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Differential associations of locus of control with anxiety, depression and life-events:

A five-wave, nine-year study to test stability and change

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Abstract

Background: The locus of control (LOC) construct has been associated with onset, course, and severity of anxiety and depression. We investigated the stability of LOC, the bidirectional relationships between LOC and symptom severity of anxiety and depression over nine years, and the influence of intermediate positive and negative life-events on these associations.

Methods: Data came from five assessment waves over nine years of 2052 subjects with an anxiety or depressive disorder or healthy controls. First, the stability of LOC (assessed with 5-item Mastery Scale) was tested. Next, associations between LOC, anxiety severity (Beck Anxiety Inventory), depression severity (Inventory of Depressive Symptomatology), and intermediate positive and negative life-events (20-item List of Threatening Experience Questionnaire) were determined with structural equation modeling.

Results: LOC was rather stable over nine years ($r = .62$), and scores increased slightly with age (i.e. became more internal). LOC yielded equal stability estimates as symptom levels of anxiety and depression did over nine years. A more external LOC predicted higher anxiety and depression severity, but did not influence the incidence of positive and negative life-events. Higher depression severity and more negative life-events predicted the development of a more external LOC, whereas more positive life-events predicted a more internal LOC. Anxiety severity had no effect on LOC.

Limitations: Life-events were assessed with self-report measures.

Conclusions: The prospective associations between LOC and meaningful changes in anxiety and depressive symptom severity and experienced life-events may yield important new insights for clinical interventions.

Keywords: mastery, anxiety symptoms, depressive symptoms, positive life-events, negative life-events

1. Introduction

Cognitive vulnerabilities play an important role in the etiology of anxiety and depressive disorders (Hong & Cheung, 2015; Jeronimus et al., 2016). One major cognitive vulnerability factor is the personality dimension locus of control (LOC), which has been defined as the “generalized attitude, belief, or expectancy regarding the nature of the causal relationship between one’s own behavior and its consequences” (Rotter, 1966). LOC covers two major components, namely, *mastery* or an individual’s sense of self-efficacy in achieving one’s goals, and *perceived constraints*, or feeling unable to overcome external factors (Lachman, 2006; Skinner, 1996). LOC thus reflects an individual’s perception about one’s ability to control their personal environment and future and is operationalized as a unidimensional scale that ranges from being internally oriented (one is able to influence actions, people, and events) to being externally oriented (what happens is just luck, fate, or chance; Rotter, 1966, 1990).

As individuals with a more external LOC experience lower control over their fate they typically experience more stress. An external LOC is a risk factor for the onset of anxiety and depressive disorders (Barlow, 2000; Beekman et al., 1998; Chorpita and Barlow, 1998; Wiersma et al., 2011), their unfavorable course (Hovens et al., 2016), and a higher severity of anxiety and depressive symptoms (Abdolmanafi et al., 2011; Archer, 1979; Benassi et al., 1988; Chorpita and Barlow, 1998; Hoehn-Saric and McLeod, 1985). This relationship between LOC and anxiety and depression was also found in longitudinal studies although follow-up times up till 12 months can be considered as limited (Hooke and Page, 2002; Struijs et al., 2013). The exact relationship between LOC and symptoms of anxiety and depression is thus of eminent importance but remains unclear due to the substantial overlap between anxiety and depression and the lack of long-term multiple-wave longitudinal studies.

