase in neuroticism at T_2 after endogenous LTDs (d = 0.49) and in neuroticism at T_4 following a deterioration in quality of life (d = 0.46). Improved life quality predicted decreases in neuroticism at T_4 (d = −0.25). Moreover, endogenous and exogenous LTDs predicted delayed increases in neuroticism at T_3 (T_{x+2}, ds = 0.44, and 0.49, respectively), while improved life quality predicted delayed increases in neuroticism at T_5 (d = 0.38 over 6 years). However, none of the aggregated life event measures affected neuroticism, and positive and negative life events between T_2 and T_3 occurred, on average, only 6 months before the assessment of neuroticism at T_3. In sum, only LTDs and changes in quality of life predicted change in neuroticism over at least four years, and their effect was small. Most variance was explained for the endogenous long-term difficulties at T_2 (R^2 = .15, SE = 0.05, $p < .001$).

Indirect effects. We used indirect effects models to gauge and quantify mediation of neuroticism and experiential effects by interposed neuroticism and experiential measures (all details can be found in the Table S2 of the supplemental materials). Results indicated that the temporal association between neuroticism at T_1 and at T_3 (β = 0.44) was partially mediated by endogenous LTDs (11%, β = 0.05, $p < .005$). Although the magnitude of this indirect experiential effect decreased over time, endogenous LTDs still explained 10% (β = 0.02, $p < .01$) of the 16-year association between neuroticism at T_1 and T_5 (β = 0.18, d = 0.47), whereas 85% was due to interposed neuroticism. Finally, neuroticism at T_4 mediated part of the effect of diminished quality of life on neuroticism at T_5 (β = 0.03, p = .05).

For experiential effects, the final model indicates that the effects of delayed endogenous LTDs on neuroticism at T_3 (over 3.5 years, β = 0.30 in final model) were 42% mediated by neuroticism at T_2 (β = 0.12, $p < .005$) and 58% unmediated ($p < .001$). The indirect experiential effects for endogenous LTDs extended to neuroticism at T_4 over 11.5 years (β = 0.17) and were mediated by direct experiential effects on neuroticism at T_2 (40%) and delayed experiential effects on neuroticism at T_3 (56%). Notably, these indirect effects on neuroticism were still significant at T_5 after more than 13.5 years (T_{x+4}, β = 0.12, all ps < .001). Finally, deteriorated life quality influenced neuroticism at T_5 indirectly via neuroticism at T_4 (β = 0.03, p = .05). Moreover, although the final model showed a small anomalous delayed increase in neuroticism at T_5 following improved quality of life (β = 0.14, $p < .01$), this was opposed by an indirect effect carried forward from neuroticism at T_4 (β = −0.09, p = .05); the net effect was negative, indicating a decrease in neuroticism at T_5 related to improved quality of life between T_3 and T_4.

Post hoc analyses. Narrow interpretation of the corresponsive principle would suggest that interposed neuroticism mediates all

