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

Jeronimus, Bertus F.

Front cover
Chapter 10

Discussion
Q8. “Despite many critiques of the practice of dividing up behaviour into the innate and the acquired, the habit dies hard”.

*Patrick Bateson, 2007 [999].*

Q9. “The truth is that our finest moments are most likely to occur when we are feeling deeply uncomfortable, unhappy or unfulfilled. For it is only in such moments, propelled by our discomfort, that we are likely to step out of our ruts and start searching for different ways or truer answers.”

*M. Scott Peck (1936-2005).*

Q10. “Undoubtedly genetic variance is a significant cause of much phenotypic variance in observed populations, but its effects may be overridden, modified, enhanced or reversed by other causes. Genes may modify the effects of other genes, and may modify the effects of the environment. Environmental events, both internal and external, may modify the effect of genes, and may modify the effects of other environmental events”.

*Richard Dawkins, 1982 [263].*

Q11. “It might be said that emotions are the weather of our lives. Some days, we experience the blue skies of happiness and the sunshine of joy. Other days, we are drenched by the rain clouds of sadness or buffeted by the hot winds of anger. How we respond adaptively to our emotional weather patterns - finding the silver lining in every dark cloud - has important consequences for our physical and mental wellbeing. Although we cannot control the weather outside, we are capable of using myriad emotion regulation strategies to take control of our internal climates. Such strategies allow us to wholly or partially alter the nature, magnitude, and duration of our emotional responses, including initiating new ones.”

*Kevin Ochsner, 2012 [1000].*

Q12. “In no class of disease treated in the hospital have the results been better than in that of Hysteria and nervous exhaustion. It is often difficult to treat such patients in their home, where their own friends are frequently their worst enemies”.

*Medical registrar in Queen Square Hospital, Annual Report, 1883 [1001].*

Q13 “Only in man does man know himself, life alone teaches each one what he is”.

*Goethe, Tasso, act 2, sc. 3.*
SUMMARY OF THE RESEARCH AIM

In this PhD thesis I investigated whether and how environmental influences predict change in neuroticism and how long such changes persist (chapter 2-4, 6 & 9) and examined the meaning of the strong prospective association between neuroticism and the common mental disorders (chapter 2, 5 & 6).

Changes in neuroticism after stressful life events (SLEs) and changes in life situation were studied in a systematic review (chapter 4) and a series of longitudinal studies (chapters 2, 3, 6 and 9). To reduce the genetic confounding of life event occurrences, we selected studies with a specific research design in this review: studies of monozygotic (MZ) twin pairs discordant for neuroticism, and longitudinal studies of within-individual changes in neuroticism in adults, in which neuroticism was measured twice, at least one year apart. Lastly, we performed a twin study of neuroticism and SLEs. Results shall be interpreted below in terms of the so-called ‘mixed model of change in neuroticism’, in which short-term state fluctuations around a person specific setpoint of neuroticism are distinguished from long-term changes in the setpoint of neuroticism itself, and in terms of the corresponsive principle, the social investment principle, and the red queen personality principle.

Additionally, I critically examined the strong prospective association between neuroticism and the common mental disorders (CMDs, viz., anxiety, depressive, and substance use disorders; chapter 5), which is often used to suggest that neuroticism is an independent etiologically informative risk factor for CMDs [52,54]. Five explanatory models for this prospective pathway have been proposed: the vulnerability model (cf. diathesis-stress, neuroticism drives processes that lead to CMDs such as the selection of SLEs), the spectrum model (neuroticism and CMDs form one dimension), the common cause model (neuroticism and CMDs share their genetic and/or environmental determinants), the scar model (higher neuroticism after having suffered from CMD-episodes), and the state model (higher neuroticism during episodes of CMD, but not after remission). In a review on population studies with at least 200 adults, in which neuroticism was assessed at \( T_1 \) and CMDs at \( T_2 \), at least one year apart, these five explanatory models were evaluated (chapter 5). Criteria were the strength of the prospective neuroticism-CMD association, item overlap, shared determinants, and differential change and stability, and treatment effects.

Finally, the two-year prospective effects of temperamental negative emotionality (viz., fear and frustration) on CMDs during adolescence were tested, because fear and frustration are developmental precursors of adult neuroticism [123,192,196,220,1002,1003].

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83. Temperamental negative emotionality captures sadness and anger, but is most manifest via frustration [196,220,1002]. Negative emotionality mobilizes avoidance behavior away from non-reward and
We tested whether this prospective association was mediated or moderated by the generation of SLEs (chapters 6-8), and tested mechanisms that could underlay increases in fear and frustration in late adolescence (chapter 7 & 8).

**THE MIXED MODEL OF CHANGE IN NEUROTICISM**

Neuroticism was introduced as a psychometric continuum that runs from emotional stability to emotional volatility, and high scores capture the tendency to experience chronic negative emotions, which are easily triggered, and difficult to control [46,50,56,60,61,63]. A recent review of ‘neuroticism setpoint models’ boiled down to a ‘mixed model of neuroticism’ [94], in which temporary fluctuations in neuroticism are distinguished from more persistent changes in an individual’s setpoint around which neuroticism levels are regulated (chapter 2 & 9). In the following paragraphs stability and change in neuroticism are discussed in terms of three time scales (i.e., hours, weeks to months and years).

**Level 1 (Hours): Emotions and Affect States**

At the most momentary level we experience positive and negative affect in response to environmental influences in the form of basic emotions (e.g., anger, frustration, fear, sadness) we share with all mammals, and which guide our everyday conduct, decision-making, and navigation of our worlds [22,124,659]84. Such core affects are...
neurophysiological states that are consciously accessible as the simplest raw unreflective feelings evident in moods and emotions [97,1004] which operate automatically and quickly (instinctive) in response to environmental influences, without effort or sense of voluntary control [124,659,659,1005,1006]. At this level brief stressors can be conceptualized, such as a short public speech, mental arithmetic [1007], an academic examination, or a job interview [1007], but also acute stress reaction and panic attacks (ICD-10)\textsuperscript{85}. High neuroticism is characterized by more intense and persistent negative emotions [21,103,251], which can be transmitted via emotional contagion [1008,1009], but each episode lasts for several hours at best [22,661].

**Level 2 (Weeks to Months): Mood and Temperament**

A secondary temporal level captures characteristic differences in behavior and emotion across multiple contexts, which form a personal density distribution with an average score over weeks and months. At this temporal level we conceptualize temperament and personality frameworks [13,15,18,22,94,110,114,119,123,192]\textsuperscript{86}, subjective wellbeing [71,152], and encounter persistent episodes of mood [78,94,222,663], specific symptoms [53,1010], and episodes of anxious or depressive illness [114,222]. A prerequisite to diagnose depression is that symptoms are present for at least two weeks [639]. Patients suffering from episodes of anxiety or depression often recover in about three months [664,1011]\textsuperscript{87}.

SLEs trigger an episode of anxiety or depression typically within maximal three months after their occurrence [345,356-358]. In our own work in chapter 2-4 we observed that most SLE-driven effects on neuroticism also recede within three to six months. SLEs have been defined as “discrete events with a beginning and ending that have negative or undesirable content” [721]. We observed in chapter 2 that short-term

\textsuperscript{85} The International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10) from the World Health Organization (WHO) [1274] classifies acute stress reaction (code F43.0) as “a transient disorder that develops in an individual without any other apparent mental disorder in response to exceptional physical and mental stress and that usually subsides within hours” but can last for 2 to 3 days.

\textsuperscript{86} William Stern (who also invented IQ) outlined in 1900 that momentary behaviors and “mental energy” were influenced by both stable person-specific dispositions and momentary factors, and advised theorists to study both [1275]. According to Stern, dispositions could be derived by measuring behaviors repeatedly to calculate their mean value, which would eliminate the momentary fluctuations around dispositions [105]. This are the first two levels of the proposed model.

\textsuperscript{87} In a study of the Dutch adult population (n= 7076) the median duration of a major depressive episode was about 3 months (50% recovered within 3 months, 63% within 6 months, 76% within 12 months, and nearly 20% had not recovered at 24 months [664]).
increases in neuroticism in terms of months were largely mediated by more symptoms of depression/anxiety. This led us to review studies that span at least one year in chapter 4 and 5. SLEs that persist for more than six months were labeled “chronic stressors” or long-term difficulties, which are characterized by an absence of a clearly defined start or endpoint [414,1012]. For example, dealing with physical or mental disability, proving care for a family member with a long-term condition, or the death of a spouse. Perhaps this lack of a known endpoint requires individuals to implement a shift in the setpoint of neuroticism as an internal adaptation.

**Level 3 (Years and Decades): The Setpoint Level**

We conceptualize the neuroticism setpoint at a third temporal level, viz., stability and change in the setpoint of the personal density distribution of negative-affective expressions across the lifespan (chapter 2, 3 & 9, *cf.* [94,114]). At this temporal scale we encounter normative mean level decreases in the setpoint of neuroticism of about \( d = 0.80 \) between age 10 and 40 [163,165], and the increasing rank-order stability for the setpoint of neuroticism until midlife [93,164,253]. Based upon our results we conceive changes in neuroticism that persisted for more than 6 months as changes in the setpoint of neuroticism (chapter 2-4 & 9). Notably, the diagnostic and statistical manual of mental disorders (DSM [639]) often requires symptoms to persist for at least 6 months to diagnose a mental disorder (*e.g.*, general anxiety disorder), to ensure that the condition outlasts transient stressors and indicates an *internal* dysfunction that operates independently of the original stressor [639,1013].

Prior studies [100,246,331] indicated that increases in the setpoint of neuroticism after severe stressors could persist over a decade. Two of these studies reported major increases in the neuroticism setpoint about two years after a stressor (\( d = 0.50 \) to 0.70, *e.g.* death of a spouse during midlife), which returned by about 60% over the next decade [100,331], as illustrated in Figure 11 below. The findings presented in Chapter 2-4 and 9 of this thesis bolster this distinction between the second and third temporal level in the mixed model of change in neuroticism (Figure 12) and are consistent with (i) behavioural-genetic evidence that a substantial part of both stability and change in the setpoint of neuroticism is explained by environmental factors [81-92] and (ii) the small but steady drop in test-retest correlations for neuroticism with increasing time intervals [164,232,236,328,411,432].

Despite the increasingly stable rank-order of our neuroticism setpoint with age [18,85,91,93,163,234-238,253], still about a third of the general population showed intra-individual decreases or increases in the setpoint of neuroticism of \( d = 0.50 \) over midlife [231,232]. The most influential environmental factors in terms of the neuroticism setpoint were unshared with siblings [18,85,91,93,234-238], and affected core aspects of our identity and status, mainly in our role as partner (marriage/divorce) and
employee (job loss, promotions), in line with the social investment principle, which is discussed below. We observed that most increases in the setpoint of neuroticism followed severe SLEs that were characterized as unpredictable, uncontrollable, unexpected, undesirable, and non-normative from a life history perspective, and regarded interpersonal stress and conflict (chapter 2-4 & 9).

**Change in the Setpoint of Neuroticism**

Though SLEs can have a strong impact in the first six months after their occurrence, their effects often receded thereafter (see chapter 2-4 & 9). This indicates that it is important to take the timing of experienced SLEs into account (see chapter 2-4 & 9, cf. [235,244]). For instance, positive life events (PLEs) have relatively strong short-term influences on neuroticism compared to negative life events (NLEs; in terms of months),
but increases in the neuroticism setpoint after deteriorated environments persisted much longer than decreases in neuroticism after positive changes in the environment (13.5 vs. 6 years), in line with the literature [384,387,388]. We proposed a balance between PLEs and NLEs in which PLEs occur more often but the impact of NLEs is about 25% larger, and more persistent (chapter 3 & 4). Moreover, the literature suggests that PLEs and NLEs have concurrent \( r = 0.10 \) to \( 0.40 \), chapter 2 & 3) and complementary sequential relationships [235, 381, 629, 640, 1014], e.g. marriage and divorce (chapter 4); but dismissal can also lead to divorce, change of residence, or loss of social contacts. PLEs can also buffer for the impact of NLEs on neuroticism, thus make people more resilient [269, 382-384, 387, 644, 1015]. Importantly, PLE-driven decreases in neuroticism persisted for years, see chapter 2-4 (cf. [339, 543, 635]). Behavior genetic studies indicate that PLEs are more person-dependent than NLEs [235], and stronger associated with socioeconomic status [67, 107, 1016, 1017]. Thus even though neuroticism is not predictive for PLEs [222, 339, 340, 629, 630], PLEs should also be considered in discussions of resilience, prevention, and politics [152, 1018].

This thesis suggests that most SLE effects recede in terms of months, but that some changes in neuroticism can persist for years [94, 223, 244] or even decades [100, 246, 1019]. Perhaps severe SLEs change the social interactions in our daily life, called proximal processes, and such changes may become amplified via feedback-processes (which we minted “malignant” and “benign” cycles in chapter 3). When a shift in neuroticism becomes anchored in changed proximal processes, such as changes in social support, resources, identity, habits, or secondary SLEs, this new setpoint may persist beyond the transient direct effects of the initial SLEs [93, 94, 117, 411, 429]. Changes in the setpoint of neuroticism may thus reflect a shift in the balance of our inner and outer worlds in order to adapt to or cope with changes in our personal atmosphere. A relative quick change in neuroticism (in terms of months) may be followed by slower external adaptation processes in the personal atmosphere, e.g. via substitution of resources [574, 665, 922], which enable the neuroticism setpoint to gravitate

88. For example, the increases in the neuroticism setpoint after deteriorated environments were about 25% stronger than the decreases in neuroticism after improved environments after 4 years, but the difference was 65% after 6 years (chapter 3).
89. Recent studies indicate that personality can change in short periods such as a few months, especially if triggered by a major life transition [244, 1276] or psychological intervention [1277, 1278]. Conceivably, each major flux in a microsystem may trigger a feedback loop and alter its network properties [116, 117]. Such effects have been observed after new romantic partner relationships or divorce [331], pre-term childbirth [430], and dismissal or promotion [410]. In chapter 4 we outlined why differences in environments may in part explain why effects of a new romantic partner, divorce, dismissal, promotion, and pre-term childbirth seem to persist in some adults, but not all. Regarding the substitution processes: The Social Production Function (SPF) theory proposes that physical and psychosocial well-being are derived from a hierarchical arranged pyramid of nested social production functions: affection, behavioral confirmation, status, comfort, and stimulation [574, 665, 922]. In the SPF framework
slowly back (in terms of years) towards the, generally more normative, neuroticism levels to which one was accustomed [93,94,100].

These speculations about balances between the neuroticism setpoint and aspect of our environments in terms of months have never been studied properly [100,244,245]. Moreover, whether and when the setpoint of neuroticism fully recedes remains unknown. The rate and extend of recovery in terms of a neuroticism setpoint is likely to differ for different individuals, just as recovery rates for disorders do. Population studies suggest that changes in setpoint levels of subjective wellbeing persisted over more than 20 years for about 30% of the adults (e.g. after the death of one’s child [403]), which suggests that comparable processes are not unlikely for neuroticism. It also remains unsolved how long changes in the neuroticism density distribution must persist before we can speak of a setpoint change, that is, where the second and third temporal levels border. Based on my studies as reported in chapter 2-4 and 9, 6 months seems reasonable.

The Normal-Disordered Boundary

It remains unclear what distinguishes setpoint change from long-term fluctuations in mental health, e.g. are they one dimension or different in kind [116,162,165,310,320,732,1020]90. In the DSM the convention is that persistent, excessive, and unrealistic worry about everyday things that lasts for at least 6 months is called a generalized anxiety disorder, rather than a substantial increase in the setpoint of neuroticism [54,301,639,1021]91. Moreover, the literature showed that episodes of Limerence - extreme longing for a loved one - can persist over a year [156,1022], while grief or bereavement after the loss of a loved one can persists for up to two years [639,1023]. Furthermore, the 10-year differential stabilities for specific and social phobia symptoms [776,1024], panic disorder [1025], and personality disorders [253,320,639], are

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90. For example, personality theory suggests behaviour to become maladaptive at both ends of trait continua [146,147,320], while personality disorders in the DSM have only one maladaptive pole [639].

91. As recently as thirty-five years ago anxiety did not exist as a diagnostic category [17,182]. Today, it is the most common form of officially classified mental illness, responsible for over a third of all costs associated with mental illnesses [639,1280].
comparable to the stability of neuroticism [164,253], thus more akin to personality than affective states. Test-retest correlations for neuroticism over comparable intervals seem in general only about a third higher than for CMD symptoms (chapter 5, Table 18). This indicates that there is more stability in CMD symptoms and more change in neuroticism than the concept of state (symptoms) and trait (neuroticism) would suggest.

The question what distinguishes high neuroticism from psychopathology remains largely unsolved. Perhaps changes in the neuroticism setpoint must become entrenched in our semantic memory and identity to persist, while mood fluctuations are stored in our episodic memory [573,732]92. It may also be that changes in the setpoint of neuroticism are functional, e.g. severe SLEs may shift the allocation of resources towards certain outcomes, viz., a more conservative ‘better-safe-than-sorry’ behavioral strategy to reduce the risk of losing at the price of limiting the chances of winning [22,24,116,145,184,1026]93. An argument for adaptiveness is not so easily made for CMDs [297,1027,1028]94. Perhaps the difference between biologically “designed” functioning - the result of natural selection - and the failure of such functioning (“dysfunction”) is the most plausible demarcation point between human normality and disorder in the medical sense [22,162,322,322,1013,1029]. The idea that CMDs

92. Our innate temperamental propensities lead to frequently repeated responses, which accumulate strength or functional value through repeated reinforcements, and become encoded as a memory that becomes as much part of the person as the genetic inclination [18,24,1004]. These habits elaborate and differentiate into cognitive and affective representations that are quickly and frequently activated, which we tend to call personality traits [18,24,192,1004]. But note that personality disorders are also egosyntonic, that is, part of the self-concept, and come without biographical breaks and identifiable lesions, while even their harm seems essentially normative, e.g. being functionally ineffective or socially annoying [128,1282]. It has been estimated that that approximately 15-19% of the adults qualify for at least one DSM-5 personality disorder [639,1283].

93. Emotional stability then may characterize a ‘win-stay position’ because individuals with adaptive or ‘favorable characteristics shall experience less need to change [123,250,329,1106].

94. The DSM distinction between mental disorders and normality is guided by two fundamental principles: a disorder indicates a “clinically significant” disturbance in psychological functioning that has negative consequences for the person, with distress, disability, and disadvantage as prime examples, and this harm has to be caused by the condition [162,322,639,1029]. Second, the disorder is a “dysfunction” which means that some internal mechanism is not functioning in the way it is naturally designed to function, which can be operationalized as a statistically unexpected and harmful consequence of a response [162,1013,1029]. Expectable responses to stress, however, and socially deviant behavior and conflict between a person and society are not generally due to a dysfunction, thus are not generally disorders [162,322,639,1029]. The negative mental states associated with high neuroticism such as sadness, despair, anxiety, fear, agitation, and anger, are normal (and expected) responses to life’s vicissitudes, like grief [149,322,1013,1029]. Only when the reaction happens without a trigger or persists unusually long (i.e. not appropriate to current circumstances in both content and scope), they might be seen as pathological [322,1018]. Albeit the DSM became the standard in the nosology and treatment of psychiatric disorders in many countries many theorists lament the inadequate scientific basis (either empirical [116,162,1223], theoretical [116,162,1284], or biological [311,321]), the rather vague gray “zone of rarity” or frontier between normality and disorder [53,116,639,732,1281,1285,1286], and the tendency to decontextualize mental distress [305,322,322,1111-1113].
may refer to the extreme poles of normal behavior [54,162,165,1030,1031] shall be discussed in terms of the spectrum model below. Perhaps neuroticism refers to individual characteristic levels of minor psychiatric symptoms [3,53,149,736] such that the distinction may be purely semantic, as has been argued for the distinction between emotion and mood [17,1032,1033].

**The Future of the Mixed Model**

Our understanding of treatment of high neuroticism may benefit from the mixed model of regulation of neuroticism, because it predicts that the neuroticism setpoint is partly (perhaps largely) embedded in the environment. In chapters 2-4 it was speculated that setpoint change coincides with altered environmental feedback loops, which normally regulate neuroticism back to its setpoint [94,100]. Therapy may help individuals to structure their lives in a way that anchors a decreased neuroticism setpoint in their environments in terms of more favorable social interactions, roles, resources, support, habits, activities, goals and cognitions, but also in terms of selecting themselves into more favorable environments via reactance and evocation processes [18,24,166,250-252,1034-1037].