LOC is usually considered to be a personality trait and therefore thought to be moderately stable over time (Rotter, 1966; Wolfle and List, 2004). LOC was indeed stable in a sample of 14-year olds over a period of three years (Kulas, 1996). Stability over a period of four years was also found in adults from the general population, although modest changes of LOC levels were found in young and very old people (Cobb-Clark and Schurer, 2013). Gradual changes towards a more external LOC over a period of eight years were also observed in a sample of women aged 32-46 years (Doherty, 1983), and in ageing samples more generally (Lachman, 2006; Lang and Heckhausen, 2001). In contrast, Nowicki et al. (2018) found that women developed a more internal LOC in the period from pregnancy to motherhood, whereas their partners developed a slightly more external LOC. In addition, a significant change towards a more internal LOC was found in middle-aged psychiatric inpatients with a depressive or anxiety disorder diagnosis after treatment with cognitive behavior therapy, and this improvement was maintained over a three-month follow-up (Page and Hooke, 2003). In sum, LOC may seem moderately stable in most people in the general population (Cobb-Clark and Schurer, 2013), but can change considerably in young and old people, patients, and during specific role transitions such as parenthood. This conclusion is in line with research on most other personality traits (Ormel et al., 2017; Roberts et al., 2006). This may be clinically relevant, as a recent meta-analysis by Roberts et al. (2017) showed that personality traits in general can be targeted and changed in psychological treatment, and this has also been reported for control beliefs (Lachman, 2006). It would therefore be worthwhile to examine the stability of LOC and its association with changes in symptom severity of anxiety and depression.

Several studies indicated that a more externally oriented LOC is associated with encountering more negative life-events (NLEs), in childhood and in adulthood (Barlow, 2000; Crandall and Lehman, 1977; Darshani, 2014; Tyson, 1981). In reverse, more NLEs were

found to be associated with a more external LOC in a sample of young parents (Nowicki et al., 2018) and in healthy young men and older women (Cobb-Clark and Schurer, 2013). However, these associations between LOC and NLEs were not found in a sample of university students (Kilmann et al., 1978). A more internal LOC, in contrast, was associated with experiencing more positive life-events (PLEs; Cobb-Clark & Schurer 2013). From these studies it appears that the bidirectional associations between LOC, NLEs/PLEs, and symptom severity of anxiety and depression are not straightforward. Theories have been developed indicating that a feeling of uncontrollability as a result of psychological vulnerabilities, developed early in childhood, mediates or moderates the association between NLEs and the emergence of anxiety and depression (Barlow, 2000; Chorpita and Barlow, 1998; Van den Heuvel et al., 1996). However, these theories are based on cross-sectional studies. To examine the associations between LOC and NLEs/PLEs, together with symptom severity of anxiety and depression, multiwave longitudinal data are required.

For our study we used a large cohort of subjects from the Netherlands Study of Depression and Anxiety (NESDA). Previous studies from NESDA found that an external LOC is predictive of, or associated with, chronicity of affective disorders (Struijs et al., 2018; Wiersma et al., 2011) or predicted an increase in depressive symptoms (Struijs et al., 2013). A longitudinal study in adults showed that an external LOC mediated the association between childhood maltreatment and the likelihood of remission of depressive and anxiety disorders (Hovens et al., 2016), whereby remission was based on the presence and severity of anxiety and depressive symptoms. The present study extends on this earlier work by explicitly addressing the stability of LOC using data from waves two, four, six, and nine years after baseline. This nine-year follow-up period provides us with the unique opportunity to gain insight into bidirectional relationships of LOC with symptom severity of anxiety and depression and the role of intermediate life-events, which is new to the literature.

The present study aimed to (a) test the stability of LOC over nine years and five assessment waves and (b) untangle the bidirectional relationships between LOC orientation, symptom levels of anxiety and depression, and PLEs and NLEs over this period. We hypothesized that i) LOC will be moderately stable over time; ii) People with a more externally oriented LOC developed more severe anxiety and depressive symptoms and reported more NLEs and fewer PLEs; iii) More severe anxiety and depressive symptoms predicted changes towards a more externally oriented LOC, fewer PLEs, and more NLEs; iv) More NLEs, in turn, predicted changes towards a more externally oriented LOC and more severe anxiety and depressive symptoms, with reversed associations for PLEs.