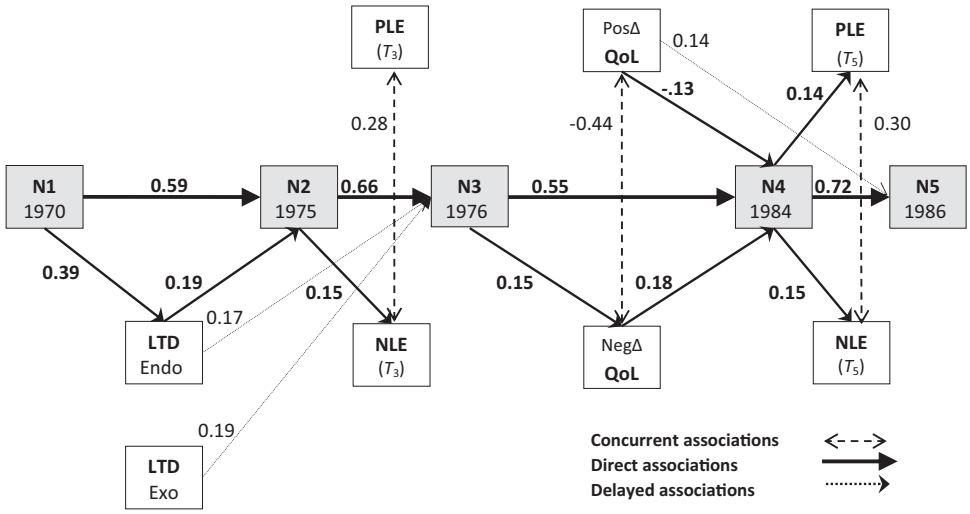

Figure 2. Final model: Mutual reinforcement between neuroticism and experiential effects over five waves and 16 years. Results are reported as standardized path estimates ($p < .05$) and can also be found in Table 4. The depicted paths are described in the Method section, the Results section, and Table 3 (and Table S3 of the supplemental materials). N_1 = neuroticism at baseline measured in 1970; LTD = long-term difficulties; Endo = endogenous; Exo = exogenous; PLE = positive life events; NLE = negative life events; PosΔQoL = improved life quality; NegΔQoL = diminished life quality; T_5 = at the fifth measurement wave. Definitions for concurrent (T_x), direct (T_{x+1}), and delayed (T_{x+2}) associations are given in the Method section.

associations between experiential measures. A fitted indirect effects model indeed showed that temporal associations between life event measures were rare. Although deteriorated life quality predicted more negative life event at T_5, this was mediated by the indirect effects of neuroticism at T_4 ($\beta = 0.03$, $p = .05$). Improved life quality had no direct effects on positive life events at T_5 ($p = .80$). However, endogenous LTDs (T_2) predicted diminished quality of life between T_3 and T_4 ($\beta = 0.21$, $d = 0.52$, $p = .01$), and this association was unmediated by neuroticism at T_2 or T_3 (both $ps > .40$). Apart from these, no other association between life event measures and mediation paths approached significance.

Discussion

This five-wave, 16-year study explored the magnitude and persistence of mutual reinforcement between neuroticism and life experiences. Our results suggest five key observations: (a) neuroticism showed high temporal stability, (b) LTDs and deteriorated life quality predicted lasting increases in neuroticism, (c) improved life quality predicted lasting decreases in neuroticism, (d) life event aggregates had no persistent impact on neuroticism, and (e) neuroticism predicted experiences more consistently than experiences predicted change in neuroticism.

These results support our hypothesis that neuroticism predicts life experiences better than life experiences predict neuroticism (Hypothesis 1), as indicated by more consistent neuroticism effects on life experiences and comparison of the largest observed effects for neuroticism versus life experiences ($d = 0.96$ vs. $d = 0.49$, respectively). Yet although neuroticism consistently predicted negative experiences (negative life events, LTDs, and deteriorated life quality), its effects on positive experiences were ambiguous. Only positive life events at T_5 were affected (which might be a chance effect). Effects of neuroticism on positive experiences are seldom reported and studied (see Lüdtke et al., 2011). Our results suggest that decreases in neuroticism may potentially evoke positive experiences. This finding merits further research. Nevertheless, our observation that, of all possible reciprocal effects in our model, experiential effects on neuroticism were more numerous than vice versa (40% and 25%, respectively, of 20 possibilities in both directions) is at odds with our first hypothesis.

Corresponsive Principle

This study aimed to clarify the rough contours of the corresponsive principle by examining several hypotheses (see the Hypotheses section above). Results indicate that LTDs and decreased quality of life increase the neuroticism set point (Hypothesis 2A) and that higher neuroticism scores predict more negative experiences (Hypothesis 2B), namely, LTDs, negative life events, and deteriorated life quality. We describe this reciprocity as a malignant cycle. Moreover, improved quality of life was associated with a decrease in the neuroticism set point (Hypothesis 3A) that, in turn, predicted more positive life events (Hypothesis 3B; although this was a variable effect). This latter finding suggests the possibility of a benign cycle (see also Jeronimus et al., 2013), in line with the corresponsive principle, which is an interesting addition to the literature. The magnitude of increases in neuroticism because of LTDs and decreased quality of life was slightly larger than the magnitude of the decreases in neuroticism evoked by improved life quality (25%), and these changes were more persistent (13.5 vs. 6 years, respectively), in line with Hypothesis 4. Together, our results support the hypothesized valence asymmetry in experiential effects that has been observed numerous times before (Baumeister et al., 2001).