The mixed model could be tested in more detail by using recent technological and statistical developments. To unravel within-person change theorists could combine trimonthly assessments of neuroticism (setpoint level) combined with high frequent sampling (viz., multiple assessments a day, repeated for multiple days) of the environmental embedding to study the personal density distribution of negative affect [73,110,1038-1040]. Intensive repeated assessments in individual’s daily life have been shown to be feasible in patients with emotional disorders [1040-1042] and informative in general population samples [71,110,661,1043,1044]. Using this approach, the momentary interaction between day-to-day environmental influences and (positive and negative) mood changes can be repeatedly, ecologically more valid, and much more reliably assessed than in research that only measures such interactions retrospectively with longer time intervals.

The repeated measurements would address the within-person heterogeneity in neuroticism that is often obscured in between-group comparisons [110,1040]. When for an individual more than 60 repeated measurements are available, these data are suitable for vector auto regression analyses [1043,1045,1046]. This kind of statistical analyses

95. Multiple studies showed that self-report measures of neuroticism change in response to therapeutic interventions [391,1287] and drug interventions [348,354]. Decreases in neuroticism over 10 months were also reported after mindfulness training [1288], the adoption of social roles [227,1289], or international travel [1290]. In many studies the researchers took steps to rule out alternative selection explanations for change in neuroticism. Finally, it has also been shown that military service [1291] or cognitive training [1037,1277] can change personality.
are particularly suited to discover causal associations between two or more variables [1047]. Moreover, other markers of neuroticism can be assessed, such as ambulatory sampling of snippets of ambient sounds [1048], individual expressions on blogs and social media [140,141,1049-1051] and behaviors [731,1052] or behavioral residue [1053].

In the future such analyses may contribute to unraveling the fabric of neuroticism, to map personal triggers for negative affect, and personal emotion regulatory mechanism to cope with the stresses of life, which may inspire theorists to craft more specific, maybe even personalized, prevention and treating strategies for decreasing neuroticism, and in this way the vulnerability for emotional disorders that is associated with high neuroticism.

**NEUROTICISM AND THE COMMON MENTAL DISORDERS**

One major drive behind this thesis was to unravel the causes for the vulnerability for common mental disorders inherent to high neuroticism, in order to prevent their to prevent their (clinical) manifestation. This is relevant as high neuroticism is the single most important risk factor in public mental health, a personal burden, and a substantial cost to society [52,54,55]. As outlined throughout this thesis, high neuroticism is associated with economic costs that exceed those of all the common mental disorders combined [52,55]. Worldwide, about 350 million people are currently depressed, and about one in four people annually suffer from mental illnesses that impair their functioning [307-309,1054]. The background against which the results of this thesis should be interpreted is that health care spending’s continue to rise, also for public mental health, which requires a reconsideration of resource allocation decisions. The observed potential for a benign transactional cycle between environments and decreases in neuroticism might stimulate prevention strategies to target the vulnerability for mental disorders inherent in neuroticism, rather than treating the subsequent manifestations of those disorders.

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96. For example, tests of the ability to remain calm under stress might be good measures of neuroticism [1052].

97. A study that sampled sounds indicated that high (vs. low) neurotic males tend to be more often outdoors (location), socialize more (activity), but less in male-only groups [1048]. Studies of social media [140,141,1049] indicate that the continuum of neuroticism associates with daily differences in terms of participation in social gatherings (e.g., church, meetings) and sports (e.g., basketball, snowboarding; see also Figure 2). On base of ratings of offices, bedrooms, or Facebook profiles the neuroticism setpoint level of the “inhabiter” seems often more accurately judged than accuracy found in zero-acquaintance research [1051,1053]. Zero-acquaintance ratings for neuroticism, in turn, tend to be much better in trait-relevant situations [731].
As indicated in the beginning of this chapter, five explanatory models have been proposed for the strong prospective association between neuroticism and CMDs: the vulnerability model, the spectrum model, the common cause model, the scar model, and the state model. The support for each of these models will be discussed in the following.

Overlap at Baseline (“Carry-over Effects”)
The spectrum model proposes that neuroticism and CMDs form one dimension. A meta-analysis of cross-sectional associations between neuroticism and CMDs showed magnitudes of Cohen’s $d$ of 0.50 for substance-use disorders, to 2.00 for some anxiety and mood disorders [281]; and over all diagnostic groups the neuroticism scores were on average $d=1.65$ higher. This overlap between neuroticism and CMDs led to many theories in which neuroticism lays at the core of emotional disorders [52,54,284,739,1021]. A comparison of longitudinal and cross-sectional associations indicated that about half of the cross-sectional association between neuroticism and the CMDs was accounted for by current mental state, except for substance use disorders (chapter 5). We discussed this effect in terms of state fluctuations around the setpoint of neuroticism in the mixed model of change above (chapter 2).

In my longitudinal studies adjustment for baseline symptoms decreased the two-year prospective effect of neuroticism on symptoms of anxiety and depression with about 75% (chapter 2, Table 7), and the two-year effect frustration on internalizing and externalizing psychopathology with about 50% (chapter 6)\(^ {98}\). Moreover, this prospective effect of neuroticism on CMDs is robust for statistical adjustment for age, sex, and socioeconomic status [741,841,1055]. In sum, both frustration and neuroticism have an independent prospective effect on CMDs, neuroticism mainly with internalizing symptoms (chapter 5), and frustration with externalizing symptoms (chapter 6).

Synchrony of Change and Content Overlap
It is often proposed that neuroticism and CMDs either form one dimension (spectrum model), or arise from shared genetic and/or environmental determinants (common cause model). A comparison of studies with short duration versus long follow-up intervals (>3.5 years) showed that the neuroticism effects on CMDs remain fairly stable over time: long-term test-retest correlations for neuroticism are about a third higher than for

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\(^ {98}\) Note that reported effects are based upon a comparison of the correlations between frustration and internalizing and externalizing symptoms before and after adjustment for baseline symptoms (thus compared to partial correlations), which are reported in Appendix Table A30. The models reported in the paper were also adjusted for endogenous and exogenous SLEs, and notably, the measures of internalizing and externalizing baseline symptoms were mutually adjusted, which lead to different measures and therefore different results than the values reported here.
CMD symptoms in general (chapter 5, Table 18). Substantial content overlap between neuroticism and internalizing measures was observed (although both are assessed on different time scales), but this was not the case for substance use disorders [3,53,732]. Furthermore, the genetic correlations between internalizing disorders and neuroticism are generally high (.58 to .82 [301,511,762]), but the environmental correlations are much lower (.05 to .27 [301,762]). Nevertheless, our review indicated that uncontrollable and high-intensity childhood stressors form a risk factor both for neuroticism and CMDs [795-798], as we also observed in chapter 2 (Table 3). Finally, there is support for a general factor of psychopathology [166,170,688,1056], e.g. a study in almost 1600 adolescent twin pairs suggested that negative emotionality or neuroticism is closely related to this general factor, also genetically, and more than with either the internalizing or externalizing cluster [1030]. Also a meta-analytic review of cognitive vulnerabilities for depression and anxiety suggested that the broad dispositional dimension of neuroticism formed a common etiological core [166]. Such findings can be seen as support for a spectrum model.

**Stress Generation**

Neuroticism may drive processes that lead to CMDs, however, such as a selection for SLEs, which is known as the vulnerability model (cf. diathesis-stress). Indeed, higher neuroticism and frustration predicted experiencing more SLEs in chapter 2-6 & 9 (d= 0.20 to 1.00), which in turn may have causal effects on CMDs. Moreover, many twin and longitudinal studies report on reciprocity between neuroticism and SLEs (see chapter 2, 3 & 9, and [125,172,235,239]). The reviewed studies in chapter 4 showed that specific SLEs were more prevalent for individuals high on neuroticism [155,222,339,340,629-631], e.g. three times more interpersonal-SLEs for the quartile with the highest neuroticism scores versus the lowest scoring quartile [340,444,626,631]. This association between neuroticism and SLEs was robust for statistical adjustment for mental health status at baseline [425]. In my own work SLEs mediated about 5% of the two-year prospective effect of neuroticism or frustration on CMDs (chapter 2 and 6), but up to 10% of the effects of neuroticism on neuroticism over 16 years (chapter 3). This indicates that stress generation effects are strong forces in the context of setpoint levels of neuroticism. This process will be discussed below in terms of the corresponsive principle, which holds that selection and evocation processes magnify pre-existing individual differences (chapter 3).

**Stress Sensitivity**

The literature suggests that patients have higher neuroticism scores during episodes of CMD, but not after remission (state model). Alternatively, neuroticism levels may remain higher after remission of a CMD-episode (scar model). By now it is clear that
patients rate themselves as more neurotic during episodes of (subthreshold) CMDs, such as depression or anxiety, compared to pre- and post-episode measurements [238,281,348,350,762,763]. In chapter 2 we outlined that this phenomenon is often interpreted in terms of is often interpreted in terms of a transient state-deviation in self-perception due to the state-effects of the patient’s current psychopathology, rather than real change in set-point-neuroticism [3,351-354]. This has been discussed in terms of the mixed model of change in neuroticism above. The support for the scar model is limited to major depression, and rather inconsistent, and may as well indicate decaying state effects (chapter 5). Moreover, scars, if any, are most likely to develop proportionally to the amount of symptoms experienced [322,1034]. When we assume that psychiatric disorder are dimensional constructs [116,1039,1057], vulnerabilities (“scars”) would develop gradually as an autocatalytic process, which could eventually lead to the onset of a full-blown episode of depression [166,675,1034,1039,1058]. This suggests that a dichotomous disorder approach would make it impossible to identify scars, because the (pre-morbid) scar would be seen as a risk-factor [1034].

**Integration of Evidence**

Regarding the models of the prospective relationship between neuroticism and CMDs, we conclude in chapter 5 that given the available evidence none of the models can account for all observations. The spectrum model could not account for the existence of risk factors that are unique to neuroticism (and not shared with CMDs, e.g. chapter 2 & 6). However, the spectrum model was supported for the internalizing cluster by overlap in item content, synchrony of change, rather similar rank-order stabilities, and strong familiarity with a general factor. The common cause model derived stronger support, mainly because of shared determinants (mainly genetic but also childhood adversity influences and cognitive vulnerabilities), and also by synchrony of change. However, common causes cannot clarify the prospective effect of neuroticism on CMDs, nor the moderation of SLE-effects on CMDs, although the magnitude of the latter effects were negligible in this thesis.

The prospective and moderation effects of neuroticism may be better explained by the vulnerability model, which can also account for mediation by SLEs, and the typical small difference between the short (<3.5 years) and long-term prospective effects of neuroticism. Moreover, the observation that baseline adjustment did not attenuate the effect of neuroticism on substance use disorders (chapter 5) supports the vulnerability model, just as the strong independent effects of frustration on psychopathology do (chapter 6). Nevertheless, the vulnerability model does not explain item overlap, shared determinants, and synchrony of change for internalizing disorders. Moreover, there is ample evidence that neuroticism levels are increased during episodes of CMDs (compared to pre- and post-episode measurements), but the state model cannot explain
the prospective neuroticism-CMDs association. Additionally, there is little support for a scar effect on the setpoint of neuroticism. In sum, the results in this thesis suggest that the strength of the support for each model differs for the specific clusters of CMDs, \textit{viz.} anxiety, depression, and substance use disorders. CMDs have a massively multifactorial etiology, and although parsimonious explanations are generally favored in science, reality may be that no single theory can satisfy all needs, albeit we can strive for a broad framework in which most needs can be addressed [116,310,1018,1039,1059].

Future theorists should formulate more specific theories that can be falsified by observations. For example, evidence that changes in neuroticism predict change in CMD risk would differentiate between the vulnerability and common cause models (chapter 5)\textsuperscript{99}. Furthermore, future studies should implement a full control for neuroticism-CMDs overlap, which most reviewed studies were unable to do. Third, theorists should explore mechanisms that mediate the prospective effect of neuroticism on CMDs (see chapter 6-8), because our understanding of these processes remains fragmentary [116,162,165,732,1060]\textsuperscript{100}. Finally, theorists should focus on modifiable aspects of our environment that predict high neuroticism, frustration, or fear, or moderate change in the development of these traits (chapter 8), to enable prevention strategies.

DECONSTRUCTING NEUROTICISM

Over the 1990s consensus emerged that the core of neuroticism refers to a propensity to experience negative affects including anxiety, fear, sadness, anger, frustration, guilt, irritability, loneliness, worry, self-consciousness, dissatisfaction, hostility, guilt, shyness, distress, and reduced self-confidence [46,50,51,56-63]. In the future it might be worthwhile to further deconstruct neuroticism in terms of these facets and their supposed purpose. The stability of most facet traits is comparable to the neuroticism trait at broad [93,253], also over 15 years [246]. The facets of neuroticism refer to rather distinct emotional categories or mechanisms, with anxiety, fear, sadness, impulsiveness, and anger/frustration as the most prominent examples [22,192,196,230]\textsuperscript{101}.

\textsuperscript{99} For example, it has been shown that each \textit{d}=0.50 increase in neuroticism per decade associated with a 40% increase in mortality, after statistical control for physical health and age [68].

\textsuperscript{100} An understanding of aetiology is also perquisite to validate clinical syndromes, and both clinician and scientists are “much more likely to succeed if the syndromes in question have been accurately identified to begin with” [1060], \textit{cf.} [307,321,1111,1223,1224,1286]. Conventionally a psychiatric syndrome refers to a cluster of symptoms - either abnormal behaviors or abnormal distressing subjective experiences or both - and a characteristic time course [1039,1060].

\textsuperscript{101} For some facets it is also debated whether neuroticism is the domain of origin. For example, irritation or anger have also been related to low agreeableness [142,643]. Guilt is also a key affective component of conscientiousness [1072]. Social anxiety seems a part of extraversion [289]. Aspects of problems of impulse control that are part of neuroticism (alongside negative affect) could also be part of con-
Sadness, for instance, is marked by negative valance and low arousal, while anxiety and fear are marked by negative valance and high arousal [659]. Anxiety motivates avoidance behaviors while anger motivates approach behaviors [698-700]. Anxiety is characterized by a sense of uncertainty while anger is characterized by certainty [22,1061]. Temporal dynamics can distinguish closely related emotions such as shame versus guilt or fear versus anxiety [22,661,1062]. Anxiety refers to inner turmoil or expected future threat while fear is a response to a real or perceived immediate threat [11,22,639,662,1062]102, e.g. anxiety enhances pain while fear inhibits pain [1062-1064]. Sadness lasts relatively long while shame, disgust, and fear tend to last relatively short [11,661,1065,1066]. Furthermore, the facets depression and anxiety seem to change somewhat less in reaction to environmental influences than angry hostility and impulsiveness do [230,238], and both categories show different responses to different SLEs [483,487,488,616].

The facets of neuroticism also show markedly different developmental trends, which suggests that they capture information about individual development that is missed by the broad neuroticism domain [230,231,637,820]. Furthermore, many facets of neuroticism (such as anger, contempt, depression, or anxiety) seem to be mediated by different neurological underpinnings [22,703,1067], are likely multiple gene-linked [17,18,1067], show specific correlates with external variables [171,789], such as depression [1068] or family functioning [1069], and facets have different implications for development and psychopathology [291,687,701-703,1062,1068]103. Another issue is that the low poles of the neuroticism facets deserve more attention as they may inform theorists about potential purposes, e.g. indifference (vs. anxiety), timidity (vs. angry-hostility), deliriousness (vs. depression), superficiality (vs. self-consciousness), conscientiousness [47,171,198,1072,1292], (high) extraversion [47,1293,1294], (low) agreeableness, and (high) openness [47,1293]. The inclusion of impulsiveness in the neuroticism domain is based on the fact that some impulsive acts are prompted by negative feelings (overindulgence), which themselves are a hallmark of neuroticism [643,1295]. Impulsive behavior in response to stress is also called negative urgency, and often takes the form of alcohol abuse or aggression, which is more common for individuals high on neuroticism [1295]. Interestingly, impulsivity can manifest itself either in action (e.g., drug abuse or shopping) or inaction (e.g., tardiness, lack of planning), see [122]. Comparable debates about overlapping traits are also held with regard to the subcomponents of subjective wellbeing [1296-1298] or positive and negative affect in general [1299]. Most wellbeing scales overlap with personality dimensions, e.g. “positive relations with others” and agreeableness [1300] or “personal growth” and openness [1300].

102. Anxiety is characterized by hypervigilance and is future-focused while fear mobilizes people for avoidance action and is present-focused (fight/flight/freeze). General anxiety disorder and depression seem anxiety-based while animal and situational phobias and panic seem fear-based disorders [291,687,791,791,1062]. Moreover, trait anxiety forms the core of the negative emotionality dimension (or neuroticism) while trait fear also seems to load strongly on the constraint dimension (cf. conscientiousness) [1062,1301].

103. For instance, depression seems more often manifested as anger and somatic symptoms in men and in experiences of stress, irritability, sadness and anxiety in women [1302,1303].
recklessness (vs. inhibition), and being hard (vs. vulnerability), while emotional stability at broad may fade into fearless psychopathy [119,146,147]104.

Additionally, it might be informative to study whether broadly acting environmental influences, such as social investment, personal growth, and self-actualization, influence all facets of neuroticism, and which experiences only influence one facet (narrow acting influences), and leave the corresponding facets unaffected [163,209,230,231,246,338,1070]105. When specific facets become more associated during specific developmental periods we can theorize that both facets became influenced by a shared factor [209,1071], such as age [209], or normative life events [227]. Decreases in associated change between facets may suggest trait differentiation [209,1071]. In sum, the neuroticism facets capture distinguishable tendencies that may differ in their effects on adjustment and outcomes [22,703,1072,1073]. As outlined in the introduction, each individual has a neuroticism chord, a polyphony of facet traits, which resonate differently, and make people feel differently, even when their average neuroticism levels may be the same.

It remains unknown what the correlations in factor analyses actually mean, and hitherto personality theory lacks a clear rationale or empirical basis to assert that one set of facets is better than another [86,101,228,672,820]106. For example, the network perspective posits that neuroticism emerges from the interactions between the facets, instead of reflecting a latent force that drives the associations between components [117,1038,1039,1042]. Furthermore, the study of facets of neuroticism may help to illuminate the mechanisms through which neuroticism affects important life outcomes [22,122,1072,1073]107. Facets suggest specific sensitivities [22,322,1070], and seem to

104. For example, individuals with cluster-C personality disorder, characterized by high anxiety, experiential avoidance, and need for safety, seem to derive more status and resources, perform better academically, and reach higher levels of education [184]. The price, however, according to some studies, is constrained access to mates among males and a fall in fertility [184,1304], and what these individuals perceive as insufficient social networks [184,1281].

105. For example, young women in the USA high on neuroticism manifest eating disorders and excessive worrying and concern [1305], whereas neurotic women in rural Ghana rather report somatic symptoms (feelings of illness) and excessive magical thinking or other non-scientific causal reasoning [1305]. The Japanese culture seems to emphasize shame as a means of social control and Japanese indeed score higher on the self-consciousness facet of neuroticism than individuals from the USA [1306,1307]. Furthermore, theorists could focus more on correlated change between personality domains over time [1308]. Earlier studies showed correlated change between neuroticism and extraversion and conscientiousness [1309], but other studies failed to observe correlated change [231,1071].

106. Personality psychology lacks a firm theoretical core or unifying and overarching personality theory such as the theory of evolution by natural selection does for biology [16]. Some theorists explicitly argue that natural selection serves this function for psychology as well [78,97,1310], in which case we still miss a framework (see chapter 1).

107. For example, extraversion is often conceived of as the tendency to experience positive affect, and to comprise the components gregariousness, dominance, and experience seeking [1311]. However, one studied showed that after control for positive affectivity the dominance and gregariousness component
react differently to therapy [1068], and are probably a more feasible target for therapy than a fundamental personality structure like neuroticism at broad [93,253]108. Finally, it is even conceivable that some facet traits are functionally adaptive, but that the total composite neuroticism is not, as a fallacy of composition.

**THE RED QUEEN PERSONALITY PRINCIPLE**

The red queen refers to a character in Lewis Carroll’s book ‘Through the Looking Glass’ who describes her country as a place where “it takes all the running you can do, to keep in the same place” [1074]. The queen does not get very far because the landscape moves with her, a process that is known as the Red Queen Theory [7,1075]. In analogy, over our life course we keep pace with a changing environment that follows a biosocial clock that shapes the fabric of our personal atmosphere (e.g. our social convoy). The biosocial life script drives the occurrence, timing, sequencing, duration, and interaction of life events and social processes that we pass across our lifespan (see chapter 4), which I minted the Red Queen personality principle. For example, with age people are pressed to decrease their neuroticism setpoint in order to keep their relative standing in their birth cohort the same, because their peers decrease with almost $d=0.80$ between age 10 and 40 [163,163-165,230,249,264,264,521].

The red queen personality principle implies that our neuroticism emerges from a balance (or mutual accommodation) we find with the changing environment in which we live and grow [100,1039,1076], as part of a complex dynamic system [25,255,460,1059,1077] that comprises all personal characteristics including our physique [177-180,1078-1080], intellect [108,211,587], gender and age [463], goals and expectations [677,678], personality [75], but also noxious environmental stimuli and our social and material resources [18,25,25,100,103,447,460,517]109. Individuals

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were no longer correlated. This suggests that extraversion, like neuroticism, is fundamentally an affective trait [1299].