2. Methods

2.1 Study sample

The Netherlands Study of Depression and Anxiety (NESDA) is a nationwide longitudinal study designed to investigate the course and consequences of depressive and anxiety disorders. At baseline the study included 2981 subjects (mean age = 41.9 years, SD = 13.0, range 18-65 years; 66.4% women), including healthy controls (n = 652; 22%) and subjects with a past or current depressive and/or anxiety disorder diagnosis (n = 2329; 78%). To represent various settings and stages of psychopathology, subjects were recruited in the general population (n = 564; 19%), in general practices (n = 1610; 54%), and in mental health organizations (n = 807; 27%). Subjects with a primary psychotic, obsessive-compulsive, bipolar or severe addiction disorder and those not being fluent in Dutch were excluded. More details of the NESDA study, its design and attrition rates have been described elsewhere (Lamers et al., 2012; Penninx et al., 2008). For the current study, data derived from repeated assessments of the same instruments at NESDA waves baseline (T_0), 2-year (T_2), 4-year (T_4), 6-year (T_6), and 9-year follow-up (T_9) were used. Included were 2052 subjects with a current

(one-month) diagnosis of anxiety disorder (social anxiety disorder, panic disorder with and without agoraphobia, agoraphobia, and generalized anxiety disorder), depressive disorder (dysthymia and major depressive disorder), or a comorbid anxiety-depressive disorder at T_0 , as well as healthy controls (no lifetime anxiety or depressive disorder diagnosis). The Composite International Diagnostic Interview, version 2.1, was used to establish the diagnoses of anxiety and depressive disorders. The investigation was carried out in accordance with the Declaration of Helsinki. The research protocol was accredited by the Ethics Committee of participating universities and written informed consent was obtained from all subjects.

2.2 Measurements

Locus of control

The LOC construct theoretically combines mastery and perceived constraints, which are closely related and largely overlapping concepts (Lachman, 2006; Skinner, 1996). Within the NESDA study, the internal-external dimension of LOC was operationalized on a continuous scale using the 5-item Mastery Scale (Pearlin and Schooler, 1978), while perceived constraints were not assessed separately. The items of the Mastery Scale are presented in Table S1. Each item had equal weight and was rated on a 5-point scale, ranging from 1 (strongly disagree) to 5 (strongly agree); the ratings were recoded in such a way that a lower score indicates a more externally oriented LOC, whereas a higher score indicates a more internally oriented LOC. The ratings were summed to calculate the total score, which ranges from 5-25. The internal consistency of the scale in the current study was good with Cronbach's $\alpha = .88$.

Anxiety symptoms

Severity of anxiety symptoms (further referred to as anxiety severity) was measured with the Beck Anxiety Inventory (BAI; Beck et al., 1988; Muntingh et al., 2011). The BAI is a self-report instrument of 21 items which assesses the overall anxiety severity. Subjects were asked to rate how much they have been bothered by each anxiety symptom over the past week on a 4-point scale, ranging from 0 (not at all) to 3 (severely, I could barely stand it). The BAI was scored by summing the ratings for all of the 21 symptoms to obtain a total score with a range from 0 to 63. Factor analysis identified a somatic and a subjective anxiety/panic subscale (Beck et al., 1988), but in our study we only used the BAI total score of all items as a homogeneous measure of anxiety severity. The BAI showed good internal consistency with Cronbach's $\alpha = .94$.

Depressive symptoms

Severity of depressive symptoms (further referred to as depression severity) was measured with the Inventory of Depressive Symptomatology Self Report (IDS-SR; Rush et al., 1996). Although the IDS-SR is found to be multifactorial with three underlying factors (Wardenaar et al., 2010), we used the total sum score as a measure of overall depression severity. The IDS-SR comprised 28 items which were rated on a 4-point scale, ranging from 0 to 3. The scores were summed to calculate the total score, which ranges from 0 to 84. Higher scores indicate higher severity. The internal consistency was good with Cronbach's $\alpha = .86$.

Positive and negative life-events

The life-events were assessed at all waves except T_0 . The number of life-events reported between two waves were assessed with the 20-item List of Threatening Experiences Questionnaire (LTE-Q) (Brugha et al., 1985; Brugha & Cragg, 1990). The LTE-Q is a self-report instrument that assesses the experience of 13 NLEs (such as illness, death of a family

member or friend, financial problems, losing a job, accidents). The LTE-Q was extended by seven PLEs (i.e., recovering of a family member from a serious illness, getting involved with a new partner, getting a new job or a promotion). PLEs and NLEs were summarized into two separate scales. The complete list of LTE-Q items is presented in Table S2. The LTE-Q has high good test-retest reliability and high interrater agreement (Brugha and Cragg, 1990).

