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Environmental influences on neuroticism : a story about emotional (in)stability

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Document Version

Publisher's PDF, also known as Version of record

Publication date:

2015

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Jeronimus, B. (2015). *Environmental influences on neuroticism : a story about emotional (in)stability*. [S.n.].

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Chapter 9

Timing of Stressful Life Events Affects Stability and Change of Neuroticism

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European Journal of Personality 2014, 28, 2, 193-200.

ABSTRACT

Neuroticism is a predictor of many health problems. To study the determinants of within-subject change in neuroticism, three hypotheses were tested: (i) subjects who experienced stressful life events (SLEs) show an increase in neuroticism; (ii) high baseline neuroticism moderated this effect; and (iii) recent SLEs had a greater impact on neuroticism than distant SLEs. Data came from the Finnish Twin Cohort. Neuroticism data were collected in 1975 and 1981 and SLEs data in 1981 ($n = 21,085$). By entering baseline neuroticism as a predictor for neuroticism at follow-up, the outcome measure was change in neuroticism. Changes in neuroticism were predicted from SLE indices or their interaction with baseline neuroticism. Timing of SLEs was taken into account by distinguishing recent from distant SLEs. To control for confounding by shared genes and environments, both within-twin pair and between-twin pair effects were tested for monozygotic and dizygotic twin pairs separately. Neuroticism's six-year stability was high ($r = .58, p < .001$). Exposure to SLEs modestly increased neuroticism ($bs > .55, p < .001$), unconfounded by shared genes. This effect was not moderated by high baseline neuroticism. Recent SLEs ($.09 < bs < .15$) had more impact than distant SLEs ($.03 < bs < .11; p < .01$). In conclusion, the findings strongly supported a model of environmentally driven SLEs causing dynamic fluctuations around a person's set point of neuroticism

INTRODUCTION

The personality trait of neuroticism is a powerful predictor of a wide variety of both mental and physical health problems [52,281]. Low neuroticism scores have even been shown to predict longevity [52]. High neuroticism scores are strongly associated with current and lifetime mental disorders, especially affective disorders [281]. Moreover, the economic burden of neuroticism to the society as a whole is enormous and exceeds those of common mental disorders [55]. Although highly relevant, the relationship between experienced stressful life events (SLEs) and fluctuations, or changes, in scores on a neuroticism inventory is an understudied topic.

Models of personality traits often conceptualize neuroticism as stable during the adult life course [985]. Indeed, the mean level of neuroticism in a population remains fairly stable after the age of 30 years, after an increase during young adulthood and followed by a slight decrease in later life [163,986]. However, meaningful changes in neuroticism at various ages have been reported as well [85,164,331,339]. Individuals are known to differ in their rate of change in neuroticism; some are more inclined to remain stable over time, but others change in their level of this trait [331]. Moreover, the rank-order stability of neuroticism from age 21 years drops steadily from .55 after a year, to .41 over 20 years, to .25 after 40 years, indicating within-subject changes in neuroticism [164,236,328]. Currently, the exact determinants causing within-subject changes in neuroticism scores are largely unknown, although the role of SLEs may be more important than assumed [328,599].

Prior studies suggested that life events can both decrease and increase neuroticism scores. For example, promotions at work and transitions into romantic relationships/marriage have been associated with decreases in neuroticism [331,484,987], whereas job loss, divorce, death of a spouse, serious accidents and severe illnesses have been associated with increases in neuroticism [172,239,331,339,346]. In the current study, we explicitly tested for change in neuroticism scores by entering baseline neuroticism as a predictor for neuroticism at follow-up. Moreover, in an additional model, moderation effects of baseline neuroticism on the association between SLEs and change in neuroticism were tested [339].