Post hoc analyses showed that experiential measures were mainly unrelated to each other and, when related, were generally

Table 4
Magnitude and Persistence of Each Significant Path in the Final Model

Type and year	Path	Estimate	*SE*	Description
Stability				
1	11	0.66***	0.04	$N_2 \rightarrow N_3$
2	31	0.72***	0.04	$N_4 \rightarrow N_5$
5	1	0.59***	0.04	$N_1 \rightarrow N_2$
8	21	0.55***	0.05	$N_3 \rightarrow N_4$
		Neuroticism		
Direct				
0.5	12	0.15*	0.06	$N_2 \rightarrow (T_3)$ NLEs
1	30	0.14*	0.06	$N_4 \rightarrow (T_5)$ PLEs
1	32	0.15**	0.06	$N_4 \rightarrow (T_5)$ NLEs
2.5	2	0.39***	0.06	$N_1 \rightarrow$ LTDendo
4	22	0.15*	0.06	$N_3 \rightarrow$ NegΔQoL
Concurrent				
0	15	0.28***	0.06	PLEs↔NLEs (T_3)
0	33	0.30***	0.06	NLEs↔PLEs (T_5)
0	25	−0.44***	0.03	NegΔQoL↔PosΔQoL (T_4)
		Experiential		
Direct				
2.5	5	0.19***	0.05	LTDendo$\rightarrow N_2$
4	26	−0.13*	0.07	PosΔQoL$\rightarrow N_4$
4	27	0.18**	0.07	NegΔQoL$\rightarrow N_4$
Delayed				
3.5	8	0.17***	0.05	LTDendo$\rightarrow N_3$
3.5	9	0.19***	0.04	LTDexo$\rightarrow N_3$
6	28	0.14**	0.05	PosΔQoL$\rightarrow N_5$

Note. *SE* = standardized estimate; LTD = long-term difficulties; endo = endogenous; exo = exogenous; N_1 = neuroticism at the first measurement wave (T_1); N_2 = neuroticism at the second measurement wave (T_2); N_3 = neuroticism at the third measurement wave (T_3); N_4 = neuroticism at the fourth measurement wave (T_4); N_5 = neuroticism at the fifth measurement wave (T_5); NegΔQoL = diminished life quality; NLEs = negative life events; PLEs = positive life events; PosΔQoL = improved life quality; T_{x+1} = at the subsequent measurement wave; T_{x+2} = the second subsequent measurement wave. All details about all variables and each type of effect are reported in the Method section. Paths are depicted in Figure 1; the estimates are standardized (see the Method section for more information).
* $p < .05$, two-tailed. ** $p < .01$. *** $p < .001$.

fully mediated by interposed neuroticism. However, we observed one exception. Endogenous LTDs at T_2 forecast deteriorated life quality at T_4, and neither neuroticism at T_2 nor neuroticism at T_3 mediated this effect. This argues against a narrow interpretation of the corresponsive principle, in which all effects are carried forward by interposed neuroticism. However, these findings require replication. The total impact on neuroticism of previous neuroticism-driven exposure to experiential effects (selection) was relatively small ($R^2 < 7\%$), in accordance with most literature (Lüdtke, Trautwein, & Husemann, 2009; Riese et al., 2014; van Os, Park, & Jones, 2001). Nevertheless, these effects were persistent and may accumulate over time (reciprocal causation) as individuals seek, shape, and evoke life events and situations that match their personality, in line with the corresponsive principle (Caspi & Shiner, 2011). This is also known as the Dickens–Flynn model (Beam & Turkheimer, 2013). Thus, such feedback loops may become a substantial long-term influence on both neuroticism and life experiences.