2.3 Statistical analysis

Descriptive statistics were used to present the baseline characteristics of the study sample. Healthy subjects and those with an anxiety disorder, depressive disorder, or comorbid anxiety-depressive disorder diagnosis were all merged and treated as one group in all analyses. Distributions of all variables were checked and found to deviate from a normal distribution. Therefore, correlations between all variables were calculated with Spearman's rho. We classified correlations (r) as very weak if between .00 and .29, weak between .30 and .49, moderate between .50 and .69, strong between .70 and .89, and very strong if above .90 (Mukaka, 2012). The stability of the LOC over time was tested with the Friedman test. Posthoc Wilcoxon signed-rank test was used to test the pairwise differences between two follow-ups. Bonferroni correction was applied, resulting in a significance level $p < .005$. Descriptives were calculated with SPSS version 23.0 (IBM SPSS statistics for windows, 2013).

Modeling procedure SEM

Structural equation modeling (SEM) was used to determine the associations between LOC, anxiety severity, depression severity, PLEs, and NLEs. SEM models were calculated using Mplus Version 6.12 (Muthén & Muthén, 2010). In a SEM model several associations between multiple variables can be estimated together using regression equations, and each

variable can be predictor and outcome simultaneously. In order to test our hypotheses, associations can be prospective or cross-sectional (as retrospective associations were excluded from the model in advance). To describe our results we distinguished four temporal dimensions (see Table 3): direct effects over one assessment interval (T_{x+1} , path 1-115) and delayed effects (T_{x+2} , T_{x+3} , T_{x+4}), which are potentially mediated by interposed variables (path 116-217). The default model in Figure 1 shows the possible associations between the variables. Only for the purpose of clarity, this default model is split up into two complementary parts, which are supposed to be projected over each other. Hence, in the analyses both diagrams in Figure 1 are combined and treated as one single model. Only the direct paths (1-115) are displayed in Figure 1, but all 217 paths were estimated and are categorized in Table 3. A detailed specification of each path can be found in the supplemental materials Table S4.

SEM models were calculated using Robust Maximum Likelihood estimation (MLR), which deals with missing data and provides robust SEs that account for the non-normality in our data. Multivariate kurtosis distorts the distribution of the chi-square (χ^2)-test statistic, which inhibits a comparison of nested models via χ^2 -difference tests with specified degrees of freedom (df, henceforth $\Delta\chi^2(\Delta df)$). MLR provides a correction factor that enables the calculation of Satorra-Bentler-corrected $\Delta\chi^2(df)_{SB}$. When the asymptotic nature of this correction led to negative $\Delta\chi^2(df)_{SB}$, we calculated only strictly positive $\Delta\chi^2(df)_{SB}$ -tests via a clone model (see Bryant and Satorra (2012) for details). Nested model modifications that improved on the model fit relative to the default model converged in the final model, and a $p < .05$ was considered to indicate significant improvement.

To test our hypotheses we first fit a parsimonious model in which all associations with a p -value $> .01$ were removed, as they are unlikely to represent a true effect (Lakens and Evers, 2014), using stepwise backward selection (based on $\Delta\chi^2(\Delta df)$ -tests) starting with the

highest p-value. Associations between variables were provided as standardized beta weights (B).

After checking the assumptions underlying the fit indices (Bentler and Chou, 1987), the models were evaluated on the basis of six fit indices: a) the chi-square ($\chi^2_{(df)}$); b) the comparative fit index ($CFI \geq .90$ for an acceptable model); c) the root mean square error of approximation ($RMSEA < .06$ in an acceptable model); d) the Tucker-Lewis index ($TLI \geq .90$ is acceptable); e) the Akaike information criterion (AIC) and f) the Bayesian information criterion (BIC). The model that best fit the data, produced the largest value on CFI and TLI, and the lowest on AIC, BIC, and RMSEA, and showed no significant $\Delta\chi^2(df)_{SB}$ -tests was considered to be our most parsimonious final model.