We investigated changes in neuroticism scores and the influence of self-reported SLEs on those changes in a six year follow-up study. Because correspondence between different methods to assess SLEs is relatively low [271], two of the most often used indices were applied. First, the 'life change unit (LCU) weights' originating from the Social Readjustment Rating Scale (SRRS) were used to calculate a weighted sum score on the basis of the extent SLEs are assumed to require adaptive behaviour after experiencing the particular SLE. The LCU weights were based on panel ratings for each SRRS event [366]. Second, because the accumulation of negative SLEs has been

shown to predict the onset of psychopathology [271,988], a weighted sum score was calculated in which the weight of each SLE was based on its prevalence. In this study, all hypotheses were tested for both indices separately.

Because variation in both neuroticism [989,990] and, although maybe counter intuitive, SLEs [412,991] have a genetic component, it is also important to take into account any potential shared genetic influences between neuroticism and SLEs. Co-twin control studies enable control for shared genetic factors in addition to shared environments. Differences in findings for monozygotic (MZ) and dizygotic (DZ) twin pairs in such studies allow for conclusions on the genetic or environmental origins of changes in neuroticism attributed to SLEs. Therefore, the analyses were carried out separately for, and the results compared between, MZ and DZ twin pairs [992].

Changes in neuroticism scores have been argued to reflect dynamic state fluctuation around an individual's specific negative affect set point or trait neuroticism level [94,114,347]. However, different models for underlying mechanisms causing changes in neuroticism scores across the adult life span have been proposed [94]. In a recent review, we [94] evaluated three neuroticism set point models. First was the immutable set point model in which SLEs may induce short-term perturbations in neuroticism but scores are deemed to return decisively to their individual's person-specific set points. However, only little support was found for this model. More evidence was found, although still inconclusive, for support for the other two models. Second was the experience-dependent model in which it is assumed that set points can change persistently when instigated by SLEs that become biologically, cognitively or environmentally embedded in an individual. Although it has been argued that this 'state-deviation' may reflect a true change in set point neuroticism [348,354], we argued in our review for a more complex third 'mixed model'. In this latter model, the mechanisms of the foregoing models were combined; SLEs are considered to induce temporally short-term rather transient state-like perturbations around a relatively stable neuroticism set point, which itself is able to change over lifetime. In sum, such set point shifts reflect changes beyond the temporary perturbations owing to SLEs and measurement error.

The impact of SLEs on affect typically decays in about three months [272,345]. If and how timing of SLEs is related to changes in neuroticism scores is largely unknown, but it is assumed to play an important role. In this study, data were available on the timing of reported SLEs. These were used to distinguish between recent (the last six months) and distant (the last five years excluding the last six months) SLEs and as predictors for changes in neuroticism scores. Although assuming that neuroticism's set point is relatively stable, dynamic fluctuations in neuroticism scores might be more strongly affected by recent, compared with more distant, SLEs. In sum, in this prospective study, we tested the hypotheses that (i) subjects who experienced SLEs showed an increase in their neuroticism scores; (ii) high baseline neuroticism scores moderated

this effect, and based on the mixed model that predicts the decaying impact of SLEs on neuroticism over time; (iii) recent SLEs had a larger impact on these changes in neuroticism scores compared with distant SLEs.

METHODS

Study Population

Data came from the Finnish Adult Twin Cohort, a large prospective population-based twin study. It includes all Finnish same-sex twin pairs born before 1958 of which both twins of a pair were alive in 1975. Data presented in this paper are from the first (T_1 , 1975) and second (T_2 , 1981) assessment waves. A more detailed description of the cohort has been described elsewhere [993,994]. Only the data of subjects who had completed both the 1975 and 1981 neuroticism surveys were used in the current study ($n = 21\,085$). Excluded subjects with incomplete neuroticism data ($n = 1132$ who participated only once) did not differ from included subjects on neuroticism scores in 1975 or 1981. Subjects were on average 41.6 years old ($SD = 14.6$, range = 24–101) in 1981, and 50% were women. Subjects were informed about the study both before and after the surveys and were able to withdraw from the study. The project was accepted by the Ethical Committee of the Department of Public Health, University of Helsinki.