108. For example, mindfulness-based cognitive therapy or cognitive behavioral therapy (CBT) may effectively desensitize networks of negative cognitions or inhibit reactivity to specific events that activate specific negative cognitive schemes [1035,1036,1312]. Such approaches may be more feasible for specific associations than for whole personality domains at broad.

109. We also have to adapt or cope with changes in the social convoy that escorts us as fellow travelers through time, viz., parents, spouse, children, friends, colleagues, neighbors, and acquaintances [73,204,445,448,1313], and also our social status and material resources change [24,107,245,1017]. No genome has an unconditionally highest fitness [25,191,1314-1317] and natural selection probably favored genes that equip a phenotype with the optimal compromise between all internal and external pressures; the point where all additional (non-essential) growth would call on the organism’s developmental budget [191,1315,1317,1318]. The setpoint of neuroticism is part of this balance or optimal compromise to maintain a dynamic steady state [460].
may change in some aspects in order to remain stable in others in a search for an approximately Pareto optimal allocation of their energy budged, given their genome and context, viz. the balance between all factors where no internal or external changes can be made without increasing the costs somewhere else [1059,1081-1084]. However, because the environment changes continuously, continuous little adaptations are required to maintain a Pareto balance.

The social investment principle or functional maturation hypothesis posits that the normative decrease in the neuroticism setpoint (about d= 0.80) is partly driven by social pressures, expectations, and contingencies that follow investment in social roles as student, partner, parent, and colleague [24,27,142,163-165,251,433,450].

110. Change from this Pareto criterion would degrade one of your objectives or desired end-states, if only because of opportunity costs [665,1092,1135]. Multi-objective problems like the one proposed often have a set of Pareto solutions - called a Pareto frontier, and the selection for “the best” balance along this frontier remains a subjective human decision, and is dependent on historical-cultural contingencies [162,683,1081,1082,1084,1110]. Change from one state to another requires energy and has a transaction cost [1039,1135]. The Red Queen Personality Principle roots in the second law of thermodynamics which holds that isolated systems spontaneously evolve towards a thermodynamic equilibrium, viz., the state of maximum entropy or chaos [97,1077,1315,1319]. Organisms differ from other natural phenomena in terms of their maintenance of a thermodynamically improbable internal organization including disequilibria with their environments in terms of temperature, acidity, water content, or electric potential, which are balances that require continuous work [97,1077,1315,1319]. Such highly ordered and functional internal states comport with the Second Law of thermodynamics because this internal organization causes a net increase in disorder in the environment [25,97]. In other words, the body is not an isolated system, and we form one unified system with our environment [25,191,255,256,263,310,991]. Heritable effects on the immune system [1320] or phenotypic expression are overshadowed by environmental effects [24,25,470,1026,1102,1124,1314,1321]. In the field of psychology this may be reflected in concepts like stress, which is transactional, and perhaps stress cannot be found in the objective circumstances themselves nor in the person but only in an ongoing interaction between both [459,460,462,1322]. The second law of thermodynamics has been postulated as the first law of psychology because it implies that the apparent organization of the psychological realm (see Figure 1) requires an explanation [97,1323]. Part of this explanation may be found with approaches that explore differences and similarities between factors (or nodes) in a dynamic system, the interactions between them, and the processes that hold the various components together [1039,1059,1076]. Network theory may help to discover fundamental laws that determine and limit the behavior of the components in such complex systems [1039,1059]. Multi-cellular creatures are ecological networks rather than single living entities [1077] and even sex indicates that we are (reproductively speaking) incomplete, and that individuality is actually non-existent [973,1077]. In some deeper sense we are one with all other living things on our planet.

111. For example, neuroticism levels are lower in twins who are reared religiously than in secular twins, which suggests that religious upbringing modifies the genetic determinants of neuroticism, such that neuroticism becomes more heritable in twins without religious upbringing [546,1324]. Furthermore, experiences and sociocultural factors can imprint sociocultural display rules that govern when, how, and to whom emotions may be expressed [123,195,198,876], e.g. boys become increasingly less emotional expressive between age four to six, while girls do not change, which suggests that cultural gender socialization dampens boys’ initial emotional expressivity [195,876,1325]. This process of environmental canalization may explain part of the variance in heritability estimates of neuroticism over populations, time, space, cultures, age, levels of social control, and part of the variation in indirect genetic effects [41,85,92,565,567,568,1101,1326].
The social investment principle is supported by associations between the timing of social events and the normative decrease in neuroticism across cultures [93,227] and the accelerations of this normative change after social investment in the family or occupational environment (chapter 4). In sum, the major social roles influence the neuroticism setpoint, especially when successes or failures in the relationship, family, and occupational domain accumulate over time [18,64,251,427,535,672].

The normative mean-level decrease in neuroticism does not preclude an increasing rank-order consistency with age [163,227]\(^{112}\). The genetic influences on the neuroticism setpoint stabilize during childhood (test-retest correlation, \(r = .75\)), and approach unity near age 30 (\(r > .90\) [93]). Environmental influences on the setpoint, in contrast, are unstable during infancy (adjusted for measurement error, \(r = .25\)), but continue to stabilize over the majority of the lifespan (at age 15, \(r = .50\)), and reach a plateau during middle age (age 45, \(r = .80\)), when the neuroticism setpoint is most stable [18,85,91,93,163,234-238,253]\(^{113}\). The increase in stability for the neuroticism setpoint with age dovetails with an increasingly stable personal environment, which suggest that most people experience fewer social pressures to change with age [85,91,93,164,236,247,445]\(^{114}\). Prior work thus showed that a substantial amount of variance in the neuroticism setpoint is truly environmentally mediated [75,93,253] and unrelated to state-like fluctuations [18,85,91-93,234-238]. About a third of the general population shows intra-individual increases or decreases of \(d = 0.50\) in the setpoint of neuroticism over midlife [231,232].

The neuroticism setpoint seems most sensitive to environmental influences that are not shared with our siblings [18,85,91,93,234-238] and relate to the core aspects of our identity and status: mainly in our role as partner (marriage/divorce) and employee (job loss, promotions), in line with the social investment theory (chapter 4). We outlined that also severe SLEs that were unpredictable, uncontrollable, unexpected, undesir-

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\(^{112}\) It is therefore important to note that continuity and change are not natural opposites [64,1327]. A true understanding of the developmental stability and change in neuroticism requires a combination of analytic approaches [1195,1328], and a focus on differential change, or individual differences in change, which combines mean-level and rank-order stability [249] as well as change in the magnitude of individual differences in neuroticism over time [1195,1329].

\(^{113}\) A clear personality structure does not appear before the preteen period [19], and as outlined in chapter 1, most trait differentiation takes place between age 12 and 18 [19,207-209]. Furthermore, the magnitudes of genetic influences on neuroticism differ across environmental contexts [453,1330]. The increasing stability of environments may in part reflect identity development and cumulative selection of specific environmental niches, which we called the personal atmosphere [93,164,230-232,434,441]. Note that substantial heritability of neuroticism does not rule out strong environmental-effects across cultures [106,427,565,567,568]. For example, in Morocco women score much higher on neuroticism than males \((d = 0.80)\), whereas in Indonesia and Botswana males score higher on neuroticism \((d > 0.10)\) than women [463].

\(^{114}\) Functional explanations for increasing stability in the setpoint of neuroticism include benefits of predictability, constraint, and positive feedback loops between state and behaviour [15,24,433].
able, and ‘off time’ from a life history perspective predicted changes in the setpoint of neuroticism, especially when they were characterized by interpersonal stress and conflict (chapter 4). In sum, if we aim to understand individual differences in the setpoint of neuroticism our social life context matters.

**Corresponsive Principle**

The corresponsive principle holds that the effect of life experiences on personality is to deepen the characteristics that lead people to those experiences in the first place, for example, via the selection of specific friends, spouses, occupations, experiences and hobbies [29,64,67,172,248,263,411,436-441,1085]. In other words, individuals with high neuroticism setpoint levels select/evoke experiences that lead to even higher setpoint levels, an accumulative model (or snowballing effect). The accumulative model is often invoked to argue that individuals respond differently to non-normative SLEs, because responses depend on their pre-existing neuroticism levels [38,399,626,666,667], and this would result in person-specific outcome patterns [249,451,554]. The corresponsive principle has been a key theory in this thesis, but has certain shortcomings as well (e.g., with regard to timing, mechanism, and specificity).

**Accumulation versus Balance Models**

As discussed above, after severe SLEs most individuals increase in their neuroticism setpoint, but this higher level tends to regress slowly back towards previous neuroticism levels over the next decade (see Figure 11). This process is not directly reconciled with an accumulative model. The corresponsive principle also lacks information about the timing of this increasingly stronger neuroticism-environment correlation, about the underlying mechanism, about differences between positive and negative effects on neuroticism (in magnitude or persistence), or their interaction [38,399,626,666,667].

Many observations in this thesis do not concur with a snowballing process (see chapter 2, 4 and 9, or [603,673,812]). For example, early life challenges may, when overcome, induce adaptations that lead to resilience to SLEs later in life, compared to individuals without a history of adversity (see chapter 2), which is known as a “steeling effect” [222,461,1086,1087]. Adjudicated individuals tend to score high on neuroticism over young adulthood, but often shift rapidly towards more normative developmental trajectories when they invest in a romantic partnership or an occupation [603]. Furthermore, neuroticism does not occur in vacuum, and individuals high on neuroticism and high on conscientiousness may develop very differently from peers high on neuroticism but low on agreeableness [99,185], while high cognitive ability may buffer for high neuroticism [211,1088,1089]. Finally, the corresponsive principle is based upon the observation that personality effects on the social environment are stronger than vice versa (chapter 3). However, recent studies, with more sophisticated
designs and instruments, suggest that neuroticism and environmental effects are more in balance than previously assumed [100,246,249].

The Red Queen personality principle is simplified explanation for all observations in terms of a balance between our neuroticism setpoint level (and other characteristics) and our personified environment. However, the Red Queen personality principle implies function, viz. benefits and costs associated with the gradient from high neuroticism to emotional stability in a given context. This balance theory predicts that a certain predisposition for neuroticism may lead to a relatively low neuroticism setpoint in favorable environments, but to higher setpoint levels in more risky and unpredictable environments [24,165,1026,1090-1092]115. This may also reflect in higher neuroticism levels during more vulnerable phases, e.g. childhood [78,98,123,195] and old age [75]. Further research should seek to determine the specific environmental conditions that influence the neuroticism setpoint and the mechanisms that drive such calibrations.

The balancing model is supported by fluctuations in the distribution of neuroticism within populations around the globe [379,449,463,565-568,1093] and over generations [41,228,333,334,434,562-564]. For example, in the south and east of Europe populations tend to score higher on neuroticism than in the other European countries [1094,1095], while Northern and Southeast populations in the United States score higher on neuroticism than Midwest and Western populations [1096-1098]116. Cultural differences have also been observed regarding inter-individual variance in neuroticism.

115. For example, during childhood [78,98,123,195]. The moment when physical changes enable children to crawl and to extend their range coincides with the emergence of separation anxiety [98,119,192]. Furthermore, it has been speculated that young women tend to score higher on neuroticism than young men in order to keep young females in groups for mutual support, i.e. neuroticism as a protective device [1318,1331], in keeping with the ‘staying-alive-theory’, which holds that it is imperative for mothers (rather than fathers) to survive long enough to take physical care of their offspring and ensure their survival to sexual maturity [78,1331,1332]. Women should therefore also be more risk-aversive than men [78,191,1331], everything else equal.

116. Populations in Japan and Argentina show relatively high national neuroticism levels and in Congo and Slovenia relatively low levels [568]. Even bordered countries can show subtle differences, e.g. monozygotic twins males in Sweden score lower on neuroticism (d= -0.19) than in Finland [172]. The most parsimonious explanation seems that environmental impediments are able to suppress genetic predispositions for neuroticism, thus “canalize” the reaction norm for neuroticism, that is, the ways in which neuroticism will be expressed [25,31,255,463,1101,1321,1333]. In other words, stable environmental conditions around an individual influence the way in which this individual expresses his inherited propensities (and therewith inter-individual variance), just as genotypes can [262,1026,1101,1321]. Note that heritability estimates capture the proportion of individual differences in neuroticism that can be attributed to actualized genetic potential but the degree of non-actualized potential remains unknown [103,453,547,1229]. The neuroticism setpoint thus refers to an average personal level of neuroticism, and not to a biological setpoint. Theorists could assess genetic covariance between specific conditions and neuroticism levels to map ‘genetic capture’, which refers to the theory that traits capture the genetic variance in condition [25,453,1317]. Perhaps personality theorists might focus more on correlations between genes and environments (see chapter 4) or environmentally contingent behavioral patterns or conditional adaptations [1090], given observations in related fields [453,470,547,1320].
and gender differences in neuroticism [463,565,1100]. The neuroticism setpoint levels and gender differences appear to increase along a continuum towards more prosperous [255,463,565,566], non-religious [546], urban [1101], and egalitarian human socio-ecologies [41,331,333,334,562-564]117. This suggests that the costs of neuroticism are condition dependent, and different environments may favour different neuroticism levels (and different facet combinations) for different segments of the human population [11,134,145,1102-1108]. Remarkably little is known about such processes [95,227,228,628,658]. Notably, not only neuroticism levels seem to differ across cultures [463,565,566], also CMDs manifest themselves differently at different continents [17,116,305,307,761,1109]118, and the incidence and impact of SLEs differs as well [608,1017,1110]. The perspective on the setpoint of neuroticism as a balance of our inner and outer worlds, in order to adapt to or cope with changes in our personal atmosphere, led to two hypotheses that have been outlined before:

A). Perhaps SLEs such as divorce or dismissal alter the proximal processes in our personal atmosphere, that is, the daily patterned interactions within our family or occupational environment, e.g. in terms of social support, resources, identity, habits, or secondary life events. These changes may spark feedback loops until these newly structured proximal processes anchor the neuroticism setpoint and prevent regression towards previous averages. Consequently the changed neuroticism setpoint can persist beyond the transient effect of the initial experience (>6 months). As outlined above, this could be tested with frequent sampling of these proximal processes.

B). Individuals may increase rapidly in neuroticism after severe SLEs (in terms of months) but this setpoint change may be followed by slower external adaptation processes in the personal atmosphere (in terms of years and decades), e.g. via substitution of resources, which enable the neuroticism setpoint to gravitate slowly back towards the - generally more normative - neuroticism levels to which one was accustomed

117. In other words, gender differences in neuroticism tend to be larger in more developed Western cultures with less traditional gender roles [463,565]. The widening gender-gap in neuroticism is accompanied by a widening sex-difference in blood pressure and height [463], which may suggest that enhanced sexual dimorphism is part of the explanation [1334]. This would align with the observation that men show more variance in neuroticism across cultures [463,565], akin to the general rule that males are most sensitive to environmental differences and selection pressures [11,78,615]. Most species reach their sex-specific optima only under favorable environmental conditions [78,78,255,1334,1335]. Increasing human sexual dimorphism has also been shown within human cultures in high versus low socioeconomic strata (SES, cf. [1336]). Moreover, this effect is mirrored in epidemiological data on gender differences in depression [1337]. Perhaps differences in the use of mental reference categories (e.g., comparison of the self with others of the same gender versus all others) can explain part of the gender or global differences in neuroticism [565], but artifact reference categories are less likely to vary consistently within countries or federations like the USA.

118. The point prevalence of anxiety disorders, for example, appears to range from 2.1% in East Asia (1.8-2.5%) to 6.1% (5.1-7.4%) in North Africa and the Middle East, and to be more common in regions with a history of recent conflict [1109].
(cf. pareto optima, perhaps a different locus along the pareto frontier). Testing this hypothesis also requires frequent sampling of the personal environment, but now in terms of months and years.

**Environmental Taxonomy**

Testing hypotheses about the balance between the neuroticism setpoint and environmental influences requires a taxonomy of environmental influences, alike personality taxonomies, to measure and compare specific environmental influences on neuroticism and changes in our personal atmosphere [103,244,245,265-267,409]119. Such a taxonomy would “complement, enrich, and spur forward the relatively advanced psychology of personality so that persons thinking, feeling, and acting in situ can be studied” [245]. In chapter 4 we outlined that the Bioecological system theory may be used as a stepping stone [103,447], but also the socioanalytic personality approach [95,409], social production functions [574,665], and the DIAMOND model [245] could be consulted. This approach may also help psychiatry to attend to context in a systematic fashion [305,322,1111-1113].

Our results suggest that it may be worthwhile to chart and dissect the transactions between environmental influences and neuroticism in more detail, and to take all surrounding factors, conditions, or external influences that affect a given individual at a given time into account. A core question remains which factors render a personal atmosphere a risk factor (vulnerability) for high neuroticism after SLEs, and which environmental factors enable for stability, and function as an insulating layer (resilience)120. In sum, we feel that a strict demarcation between the individual and the environment is largely based on statistical methodology (chapter 4). Improvements in social and psychological functioning require a consideration of both the person and the personified environment in which the person lives an grows, and which function as one unified and highly personalized system.

119. Our human nature has been carefully crafted by cumulative natural selection for use by a social, bipedal, African ape, which assesses probabilistic risks against the background of our subjective timescale (median lifespan) and conditions that may happen to us individually or the narrow social circle around us, which makes us more sensitive to specific threats and less to others [7,78,96,97,152,191,613,1315,1338-1340]. The literature suggests that environmental cues that signal loss of limited reproductive resources (spouse, friend, skills, appearance, roles, and status), loss of social competition for these resources (and threat thereof) reliably elicit increases in neuroticism. This might have been an adaptive response because loss is reliably related to threat/danger, and vice versa. The goal might have been to avoid similar or additional (immediate) losses [7,78,96,97,152,191,613,1315,1338-1340].

120. The extent to which specific experiences can be regarded as generally noxious, or only noxious for specific individuals, is largely a function of our level of abstraction, thus we might aim for characteristics or properties (types of signals or symbols) that predict change in neuroticism, while the exact attributes themselves may differ across time and context and for different people, cf. the concept of a secondary reinforcer [16,22,78,96,260,460,612,1310].
CONCLUSION

Our work showed that life event driven changes in the neuroticism setpoint persisted for years to decades. Neuroticism refers to negative affect accompanied by the pervasive perception that the world is a dangerous and threatening place and beliefs about one’s inability to cope with this environment [53,54,62,63,238,1114,1115]. Albeit we should be careful to ensure that treatment for high neuroticism has no consequences that would make the cure worse than the ailment, prior work has shown that interventions treating high neuroticism are feasible [351,354,355,391,726-728,1037,1116], both psychological and pharmacological ($d= 0.40$ to $1.25$). Therapists could focus on cognitive vulnerabilities that are associated with neuroticism such as a pessimistic inferential style, dysfunctional attitudes, ruminantive style, anxiety sensitivity, intolerance of uncertainty, and fear of negative evaluation [32,54,166,1117]. Therapy may help individuals to structure their lives and social interactions in ways that anchors a decrease in neuroticism in their daily environment. This thesis suggests that a decrease in the setpoint of neuroticism would impact on most life domains and would certainly be worth the bother in terms of effort and time. Perhaps it is most cost-effective to implement population-wide intervention strategies at school during childhood [1114,1118,1119], e.g. by teaching children that their personality has the potential to change [1120], and to provide coping skills before symptoms of anxiety or depression cascade into other spheres of functioning during adolescence, and at a point when the environmental anchors of the neuroticism setpoint seem most malleable.

This thesis showed that high neuroticism is an important prospective indicator of risk for the development of full-blown psychological disorders, and can, to some extent, be viewed as sub-threshold psychopathology. We conclude that the neuroticism setpoint is environmentally embedded, sensitive to stressful life events, consequential, and more malleable than researchers originally believed.
Figure 14. A summary of this thesis
REFERENCES

References


108. Ackerman PL, Heggestad ED. (1997) Intelligence, personality, and interests: Evidence for overlapping traits. Psychol Bull 121: 219-245.


References | 271


References

References


References


References


References


References


References


References

1142. Van Leeuwen, Edwin J. C., Cronin KA, Schütte S, Call J, Haun DBM. (2013) Chimpanzees (pan troglodytes) flexibly adjust their behaviour in order to maximize payoffs, not to conform to majorities. PLoS ONE 8, 1., e80945. doi:10.1371/journal.pone.0080945


References


Figure A1. Histogram of the distribution of neuroticism at the third assessment wave (chapter 3).

Figure A2. Histogram of the distribution of exogenous long-term difficulties at the second assessment wave (chapter 3).
Figure A3. The association between temperamental frustration (plotted as standardized values and grouped in parcels of 33.3%) at baseline, Endogenous or Exogenous SLEs, and Psychopathology at follow-up (chapter 6).