INSERT-FIGURE-1-ABOUT-HERE

3. Results

3.1 Descriptives of the study sample

The characteristics of the study sample at baseline are displayed in Table 1. The descriptives of the variables used in our analyses are provided in Table 2. All correlations between all variables are represented in the supplemental materials Table S3 and can be summarized as follows: All variables showed very weak to weak correlations with gender ($r \leq .11$), education level ($r \leq -.23$), and age ($r \leq .14$). Anxiety severity and depression severity showed moderate to strong correlation ($r = .55$ to $.83$) and both showed moderate to strong inverse correlation with LOC (anxiety: $r = -.60$ to $-.46$ and depression: $r = -.72$ to $-.53$). LOC showed very weak correlation with PLEs ($r = -.00$ to $.16$) and NLEs ($r = -.19$ to $-.09$). Anxiety and depression severity showed very weak to weak correlation with subsequent PLES (anxiety: $r = -.11$ to $.03$ and depression: $r = -.17$ to $-.04$) and NLEs (anxiety: $r = .11$ to $.20$ and depression: $r = .14$ to $.22$).

LOC exhibited a mean-level change between T_0 and T_9 (Table 2) as the scores increased significantly over the five assessment waves ($\chi^2_{(4)}=184.94, p < .001$). LOC scores at T_0 and T_2 differed from later LOC scores, but LOC scores at $T_4, T_6,$ and T_9 did not differ statistically from each other. More details can be found in the supplemental materials.

INSERT-TABLE-1-and TABLE-2-ABOUT-HERE

3.2 Final model

The measurements of the associations and predictive values of the variables at T_0 were replicated at the other four waves, ensuring a model with five comparable assessment waves. From our default model 132 non-significant paths could be removed without a significant decrease in model fit, yielding a parsimonious Final model with significant paths only, as

presented in Table 3. A graphical representation of the Final model is depicted in the supplemental materials Figure S2. The contribution of each individual path is specified in supplemental materials Table S4. Compared to the default model, the final model had the same CFI (.94), higher TLI (.92) and lower RMSEA (.06), AIC (184,799) and BIC (185,536) values; the overall fit of the final model was satisfactory and not statistically different from the fit of the default model ($\chi^2_{(132)} = 156.42, p = .07$). Details about the fit indices are given in supplemental materials Table S5. Our hypotheses were tested using this Final model.

Temporal and concurrent associations

Table 3 shows that for each variable the temporal associations between the measurements were approximately stable, with coefficients varying between 0.50 and 0.69 (for LOC, anxiety severity, and depression severity), between 0.39 and 0.47 (for PLEs), and between 0.24 and 0.29 (for NLEs). There were inverse concurrent associations between LOC and anxiety severity, and between LOC and depression severity, which means that higher LOC scores were associated with less severe symptoms of anxiety and depression (and vice versa). The concurrent associations between anxiety severity and depression severity were positive, which means that higher anxiety severity co-occurred with higher depression severity. Subjects who experienced more PLEs also reported more NLEs.

LOC predicting anxiety severity, depression severity, PLEs, and NLEs

Table 3 shows that a more external LOC at T_0 and T_2 predicted higher anxiety severity and depression severity. LOC at T_4 and T_6 had no association with anxiety severity nor with depression severity at T_6 and T_9 respectively. LOC was generally not predictive of PLEs and NLEs in subsequent waves, with an exception for LOC at T_0 , which was positively associated with PLEs and negatively associated with NLEs at T_2 .

Anxiety severity predicting LOC, depression severity, PLEs, and NLEs

Table 3 shows that anxiety severity did not predict LOC. Anxiety severity at the first two waves was not predictive of depression severity in the next wave, but associations with depression severity at waves T_4 and T_6 were statistically significant. Anxiety severity was not predictive of PLEs or NLEs.