Measures

Neuroticism

At both T_1 and T_2 , neuroticism was assessed with the same self-report short form of the Eysenck Personality Inventory [85]. This scale consists of 10 items. Subjects had to answer in a yes (= 1)/no (= 0) format (theoretical range of the sum score = 0–10). Missing items were replaced by the mean value of the remaining scale items. Up to two missing items were allowed when calculating the neuroticism sum scores. The Cronbach's alphas were .73 (T_1) and .71 (T_2).

Stressful Life Events

Assessment of stressful life events The SLEs experienced in the period between T_1 and T_2 were assessed at T_2 , by means of a self-report questionnaire. Subjects were asked to indicate which SLEs they had encountered and to specify the timing of the events as never, during the last six months, during the last five years or earlier. An extensive description of this 21-item life-event inventory and its development is given in an earlier publication [995]. We formulated two different weighted summary indices on

the basis of these reported life events. Weights for the individual SLE items are given in Table 32.

The first index was a weighted life change sum score (the Holmes and Rahe index). For each of the 17 Holmes and Rahe life change-related events, the SLE-specific Holmes and Rahe's importance weight was used [1995]. Of the 21085 subjects with

Table 32. Prevalence (%) of Recent and Distant Stressful Life-Events (SLEs) of Each SLE Reported. The Holmes and Rahe Weights, and the Weights Based on the Inverse of the Life-Time Prevalence for the Selected Negative SLEs are Given.

Stressful Life-events	Prevalence SLEs (%)		Stressful Life-event index	
	Recent	Distant	Holmes and Rahe ^b	Inverse prevalence Negative SLEs ^c
Death of spouse	0.3	1.4	100	.9529
Divorce or separation	1.1	3.6	69	.9107
Disease or injury causing over three weeks work disability	5.4	12.8	53	.7310
Death of close relative or good friend	11.8	31.6	50	.3365
Loss of job	2.4	5.1	47	.8911
Marked change in the health of a family member (not death) ^a	7.0	10.6	44	.7671
Increase in family size	5.4	19.3	39	
Difficulties of a sexual nature	7.8	7.5	39	.8112
Marked worsening in financial situation	9.8	8.2	38	.7684
Change to another kind of work	6.9	16.5	36	
Marked increase in difficulties with spouse (not divorce)	5.7	6.3	35	.8460
Bank loan equivalent to over six month's earnings	10.1	21.8	31	
Marked increase of responsibility at work	11.0	17.2	29	
Departure of family member from home	5.3	11.1	29	
Marked difficulties with superiors, colleagues or subordinates at work	5.2	4.7	23	.8753
Marked increase in work load ^a	15.1	18.0	20	.6179
Change of home (residence)	12.4	34.5	20	

Note. Recent stressful life-events (SLEs) were defined as having encountered SLEs during the last six months. Distant SLEs were defined as having encountered SLEs during the last five years (excluding the events reported during the last six months). Negative SLEs were selected by having the 17 Holmes and Rahe items rated by four experts on SLEs on being negative, positive or ambiguous. There was consensus among the raters on 9 items as being negative and there was disagreement on the 2 items marked with ^a (see method section for further details). ^bThe weight of an individual SLE on the Holmes and Rahe scale (Holmes and Rahe, 1967); ^c The weight of an individual negative SLE based on the prevalence (calculated as the total number of reported SLEs) in the current sample (see method section for further details).

complete neuroticism data, 1679 (7.96%) had more than three items missing on this first SLE index and were excluded, leaving 19406 subjects for the analyses. Analogous to Lillberg et al. [1995], if subjects had ≤ 3 missing SLE items, these were coded as 0 (*i.e.* not experienced).

The second SLE index was a weighted negative SLE sum score (the Negative SLE index). Negative SLEs were selected by having the 17 Holmes and Rahe items rated by four experts on SLEs on being negative, positive or ambiguous (Cronbach's alpha = .96). There was consensus among the raters on nine items as being negative and disagreement on two items (see note below Table 32 for further details). Robustness analyses using either 9 or 11 items for the calculation of this index showed that the findings were highly comparable, and therefore, the findings of the 11-item sum score were reported and discussed in the paper.