Figure A4. The association between temperamental frustration at baseline, Endogenous or Exogenous SLEs, and change in psychopathology between baseline and follow-up, reported in standardized values, and grouped in thirds (parcels of 33.3%) (chapter 6).
Figure A5. The association between Frustration, concurrent Psychopathology \( (T_1, \text{left}) \), or Psychopathology at follow-up \( (T_2, \text{right}) \), reported in standardized values, and grouped in thirds (parcels of 33.3\%) (chapter 6)

Figure A6. The association between Frustration and Psychopathology at follow-up \( (T_2) \) for women (left) or for men (right), reported in standardized values, and grouped in thirds (parcels of 33.3\%) (chapter 6)
### Table A1. Regression with neuroticism at $T_2$ as outcome, and neuroticism at $T_1$ (N1), gender and age, and interactions thereof with weighted LE-measures as predictors (chapter 2).

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Beta (P)</th>
<th>Beta (P)</th>
<th>Beta (P)</th>
<th>Beta (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1</td>
<td>.763 (.001)</td>
<td>.763 (.001)</td>
<td>.763 (.001)</td>
<td>.763 (.001)</td>
</tr>
<tr>
<td>Gender</td>
<td>.013 (.328)</td>
<td>.032 (.727)</td>
<td>.017 (.779)</td>
<td>.020 (.871)</td>
</tr>
<tr>
<td>Age</td>
<td>-.022 (.139)</td>
<td>.000 (.996)</td>
<td>-.010 (.871)</td>
<td>.002 (.976)</td>
</tr>
<tr>
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<td>.050 (.001)</td>
<td>.050 (.001)</td>
<td>.050 (.001)</td>
</tr>
<tr>
<td>Recent PLEs</td>
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<td>-.043 (.001)</td>
<td>-.055 (.376)</td>
<td>-.055 (.376)</td>
</tr>
<tr>
<td>Distant NLEs</td>
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<td>.043 (.001)</td>
<td>.043 (.001)</td>
<td>.043 (.001)</td>
</tr>
<tr>
<td>Distant PLEs</td>
<td>-.077 (.001)</td>
<td>-.077 (.001)</td>
<td>-.105 (.129)</td>
<td>-.105 (.129)</td>
</tr>
<tr>
<td>Gender*Age</td>
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<td>-.012 (.858)</td>
<td>-.028 (.644)</td>
<td>-.012 (.858)</td>
</tr>
<tr>
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<td>.007 (.880)</td>
<td>.007 (.880)</td>
<td>.007 (.880)</td>
</tr>
<tr>
<td>Gender*recent PLEs</td>
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<td>.058 (.264)</td>
<td>.058 (.264)</td>
<td>.058 (.264)</td>
</tr>
<tr>
<td>Gender*distant NLEs</td>
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<td>-.013 (.802)</td>
<td>-.013 (.802)</td>
<td>-.013 (.802)</td>
</tr>
<tr>
<td>Gender*distant PLEs</td>
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<td>-.004 (.940)</td>
<td>-.004 (.940)</td>
<td>-.004 (.940)</td>
</tr>
<tr>
<td>Age*recent NLEs</td>
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<td>-.043 (.337)</td>
<td>-.043 (.337)</td>
<td>-.043 (.337)</td>
</tr>
<tr>
<td>Age*recent PLEs</td>
<td>-.047 (.231)</td>
<td>-.047 (.231)</td>
<td>-.047 (.231)</td>
<td>-.047 (.231)</td>
</tr>
<tr>
<td>Age*distant NLEs</td>
<td>-.042 (.364)</td>
<td>-.042 (.364)</td>
<td>-.042 (.364)</td>
<td>-.042 (.364)</td>
</tr>
<tr>
<td>Age*distant PLEs</td>
<td>.031 (.425)</td>
<td>.031 (.425)</td>
<td>.031 (.425)</td>
<td>.031 (.425)</td>
</tr>
</tbody>
</table>

*Note. N= 2356, significant beta’s in bold (p< .001).*

### Table A2. Correlations between Neuroticism (N), Depression (D), or Anxiety (A) at baseline ($T_1$) and follow-up ($T_2$), and partial correlations adjustment (Adj.) for one-another, or for stressful life events (SLEs) between $T_1$ and $T_2$ (mediation), and the magnitude of the adjustment as percentage (%) of the total association (chapter 2).

<table>
<thead>
<tr>
<th>$T_1$</th>
<th>$T_2$</th>
<th>$r$</th>
<th>Adj. for A1 (%)</th>
<th>Adj. for D1 (%)</th>
<th>Adj. for N1 (%)</th>
<th>Adj. for both (%)</th>
<th>Adj. for SLEs (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1</td>
<td>N2</td>
<td>.78</td>
<td>.68 (13%)</td>
<td>.59 (24%)</td>
<td>.59 (24%)</td>
<td>.77 (1%)</td>
<td>.77 (1%)</td>
</tr>
<tr>
<td>D2</td>
<td>.60</td>
<td>.36 (40%)</td>
<td>.12 (81%)</td>
<td>.11 (82%)</td>
<td>.60 (30%)</td>
<td>.60 (30%)</td>
<td>.60 (30%)</td>
</tr>
<tr>
<td>A2</td>
<td>.51</td>
<td>.13 (75%)</td>
<td>.10 (81%)</td>
<td>.05 (91%)</td>
<td>.50 (91%)</td>
<td>.50 (91%)</td>
<td>.50 (91%)</td>
</tr>
<tr>
<td>D1</td>
<td>D2</td>
<td>.72</td>
<td>.51 (29%)</td>
<td>.50 (30%)</td>
<td>.40 (44%)</td>
<td>.70 (44%)</td>
<td>.70 (44%)</td>
</tr>
<tr>
<td>A1</td>
<td>A2</td>
<td>.69</td>
<td>.44 (36%)</td>
<td>.55 (21%)</td>
<td>.43 (37%)</td>
<td>.67 (37%)</td>
<td>.67 (37%)</td>
</tr>
</tbody>
</table>

*Note. n= 2347, all associations significant at $p< .01$, two-sided.*
Table A3. Neuroticism items of the Amsterdamse Biografische Vragenlijst (chapter 3).

1. Do you often feel lonely?
2. Does it often happen that you make a decision too late?
3. Do you ever feel unhappy without knowing why?
4. Are you often kept awake by troubles?
5. Are you usually so burdened by disappointments that you cannot put them out of your mind?
6. Do you ever have nightmares?
7. Do you sometimes have the feeling that your life is meaningless and pointless?
8. Do you sometimes feel that when it matters you are on your own in life?

Table A4. All distinguished paths and their labels (chapter 3).

<table>
<thead>
<tr>
<th>Path</th>
<th>Description</th>
<th>Type</th>
<th>Path</th>
<th>Description</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>N₁→N₂</td>
<td>T₁₂₁</td>
<td>N*</td>
<td>Stability</td>
<td>E*</td>
</tr>
<tr>
<td>2</td>
<td>N₁→LTĐendo</td>
<td>T₁₂₀</td>
<td>N*</td>
<td>Direct</td>
<td>E*</td>
</tr>
<tr>
<td>3</td>
<td>N₁→PLEs (T₃)</td>
<td>T₁₂₂</td>
<td>N</td>
<td>Delayed</td>
<td>E*</td>
</tr>
<tr>
<td>4</td>
<td>N₁→NLEs (T₃)</td>
<td>T₁₂₁</td>
<td>N</td>
<td>Delayed</td>
<td>E*</td>
</tr>
<tr>
<td>5</td>
<td>LTĐendo→N₁</td>
<td>T₀₁₁</td>
<td>E</td>
<td>Direct</td>
<td>N*</td>
</tr>
<tr>
<td>6</td>
<td>LTĐexo→N₂</td>
<td>T₁₂₂</td>
<td>E</td>
<td>Delayed</td>
<td>N</td>
</tr>
<tr>
<td>7</td>
<td>LTĐendo↔LTĐexo</td>
<td>T₀</td>
<td>E</td>
<td>Concurrent</td>
<td>N*</td>
</tr>
<tr>
<td>8</td>
<td>LTĐendo→N₃</td>
<td>T₁₂₁</td>
<td>E</td>
<td>Delayed</td>
<td>N*</td>
</tr>
<tr>
<td>9</td>
<td>LTĐexo→N₃</td>
<td>T₁₂₁</td>
<td>E</td>
<td>Delayed</td>
<td>N</td>
</tr>
<tr>
<td>10</td>
<td>N₁→PLEs (T₃)</td>
<td>T₀₁₁</td>
<td>N</td>
<td>Direct</td>
<td>N*</td>
</tr>
<tr>
<td>11</td>
<td>N₂→N₃</td>
<td>T₁₂₁</td>
<td>N*</td>
<td>Stability</td>
<td>N</td>
</tr>
<tr>
<td>12</td>
<td>N₂→NLEs (T₃)</td>
<td>T₁₂₁</td>
<td>N</td>
<td>Delayed</td>
<td>N*</td>
</tr>
<tr>
<td>13</td>
<td>N₂→PosΔQoL</td>
<td>T₁₂₁</td>
<td>N</td>
<td>Delayed</td>
<td>N</td>
</tr>
<tr>
<td>14</td>
<td>N₂→NegΔQoL</td>
<td>T₁₂₂</td>
<td>N</td>
<td>Delayed</td>
<td>N</td>
</tr>
<tr>
<td>15</td>
<td>(T₃)PLEs↔NLEs</td>
<td>T₀</td>
<td>E</td>
<td>Concurrent</td>
<td>N</td>
</tr>
<tr>
<td>16</td>
<td>(T₃)PLEs→N₃</td>
<td>T₀₁₁</td>
<td>E</td>
<td>Direct</td>
<td>N</td>
</tr>
<tr>
<td>17</td>
<td>(T₃)NLEs→N₃</td>
<td>T₀₁₁</td>
<td>E</td>
<td>Direct</td>
<td>N</td>
</tr>
<tr>
<td>18</td>
<td>(T₃)PLEs→N₄</td>
<td>T₀₁₁</td>
<td>E</td>
<td>Delayed</td>
<td>N</td>
</tr>
<tr>
<td>19</td>
<td>(T₃)NLEs→N₄</td>
<td>T₀₂₂</td>
<td>E</td>
<td>Delayed</td>
<td>N</td>
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<td>20</td>
<td>N₁→PosΔQoL</td>
<td>T₀₁₁</td>
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<td>Direct</td>
<td>N</td>
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<tr>
<td>21</td>
<td>N₁→N₄</td>
<td>T₀₁₁</td>
<td>N</td>
<td>Stability</td>
<td>N</td>
</tr>
<tr>
<td>22</td>
<td>N₁→NegΔQoL</td>
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<td>Delayed</td>
<td>N</td>
</tr>
<tr>
<td>23</td>
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<td>Delayed</td>
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<tr>
<td>24</td>
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<td>Delayed</td>
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<tr>
<td>25</td>
<td>PosΔQoL↔NegΔQoL</td>
<td>T₀</td>
<td>E</td>
<td>Concurrent</td>
<td>N</td>
</tr>
</tbody>
</table>

Note. The bold paths 1-35 are depicted in Figure 3, but all paths are described in the method section (including 36-48).

Legend: E= Environment; N= Neuroticism; N₁= neuroticism at T₁; N₂ at T₃ etc.; LTDs= long-term difficulties, Endo= endogenous, Exo= exogenous, PosΔQoL= improved life quality; NegΔQoL= diminished life quality.

*=estimated in the Final model (see Appendix Table A5 or the significant paths). The method section gives all details.
Table A5. Model testing and path estimates of neuroticism and environment effects (chapter 3).

<table>
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<tr>
<th>Effects</th>
<th>Span</th>
<th>Df</th>
<th>Log Likelihood</th>
<th>ΔX^2 (P value)</th>
<th>AIC</th>
<th>RMSEA</th>
<th>Path Description</th>
<th>Log Likelihood</th>
<th>CF</th>
<th>Cd</th>
<th>TRd</th>
<th>P value</th>
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<td>Baseline</td>
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<td>1.29</td>
<td>14847</td>
<td>15065</td>
<td>0.072</td>
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<td></td>
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<tr>
<td>Stability</td>
<td>Tx+1</td>
<td>47</td>
<td>-7638.09</td>
<td>1.27</td>
<td>523.41*</td>
<td>15386</td>
<td>0.198</td>
<td>1* N₁→N₂</td>
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<td>1.25</td>
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<td>(595.01)</td>
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<td>15589</td>
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<tr>
<td>Exogenous</td>
<td>T_x</td>
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<td>0.71*</td>
<td>14848</td>
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<td>36* N₁→LTDexo (T₂)</td>
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<td>1.60</td>
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<tr>
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<td>1.33</td>
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### Table A5. Model testing and path estimates of neuroticism and environment effects (chapter 3). (continued)

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<tr>
<th>Effects</th>
<th>Span</th>
<th>DF (Δdf)</th>
<th>Log Likelihood (X²)</th>
<th>CF (Cd)</th>
<th>ΔX²sb</th>
<th>Path Description</th>
<th>AIC</th>
<th>BIC</th>
<th>Path</th>
<th>Description</th>
<th>Log Likelihood</th>
<th>CF</th>
<th>Cd</th>
<th>TRd</th>
<th>P value</th>
</tr>
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<tbody>
<tr>
<td><strong>Environment</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Concurrent</td>
<td>$T_{c0}$</td>
<td>47</td>
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<td>1.31</td>
<td>71.19*</td>
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<td>LTDendo ↔ LTDexo</td>
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<td>1.38</td>
<td>0.34</td>
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<td>$T_{c1}$</td>
<td>51</td>
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<td>35.93*</td>
<td>14883</td>
<td>0.083</td>
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<td>LTDendo → N₂</td>
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<td>1.38</td>
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<td>(1.43)</td>
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<td>15071</td>
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<td>1.32</td>
<td>30.77*</td>
<td>14881</td>
<td>0.084</td>
<td>8°</td>
<td>LTDendo → N₁</td>
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<td>LTDexo → N₁</td>
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**Table A5.** Model testing and path estimates of neuroticism and environment effects (chapter 3). (continued)

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<tr>
<th>Effects</th>
<th>Span Df (Δdf)</th>
<th>Log Likelihood (X²)</th>
<th>CF (Ca)</th>
<th>ΔX²sb (P value)</th>
<th>AIC BIC</th>
<th>RMSEA</th>
<th>Path Description</th>
<th>Path</th>
<th>Log Likelihood</th>
<th>CF Cd TRd Pvalue</th>
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<td><strong>Final Model</strong></td>
<td>60 (17)</td>
<td>-7374.19 1.38</td>
<td>(126.19) (1.60)</td>
<td>12.04</td>
<td>14832 14987</td>
<td>0.061</td>
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<td>Catch-35 model</td>
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<td>-7371.98 1.38</td>
<td>(122.92) (1.20)</td>
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<td>14830</td>
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Note. *two-tailed significance p< .05; † Change in X² from the baseline model; ‡ Path as depicted in Figure 3 (and Appendix Table A4 and A7); § = change in X²sb (Δdf) compared to baseline model; df= degrees of freedom; LTDendo= endogenous long-term difficulties; LTDexo= exogenous long-term difficulties; NLEs= negative life events; PLEs= positive life events; PosAQoL= improved quality of life; NegAQoL= diminished life quality; Tα,β= at subsequent measurement wave; Tα,γ= over two intervals; Tα,δ= over three intervals; The method section gives details on the variables in the description row, the effect types, and model-fit indices BIC, AIC, and RMSEA. The sequence wherein paths of the between model are constrained to derive at the final model can be found in Appendix Table A7.

Modeling robustness check. A valid interpretation of the fit indices requires a sample-size/free-parameter ratio >5 [423], this was >7 in our study. We checked robustness using MLR-models with Tα improved or diminished life quality as categorical (either 0, 1, or 2) and all other LE-variables as censored from below (floor-effect). Doing so neither allowed for comparison of nested models (via ΔX²sb (Δdf) tests), nor for concurrent associations, nor for standardized results (impeding path comparison within a model); hence, would not serve our purposes. Furthermore, z-standardizations of data before modeling would impact the covariance metric, and thereby the integrity of relative mean-level change estimations [1121], while the heterogeneity of LE-measures impeded transformations to an across time grand mean and SD reference. However, comparison of so derived model indices (and unstandardized estimates) references the robustness of our primary findings.

Model "catch-35" wherein the direct effect of NLEs (T5) on N3 was estimated freely, instead of fixation at zero, fitted slightly better than the Final model (X²sb(1)=3.674, p= .0553, -1 df, +2 BIC, -2 AIC, rest identical). However, path 35 itself would have been non-significant (β=0.043, SE=0.02, p=.07). Furthermore, robustness checks like a linear (ML) estimation of the Final model fitted slightly worse (see Appendix Table A5) but showed all paths, comparable estimates, and somewhat higher p-values. However, the model selection procedure would have been slightly different; whereas the effect from N4 on PLEs/NLEs at T5 would have been discarded in the Final model, but the effect of NLEs at T5 on N3 would have been significant (β= 0.10, p<.05) and incorporated. Finally, the Final model with improved and diminished life quality (QoL) fitted as categorical (montecarlo) required deletion of path 25, their correlation. Results showed a non-significant direct E-effect of improved life quality on N4, but increased N4 for participants who experienced diminished life quality (OR=1.19, d= 0.11). Theoretically, this is an unlikely result, given that improved life quality associated r =-.21 (p<.001) with N4 (see Table 8) and the β= -.013 (p< .05) in the Final model. Second, the deleted association between improved and diminished life quality measures was estimated at r = -.49 (see Table 8) and β= -.44 in the Final model (both p<.001). Together, this supports the presented Final model as the best representation of the data.
Table A6. Indirect effects of neuroticism, positive and negative life events (PLEs/NLEs), long-term difficulties (LTDs), and improved or diminished life quality (QoL) (chapter 3).

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Outcome</th>
<th>Span</th>
<th>Years</th>
<th>Type effect</th>
<th>Effects</th>
<th>SE</th>
<th>P value</th>
<th>Mediators</th>
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<tbody>
<tr>
<td>N₁</td>
<td>N₁</td>
<td>T₁+₂</td>
<td>6</td>
<td>Indirect</td>
<td>0.439*</td>
<td>0.039</td>
<td>&lt;.001</td>
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<td></td>
<td></td>
<td>0.391*</td>
<td>0.042</td>
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<td>N₁</td>
</tr>
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<td></td>
<td></td>
<td></td>
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<td>0.016</td>
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<td>(T₂)LTDendo, N₂</td>
</tr>
<tr>
<td>N₁</td>
<td>N₁</td>
<td>T₁+₃</td>
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<td>Indirect</td>
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<td>0.033</td>
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<td>0.009</td>
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<td>T₁+₄</td>
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<td>0.028</td>
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<td>0.050</td>
<td>.007</td>
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<td>-0.093*</td>
<td>0.047</td>
<td>.505</td>
<td>N₄</td>
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### Table A6. Indirect effects of neuroticism, positive and negative life events (PLEs/NLEs), long-term difficulties (LTDs), and improved or diminished life quality (QoL) (chapter 3). (continued)

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<th>Type effect</th>
<th>Effects</th>
<th>SE</th>
<th>P value</th>
<th>Mediators</th>
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<td>Tₛ₋₂</td>
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<td>.007</td>
<td>N₁</td>
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<td></td>
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<td>Indirect</td>
<td>0.132*</td>
<td>0.049</td>
<td>.007</td>
<td>N₁</td>
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<td>Tₛ₋₂</td>
<td>7.5</td>
<td>Total</td>
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<td>Tₛ₋₂</td>
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<td>PLEs (T₃)</td>
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<td></td>
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<td>.942</td>
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<td>0.067</td>
<td>.790</td>
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<td>.119</td>
<td>N₁</td>
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Note. *p* = two-tailed significance (Ns.= non-significant); †= path as depicted in Figure 3 or described in the method section; N₁= neuroticism at T₁ (N₂ at T₂ etc); LTDs= long-term difficulties, Endo= endogenous, Exo= exogenous, PosΔQoL= improved quality of life; NegΔQoL= diminished life quality. The method section gives all details.