Depression severity predicting LOC, anxiety severity, PLEs, and NLEs

Table 3 shows that higher depression severity predicted a more external LOC. Depression severity positively predicted anxiety severity at all waves, that is higher depression severity predicted higher anxiety severity. Depression severity was generally not predictive of PLEs (except at T_4 and a delayed effect at T_0). Depression severity positively predicted NLEs at T_0 and T_2 , but no associations were found between depression severity at T_4 and NLEs at T_6 , and depression severity at T_6 and NLEs at T_9 . However, two delayed positive association paths were found: depression severity at T_0 with NLEs at T_6 and T_9 .

PLEs and NLEs predicting LOC, anxiety severity, and depression severity

Table 3 shows that PLEs at T_2 , T_4 , and T_9 (but not at T_6) predicted a more internal LOC at the next wave. More PLEs at T_2 and T_6 predicted a decrease in anxiety severity. Further, more PLEs predicted a decrease in depression severity at T_2 , T_4 , and T_9 (but not at T_6). More PLEs at T_2 and T_4 (but not at T_6) were predictive of more NLEs in the next wave.

More NLEs predicted a more external LOC, and more severe symptoms of anxiety and depression at all waves (except for T_9 , where no association of NLEs with anxiety severity was found). NLEs were generally not predictive of PLEs, except at T_6 where more NLEs

predicted fewer PLEs at T_9 . Overall, the direct effects of NLEs were much more consistent than the effects of PLEs.

Posthoc tests

Based on the analyses and results, we performed posthoc analyses, in order to better interpret the results. The stability of LOC in terms of Spearman correlations dropped from .72 over 2 years to .62 over 9 years, which is comparable to the stabilities of anxiety severity (.74, .63) and depression severity (.72, .65), see Table S3 (all Spearman correlations, $p < .001$). This moderate LOC stability may partly reflect group-level decreases in anxiety severity or depression severity. However, when we calculated partial Spearman correlations for LOC adjusted for baseline levels of anxiety severity (.60, .49) or depression severity (.51, .41) the stabilities were even lower (all $p < .001$), which does not support the perspective that heightened baseline symptom levels weaken the LOC stability.

INSERT-TABLE-3-ABOUT-HERE

4. Discussion

In the current study we aimed to test the stability of LOC and the associations between changes in LOC and severity of anxiety and depression symptoms, as well as the role of the intermediate life-events. LOC showed moderate to strong test-retest stabilities over nine years, during which subjects developed a slightly more internally oriented LOC, on average. This stability of $r = .62$ is comparable to stabilities of neuroticism ($r = .61$, Jeronimus et al., 2014) and other personality concepts (e.g., Roberts & DelVecchio, 2000; Wortman et al., 2012) over similar follow-up periods, in studies of population samples. Although LOC was not predictive of life experiences, PLEs predicted the development of a more internal LOC whereas NLEs predicted a more external LOC. These results are discussed in more detail below.

The development towards a more internally oriented LOC in our study is largely in line with the study of Page & Hooke (2003), who reported a more internal LOC over time in a sample of patients with an anxiety or depressive disorder receiving treatment. The participants in our study with an anxiety or depressive disorder at baseline also improved in terms of symptom severity (see Rhebergen et al., 2011). However, our posthoc analyses showed that this mean-level change in LOC was not just a reflection of higher baseline versus follow-up symptom levels, and can thus not be entirely attributed to recovery from illness over nine years. In other words, we observed normative developmental changes in LOC over nine years of adult life, and these ageing processes are relevant to discuss. Earlier studies showed that people's perceptions of control typically show modest mean-level changes over the lifespan. On average, most young adults develop a more internal LOC, followed by a slow shift towards a more external LOC from middle age to retirement age, after which their sense of control stabilizes or increases slightly (Cobb-Clark and Schurer, 2013; Specht et al., 2013). The lifespan theory of control postulates that these normative changes in LOC reflect

changing capacities to influence outcomes in the external world and shifts in our preferences and the goals we value across the life cycle (Brändstadter and Rothermund, 1994; Lang and Heckhausen, 2001). Personal goals provide our lives with sense and meaning when we feel that their attainment is within our span of control, whereas unattainable goals propel feelings of helplessness and depression (Brandtstädter and Rothermund, 1994).