On the basis of prior findings that the impact of SLEs with low frequency is higher compared with those with high frequency [367], the weights for the Negative SLE index were calculated as the inverse of the lifetime prevalence (1 - prevalence) of each selected negative SLE within our cohort. Prevalence was defined as ever having experienced the specific SLE. Of the 21085 subjects with complete neuroticism data, 1705 (8.09%) subjects had more than two items missing on this second index and were excluded, leaving 19,380 subjects for the analyses. If subjects had ≤ 2 missing SLE items, these were coded as 0 (*i.e.* not experienced). In order to take the timing of the reported SLEs into account, both SLE indices were calculated for the (i) recent SLEs reported as experienced during the last 6 months preceding T_2 and (ii) distant SLEs reported as experienced during the last 5 years (excluding the events reported during the last 6 months). Previous studies have indicated that the impact of SLEs on affect typically decays in about 3 months [272,345], which supports the validity of using the last 6 months as a cutoff for recent SLEs.

Statistical Analysis

The distribution of neuroticism scores was approximately normal. All variables were standardized to a mean of 0 and an SD of 1 to obtain internally comparable regression (or beta) coefficients. Interaction terms were created by multiplying the z-transformed neuroticism and SLE indices.

Means (SD) of the study variables were calculated. Differences were analysed by (paired) *t*-tests and associations by Spearman correlations. Changes in neuroticism scores in 1981 after the 6-year follow-up due to experienced recent and distant SLEs were tested for the Holmes and Rahe and Negative SLE indices. Age and gender were entered as predictors in all models to adjust for their potential confounding influences. In the first model, neuroticism in 1981 was the dependent variable predicted by neuroticism in 1975, and recent and distant SLEs. By entering baseline neuroticism as

a predictor for follow-up neuroticism, the results can be interpreted as changes in neuroticism scores. In the second model, to test whether baseline neuroticism moderated the association of SLEs with neuroticism in 1981, a Neuroticism1975_SLEs interaction term was added to the model described earlier.

A specialized regression method for twin data, based on mixed models, was used to allow for the simultaneous examination of both within-twin pair and between-twin pair effects of SLEs on change in neuroticism scores [992]. The within-twin pair effect represents an effect on change in neuroticism controlled for confounding influences that are shared within a twin pair. The between-twin pair effects represent a variation in the change of neuroticism that can be explained by the variation in the twin-pair mean of SLEs. The Wald test was used to test whether the within-twin pair and between-twin pair effects differed from each other [992]; non-significant differences indicated that effects are unconfounded by influences shared by the twin pairs. In such a case, the distinction between within-twin pair and between-twin pair effects did not provide additional information, compared with a regular regression analyses in which the paired nature of the data was taken into account.

Analyses were performed for within-twin pair and between-twin-pair effects of MZ and DZ twin pairs separately to benefit from the informative twin design. This design relies on the assumption that MZ twins are genetically identical, whereas DZ pairs share on average 50% of their segregated genes, and both twins of a pair share part of their environment (*e.g.*, family), and are also exposed to influences that are not shared by a twin pair (individual-specific or unique environmental influences). In within-pair analyses, the potentially confounding impact of genetic makeup on the relationship between SLEs and change in neuroticism is reduced for DZ pairs and eliminated in MZ pairs. Furthermore, differences in findings for MZ and DZ twin pairs allow for conclusions on the genetic or environmental origins of the changes in neuroticism, which can be attributed to SLEs. If SLEs and change in neuroticism shared their genetic origins, the within-twin pair regression coefficient was expected to be larger for DZ (only 50% controlled) than for MZ pairs (100% controlled). Analyses were performed with Stata software (version 11.1/SE; StataCorp LP, College Station, TX, USA). In this large sample, a $p < .01$ was considered statistically significant.