### Table A7. Path Constraints to derive the Final Model (chapter 3).

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<th>Path</th>
<th>P-value</th>
<th>Effect</th>
</tr>
</thead>
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<td>1</td>
<td>29 NegΔQoL→N₅</td>
<td>.84</td>
<td>E Direct</td>
</tr>
<tr>
<td>2</td>
<td>34 PLE (T₃)→N₅</td>
<td>.81</td>
<td>E Direct</td>
</tr>
<tr>
<td>3</td>
<td>18 PLE (T₃)→N₄</td>
<td>.77</td>
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<td>4</td>
<td>7 LTDendo↔LTDexo</td>
<td>.53</td>
<td>E Concurrent</td>
</tr>
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</tr>
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<td>6</td>
<td>16 PLE (T₃)→N₅</td>
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<tr>
<td>7</td>
<td>17 NLE (T₃)→N₅</td>
<td>.24</td>
<td>E Direct</td>
</tr>
<tr>
<td>8</td>
<td>19 NLE (T₃)→N₄</td>
<td>.20</td>
<td>E Delayed</td>
</tr>
<tr>
<td>9</td>
<td>10 N₂→PLE (T₃)</td>
<td>.13</td>
<td>N Direct</td>
</tr>
<tr>
<td>10</td>
<td>6 LTDexo→N₂</td>
<td>.12</td>
<td>E Direct</td>
</tr>
<tr>
<td>11</td>
<td>35 NLE (T₃)→N₅</td>
<td>.08</td>
<td>E Direct</td>
</tr>
</tbody>
</table>

Note. LTDendo= endogenous long-term difficulties; LTDexo= exogenous long-term difficulties; N₁= neuroticism at baseline, N₂ at follow up; NLEs= negative life events; PLEs= positive life events; PosΔQoL= improved life quality; NegΔQoL= diminished life quality. The method section gives definitions of each type of effects.
<table>
<thead>
<tr>
<th>Neuroticism</th>
<th>Intercept</th>
<th>SE</th>
<th>Residual Variance</th>
<th>SE</th>
<th>$R^2$</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>$N_2$</td>
<td>2.02**</td>
<td>0.26</td>
<td>0.53**</td>
<td>0.05</td>
<td>0.47**</td>
<td>0.05</td>
</tr>
<tr>
<td>$N_3$</td>
<td>1.31**</td>
<td>0.22</td>
<td>0.41**</td>
<td>0.05</td>
<td>0.60**</td>
<td>0.05</td>
</tr>
<tr>
<td>$N_4$</td>
<td>2.47**</td>
<td>0.29</td>
<td>0.60**</td>
<td>0.06</td>
<td>0.40**</td>
<td>0.06</td>
</tr>
<tr>
<td>$N_5$</td>
<td>0.83*</td>
<td>0.29</td>
<td>0.50**</td>
<td>0.05</td>
<td>0.50**</td>
<td>0.05</td>
</tr>
<tr>
<td>LTD Endo ($T_2$)</td>
<td>-1.11***</td>
<td>0.30</td>
<td>0.85**</td>
<td>0.05</td>
<td>0.15**</td>
<td>0.05</td>
</tr>
<tr>
<td>LTD Exo ($T_3$)</td>
<td>0.78**</td>
<td>0.04</td>
<td></td>
<td></td>
<td></td>
<td>0.00</td>
</tr>
<tr>
<td>PLEs ($T_3$)</td>
<td>0.88**</td>
<td>0.04</td>
<td></td>
<td></td>
<td></td>
<td>0.00</td>
</tr>
<tr>
<td>NLEs ($T_3$)</td>
<td>0.21</td>
<td>0.33</td>
<td>0.98**</td>
<td>0.02</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Pos$\Delta$QoL ($T_4$)</td>
<td>0.84**</td>
<td>0.05</td>
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<td></td>
<td></td>
<td>0.00</td>
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<tr>
<td>Neg$\Delta$QoL ($T_4$)</td>
<td>-0.24</td>
<td>0.31</td>
<td>0.98**</td>
<td>0.02</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>PLEs ($T_5$)</td>
<td>0.08</td>
<td>0.33</td>
<td>0.98**</td>
<td>0.02</td>
<td>0.00</td>
<td></td>
</tr>
<tr>
<td>NLEs ($T_5$)</td>
<td>0.05</td>
<td>0.30</td>
<td>0.98**</td>
<td>0.02</td>
<td>0.02</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*Note.* Two-sided significance **$= p < .001$, *$= p < .005$. LTDendo= endogenous long-term difficulties; LTDexo= exogenous long-term difficulties; $N_1$= neuroticism at baseline, $N_2$ at follow up; NLEs= negative life events; PLEs= positive life events; Pos$\Delta$QoL= improved life quality; Neg$\Delta$QoL= diminished life quality; SE= standard error; $R^2$= proportion explained variance.
Table A9. Frustration Model Fit Indices (chapter 6).

<table>
<thead>
<tr>
<th></th>
<th>Baseline Model</th>
<th>Frustration Model</th>
<th>Baseline Model</th>
<th>Change Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>957</td>
<td>957</td>
<td>957</td>
<td>957</td>
</tr>
<tr>
<td>Variables</td>
<td>7</td>
<td>6</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>( \chi^2 )</td>
<td>1100.17</td>
<td>1196.45</td>
<td>1300.49</td>
<td>1586.88</td>
</tr>
<tr>
<td>df</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Loglikelihood model (H₁)</td>
<td>-10779.25</td>
<td>-10779.25</td>
<td>-10967.44</td>
<td>-10967.44</td>
</tr>
<tr>
<td>CFI</td>
<td>0.41</td>
<td>0.36</td>
<td>0.46</td>
<td>0.34</td>
</tr>
<tr>
<td>TLI</td>
<td>-0.27</td>
<td>-0.21</td>
<td>-0.26</td>
<td>-0.16</td>
</tr>
<tr>
<td>RMSEA</td>
<td>0.40</td>
<td>0.39</td>
<td>0.39</td>
<td>0.37</td>
</tr>
<tr>
<td>AIC</td>
<td>26557.65</td>
<td>26556.84</td>
<td>26941.59</td>
<td>26939.39</td>
</tr>
<tr>
<td>BIC</td>
<td>26654.93</td>
<td>26649.25</td>
<td>27068.05</td>
<td>27051.26</td>
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<tr>
<td>( \chi^2 ) independence model</td>
<td>1858.51</td>
<td>1858.51</td>
<td>2404.48</td>
<td>2404.48</td>
</tr>
<tr>
<td>df</td>
<td>15</td>
<td>15</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>Loglikelihood ( H_0 )</td>
<td>-13258.83</td>
<td>-13259.42</td>
<td>-13444.80</td>
<td>-13446.70</td>
</tr>
<tr>
<td>Scaling factor (TRd)</td>
<td>1.7314</td>
<td>1.74</td>
<td>1.58</td>
<td>1.64</td>
</tr>
<tr>
<td>Free parameters</td>
<td>20</td>
<td>19</td>
<td>26</td>
<td>23</td>
</tr>
<tr>
<td>( \Delta df )</td>
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<td></td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>TRd</td>
<td>0.74</td>
<td></td>
<td>3.56</td>
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</tr>
<tr>
<td>( p )</td>
<td>.39</td>
<td></td>
<td>.31</td>
<td></td>
</tr>
</tbody>
</table>

Note. \( \chi^2 \) = Chi-square; \( df \) = degrees of freedom; AIC = Akaike information criterion (information entropy); BIC = Bayesian information criterion (most sensitive to parsimony); CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root-mean-square error of approximation. Note that the secondary fit-indices (CFI, TLI, RMSEA) could not be estimated reliably, see method section, as well as for information about all variables, model-fit indices, and modelling procedures. \( \Delta df \) = degrees of freedom between both models; \( \chi^2 \) = result of Santorra-Bentler corrected chi-square difference test given the mentioned difference in \( df \); \( p \) = significance test. Each baseline model is unstructured, see Figure 7 for the frustration model, and Figure 8 for the change model (chapter 6).
### Table A10. Path Coefficients for Frustration Model (chapter 6)

<table>
<thead>
<tr>
<th>Path</th>
<th>Baseline Model</th>
<th></th>
<th>Frustration Model</th>
<th></th>
<th>[95% CI]</th>
<th></th>
<th>Baseline Model</th>
<th></th>
<th>Change in Psychopathology Model</th>
<th></th>
<th>[95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>Fr ↔ Psych T₁</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prospective</td>
<td>Fr → Psych T₂</td>
<td>0.22***</td>
<td>0.21***</td>
<td>0.15 to 0.27</td>
<td>0.11***</td>
<td>0.11***</td>
<td>0.05 to 0.17</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psych T₁ → Psych T₂</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Stress Generation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr → Endo SLEs</td>
<td>0.13***</td>
<td>0.14***</td>
<td>0.07 to 0.21</td>
<td>0.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr → Exo SLEs</td>
<td>0.07*</td>
<td>0.07*</td>
<td>0.00 to 0.14</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endo SLEs → Psych T₂</td>
<td></td>
<td>0.24***</td>
<td>0.24***</td>
<td>0.17 to 0.30</td>
<td>0.21***</td>
<td>0.21***</td>
<td>0.14 to 0.27</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endo SLEs → Endo SLEs</td>
<td></td>
<td>0.22***</td>
<td>0.24***</td>
<td>0.18 to 0.30</td>
<td>0.18 to 0.30</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exo SLEs → Psych T₂</td>
<td></td>
<td>0.33***</td>
<td>0.34***</td>
<td>0.20 to 0.48</td>
<td>0.25***</td>
<td>0.26***</td>
<td>0.15 to 0.36</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Stress Sensitivity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Fr*Endo → Psych T₂</td>
<td>0.21**</td>
<td>0.28***</td>
<td>0.20 to 0.35</td>
<td>0.19</td>
<td>0.19</td>
<td>0.12 to 0.26</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr*Exo → Psych T₂</td>
<td>-0.25**</td>
<td>-0.27***</td>
<td>-0.10 to -0.43</td>
<td>-0.24**</td>
<td>-0.25**</td>
<td>-0.12 to -0.37</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. T₁= baseline, T₂= follow-up; df= degrees of freedom; CI= confidence interval; Endo=endogenous SLEs; Exo= exogenous SLEs; Fr= frustration; Psych= psychopathology; SLEs= stressful life events; X²= Chi-square. All paths in both final models were tested (in the Jöreskog tradition, see Table A12), and all paths added significantly to model fit (all p< .0001). The method section provides information about all variables, model-fit indices, and modelling procedures. Each baseline model is unstructured, see Figure 7 for the frustration model and Figure 8 for the change model (chapter 6). Significance ***p< .001, **p< .01, *p< .05, two-tailed.
### Table A11. Path Constraints in the Frustration model (chapter 6)

<table>
<thead>
<tr>
<th>#</th>
<th>Path</th>
<th>Variable in model</th>
<th>Test of nested model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$p$</td>
<td>$\beta$</td>
</tr>
<tr>
<td>Baseline model</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>SLEs Endo $\rightarrow$ Psych $T_2$</td>
<td>.93</td>
<td>0.01</td>
</tr>
<tr>
<td>2</td>
<td>Fr $\rightarrow$ SLEs Exo</td>
<td>.62</td>
<td>0.02</td>
</tr>
<tr>
<td>3</td>
<td>Fr $\rightarrow$ SLEs Endo</td>
<td>.05</td>
<td>0.07</td>
</tr>
<tr>
<td>Change model</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>SLEs Endo $\rightarrow$ Psych $T_2$</td>
<td>.37</td>
<td>0.07</td>
</tr>
</tbody>
</table>

**Note.** If the $X^2$ difference value is not significant, this indicates that constraining the parameters of the nested model did not significantly worsen the fit of the model, which indicates measurement invariance of the parameters constrained to be equal in the nested model. The frustration model is visualized in Figure 7 and the change model in Figure 8. All $X^2$-tests with $\Delta df=1$ are significant from 3.84 ($p<.05$), 6.64 ($p<.01$), and 10.83 ($p<.001$).

### Table A12. Jöreskog tests for all paths in Final Models (chapter 6)

<table>
<thead>
<tr>
<th></th>
<th>Frustration Model</th>
<th>Change in Psychopathology Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\beta$</td>
<td>$\Delta X^2$</td>
</tr>
<tr>
<td>Baseline</td>
<td>Fr $\leftrightarrow$ Psych $T_1$</td>
<td>0.27***</td>
</tr>
<tr>
<td>Prospective</td>
<td>Fr $\rightarrow$ Psych $T_2$</td>
<td>0.21***</td>
</tr>
<tr>
<td></td>
<td>Psych $T_1$ $\rightarrow$ Psych $T_2$</td>
<td>0.49***</td>
</tr>
<tr>
<td>Stress Generation</td>
<td>Fr $\rightarrow$ Endo SLEs</td>
<td>0.14***</td>
</tr>
<tr>
<td></td>
<td>Fr $\rightarrow$ Exo SLEs</td>
<td>0.07*</td>
</tr>
<tr>
<td></td>
<td>Psych $T_1$ $\rightarrow$ Endo SLEs</td>
<td>0.24***</td>
</tr>
<tr>
<td></td>
<td>Psych $T_1$ $\rightarrow$ Exo SLEs</td>
<td>0.20***</td>
</tr>
<tr>
<td></td>
<td>Endo $\leftrightarrow$ Exo SLEs</td>
<td>0.21***</td>
</tr>
<tr>
<td></td>
<td>Endo SLEs $\rightarrow$ Psych $T_2$</td>
<td>0.26***</td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td>Fr*Endo $\rightarrow$ Psych $T_2$</td>
<td>0.19***</td>
</tr>
<tr>
<td></td>
<td>Fr*Exo $\rightarrow$ Psych $T_2$</td>
<td>-0.25***</td>
</tr>
</tbody>
</table>

**Note.** Significant $X^2$ differences indicate that constraining this parameter to zero in a nested structure had significantly worsened the fit of the model. The frustration model is visualized in Figure 7 and the change model in Figure 8 (chapter 6). All tests with $\Delta df=1$, which are significant from 3.84 ($p<.05$), 6.64 ($p<.01$), and 10.83 ($p<.001$).
Table A13. Change in psychopathy model: All Indirect Effects Between Frustration and Psychopathy at Follow-up (chapter 6)

<table>
<thead>
<tr>
<th>Source</th>
<th>Beta</th>
<th>95% CI</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total effect</td>
<td>0.26***</td>
<td>0.19 to 0.32</td>
<td>100.0%</td>
</tr>
<tr>
<td>Total direct effect</td>
<td>0.11***</td>
<td>0.11 to 0.17</td>
<td>43.5%</td>
</tr>
<tr>
<td>Total indirect effect</td>
<td>0.14***</td>
<td>0.11 to 0.18</td>
<td>56.5%</td>
</tr>
<tr>
<td>Psychopathology $T_1$</td>
<td>0.13***</td>
<td>0.09 to 0.17</td>
<td>51.4%</td>
</tr>
<tr>
<td>Psychopathology $T_1$*Exogenous SLEs</td>
<td>0.01**</td>
<td>0.01 to 0.02</td>
<td>5.1%</td>
</tr>
</tbody>
</table>

Note. SLEs = Stressful Life Events; $T_1$ = baseline; CI = Confidence Interval. The model is visualized in Figure 8. The method section provides information about all variables, model-fit indices, and modelling procedures. Significance *** $p < .001$, ** $p < .01$, * $p < .05$, two-tailed.

Table A14. Fit indices Models Internalizing Tendencies (chapter 6)

<table>
<thead>
<tr>
<th></th>
<th>Baseline Model</th>
<th>Internalising Model</th>
<th>Baseline Model</th>
<th>Change Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>957</td>
<td>957</td>
<td>957</td>
<td>957</td>
</tr>
<tr>
<td>Variables</td>
<td>6</td>
<td>6</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>$X^2$</td>
<td>1044.28</td>
<td>1326.40</td>
<td>1253.99</td>
<td>1686.45</td>
</tr>
<tr>
<td>$df$</td>
<td>7</td>
<td>10</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>Loglikelihood model ($H_1$)</td>
<td>-10172.59</td>
<td>Equal</td>
<td>-10005.01</td>
<td>Equal</td>
</tr>
<tr>
<td>CFI</td>
<td>0.40</td>
<td>0.24</td>
<td>0.46</td>
<td>0.27</td>
</tr>
<tr>
<td>TLI</td>
<td>-0.28</td>
<td>-0.14</td>
<td>-0.27</td>
<td>-0.10</td>
</tr>
<tr>
<td>RMSEA</td>
<td>0.39</td>
<td>0.37</td>
<td>0.38</td>
<td>0.35</td>
</tr>
<tr>
<td>AIC</td>
<td>25342.38</td>
<td>25344.22</td>
<td>25018.59</td>
<td>25016.74</td>
</tr>
<tr>
<td>BIC</td>
<td>25439.66</td>
<td>25426.90</td>
<td>25145.04</td>
<td>25118.88</td>
</tr>
<tr>
<td>$X^2$ independence model</td>
<td>1749.01</td>
<td>Equal</td>
<td>2306.70</td>
<td>Equal</td>
</tr>
<tr>
<td>$df$</td>
<td>15</td>
<td>Equal</td>
<td>21</td>
<td>Equal</td>
</tr>
<tr>
<td>Loglikelihood $H_0$</td>
<td>-12651.19</td>
<td>-12655.11</td>
<td>-12483.29</td>
<td>-12487.37</td>
</tr>
<tr>
<td>Scaling factor (TRd)</td>
<td>1.71</td>
<td>1.76</td>
<td>1.56</td>
<td>1.66</td>
</tr>
<tr>
<td>Free parameters</td>
<td>20</td>
<td>17</td>
<td>26</td>
<td>21</td>
</tr>
</tbody>
</table>

Note. $X^2$ = Chi-square; $df$ = degrees of freedom; AIC = Akaike information criterion (information entropy); BIC = Bayesian information criterion (most sensitive to parsimony); CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root-mean-square error of approximation. Note that the secondary fit-indices (CFI, TLI, RMSEA) could not be estimated reliably, see method section (also for information about all variables, model-fit indices, and modelling procedures). $\Delta df$ = degrees of freedom between both models; $X^2$ = result of Santorra-Bentler corrected (TRd) chi-square difference test given the mentioned difference in $df$; $p$ = significance test. Each baseline model is unstructured; the nested internalizing model is structured like Figure 7, the nested change model like Figure 8 (chapter 6).
Table A15. Path coefficients Internalizing Models (chapter 6)

<table>
<thead>
<tr>
<th>Path</th>
<th>Baseline Model</th>
<th>Internalizing Model</th>
<th>Baseline Model</th>
<th>Change Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Beta</td>
<td>Beta</td>
<td>[95% CI]</td>
<td>Beta</td>
</tr>
<tr>
<td>Baseline</td>
<td>Fr ↔ Internalizing $T_1$</td>
<td>0.11**</td>
<td>0.11***</td>
<td>0.04 to 0.19</td>
</tr>
<tr>
<td>Prospective</td>
<td>Fr ↔ Internalizing $T_2$</td>
<td>0.06</td>
<td>-0.01</td>
<td></td>
</tr>
<tr>
<td>Internalizing $T_1$ → $T_2$</td>
<td>0.54***</td>
<td>0.55***</td>
<td>0.51 to 0.60</td>
<td></td>
</tr>
<tr>
<td>Stress Generation</td>
<td>Fr → Endo SLEs</td>
<td>0.13***</td>
<td>0.13***</td>
<td>0.06 to 0.20</td>
</tr>
<tr>
<td>Fr → Exo SLEs</td>
<td>0.07*</td>
<td>0.07</td>
<td>0.01 to 0.14</td>
<td>0.06</td>
</tr>
<tr>
<td>Internalizing $T_1$ → Endo SLEs</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internalizing $T_1$ → Exo SLEs</td>
<td>0.14***</td>
<td>0.15***</td>
<td>0.09 to 0.21</td>
<td></td>
</tr>
<tr>
<td>Endo ↔ Exo SLEs</td>
<td>0.24***</td>
<td>0.24***</td>
<td>0.17 to 0.30</td>
<td>0.24***</td>
</tr>
<tr>
<td>Endo SLEs → Internalizing $T_2$</td>
<td>-0.13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exo SLEs → Internalizing $T_2$</td>
<td>0.29***</td>
<td>0.23***</td>
<td>0.09 to 0.36</td>
<td>0.18***</td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td>Fr*Endo → Internalizing $T_2$</td>
<td>0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr*Exo → Internalizing $T_2$</td>
<td>-0.24**</td>
<td>-0.16***</td>
<td>-0.02 to -0.31</td>
<td>-0.19***</td>
</tr>
</tbody>
</table>

Note. $T_1$: baseline, $T_2$: follow-up; $df$= degrees of freedom; CI= confidence interval; Endo=endogenous SLEs; Exo= exogenous SLEs; Fr= frustration; SLEs= stressful life events; $X^2$= Chi-square. All paths in both final models were tested in the Jöreskog tradition and added significantly to model fit (all $p< .0001$), while absent paths were constrained to zero (see Table A11). The method section provides information about all variables, model-fit indices, and modelling procedures, but in short: each baseline model is unstructured, the nested internalizing model is structured like Figure 7, the nested change model like Figure 8 (see chapter 6). Significance *** $p< .001$, ** $p< .01$, * $p< .05$, two-tailed.