Mixed Model of Change in Neuroticism

This study's key findings can be interpreted in terms of the mixed model of change in neuroticism, postulated by Ormel, Riese, and Rosmalen (2012). The mixed model refers to the idea that experiential influences commonly induce temporary fluctuations around the neuroticism set point but that the set point itself may also change over time. We regard changes in neuroticism that persist for many years (>13.5) to be indicative of neuroticism set point change. In line with the mixed model, aggregated life event measures showed no effect on the neuroticism set point in our model. Moreover, because neuroticism at T_3 was assessed, on average, only 6 months after the positive and negative life events occurred, life event effects must reflect only short-term fluctuations. This agrees with the literature (Ormel et al., 2012; Riese et al., 2014). Notably, this lack of impact cannot be explained by the assumption that life event aggregates tend to measure exogenous influences because exogenous LTDs impacted neuroticism at T_4 over 3.5 years.

Only major life change (LTDs and changed life quality) appeared to influence the neuroticism set point, whereas aggregated life event measures evoked, at best, short-term fluctuations in neuroticism. This agrees with other literature reporting long-term negative associations between change in life quality and neuroticism (Jonassaint, Siegler, Barefoot, Edwards, & Williams, 2011). These conclusions support the notion that patterns of increasing stability of neuroticism over time coincide with an increasingly stable environment, whereas change in neuroticism is associated with changes in the environment. This is known as the parallel-continuities hypothesis, in which personality is changed to cope with or adapt to new environmental conditions (Caspi & Shiner, 2011). Further research should seek to determine the specific environmental conditions that influence the neuroticism set point and the mechanisms that drive that influence.

Fine Structure of the Individual's Environment

The experiences we measured in this study accounted for 4%–7% of the change in neuroticism at each wave. However, individual change in neuroticism over long periods was substantial (test–retest reliability over 16 years, observed $r = .41$), in line with the literature (Ormel et al., 2013). Part of this change undoubtedly reflects measurement error or new genetic expression, but twin studies suggest that most change must somehow reflect systematic experiential influences (Kandler et al., 2010; Laceulle et al., 2013; Riese et al., 2014). Major life events do not explain more than about 10% of the change in neuroticism, which suggests that most change in neuroticism must have other sources that remain largely unknown.

More change in neuroticism may be explained if researchers map changes in the whole human socioecology, conceived by Bronfenbrenner (2005) as a network of hierarchically nested, interconnected systems. At the basic level, individuals are embedded in microsystems such as their family of origin, friends or peer group, school, workplace, partner relationship (family of destination), and neighborhood. These microsystems form the social context in which individuals experience enduring patterned activities, social roles, and lasting interpersonal relationships (as partner, parent, worker, or friend). Within microsystems, individuals encounter sociocultural webs of expectations, commitments, and

obligations, and the immediate interactions (proximal processes) are most profoundly affected by traits like neuroticism (Bronfenbrenner, 2005; Nettle, 2007; Roberts, Wood, & Smith, 2005).

In this article, we have emphasized that individuals shape and evoke the microsystems they inhabit (Caspi et al., 2005; Caspi & Shiner, 2011). Microsystems contain the proximal processes that drive the dynamic feedback loops through which genotypes express themselves as phenotypes (Bronfenbrenner, 2005; Buss, 2011; Ridley, 2003), for example, the work- and family-role-related influences that drive the maturational decrease in neuroticism between the ages of 25 and 45 years (Bleidorn et al., 2013; Roberts & Wood, 2006; Roberts et al., 2005). Once enduring experiential effects alter proximal processes, it may spark autocatalytic amplification (which we have called malignant and benign cycles) that can result in a system shift (Cramer et al., 2012). With this system shift, a new neuroticism set point persists beyond the transient direct effects of the initial experience (Fraley & Roberts, 2005; Ormel et al., 2012; Turkheimer & Waldron, 2000).

Once proximal processes within a system change, feedback loops may prevent regression to the old neuroticism set point, perhaps through secondary experiences (positive or negative) or spillover between microsystems. The system shift will persist if the new set point becomes anchored in newly structured proximal processes, such as change in social support, resources, identity, habits, or life events. Conceivably, each major flux in a microsystem may trigger a feedback loop and alter the network properties. Such effects have been observed after the formation of new romantic partner relationships or divorce (Mroczek & Spiro, 2003), preterm childbirth (Kersting et al., 2004), and dismissal or promotion (Sutin et al., 2009), for example.