RESULTS

Mean neuroticism scores slightly decreased during the 6-year follow-up [M1975 = 4.35, (SD= 2.51), M1981= 4.09 (SD= 2.43), $p < .001$], but neuroticism showed high temporal stability given the imperfect reliability of the questionnaires ($r = .58$, $p < .001$). Recent SLEs correlated negatively, or not at all with distant SLEs ($r = .06$, $p < .001$ for

the Holmes and Rahe index; $r = .002, p = .79$ for the Negative SLE index). Baseline neuroticism was associated with both recent and distant SLEs exposure ($r = .12$ and $r = .04$ for the Holmes and Rahe index, $r = .14$ and $r = .08$ for the Negative SLE index, respectively). Recent and distant SLEs in turn were associated with neuroticism at follow-up somewhat more strongly ($r = .15$ and $r = .05$ for the Holmes and Rahe index, $r = .19$ and $r = .11$ for the Negative SLE index, respectively; all $p < .001$). The correlations between the two SLE indices were $r = .84$ and $r = .81$ ($p < .001$) for the recent and distant SLE indices, respectively.

Table 33. Within- and Between- Twin Pair Estimates of Standardized Regression Coefficients [and 95% Confidence Intervals] Representing Linear Associations Between Changes in Neuroticism Scores and the Holmes and Rahe Index and the Negative Stressful Life-Event Index, respectively, for Monozygotic (MZ) and Dizygotic (DZ) Twin Pairs Separately. Analyses Were Controlled for Gender and Age

Predictor	MZ twins			DZ twins			
	β	95% CI	p	β	95% CI	p	
Holmes Rahe index							
Neuroticism 1975	.56	[.54, .59]	<.001	.56	[.55, .58]	<.001	
Recent SLEs	Between-pair	.13	[.10, .16]	<.001	.11	[.09, .13]	<.001
	Within-pair	.10	[.07, .13]	<.001	.09	[.07, .12]	<.001
Neuroticism 1975 x Recent SLEs	Between-pair	.02	[-.003, .05]	.08	-.01	[-.04, .006]	.18
	Within-pair	.04	[.008, .07]	.013	.02	[-.004, .04]	.11
Distant SLEs	Between-pair	.07	[.04, .10]	<.001	.03	[.01, .05]	.004
	Within-pair	.05	[.01, .08]	.005	.05	[.03, .07]	<.001
Neuroticism 1975 x Distant SLEs	Between-pair	-.003	[-.03, .03]	.86	.006	[-.02, .03]	.60
	Within-pair	-.002	[-.03, .03]	.92	.03	[.003, .05]	.03
Negative Stressful Life-event index							
Neuroticism 1975	.55	[.53, .57]	<.001	.55	[.53, .56]	<.001	
Recent SLEs	Between-pair	.15	[.12, .18]	<.001	.14	[.12, .16]	<.001
	Within-pair	.11	[.08, .14]	<.001	.13	[.11, .15]	<.001
Neuroticism 1975 x Recent SLEs	Between-pair	.007	[-.02, .03]	.60	-.01	[-.03, .009]	.25
	Within-pair	.03	[.004, .07]	.03	.008	[-.01, .03]	.49
Distant SLEs	Between-pair	.11	[.08, .14]	<.001	.07	[.05, .09]	<.001
	Within-pair	.07	[.04, .10]	<.001	.08	[.06, .10]	<.001
Neuroticism 1975 x Distant SLEs	Between-pair	.01	[-.02, .04]	.41	.01	[-.009, .04]	.23
	Within-pair	-.010	[-.04, .02]	.53	.02	[-.003, .04]	.10

Note. Recent stressful life-events (SLEs) were defined as having encountered SLEs during the last six months. Distant SLEs were defined as having encountered SLEs during the last five years (excluding the SLEs reported during the last six months). MZ = monozygotic twins; DZ = dizygotic twins. In the analyses with the Holmes and Rahe index: $n_{MZ} = 5801, n_{DZ} = 12343, n_{unknown\ zygosity} = 1262$. In the analyses with the Negative Stressful Life-events index: $n_{MZ} = 5790, n_{DZ} = 12326, n_{unknown\ zygosity} = 1264$.