Table A16. Path Constraints Internalizing Model (chapter 6)

<table>
<thead>
<tr>
<th>#</th>
<th>Path</th>
<th>Variable in model</th>
<th>Test of nested model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$p$</td>
<td>$\beta$</td>
</tr>
<tr>
<td>Frustration Model</td>
<td>Fr → Internalizing $T_2$</td>
<td>.11</td>
<td>0.06</td>
</tr>
<tr>
<td>1.</td>
<td>Fr* SLEs Endo</td>
<td>.23</td>
<td>0.12</td>
</tr>
<tr>
<td>2.</td>
<td>SLEs Endo → Internalizing $T_2$</td>
<td>.17</td>
<td>-0.05</td>
</tr>
<tr>
<td>Change in Internalizing Model</td>
<td>Fr → Internalizing $T_2$</td>
<td>.86</td>
<td>-0.01</td>
</tr>
<tr>
<td>1.</td>
<td>Internalizing $T_1$ → SLEs Endo</td>
<td>.76</td>
<td>0.01</td>
</tr>
<tr>
<td>2.</td>
<td>Fr*Endo → Internalizing $T_2$</td>
<td>.13</td>
<td>0.10</td>
</tr>
<tr>
<td>3.</td>
<td>SLEs Endo → Internalizing $T_2$</td>
<td>.19</td>
<td>-0.04</td>
</tr>
<tr>
<td>4.</td>
<td>Fr* SLEs Exo</td>
<td>.09</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Note. Significant $X^2$ differences indicate that constraining this parameter to zero in a nested structure had significantly worsened the fit of the model. The nested internalizing model is structured like Figure 7, the nested change model like Figure 8. All tests with $\Delta df= 1$, which are significant from 3.84 ($p< .05$), 6.64 ($p< .01$), and 10.83 ($p< .001$).
Table A17. Model Fit Indices Externalizing Model (chapter 6)

<table>
<thead>
<tr>
<th>Model</th>
<th>Baseline Model</th>
<th>Externalizing Model</th>
<th>Baseline Model</th>
<th>Change Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>957</td>
<td>957</td>
<td>957</td>
<td>957</td>
</tr>
<tr>
<td>Variables</td>
<td>6</td>
<td>6</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>$\chi^2$</td>
<td>1060.41</td>
<td>1330.76</td>
<td>1264.15</td>
<td>1669.19</td>
</tr>
<tr>
<td>df</td>
<td>7</td>
<td>10</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>Loglikelihood model ($H_1$)</td>
<td>-9940.65</td>
<td>-9940.65</td>
<td>-9631.18</td>
<td>Equal</td>
</tr>
<tr>
<td>CFI</td>
<td>0.41</td>
<td>0.26</td>
<td>0.44</td>
<td>0.26</td>
</tr>
<tr>
<td>TLI</td>
<td>-0.27</td>
<td>-0.11</td>
<td>-0.31</td>
<td>-0.11</td>
</tr>
<tr>
<td>RMSEA</td>
<td>0.40</td>
<td>0.37</td>
<td>0.38</td>
<td>0.35</td>
</tr>
<tr>
<td>AIC</td>
<td>24890.77</td>
<td>24887.39</td>
<td>24285.96</td>
<td>24281.77</td>
</tr>
<tr>
<td>BIC</td>
<td>24988.05</td>
<td>24970.07</td>
<td>24412.42</td>
<td>24383.91</td>
</tr>
<tr>
<td>$\chi^2$ independence model</td>
<td>1799.40</td>
<td>1499.40</td>
<td>2261.12</td>
<td>Equal</td>
</tr>
<tr>
<td>df</td>
<td>15</td>
<td>15</td>
<td>21</td>
<td>Equal</td>
</tr>
<tr>
<td>Loglikelihood $H_0$</td>
<td>-12425.39</td>
<td>-12426.69</td>
<td>-12116.98</td>
<td>-12119.89</td>
</tr>
<tr>
<td>Scaling factor (TRd)</td>
<td>1.72</td>
<td>1.75</td>
<td>1.61</td>
<td>1.69</td>
</tr>
<tr>
<td>Free parameters</td>
<td>20</td>
<td>17</td>
<td>26</td>
<td>21</td>
</tr>
<tr>
<td>$\Delta$df</td>
<td>3</td>
<td></td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>TRd</td>
<td>1.71</td>
<td></td>
<td></td>
<td>4.59</td>
</tr>
<tr>
<td>$p$</td>
<td>.64</td>
<td></td>
<td></td>
<td>.47</td>
</tr>
</tbody>
</table>

Note. X^2 = Chi-square; df = degrees of freedom; AIC= Akaike information criterion (information entropy); BIC= Bayesian information criterion (most sensitive to parsimony); CFI= comparative fit index; TLI= Tucker-Lewis index; RMSEA= root-mean-square error of approximation. Note that the secondary fit-indices (CFI, TLI, RMSEA) could not be estimated reliably, see method section (also for information about all variables, model-fit indices, and modelling procedures). $\Delta$df= degrees of freedom between both models; $\chi^2$ = result of Santorra-Bentler corrected (TRd) chi-square difference test given the mentioned difference in df; p= significance test. Each baseline model is unstructured, the nested externalizing model is structured like Figure 7, the nested change model like Figure 8.
### Table A18. Path Coefficients in Change in Externalizing Tendencies Model (chapter 6)

<table>
<thead>
<tr>
<th>Path</th>
<th>Baseline Model</th>
<th>Externalizing Model</th>
<th>Baseline Model</th>
<th>Externalizing Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Beta</td>
<td>Beta</td>
<td>Beta</td>
<td>Beta</td>
</tr>
<tr>
<td></td>
<td>[95% CI]</td>
<td>[95% CI]</td>
<td>[95% CI]</td>
<td>[95% CI]</td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
<td></td>
<td>0.20***</td>
<td>0.20***</td>
</tr>
<tr>
<td>Prospective</td>
<td>0.14***</td>
<td>0.16***</td>
<td>0.10 to 0.23</td>
<td>0.08*</td>
</tr>
<tr>
<td>Externalizing T₁ → T₂</td>
<td>0.24***</td>
<td>0.26***</td>
<td>0.17 to 0.30</td>
<td>0.23***</td>
</tr>
<tr>
<td>Stress Generation</td>
<td>0.24***</td>
<td>0.24***</td>
<td>0.36</td>
<td>0.16*</td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td>0.07***</td>
<td>0.07***</td>
<td>0.21</td>
<td>0.19***</td>
</tr>
<tr>
<td>Exo SLEs → Externalizing T₁</td>
<td>-0.03</td>
<td>-0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr*Exo → Externalizing T₂</td>
<td>0.28</td>
<td>0.10</td>
<td>1.24</td>
<td>1</td>
</tr>
<tr>
<td>Fr*Endo → Externalizing T₂</td>
<td>0.09</td>
<td>0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr*Endo SLEs → Externalizing T₂</td>
<td>0.28</td>
<td>0.10</td>
<td>1.24</td>
<td>1</td>
</tr>
</tbody>
</table>

*Note.* T₁= baseline, T₂= follow-up; df= degrees of freedom; CI= confidence interval; Endo=endogenous SLEs; Exo= exogenous SLEs; Fr= frustration; SLEs= stressful life events; X²= Chi-square. All paths in both final models were tested in the Jöreskog tradition and added significantly to model fit (all p < .0001), while absent paths were constrained to zero (see Table A16). The method section provides information about all variables, model-fit indices, and modelling procedures, but in short: each baseline model is unstructured, the nested externalizing model is structured like Figure 7, the nested change model like Figure 8 (chapter 6). Significance: ***p < .001, **p < .01, *p < .05, two-tailed.

### Table A19. Constraints in Externalizing Model (chapter 6)

<table>
<thead>
<tr>
<th># Path</th>
<th>Variable in model</th>
<th>Test of nested model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p</td>
<td>β</td>
</tr>
<tr>
<td><strong>Frustration Model</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. SLEs Exo → Externalizing T₂</td>
<td>.70</td>
<td>-0.03</td>
</tr>
<tr>
<td>2. Fr*SLEs → Externalizing T₂</td>
<td>.66</td>
<td>0.02</td>
</tr>
<tr>
<td>3. Fr*SLEs → Externalizing T₂</td>
<td>.28</td>
<td>0.10</td>
</tr>
<tr>
<td><strong>Change in Externalizing Model</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. SLEs Exo → Externalizing T₂</td>
<td>.88</td>
<td>-0.01</td>
</tr>
<tr>
<td>2. Fr*Exo SLEs → Externalizing T₂</td>
<td>.85</td>
<td>0.01</td>
</tr>
<tr>
<td>3. Fr*Endo SLEs → Externalizing T₂</td>
<td>.55</td>
<td>0.05</td>
</tr>
<tr>
<td>4. Fr → SLEs Exo</td>
<td>.14</td>
<td>0.06</td>
</tr>
<tr>
<td>5. Fr → SLEs Endo</td>
<td>.09</td>
<td>0.36</td>
</tr>
</tbody>
</table>

*Note.* All X²-tests with Δdf= 1 are significant from 3.84 (p < .05), 6.64 (p < .01), and 10.83 (p < .001). If the X² difference value is not significant, the parameters of the nested model did not significantly worsen the fit of the model, indicating measurement invariance of the parameters constrained to zero in the nested model. The nested internalizing model is structured like Figure 7, the nested change model like Figure 8 (chapter 6).
Table A20. Fit indices for Gender-stratified Frustration Models (chapter 6)

<table>
<thead>
<tr>
<th></th>
<th>Psychopathology</th>
<th>Internalizing</th>
<th>Externalizing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Final</td>
<td>Baseline</td>
</tr>
<tr>
<td>N</td>
<td>957</td>
<td>957</td>
<td>957</td>
</tr>
<tr>
<td>Women</td>
<td>523</td>
<td>523</td>
<td>523</td>
</tr>
<tr>
<td>Variables</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>$\chi^2$</td>
<td>1460.85</td>
<td>2657.20</td>
<td>1484.31</td>
</tr>
<tr>
<td>df</td>
<td>18</td>
<td>42</td>
<td>18</td>
</tr>
<tr>
<td>Loglikelihood model (H1)</td>
<td>-10876.63</td>
<td>-9832.81</td>
<td>-9531.99</td>
</tr>
<tr>
<td>CFI</td>
<td>0.45</td>
<td>0.21</td>
<td>0.43</td>
</tr>
<tr>
<td>TLI</td>
<td>-0.29</td>
<td>-0.04</td>
<td>-0.34</td>
</tr>
<tr>
<td>RMSEA</td>
<td>0.41</td>
<td>0.37</td>
<td>0.41</td>
</tr>
<tr>
<td>AIC</td>
<td>26847.94</td>
<td>26836.19</td>
<td>24766.27</td>
</tr>
<tr>
<td>BIC</td>
<td>27100.85</td>
<td>27021.01</td>
<td>25019.19</td>
</tr>
<tr>
<td>$\chi^2$ independence model</td>
<td>2657.20</td>
<td>2598.09</td>
<td>2551.27</td>
</tr>
<tr>
<td>df</td>
<td>42</td>
<td>42</td>
<td>42</td>
</tr>
<tr>
<td>Loglikelihood H0</td>
<td>-13371.97</td>
<td>-13380.09</td>
<td>-12331.14</td>
</tr>
<tr>
<td>Scaling-factor (TRd)</td>
<td>1.53</td>
<td>1.70</td>
<td>1.55</td>
</tr>
<tr>
<td>Free parameters</td>
<td>52</td>
<td>52</td>
<td>52</td>
</tr>
<tr>
<td>$\Delta$df</td>
<td>14</td>
<td>16.99</td>
<td>16</td>
</tr>
<tr>
<td>TRd</td>
<td>15.21</td>
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<td>17.10</td>
</tr>
<tr>
<td>$p$</td>
<td>.36</td>
<td>.20</td>
<td>.38</td>
</tr>
</tbody>
</table>

Note. $N$ = number of participants; $\chi^2$ = Chi-square; df = degrees of freedom; AIC = Akaike information criterion (information entropy); BIC = Bayesian information criterion (most sensitive to parsimony); CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root-mean-square error of approximation. Note that the secondary fit-indices (CFI, TLI, RMSEA) could not be estimated reliably, see method section (as well as for information about all variables, model-fit indices, and modelling procedures). $\Delta$df = degrees of freedom between both models; $\chi^2$ = result of Santorra-Bentler corrected (TRd) chi-square difference test given the mentioned difference in df; $p$ = significance test. Each baseline model is unstructured, the structure of the nested final models are visualized in Figure 8.
Table A21. Path Coefficients for Gender-stratified Psychopathology Model (chapter 6)

<table>
<thead>
<tr>
<th>Paths</th>
<th>Baseline</th>
<th>Final</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>Baseline</td>
<td>Fr ↔ Psych T₁</td>
<td>0.25***</td>
</tr>
<tr>
<td>Prospective</td>
<td>Fr → Psych T₂</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>Psych T₁ → Psych T₂</td>
<td>0.51***</td>
</tr>
<tr>
<td>Stress Generation</td>
<td>Fr → Endo SLEs</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>Fr → Exo SLEs</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Psych T₁ → Endo SLEs</td>
<td>0.19***</td>
</tr>
<tr>
<td></td>
<td>Psych T₁ → Exo SLEs</td>
<td>0.18***</td>
</tr>
<tr>
<td></td>
<td>Endo ↔ Exo SLEs</td>
<td>0.21***</td>
</tr>
<tr>
<td></td>
<td>Endo SLEs → Psych T₂</td>
<td>-0.03</td>
</tr>
<tr>
<td></td>
<td>Exo SLEs → Psych T₂</td>
<td>0.29***</td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td>Fr*Endo → Psych T₂</td>
<td>0.30***</td>
</tr>
<tr>
<td></td>
<td>Fr*Exo → Psych T₂</td>
<td>-0.30***</td>
</tr>
</tbody>
</table>

*Note.* T₁ = baseline, T₂ = follow-up; df = degrees of freedom; CI = confidence interval; Endo = endogenous SLEs; Exo = exogenous SLEs; Fr = frustration; SLEs = stressful life events; X² = Chi-square. All paths in both final models were tested in the Jöreskog tradition and added significantly to model fit (all p < .0001), while absent paths were constrained to zero (see Table A22). The method section provides information about all variables, model-fit indices, and modelling procedures, but in short: each baseline model is unstructured, the nested final model is structured alike Figure 8. Significance ***p < .001, **p < .01, *p < .05, two-tailed.
### Table A22. Constraints and Jöreskog tests of gender-equality in Psychopathology Model (chapter 6)

<table>
<thead>
<tr>
<th>Variable in model</th>
<th>Test of nested model</th>
<th>And gender model differences</th>
<th>Step</th>
<th>p</th>
<th>β</th>
<th>p</th>
<th>β</th>
<th>ΔX^2</th>
<th>Δdf</th>
<th>p</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr ↔ Psych T₁</td>
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<td>.001</td>
<td>.25</td>
<td>.001</td>
<td>.30</td>
<td>.030</td>
<td>1</td>
<td>.86</td>
<td>Equal</td>
</tr>
<tr>
<td>Prospective</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>.06</td>
<td>.001</td>
<td>.16</td>
<td>1.716</td>
<td>1</td>
<td>.19</td>
<td>Equal</td>
</tr>
<tr>
<td>Stress Generation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>Fr → Endo SLEs</td>
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<td>.30</td>
<td>.05</td>
<td>3.013</td>
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<td>.22</td>
<td>Redundant</td>
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<td>.001</td>
<td>.51</td>
<td>.001</td>
<td>.45</td>
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<td>1</td>
<td>.81</td>
<td>Equal</td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr*Endo → Psych T₁</td>
<td></td>
<td></td>
<td>6</td>
<td>.001</td>
<td>.26</td>
<td>.01</td>
<td>.12</td>
<td>7.192</td>
<td>1</td>
<td>.01</td>
<td>Genders differ</td>
</tr>
</tbody>
</table>

*Note.* If the X^2^ difference value is not significant, constraining the parameters of the nested model did not significantly worsen the fit of the model, which indicates measurement invariance of the parameters constrained to be equal in the nested model. The “Step” column indicates the sequence of model modification, in which we started with the worst fitting paths. All X^2^-tests with Δdf=1 are significant from 3.84 (p<.05), 6.64 (p<.01), and 10.83 (p<.001).

### Table A23. Change in internalizing tendencies model stratified by gender (chapter 6)

<table>
<thead>
<tr>
<th>Variable in model</th>
<th>Baseline</th>
<th>Final</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>Fr ↔ Internalizing T₁</td>
<td>0.11♀</td>
</tr>
<tr>
<td>Prospective</td>
<td>Fr → Internalizing T₂</td>
<td>-0.00</td>
</tr>
<tr>
<td>Stress Generation</td>
<td>Fr → Endo SLEs</td>
<td>0.12♀</td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td>Fr*Endo → Internalizing T₁</td>
<td>0.15♀</td>
</tr>
</tbody>
</table>

*Note.* T₁= baseline, T₂= follow-up; df= degrees of freedom; Endo=endogenous SLEs; Exo=exogenous SLEs; Fr= frustration; Psych= psychopathology; SLEs= stressful life events; X^2^= Chi-square. The method section provides information about all variables, model-fit indices, and modelling procedures. All paths in both final models were tested (Jöreskog tradition), and all paths added significantly to model fit (all p<.0001). Significance ***p<.001, **p<.01, *p<.05, †= .052, two-tailed.
Table A24. Constraints and Jöreskog Tests of Gender-Equality for Change in Internalizing Tendencies Model Stratified by Gender (chapter 6)

<table>
<thead>
<tr>
<th>Variable in model</th>
<th>Test of nested model</th>
<th>Step</th>
<th>Variable</th>
<th>p</th>
<th>β</th>
<th>p</th>
<th>β</th>
<th>ΔX²</th>
<th>Δdf</th>
<th>p</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td></td>
<td>14.</td>
<td>Fr ↔ Internalizing T₁</td>
<td>.03</td>
<td>.11</td>
<td>.02</td>
<td>.13</td>
<td>.03</td>
<td>1</td>
<td>.86</td>
<td>Genders equal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15.</td>
<td></td>
<td>10.92</td>
<td>2</td>
<td>.00</td>
<td>Path required</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prospective</td>
<td></td>
<td>1.</td>
<td>Fr → Internalizing T₂</td>
<td>.96</td>
<td>-.00</td>
<td>.43</td>
<td>-.04</td>
<td>0.63</td>
<td>2</td>
<td>.73</td>
<td>Redundant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>16.</td>
<td>Internalizing T₁ → T₂</td>
<td>.001</td>
<td>.51</td>
<td>.001</td>
<td>.51</td>
<td>1.36</td>
<td>1</td>
<td>.24</td>
<td>Genders equal</td>
</tr>
<tr>
<td>Stress Generation</td>
<td></td>
<td>17.</td>
<td></td>
<td>222.36</td>
<td>2</td>
<td>.001</td>
<td>Path required</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td></td>
<td>18.</td>
<td>Fr*Endo → Internalizing T₂</td>
<td>.06</td>
<td>.10</td>
<td>.005</td>
<td>.13</td>
<td>.08</td>
<td>1</td>
<td>.78</td>
<td>Genders equal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>19.</td>
<td></td>
<td>11.74</td>
<td>2</td>
<td>.003</td>
<td>Path required</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3.</td>
<td>Fr → Exo SLEs</td>
<td>.25</td>
<td>.06</td>
<td>.14</td>
<td>.07</td>
<td>3.43</td>
<td>2</td>
<td>.18</td>
<td>Redundant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8.</td>
<td>Internalizing T₁ → Endo SLEs</td>
<td>.11</td>
<td>1.00</td>
<td>.00</td>
<td>-3.69</td>
<td>23.96</td>
<td>1</td>
<td>.001</td>
<td>Genders differ</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9.</td>
<td></td>
<td>20.29</td>
<td>2</td>
<td>.001</td>
<td>Path required</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>6.</td>
<td>Internalizing T₁ → Exo SLEs</td>
<td>.01</td>
<td>.01</td>
<td>.81</td>
<td>.00</td>
<td>.03</td>
<td>1</td>
<td>.87</td>
<td>Genders equal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7.</td>
<td></td>
<td>4.23</td>
<td>2</td>
<td>.12</td>
<td>Redundant</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>20.</td>
<td>Endo ↔ Exo SLEs</td>
<td>.001</td>
<td>.23</td>
<td>.001</td>
<td>.27</td>
<td>0.00</td>
<td>1</td>
<td>1.00</td>
<td>Genders equal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>21.</td>
<td></td>
<td>46.62</td>
<td>2</td>
<td>.001</td>
<td>Path required</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.</td>
<td>Endo SLEs → Internalizing T₂</td>
<td>.29</td>
<td>-.06</td>
<td>.08</td>
<td>-.23</td>
<td>3.78</td>
<td>2</td>
<td>.15</td>
<td>Redundant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12.</td>
<td>Exo SLEs → Internalizing T₂</td>
<td>.002</td>
<td>.19</td>
<td>.73</td>
<td>.04</td>
<td>1.31</td>
<td>1</td>
<td>.25</td>
<td>Genders equal</td>
</tr>
<tr>
<td>Stress Sensitivity</td>
<td></td>
<td>13.</td>
<td></td>
<td>6.88</td>
<td>2</td>
<td>.03</td>
<td>Path required</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10.</td>
<td>Fr*Endo → Internalizing T₂</td>
<td>.10</td>
<td>.09</td>
<td>.00</td>
<td>-.15</td>
<td>12.58</td>
<td>1</td>
<td>.001</td>
<td>Genders differ</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11.</td>
<td></td>
<td>10.64</td>
<td>2</td>
<td>.005</td>
<td>Path required</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>4.</td>
<td>Fr*Exo → Internalizing T₂</td>
<td>.001</td>
<td>-.24</td>
<td>.85</td>
<td>.03</td>
<td>4.26</td>
<td>1</td>
<td>.04</td>
<td>Genders differ</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.</td>
<td></td>
<td>11.35</td>
<td>2</td>
<td>.00</td>
<td>Path required</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Fr= frustration; Endo= endogenous; Exo= exogenous; SLEs= stressful life events; T₁= baseline (age 16); T₂= follow-up (age 19). If the X² difference value is not significant, constraining the parameters of the nested model did not significantly worsen the fit of the model, which indicates measurement invariance of the parameters constrained to be equal in the nested model. The “Step” column indicates the sequence of model modification. All X²-tests with ∆df= 1 are significant from 3.84 (p< .05), 6.64 (p< .01), and 10.83 (p< .001).
Table A25. Gender stratified change in externalizing tendencies model (chapter 6)