The aggregate of all microsystems is called the mesosystem. The mesosystem changes via normative experiences, for example, entrance events (e.g., beginning school, work, relationships, or parenthood), exit events (e.g., divorce or dismissal), and accidents or disease (Bleidorn et al., 2013; Bronfenbrenner, 2005). The enduring difficulties or changes in an individual's quality of life that we studied may result in microsystem spillover and eventually affect the entire mesosystem and its many related proximal processes. For example, dismissal can lead to divorce, change of residence, or loss of social contacts. Experiential effects on the stability of neuroticism can be understood similarly (see the introduction). Individuals tend to shape their mesosystem in a way that promotes an increasing stability of the neuroticism set point with age (Caspi et al., 2005; Fraley & Roberts, 2005; Roberts & DelVecchio, 2000). Finally, different mesosystem characteristics and the unique combination of traits in each individual may explain individual differences in the rate of change in neuroticism (Billstedt et al., 2014; Bleidorn et al., 2009; Mroczek & Spiro, 2003; Small, Hertzog, Hultsch, & Dixon, 2003; Soto & John, 2012). These developmental perspectives on individual variation could be tested with a substantial sample of individuals whose day-to-day experience and surroundings are measured in time series at different points throughout their life (Molenaar, 2008).

Limitations and Future Directions

Our study results should be interpreted in light of the following strengths and limitations. Retrospective self-reports of life events inherently incorporate response components that may be influenced by current mental state, such as cognition, appraisal, interpretation, and recall (Dohrenwend, 2006; Monroe, 2008; Schwarz, 2007). Our panel ratings (see the Method section) address this bias by producing life event ratings independent of the respondent. Second, a maximum-likelihood application on multivariate data is, at best, only an approximation of reality, and "even the best possible model fit may not protect one from meaningless results" (Bentler & Chou, 1987). We believe, however, that our sample came from a relevant population for evaluating the postulated hypothesis and that the final model contained sufficient degrees of freedom. Moreover, we interviewed 296 participants five times over 16 years and assessed life events, LTDs, and life situation change with interviews and panel ratings, the current gold standard (Dohrenwend, 2006; Monroe, 2008). This methodology allowed for empirical testing of the corresponsive principle (reciprocal causation) that most studies are poorly suited to undertake. The small mean-level fluctuations we reported presumably reflect sociocultural events, random fluctuations, and measurement error (Roberts et al., 2006).

Finally, our use of different measures at different time periods (due to financial and methodological constraints) may have been confusing and perhaps accounts for part of the differences in effect sizes. However, we think it is unlikely that it has produced a major bias. Moreover, the use of different measures resulted in an unintended advantage as well: We now could compare the associations between different types of measures of change in a participant's life situation and change in neuroticism.

Conclusion

The results of this study indicate that neuroticism, an enduring personality trait, consistently predicts negative experiences, whereas its effects on positive experiences are variable. Long-term difficulties and deteriorated life quality predicted small but persistent increases in neuroticism, whereas improved life quality predicted small long-term decreases in neuroticism. This suggests set point change. However, positive and negative life event aggregates had no effect on neuroticism. Together, these results align with the corresponsive principle, the plasticity principle, and the mixed model of neuroticism, which distinguishes temporary changes in neuroticism from persistent changes in an individual's set point. Our results emphasize a mutually reinforcing relationship between phenotype and environment. Consequently, future studies of neuroticism should focus on the structure of the environment that individuals inhabit (their mesosystem topologies) and ideographically track the continuing effects of specific life-changing experiences or changes in quality of life over a lifetime (see Friedman & Martin, 2011). Finally, we hope that the potential we observed for a benign cycle helps to create prevention strategies to target the vulnerability to mental disorders inherent in neuroticism rather than treating the subsequent manifestations of those disorders.

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Received April 25, 2013
Revision received March 7, 2014
Accepted April 24, 2014 ■