As indicated by the largely overlapping 95% confidence intervals (CIs), findings for the analyses using the Holmes and Rahe and Negative SLE indices as well as findings for the MZ and DZ twin pairs were highly comparable (Table 33). When change in neuroticism was predicted by the number of reported recent and distant SLEs, recent SLEs showed a stronger effect compared with the distant SLEs. However, there was no evidence that SLEs elicited more change in neuroticism scores among the subjects with higher baseline neuroticism scores, as indicated by the lack of significant Neuroticism1975_SLEs interactions. Moreover, the nonsignificant Wald tests ($.05 < p < .72$) indicated that the addition of within-twin pair and between twin-pair factors did not improve the fit of the model (also illustrated in Table 33 by the overlapping 95% CIs of the coefficients for the between-pair and within-pair differences coefficients).

On the basis of these findings, post hoc analyses without within-twin pair and between-twin pair effects collapsed over MZ and DZ twin pairs were performed. In these post hoc analyses, robust estimators of variance when estimating standard errors (cluster option in Stata [1996]) were used and gave indeed similar findings as the foregoing analyses. In these latter analyses, the stronger effect of recent SLEs compared with the distant SLEs was significant, as indicated by the non-overlapping 95% confidence interval of the coefficients (Table 34).

Table 34. Estimates of Standardized Regression Coefficients [and 95% Confidence Intervals] Representing Linear Associations Between Changes in Neuroticism Scores and the Holmes and Rahe Index and the Negative Stressful Life-Event Index. Analyses Were Controlled for Gender and Age

Predictor	β	95% CI	p	β	95% CI	p
Holmes Rahe index						
Neuroticism 1975	.57	[.56, .58]	<.001	.57	[.56, .58]	<.001
Recent SLEs	.11	[.10, .12]	<.001	.11	[.10, .12]	<.001
Neuroticism 1975 x Recent SLEs				.01	[.001, .02]	.03
Distant SLEs	.05	[.04, .06]	<.001	.05	[.04, .06]	<.001
Neuroticism 1975 x Distant SLEs				.01	[.001, .02]	.09
Negative Stressful Life-event index						
Neuroticism 1975	.56	[.55, .57]	<.001	.56	[.54, .57]	<.001
Recent SLEs	.14	[.13, .15]	<.001	.14	[.12, .15]	<.001
Neuroticism 1975 x Recent SLEs				.008	[-.003, .02]	.16
Distant SLEs	.08	[.07, .09]	<.001	.08	[.07, .09]	<.001
Neuroticism 1975 x Distant SLEs				.01	[.0002, .02]	.05

Note. Recent stressful life-events (SLEs) were defined as having encountered SLEs during the last six months. Distant SLEs were defined as having encountered SLEs during the last five years (excluding the SLEs reported during the last six months). In the analyses with the Holmes and Rahe index: $n = 19406$; in the analyses with the Negative Stressful Life-events index: $n = 19380$.

DISCUSSION

In this six-year follow-up study among adults, neuroticism showed high temporal stability. Nevertheless, individuals who experienced SLEs also experienced increased neuroticism, independent of their baseline neuroticism scores. Results for the two different SLE indices were largely similar and lead to the same conclusions. Recent SLEs had a larger impact on neuroticism than distant SLEs, suggesting that the effect of SLEs decayed over time. This indicated that it is important to take the timing of experienced SLEs into account. Moreover, neither differences between within-twin pair and between twin pair effects nor differences across MZ and DZ twin pairs were found, which indicated that the effects of SLEs on change in neuroticism was solely due to influences that were not shared by a twin pair, also known as unique environmental influences.

These findings should be interpreted in light of the following strengths and limitations. The strengths included that our distinction between recent and distant SLEs provided the opportunity to examine the temporal effects of SLEs on change in neuroticism scores. A further strength was that this sample is based on data from a national population cohort representative of the Finnish adult population. Moreover, the very large sample size gave enough power to test more advanced models and detect small-effect sizes. SLEs only had a small effect on change in neuroticism scores, which was comparable with findings reported by others [222,292]. Limitations of the study were that neuroticism was measured only twice and SLEs only once and not before the first neuroticism measurement (see also next paragraph). Moreover, we did not test or discuss possible effects of genotype–environment correlations as carried out by others [29,235,997] because this, although also very interesting and relevant, was beyond the scope of the current paper.