<table>
<thead>
<tr>
<th>Path</th>
<th>Baseline</th>
<th>Final</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>♀</td>
<td>♂</td>
</tr>
<tr>
<td><strong>Baseline</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr ↔ Externalizing $T_1$</td>
<td>0.21**</td>
<td>0.20***</td>
</tr>
<tr>
<td><strong>Prospective</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr → Externalizing $T_2$</td>
<td>0.03</td>
<td>0.15***</td>
</tr>
<tr>
<td>Externalizing $T_1$ → $T_2$</td>
<td>0.47***</td>
<td>0.33***</td>
</tr>
<tr>
<td><strong>Stress Generation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr → Endo SLEs</td>
<td>0.09</td>
<td>0.06</td>
</tr>
<tr>
<td>Fr → Exo SLEs</td>
<td>0.03</td>
<td>0.07</td>
</tr>
<tr>
<td>Externalizing $T_1$ → Endo SLEs</td>
<td>0.15***</td>
<td>0.36***</td>
</tr>
<tr>
<td>Externalizing $T_1$ → Exo SLEs</td>
<td>0.13**</td>
<td>0.05</td>
</tr>
<tr>
<td>Endo ↔ Exo SLEs</td>
<td>0.22***</td>
<td>0.26***</td>
</tr>
<tr>
<td>Endo SLEs → Externalizing $T_2$</td>
<td>0.08</td>
<td>0.30***</td>
</tr>
<tr>
<td>Exo SLEs → Externalizing $T_2$</td>
<td>-0.02</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Stress Sensitivity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fr*Endo → Externalizing $T_2$</td>
<td>0.08</td>
<td>-0.05</td>
</tr>
<tr>
<td>Fr*Exo → Externalizing $T_2$</td>
<td>0.08</td>
<td>-0.06</td>
</tr>
</tbody>
</table>

Note. $T_1$= baseline (age 16), $T_2$= follow-up (age 19); df= degrees of freedom; Endo=endogenous; Exo=exogenous; Fr=frustration; SLEs=stressful life events; $X^2$= Chi-square. The method section provides information about all variables, model-fit indices, and modelling procedures. All paths in both final models were tested (Jöreskog tradition), and all paths added significantly to model fit (all $p<.0001$). Significance *** $p<.001$, ** $p<.01$, * $p<.05$, two-tailed.
Table A26. Gender stratified change in externalizing tendencies model: Constraints and Jöreskog tests of gender-equality (chapter 6)

<table>
<thead>
<tr>
<th>Step</th>
<th>Variable in model</th>
<th>Test of nested model</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Fr ↔ Externalizing $T_1$</td>
<td>$\Delta X^2$ = 1.73, $p = .165$, Genders equal</td>
</tr>
<tr>
<td>7</td>
<td>Fr $\rightarrow$ Externalizing $T_2$</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>8</td>
<td>Externalizing $T_1$ $\rightarrow$ $T_2$</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>3</td>
<td>Fr $\rightarrow$ Endo SLEs</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>2</td>
<td>Fr $\rightarrow$ Exo SLEs</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>9</td>
<td>Externalizing $T_1$ $\rightarrow$ Endo SLEs</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>10</td>
<td>Externalizing $T_1$ $\rightarrow$ Exo SLEs</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>11</td>
<td>Endo $\leftrightarrow$ Exo SLEs</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>12</td>
<td>Endo SLEs $\rightarrow$ Externalizing $T_2$</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>13</td>
<td>Exo SLEs $\rightarrow$ Externalizing $T_2$</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>5</td>
<td>Fr*Endo $\rightarrow$ Externalizing $T_2$</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
<tr>
<td>4</td>
<td>Fr*Exo $\rightarrow$ Externalizing $T_2$</td>
<td>$\Delta X^2$ = 1.27, $p = .27$, Genders equal</td>
</tr>
</tbody>
</table>

Note. Fr = frustration; Endo = endogenous; Exo = exogenous; SLEs = stressful life events; $T_1$ = baseline (age 16); $T_2$ = follow-up (age 19). If the $X^2$ difference value is not significant, constraining the parameters of the nested model did not significantly worsen the fit of the model, which indicates measurement invariance of the parameters constrained to be equal in the nested model. The “Step” column indicates the sequence of model modification. All $X^2$-tests with $\Delta df = 1$ are significant from 3.84 ($p < .05$), 6.64 ($p < .01$), and 10.83 ($p < .001$).
Table A27. Proportion explained variance ($R^2$) in outcome predicted by frustration (chapter 6)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Wave</th>
<th>Full sample R2</th>
<th>Test</th>
<th>Women R2</th>
<th>Test</th>
<th>Men R2</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychopathology</td>
<td>$T_1$</td>
<td>0.07***</td>
<td>$F(775)=55.37$</td>
<td>0.06***</td>
<td>$F(424)=27.18$</td>
<td>0.08***</td>
<td>$F(350)=32.15$</td>
</tr>
<tr>
<td>Internalizing tendencies</td>
<td>$T_1$</td>
<td>0.01***</td>
<td>$F(775)=9.16$</td>
<td>0.01*</td>
<td>$F(424)=4.59$</td>
<td>0.01*</td>
<td>$F(350)=5.97$</td>
</tr>
<tr>
<td>Externalizing tendencies</td>
<td>$T_1$</td>
<td>0.04***</td>
<td>$F(775)=31.35$</td>
<td>0.04***</td>
<td>$F(424)=18.98$</td>
<td>0.04***</td>
<td>$F(350)=14.18$</td>
</tr>
<tr>
<td>Severity marker</td>
<td>$T_1$</td>
<td>0.06***</td>
<td>$F(809)=52.72$</td>
<td>0.06***</td>
<td>$F(424)=25.68$</td>
<td>0.08***</td>
<td>$F(350)=31.99$</td>
</tr>
<tr>
<td>Endogenous SLEs</td>
<td>$T_1$, $T_2$</td>
<td>0.01***</td>
<td>$F(809)=12.49$</td>
<td>0.01*</td>
<td>$F(440)=6.11$</td>
<td>0.01*</td>
<td>$F(368)=6.28$</td>
</tr>
<tr>
<td>Exogenous SLEs</td>
<td>$T_1$, $T_2$</td>
<td>0.00</td>
<td>$F(809)=3.69$</td>
<td>0.00</td>
<td>$F(440)=1.52$</td>
<td>0.00</td>
<td>$F(368)=1.95$</td>
</tr>
<tr>
<td>Psychopathology</td>
<td>$T_2$</td>
<td>0.06***</td>
<td>$F(798)=7.61$</td>
<td>0.03***</td>
<td>$F(435)=16.13$</td>
<td>0.09***</td>
<td>$F(362)=37.12$</td>
</tr>
<tr>
<td>Internalizing tendencies</td>
<td>$T_2$</td>
<td>0.00</td>
<td>$F(798)=0.24$</td>
<td>0.00</td>
<td>$F(435)=0.03$</td>
<td>0.00</td>
<td>$F(362)=0.07$</td>
</tr>
<tr>
<td>Externalizing tendencies</td>
<td>$T_2$</td>
<td>0.04***</td>
<td>$F(798)=32.44$</td>
<td>0.03***</td>
<td>$F(435)=13.53$</td>
<td>0.06***</td>
<td>$F(362)=21.90$</td>
</tr>
</tbody>
</table>

Note: Results were derived with linear regression analyses in SPSS 20. We report the adjusted $R^2$. $F(df)=\text{the result of a } F\text{-test with a certain degrees of freedom (df). Significance }^{***}p<.001,^{**}p<.01,^*p<.05\text{, two-tailed.}$

Table A28. Spearman correlations of the study variables stratified by gender: Males below, women on top (chapter 6).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wave</th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
<th>4.</th>
<th>5.</th>
<th>6.</th>
<th>7.</th>
<th>8.</th>
<th>9.</th>
<th>10.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>$T_1$</td>
<td>-0.04</td>
<td>-0.03</td>
<td>-0.01</td>
<td>-0.04</td>
<td>-0.05</td>
<td>-0.04</td>
<td>-0.02</td>
<td>-0.03</td>
<td>-0.00</td>
<td></td>
</tr>
<tr>
<td>2. Frustration</td>
<td>$T_1$</td>
<td>-0.00</td>
<td>-0.25</td>
<td>-0.17</td>
<td>-0.25</td>
<td>-0.12</td>
<td>-0.06</td>
<td>-0.19</td>
<td>-0.13</td>
<td>-0.22</td>
<td></td>
</tr>
<tr>
<td>3. Psychopathology</td>
<td>$T_1$</td>
<td>-0.04</td>
<td>0.29***</td>
<td>-0.89***</td>
<td>0.78***</td>
<td>0.24***</td>
<td>0.19***</td>
<td>0.65***</td>
<td>0.59***</td>
<td>0.58***</td>
<td></td>
</tr>
<tr>
<td>4. Internalizing</td>
<td>$T_1$</td>
<td>-0.02</td>
<td>0.23***</td>
<td>0.82***</td>
<td>0.39***</td>
<td>-0.24</td>
<td>0.21***</td>
<td>0.48***</td>
<td>0.33***</td>
<td>0.58***</td>
<td></td>
</tr>
<tr>
<td>5. Externalizing</td>
<td>$T_1$</td>
<td>-0.05</td>
<td>0.24***</td>
<td>0.82***</td>
<td>0.39***</td>
<td>-0.24</td>
<td>0.21***</td>
<td>0.48***</td>
<td>0.33***</td>
<td>0.58***</td>
<td></td>
</tr>
<tr>
<td>6. Endogenous SLEs</td>
<td>$T_1$</td>
<td>0.07</td>
<td>0.13</td>
<td>0.22***</td>
<td>0.05</td>
<td>0.32**</td>
<td>-0.23**</td>
<td>0.33**</td>
<td>0.27**</td>
<td>0.34**</td>
<td></td>
</tr>
<tr>
<td>7. Exogenous SLEs</td>
<td>$T_1$, $T_2$</td>
<td>0.03</td>
<td>0.07</td>
<td>0.06</td>
<td>0.04</td>
<td>0.06</td>
<td>0.23***</td>
<td>-0.21**</td>
<td>0.18**</td>
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<td>10. Externalizing</td>
<td>$T_2$</td>
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<td>0.16**</td>
<td>0.89***</td>
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</table>

Note. $N=957$ (women=0, $N=523, 54.6\%$). $Sx=$ symptoms; SLEs= stressful life events; $T_1=$ baseline, $T_2=$ follow-up. Significance $^{***}p<.001,^{**}p<.01,^*p<.05\text{, two-tailed.}$
Table A29. Elaborated Correlation table: Spearman correlations in the lower half, Pearson correlations in the upper half (chapter 6).

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<th>Wave</th>
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</tbody>
</table>

Note. N= 957 (women= 0, N=523 (54.6%). T0 refers to the first TRAILS wave (age 11); T1 refers to the study baseline (age 16); T2 refers to follow-up (age 19). Notably, the internalizing and externalizing psychopathology measures presented are not mutually adjusted (in contrast to the rest of the paper, cf. Table 21). Significance ***p< .001, **p< .01, *p< .05, two-tailed.

Table A30. Correlations between baseline (T1) frustration and internalizing and externalizing symptoms at follow-up (T2), and partial correlations adjusted internalizing and externalizing symptoms at T1 (chapter 6).

<table>
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<tr>
<th></th>
<th>Unadjusted</th>
<th>Adjusted for T1 Internalizing (% reduction)</th>
<th>Adjusted for T1 Externalizing (% reduction)</th>
<th>Adjusted for both (% reduction)</th>
</tr>
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<tbody>
<tr>
<td>Psychopathology</td>
<td>.244***</td>
<td>.183*** (25%)</td>
<td>.168*** (31%)</td>
<td>.131*** (46%)</td>
</tr>
<tr>
<td>Internalizing</td>
<td>.178***</td>
<td>.094** (47%)</td>
<td>.128** (28%)</td>
<td>.080* (55%)</td>
</tr>
<tr>
<td>Externalizing</td>
<td>.264***</td>
<td>.227*** (14%)</td>
<td>.168*** (36%)</td>
<td>.155*** (41%)</td>
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</table>

Note. N= 799, all associations significant at ***p< .001, **p< .01, *p< .05, two-tailed.
Table A31. Mediation analyses of the effect of temperamental facets on conflicts in the romantic relationship domain for adolescents who reported at least one romantic relationship; all analyses adjusted for gender (chapter 7).

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<th>Adolescents with ≥1 Romantic Relationship</th>
<th></th>
<th></th>
<th>95% CI</th>
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<td>Indirect effects</td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>beta</td>
<td>SE</td>
<td>p</td>
<td>R²</td>
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<td>Partner</td>
<td>Parental</td>
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<td>.030</td>
<td>.13</td>
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<td>.002</td>
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<td>Partner</td>
<td>Parental</td>
<td>.016</td>
<td>.033</td>
<td>.64</td>
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<td>.041</td>
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*Note.* The sample is *n* = 703. R² refers to the adjusted proportion explained variance. Values in **bold** represent significant associations (*p*<.05).
Table A32. Single event analyses (chapter 7)

<table>
<thead>
<tr>
<th>Facet</th>
<th>Conflict</th>
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<th>Perceived affection</th>
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<th>Indirect Effect</th>
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<td>SE</td>
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<td>Peer</td>
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Table A32. Single event analyses (chapter 7) (continued)

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<th>Perceived affection</th>
<th>Direct Effect</th>
<th>Indirect Effect</th>
</tr>
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<td>SE</td>
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<td></td>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>-.583</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Parental</td>
<td>.682</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>-1.08</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Peer</td>
<td>-.072</td>
</tr>
<tr>
<td>Effortful</td>
<td>Parents</td>
<td>Thrown out of parental home</td>
<td>Parental</td>
<td>-.393</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td>Parental</td>
<td>.655</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.065</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>-.134</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Parental</td>
<td>.022</td>
</tr>
</tbody>
</table>

Note. The conflicts in the parent domain were being “thrown out of parental home” (n= 20), “having a serious fight with parents” (n= 92), and “running away from home” (n= 52); conflicts in the peer domain were “losing a good friend because of fight or argument” (n= 128) and “being bullied” (n= 256); and conflicts in the partner domain were “being dumped” (n= 204) and “breaking up (self) after a relationship” (n= 538).
### Table A33. Bootstrap results for indirect relationships: bias-corrected and accelerated CI’s, all adjusted for gender (chapter 7)

<table>
<thead>
<tr>
<th>Facet</th>
<th>Conflict</th>
<th>Affection</th>
<th>Independent facet effects (Boot SE 95% CI)</th>
<th>Facet effects adjusted for all other facets (Direct effects)</th>
<th>Indirect effects (SE 95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fear</td>
<td>Parents</td>
<td>Parental</td>
<td>-0.02 (-0.03 to -0.01)</td>
<td>-0.02 (-0.01 to -0.01)</td>
<td>-0.008 (-0.01 to -0.005)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.000 (.001 to .002)</td>
<td>.018 (.01 to .024)</td>
<td>.04 (.001 to .004)</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>Parental</td>
<td>-0.01 (-0.01 to -0.01)</td>
<td>.013 (.02 to .024)</td>
<td>.04 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>-0.01 (-0.01 to -0.01)</td>
<td>.011 (.02 to .024)</td>
<td>.04 (.003 to .003)</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>Parental</td>
<td>-0.02 (-0.03 to -0.01)</td>
<td>.011 (.02 to .024)</td>
<td>.04 (.003 to .003)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.001 (.001 to .002)</td>
<td>.018 (.01 to .024)</td>
<td>.04 (.004 to .004)</td>
</tr>
<tr>
<td>Frustration</td>
<td>Parents</td>
<td>Parental</td>
<td>.009 (.009 to .01)</td>
<td>.02 (.01 to .024)</td>
<td>.04 (.003 to .003)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.003 (.001 to .002)</td>
<td>.013 (.02 to .024)</td>
<td>.04 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>Parental</td>
<td>.004 (.005 to .005)</td>
<td>.017 (.02 to .024)</td>
<td>.04 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.009 (.009 to .01)</td>
<td>.02 (.01 to .024)</td>
<td>.04 (.004 to .004)</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>Parental</td>
<td>.009 (.009 to .01)</td>
<td>.02 (.01 to .024)</td>
<td>.04 (.004 to .004)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.013 (.004 to .005)</td>
<td>.02 (.01 to .024)</td>
<td>.04 (.004 to .004)</td>
</tr>
<tr>
<td>Affiliation</td>
<td>Parents</td>
<td>Parental</td>
<td>.009 (.009 to .01)</td>
<td>.02 (.01 to .024)</td>
<td>.04 (.003 to .003)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.001 (.001 to .002)</td>
<td>.013 (.02 to .024)</td>
<td>.04 (.004 to .004)</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>Parental</td>
<td>.005 (.005 to .005)</td>
<td>.017 (.02 to .024)</td>
<td>.04 (.004 to .004)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.011 (.006 to .026)</td>
<td>.017 (.02 to .024)</td>
<td>.04 (.005 to .005)</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>Parental</td>
<td>.010 (.005 to .022)</td>
<td>.02 (.01 to .024)</td>
<td>.04 (.006 to .006)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.013 (.005 to .025)</td>
<td>.011 (.02 to .024)</td>
<td>.04 (.005 to .005)</td>
</tr>
<tr>
<td>Shyness</td>
<td>Parent</td>
<td>Parental</td>
<td>.001 (.002 to .003)</td>
<td>.017 (.01 to .02)</td>
<td>.03 (.001 to .001)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.001 (.001 to .002)</td>
<td>.013 (.01 to .02)</td>
<td>.03 (.001 to .001)</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>Parental</td>
<td>.000 (.002 to .003)</td>
<td>.018 (.01 to .02)</td>
<td>.03 (.001 to .001)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.006 (.006 to .017)</td>
<td>.018 (.01 to .02)</td>
<td>.03 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>Parental</td>
<td>.001 (.002 to .003)</td>
<td>.055 (.025 to .03)</td>
<td>.03 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.007 (.004 to .017)</td>
<td>.025 (.02 to .03)</td>
<td>.03 (.003 to .003)</td>
</tr>
<tr>
<td>Surgency</td>
<td>Parents</td>
<td>Parental</td>
<td>.000 (.002 to .003)</td>
<td>.028 (.013 to .04)</td>
<td>.04 (.003 to .003)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.000 (.001 to .002)</td>
<td>.013 (.01 to .02)</td>
<td>.04 (.004 to .004)</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>Parental</td>
<td>.000 (.002 to .003)</td>
<td>.018 (.01 to .02)</td>
<td>.04 (.005 to .005)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.001 (.003 to .009)</td>
<td>.018 (.01 to .02)</td>
<td>.04 (.006 to .006)</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>Parental</td>
<td>.000 (.002 to .003)</td>
<td>.089 (.022 to .03)</td>
<td>.03 (.001 to .001)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.002 (.003 to .004)</td>
<td>.04 (.01 to .02)</td>
<td>.04 (.002 to .002)</td>
</tr>
<tr>
<td>Effortful</td>
<td>Control</td>
<td>Parental</td>
<td>.003 (.003 to .011)</td>
<td>-0.07 (.018 to .00)</td>
<td>.04 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.001 (.003 to .004)</td>
<td>.018 (.00 to .008)</td>
<td>.04 (.001 to .001)</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>Parental</td>
<td>.003 (.004 to .013)</td>
<td>-0.05 (.025 to .04)</td>
<td>.04 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.005 (.003 to .014)</td>
<td>.025 (.01 to .02)</td>
<td>.04 (.003 to .003)</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>Parental</td>
<td>.007 (.004 to .018)</td>
<td>.027 (.031 to .03)</td>
<td>.03 (.002 to .002)</td>
</tr>
<tr>
<td></td>
<td>Peer</td>
<td>Parental</td>
<td>.008 (.004 to .01)</td>
<td>.017 (.01 to .017)</td>
<td>.03 (.002 to .002)</td>
</tr>
</tbody>
</table>

**Note.** Values in **bold** represent significant associations (p<.05). All models were adjusted for gender, but the adjusted models were also adjusted for the five other temperamental facets. The sample is n= 1154, R² refers to the adjusted proportion explained variance.
Table A34. Spearman partial rho coefficients either adjusted for gender (main effects) or also adjusted for perceived relationship affection (PA) (chapter 7).

<table>
<thead>
<tr>
<th>Facet</th>
<th>Conflict</th>
<th>Total effect</th>
<th>Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$r_p$</td>
<td>$p$</td>
</tr>
<tr>
<td>Fear</td>
<td>Parents</td>
<td>.032</td>
<td>.27</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>.032</td>
<td>.27</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>.061</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>.061</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>-.018</td>
<td>.53</td>
</tr>
<tr>
<td>Frustration</td>
<td>Parents</td>
<td>.133</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>.092</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>.086</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>.033</td>
<td>.25</td>
</tr>
<tr>
<td>Affiliation</td>
<td>Parents</td>
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<td>.49</td>
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<td></td>
<td>Peers</td>
<td>.016</td>
<td>.59</td>
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<td>.39</td>
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<td>.17</td>
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<td>Parents</td>
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<td>.23</td>
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<td>.005</td>
<td>.87</td>
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<tr>
<td></td>
<td>Peers</td>
<td>.000</td>
<td>.99</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>-.111</td>
<td>.00</td>
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<tr>
<td>Surgency</td>
<td>Parents</td>
<td>.048</td>
<td>.10</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>-.008</td>
<td>.80</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>-.007</td>
<td>.80</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>.146</td>
<td>.00</td>
</tr>
<tr>
<td>Effortful</td>
<td>Parents</td>
<td>-.135</td>
<td>.00</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>-.096</td>
<td>.00</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>-.091</td>
<td>.00</td>
</tr>
<tr>
<td></td>
<td>Partner</td>
<td>-.034</td>
<td>.24</td>
</tr>
<tr>
<td></td>
<td>Peers</td>
<td>-.040</td>
<td>.17</td>
</tr>
</tbody>
</table>

Note. All effects in **bold** were significant at $p<.05$. Sample sizes differed slightly for total effects ($n=1193$) and effects adjusted for parental affection ($n=1158$) and peer affection ($n=1179$). PA= perceived affection; $n$= number of participants; $p$= significance; $r_p$= non-parametric partial correlation; %Δ= percentage change in correlation after additional adjustment for perceived affection versus only adjustment for gender, suggesting mediation effects. Spearman partial rho ($r_{sp}$) coefficients are robust against data that deviates from the normal distribution (see [419]).
Table A35. Biserial correlations between temperament and presence of at least one romantic relationship (0, 1) (chapter 7).

<table>
<thead>
<tr>
<th>Temperament</th>
<th>$r_b$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fear</td>
<td>-.01</td>
<td>.83</td>
</tr>
<tr>
<td>Frustration</td>
<td>-.00</td>
<td>.93</td>
</tr>
<tr>
<td>Affiliation</td>
<td>.10</td>
<td>.001</td>
</tr>
<tr>
<td>Shyness</td>
<td>-.15</td>
<td>.001</td>
</tr>
<tr>
<td>Surgency</td>
<td>.12</td>
<td>.001</td>
</tr>
<tr>
<td>Effortful control</td>
<td>.01</td>
<td>.87</td>
</tr>
</tbody>
</table>

Note. $N=1975$, Bootstrapped Pearson correlations ($k=1000$, BCA, 95% CI, or point-biserial correlations) were adjusted to derive biserial correlations because romantic relationships are a continuous dichotomy, viz. $r_b = (r_{pb} \times \sqrt{pq}) / y$, in which $p$ is the largest proportion, $q$ the smallest, and $y$ the ordinate of their balance point in the normal distribution [1122,1123].

Table A36. Frequencies for School Progress and Socioeconomic status (SES) Stratified over Relative Age Distribution in Quartiles (chapter 8)

<table>
<thead>
<tr>
<th>Relative age:</th>
<th>1 to 3</th>
<th>4 to 6</th>
<th>7 to 9</th>
<th>10 to 12</th>
<th>Relative Old</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth month:</td>
<td>Sept, Aug, July</td>
<td>June, May, April</td>
<td>March, Feb, Jan</td>
<td>Dec, Nov, Oct</td>
<td></td>
</tr>
<tr>
<td>Total sample</td>
<td>2230</td>
<td>624</td>
<td>569</td>
<td>537</td>
<td>500</td>
</tr>
<tr>
<td>Normative development</td>
<td>1681</td>
<td>75.4%</td>
<td>63.8%</td>
<td>77.5%</td>
<td>82.5%</td>
</tr>
<tr>
<td>Repeated grade</td>
<td>377</td>
<td>16.9%</td>
<td>29.6%</td>
<td>15.8%</td>
<td>11.4%</td>
</tr>
<tr>
<td>Skipped grade</td>
<td>48</td>
<td>2.2%</td>
<td>0.3%</td>
<td>0.7%</td>
<td>1.3%</td>
</tr>
<tr>
<td>Special education</td>
<td>124</td>
<td>5.5%</td>
<td>6.3%</td>
<td>6.0%</td>
<td>4.8%</td>
</tr>
<tr>
<td>Low SES quartile</td>
<td>547</td>
<td>25.0%</td>
<td>24.7%</td>
<td>26.0%</td>
<td>27.2%</td>
</tr>
<tr>
<td>High SES quartile</td>
<td>547</td>
<td>25.0%</td>
<td>24.7%</td>
<td>26.0%</td>
<td>27.2%</td>
</tr>
</tbody>
</table>

Note. $N=2230$ (50.8% women); %= percentage.
Table A37. Relative Age Effects on Multiple Domains, Adjusted for Actual Age at Testing and Socioeconomic Status (SES) of the Family of Origin, for Adolescents with a Normative School Progress (\(n=1681\)), and Adolescents who Repeated a Grade (\(n=377\)) (chapter 8).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wave</th>
<th>Relative age effect</th>
<th>95% CI</th>
<th>Relative age effect</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>( r_p )</td>
<td>B</td>
<td>( r_p )</td>
<td>B</td>
</tr>
<tr>
<td>Length (cm)</td>
<td>2</td>
<td>-0.04</td>
<td>-0.10 (-0.24 to 0.04)</td>
<td>-0.01</td>
<td>0.09 (-0.25 to 0.42)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>2</td>
<td>-0.04</td>
<td>-0.13 (-0.30 to 0.05)</td>
<td>0.08</td>
<td>0.60* (0.02 to 1.11)</td>
</tr>
<tr>
<td>BMI</td>
<td>2</td>
<td>-0.02</td>
<td>-0.02 (-0.07 to 0.03)</td>
<td>0.10</td>
<td>0.19* (0.03 to 0.36)</td>
</tr>
<tr>
<td>Pubertal status</td>
<td>2</td>
<td>-0.02</td>
<td>-0.04 (-0.10 to 0.02)</td>
<td>0.08</td>
<td>0.07 (-0.07 to 0.19)</td>
</tr>
<tr>
<td>Intellectual Development</td>
<td>2</td>
<td>0.04</td>
<td>0.06 (-0.01 to 0.13)</td>
<td>-0.12</td>
<td>-0.19* (-0.36 to -0.02)</td>
</tr>
<tr>
<td>Sport Competence</td>
<td>2</td>
<td>0.01</td>
<td>0.00 (-0.01 to 0.02)</td>
<td>-0.01</td>
<td>-0.00 (-0.03 to 0.03)</td>
</tr>
<tr>
<td>Fear</td>
<td>1</td>
<td>0.01</td>
<td>0.01 (-0.01 to 0.02)</td>
<td>0.09</td>
<td>0.04 (-0.00 to 0.08)</td>
</tr>
<tr>
<td>Frustration</td>
<td>1</td>
<td>0.05</td>
<td>0.02 (-0.00 to 0.03)</td>
<td>0.04</td>
<td>0.02 (-0.03 to 0.06)</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>1</td>
<td>0.01</td>
<td>0.00 (-0.02 to 0.02)</td>
<td>0.18***</td>
<td>0.02** (0.02 to 0.11)</td>
</tr>
<tr>
<td>ΔFear</td>
<td>1-3</td>
<td>-0.06*</td>
<td>-0.02 (-0.04 to 0.00)</td>
<td>-0.11</td>
<td>-0.06 (-0.13 to 0.02)</td>
</tr>
<tr>
<td>ΔFrustration</td>
<td>1-3</td>
<td>-0.01</td>
<td>0.00 (-0.03 to 0.02)</td>
<td>-0.04</td>
<td>-0.03 (-0.09 to 0.04)</td>
</tr>
<tr>
<td>ΔDepressive symptoms</td>
<td>1-3</td>
<td>0.01</td>
<td>0.00 (-0.02 to 0.02)</td>
<td>-0.08</td>
<td>-0.03 (-0.09 to 0.04)</td>
</tr>
</tbody>
</table>

Note. *\( p = .052; Δ= change between \(T_1\) (age 11) and \(T_3\) (Age 16); BMI= body mass index; \( r_p \)= partial correlations between relative age and outcome, adjusted for real age at time of testing. Regression estimates were bootstrapped (\(k=10,000\), bias corrected intervals), and indicate change in outcome per month in relative age, after adjustment for age at testing. Note that for change variables we also adjusted for change in age between \(T_1\) and \(T_3\). Details on all measures and procedures can be found in the method section. All correlations between all variables are given in Table 29. Significance: ***\( p<.001\), **\( p<.01\), *\( p<.05\), two-tailed.

Table A38. Relative Age Effects on School Progress (Normative Development is Reference) (chapter 8).

<table>
<thead>
<tr>
<th>Binary Outcome</th>
<th>Adj. for real age(^a)</th>
<th>Adj. for real age &amp; SES(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>(95% CI)</td>
</tr>
<tr>
<td>Repeated grade</td>
<td>0.83***</td>
<td>(0.80 to 0.86)</td>
</tr>
<tr>
<td>Skipped grade</td>
<td>1.47***</td>
<td>(1.30 to 1.67)</td>
</tr>
<tr>
<td>Special education</td>
<td>0.96</td>
<td>(0.91 to 1.01)</td>
</tr>
</tbody>
</table>

Note. N= 2230 (50.8% women). *The odds are based on bootstrapping (\(k=10,000\)); \(^b\) odds are not based on bootstrapping. CI= bias corrected confidence interval; OR= Ratio of the probability that an event will happen to all possible cases for that event. Adolescents who repeated a grade were almost three times more often from the low than the high SES quartile (33.4% vs. 12.7%), but those who skipped a grade were three times more often from the high SES quartile (10.6% vs. 36.2%), see Appendix Table A36. Significance ***\( p<.001\), **\( p<.01\), *\( p<.05\), two-tailed.
Table A39. Relative Age as the Predictor of the Odds of Being Popular or Rejected (Normative Development is Reference) in Adolescents with a Normative School Progress and Adolescents who Repeated a Grade, Adjusted for Real Age at Testing (chapter 8).

<table>
<thead>
<tr>
<th>School progress</th>
<th>Social Status</th>
<th></th>
<th>OR</th>
<th>(95% CI)</th>
<th>p</th>
<th>B</th>
<th>(95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normative</td>
<td>Popular</td>
<td>-.00</td>
<td>1.00</td>
<td>(0.94 to 1.06)</td>
<td>.90</td>
<td>-0.00</td>
<td>(-0.07 to 0.06)</td>
</tr>
<tr>
<td></td>
<td>Rejected</td>
<td>.09***</td>
<td>1.12</td>
<td>(1.05 to 1.19)</td>
<td>.00</td>
<td>0.11</td>
<td>(0.04 to 0.18)</td>
</tr>
<tr>
<td>Repeated a grade</td>
<td>Popular</td>
<td>.04</td>
<td>1.11</td>
<td>(0.83 to 1.49)</td>
<td>.55</td>
<td>0.11</td>
<td>(-0.53 to 0.66)</td>
</tr>
<tr>
<td></td>
<td>Rejected</td>
<td>.02</td>
<td>1.04</td>
<td>(0.85 to 1.28)</td>
<td>.67</td>
<td>0.04</td>
<td>(-0.20 to 0.24)</td>
</tr>
</tbody>
</table>

Note. N= 2230 (50.8% women). In total 137 (8.1%) adolescents with a normative school progress were rated as popular and 132 (7.9%) as rejected. Regression estimates are based on bootstrapping (k= 10,000), the confidence intervals (95% CI) bias corrected; OR= Ratio of the probability that an event will happen to all possible cases for that event; \( r_p \)= partial correlations between relative age and outcome, adjusted for real age at time of testing. The values for the presented correlations and regression remained almost identical after additional adjustment for family SES. Note that the estimations for the group who repeated a grade are based on very few subjects (see Appendix Table A36), therefore rather unreliable thus not reported in the manuscript. Significance ***=p<.001, **=p<.01, *=p<.05, two-tailed.

Table A40. Social Status Stratified over Relative Age Position in Quartiles and School Progress (chapter 8).

<table>
<thead>
<tr>
<th>Normative school progress</th>
<th>Repeated a grade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Relatively young</td>
<td>37</td>
</tr>
<tr>
<td>Second</td>
<td>27</td>
</tr>
<tr>
<td>Third</td>
<td>29</td>
</tr>
<tr>
<td>Relatively old</td>
<td>39</td>
</tr>
<tr>
<td>Total</td>
<td>132</td>
</tr>
</tbody>
</table>

Note. The frequencies of subjects with a rejected and popular status stratified for adolescents with a normative school progress and for the group of adolescents who repeated a grade. n= number of subjects.

Table A41. Quartiles of Socioeconomic Status (SES) stratified over School Progress (chapter 8).

<table>
<thead>
<tr>
<th></th>
<th>Normative</th>
<th>Repeated a grade</th>
<th>Skipped a grade</th>
<th>Special education</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Low SES</td>
<td>65.3%</td>
<td>357</td>
<td>22.7%</td>
<td>124</td>
</tr>
<tr>
<td>Second Q</td>
<td>72.0%</td>
<td>394</td>
<td>21.4%</td>
<td>117</td>
</tr>
<tr>
<td>Third Q</td>
<td>77.1%</td>
<td>422</td>
<td>15.2%</td>
<td>83</td>
</tr>
<tr>
<td>High SES</td>
<td>87.6%</td>
<td>479</td>
<td>8.6%</td>
<td>47</td>
</tr>
<tr>
<td></td>
<td>1652</td>
<td>371</td>
<td>47</td>
<td>118</td>
</tr>
</tbody>
</table>

Note. n= number of subjects; Q= Quartile. The adolescents who repeated a grade were almost three times more often from the lowest than the highest SES quartile (n= 124 vs. 47 or 33.4% vs. 12.7% of 371), but adolescents who skipped a grade were three times more often from the highest than the lowest SES quartile (36.2% vs. 10.6%).
OMGEVINGSINVLOEDEN OP NEUROTICISME: EEN STUDIE VAN EMOTIONELE (IN)STABILITEIT

Een hoog niveau van neuroticisme is een belangrijke risico factor voor zowel psychische als lichamelijke problemen. Bovendien is neuroticisme niet alleen geassocieerd met problemen op het gebied van persoonlijk welbevinden maar ook met substantiële kosten voor de maatschappij: Studies hebben laten zien dat de totale extra kosten van de hoogst scorende 25% van de populatie ongeveer 1 miljard euro per 1 miljoen Nederlanders bedragen. Dat is bijna 2.5 maal zoveel als de extra kosten van angststoornissen en depressies samen. Het is daarom belangrijk om zowel de oorzaken als de gevolgen van neuroticisme beter te begrijpen.

Gedrags-genetische studies suggereren dat ongeveer de helft van de verschillen tussen mensen in neuroticisme veroorzaakt wordt door omgevingsinvloeden. In dit proefschrift heb ik onderzocht welke omgevingsinvloeden vooraf gaan aan een verandering in neuroticisme, en hoe lang zulke veranderingen stand houden. Hiertoe heb ik een aantal longitudinale studies uitgevoerd waarin ik het verband tussen levensgebeurtenissen en veranderingen in neuroticisme heb onderzocht. Ook heb ik de literatuur samengevat over verschillen in neuroticisme in eeneiige tweelingen. Voordeel van onderzoek met eeneiige tweelingen is dat de genetische invloed op het meemaken van levensgebeurtenissen kleiner is dan in studies met andere populaties. Daarnaast heb ik in kaart gebracht welke levensgebeurtenissen bij volwassen voorafgaan aan veranderingen in neuroticisme. De resultaten laten zien dat stressvolle levensgebeurtenissen (en in het bijzonder sociale stress en conflict), die gekarakteriseerd kunnen worden als onvoorspelbaar, oncontroleerbaar, onverwacht, onwenselijk, of niet passend bij de normale levensloop, vaak worden gevolgd door een langdurige toename in neuroticisme. Echter, positieve levensveranderingen kunnen ook leiden tot een afname van neuroticisme.

Tenslotte heb ik de betekenis onderzocht van de, in eerder onderzoek gevonden, sterke relatie tussen neuroticisme en veelvoorkomende psychische problemen, zoals angst, depressie, en middelenmisbruik. Hiervoor heb ik zowel een aantal longitudinale studies uitgevoerd als bevolkingsstudies in kaart gebracht waarin neuroticisme veelvoorkomende psychische problemen voorspelt. De resultaten laten zien dat hoog neuroticisme inderdaad een belangrijke voorspeller is voor de ontwikkeling van veelvoorkomende psychische problemen, en tot op zekere hoogte gezien kan worden als een maat voor meer milde problemen.

De conclusie van dit proefschrift is dat het neuroticisme niveau van mensenmede afhankelijk is van zijn of haar omgeving, gevoelig is voor positieve en negatieve levensgebeurtenissen, consequenties heeft voor psychische gezondheid, en veranderlijker is dan lang werd gedacht. Mogelijk kunnen we beter nadenken over behandelingen die zich richten op hoog neuroticisme, in plaats van op haar afzonderlijke negatieve uitkomsten en klinische manifestaties.
CURRICULUM VITAE

Bertus Filippus Jeronimus was born on 31 January 1984 in Frisia, where he lived with his parents, brother and sister. He finished his pre-university secondary education in 2002. Subsequently he studied clinical and developmental psychology and Dutch law at the University of Groningen. Between 2009 and 2014 Bertus worked on this PhD thesis at the Interdisciplinary Center Psychopathology and Emotion regulation (ICPE), part of the department of Psychiatry of the University Medical Center Groningen. Together with Prof.dr. Hans Ormel, dr. Harriëtte Riese, and Prof. dr. Tineke Oldehinkel he studied the association between life events, negative-affective temperament and the personality trait neuroticism. From 2014 onwards he works as a postdoctoral researcher at the ICPE in collaboration with Prof. dr. Peter de Jonge and the Espria Academy to study predictors of subjective wellbeing.
‘Environmental Influences on Neuroticism’

High neuroticism is the single most important risk factor in public mental health, a personal burden, and a substantial cost to society. About half of the individual differences in neuroticism have a non-genetic origin. In this PhD thesis I investigated whether and how environmental influences associate with change in neuroticism, and how long such changes persist. To do so, I performed a series of longitudinal studies. Additionally, I reviewed studies on monozygotic twin pairs discordant for neuroticism (a design that reduces genetic confounding of life event occurrences) and longitudinal studies on within-individual changes in neuroticism in adults. Results indicate that increases in the setpoint of neuroticism tend to follow stressful life events (especially social stress and conflict) that can be characterized as unpredictable, uncontrollable, unexpected, undesirable, and non-normative from a life history perspective.

Additionally, I critically examined the meaning of the previously reported strong prospective association between neuroticism and the common mental disorders (CMDs, viz. anxiety, depression, and substance use disorders). This was done in a series of longitudinal studies and a review of population studies in which neuroticism predicts CMDs. The key observation is that high neuroticism is, indeed, an important prospective indicator of risk for the development of full-blown psychological disorders, and can, to some extent, be viewed as sub-threshold psychopathology.

In conclusion, this thesis indicates that the neuroticism setpoint is embedded in the environment, sensitive to both positive and negative life events, consequential for future mental health, and more malleable than researchers originally believed.