Findings of the current study provided evidence that an adult's relatively stable neuroticism score increased in response to SLEs. The increase was at least partially temporary. With the decay of the impact of experienced SLEs, neuroticism seemed to return towards an individual's set point. Our findings were in line with the 'mixed model' in which neuroticism scores are presumed to result from both a relatively stable set point and temporary experiencedependent changes around this set point [94]. However, because it remained unclear whether the small impact of distant SLEs further decayed, the data were inconclusive regarding the potential of SLEs to shift a person's set point permanently. Long-term longitudinal studies with at least three assessment waves are needed to test this hypothesis in more detail [94].

Analogous to a prior study [339], baseline neuroticism was associated with subsequent SLEs. This finding suggested that individuals with high neuroticism scores selected themselves into environments in which they experience most likely person-

dependent SLEs [380] or reported more SLEs owing to their recall bias for negative memories and SLEs [33,155,998]. Although not absent, this kind of bias might be smaller when using an interactive interview [270,385]. However, compared with questionnaires, interviews are more time-consuming and costly and will thus lead to smaller samples and fewer studies [377]. Moreover, individuals may be less likely to report embarrassing information [386]. There have also been attempts to improve the validity of questionnaire-based self-reports of SLEs, for example, through ratings of independent experts on their level of objectivity and stressfulness [629]. However, no consensus on the best method to assess life events has been established yet.

In the current study, similar results were found for the two different SLE indices used. Indeed, the indices corresponded with each other in that they did not only yield the same number and types of SLEs experienced but were also both constructed from the same pool of questionnaire items of which the same two items (*viz.*, ‘death of spouse’ and ‘divorce or separation’) received the highest weights in both indices. The major difference between the two indices was the origin of the weights of the individual SLE used for the weighted sum scores. The weights of the Negative SLE index were based on the prevalence of the SLEs in a very large sample ($n > 20000$) of the Finnish population studied in the late 1970s/early 1980s. The weights of the Holmes and Rahe index were based on severity ratings by respondents from a US sample ($n = 394$) studied in the mid-1960s who had experienced the event [366]. Despite the differential origins of the weights for the SLEs, the comparableness of the results for the two indices attested to the robustness of our findings, although it should be noted that it is difficult to speculate to what extent the findings, based on data assessed more than 30 years ago, are still valid nowadays.

Future studies are needed to gain insight into the specific SLEs that induce changes in neuroticism and thus its (temporal) change in vulnerability to develop psychopathology. Another step forward would be to take the underlying facets of neuroticism (*e.g.*, impulsivity and anxiety) into account. Facets have been shown to be selectively affected [346] by SLEs and might possibly also be differentially affected by recent and distant SLEs. Moreover, there is some evidence that controllable stress paired with adequate social support tended to reduce neuroticism scores [636]. This topic is beyond the scope of our study but warrants attention in future studies. In particular, because most SLEs cannot be avoided, successful coping with them could prevent both increases in neuroticism scores and its concurrent increase in vulnerability to develop (psycho-)pathology. Finally, future research should focus on favourable changes in neuroticism scores (decrease) due to positive life events. There is some evidence for this [483], and a recent study showed that its effect seemed stronger compared with negative SLEs, at least in a partly clinical sample [222].

In conclusion, our results supported the notion that neuroticism is a fairly stable personality trait. Negative SLEs produced modest increases in neuroticism, but these changes, at least partially, decayed over time. It remained unknown whether the SLE effect will either ultimately decay completely or can even induce a lasting set point change that persists beyond the temporary perturbations due to SLE effects. However, the findings strongly supported a model of SLEs driving dynamic fluctuations around a person's setpoint of neuroticism. Our findings suggested that these fluctuations were not driven by baseline neuroticism scores or genetic influences but mostly by environmental influences of SLEs.

Acknowledgements

This work was supported in parts by grants from the Academy of Finland Center of Excellence in Complex Disease Genetics (Grants 213506 and 129680).