At baseline our study sample comprised persons between 18-65 years of which many experienced high mood symptom levels. The observed modest change in LOC towards a more internal orientation may therefore be best interpreted as originating from a mix of normative (healthy) ageing and disorder symptom related changes, and future studies may disentangle such processes in more detail. It has consistently been found that a high sense of control is associated with being happy, healthy, wealthy, and wise (Infurna et al., 2013; Lachman, 2006). Inconsistencies between our results and the studies reviewed in the introduction may result from i) the average age and socioeconomic status of the subjects in our sample (e.g. financial scarcity has been associated with lower perceived control, Kraus et al., 2009), ii) our study design with five repeated measurements over a long period of time and/or iii) the use of a mixed sample that included both mentally healthy and affected subjects.

The observation that the stability estimates for LOC versus anxiety and depression severity did not differ substantially aligns with the conclusion in the review of Ormel et al. (2013) that the longitudinal stabilities of state and trait constructs are in general more comparable than usually assumed. This shift in perspective is also reflected in the change from the DSM-IV multi-axial system to a non-axial system in DSM-5. Due to the lack of fundamental differences between the axes, especially between clinical disorders (Axis I) and personality disorders (Axis II), anxiety and depressive disorders were placed together with personality disorders during the latest revision on the DSM (Kress et al., 2014; Røysamb et

al., 2011). The similarities between the stability estimates of LOC, anxiety severity, and depression severity underscores this shift towards a nonaxial system.

In line with previous NESDA studies (Hovens et al., 2016; Struijs et al., 2013, 2018; Wiersma et al., 2011), we found that a more external LOC was predictive of more severe anxiety and depressive symptoms, with regression coefficients between -0.08 and -0.17. In our study, this predictive value disappeared after four years, when LOC became more internally oriented on average. However, this mean-level shift in LOC is modest and cannot fully account for the loss of predictive value for symptoms at later follow-up waves. Although speculative, the predictive value of a more internal LOC on anxiety severity and depression severity may have been overshadowed by indirect effects, such as mediation via self-esteem (Yu and Fan, 2016) or personality factors (Jeronimus et al., 2016), as elaborated upon below. The assessment of such indirect effects was beyond the scope of this study and may be explored in future work.

Over the nine year follow-up, LOC could be predicted by the other variables. While anxiety severity *did not* predict LOC, depression severity *did* predict LOC at all waves. There seems to be a clear difference between anxiety and depression as depression predicted anxiety at all waves, whereas anxiety only predicted depression at waves 3 and 4. Furthermore, in the first two waves depression predicted NLEs, while anxiety did not predict NLEs at all. In the past in-depth discussions took place about the similarities and differences between anxiety and depressive disorders (e.g., Andrews et al. 2008; Beesdo et al. 2010; Goldberg et al. 2009; Hettema, 2008; Watson, 2005). Despite the high correlation and comorbidity of anxiety and depressive disorders (see Brown & Barlow, 2009; Schoevers et al., 2008; Shankman & Klein, 2003), insufficient arguments were found to merge both disorders. In our study, in a model accounting for the overlap between the severity of anxiety and depressive symptoms (as we

did not study disorders), their unique variance yielded unique predictions, which we think endorse the conclusion that anxiety and depression are partly different entities.

With regard to life-events, LOC did not predict the number of PLEs or NLEs (in line with work by Cobb-Clark and Schurer, 2013), although concurrently, life-events were predictive of LOC. In line with previous work our results indicate that NLEs precede changes towards a more external LOC (Nowicki et al., 2018). Life-events also predicted changes in severity of anxiety and depression symptoms, in line with previous reports (Kinderman et al., 2013; Spinhoven et al., 2011). Our finding that the associations between LOC and life-events were not bidirectional suggests that LOC can be a mediator between life-events and symptom severity. This idea diverges from the conceptual model outlined by Barlow (2000), and Chorpita and Barlow (1998) in which they presume that a low sense of controllability in childhood mediates the experience of life-events and the subsequent development of anxiety and depression, whereas in adults a low sense of control moderates (and not mediates) the impact of aversive experience. Others showed that a strong sense of control positively moderates the impact of PLEs on affect (Lang and Heckhausen, 2001). Obviously, LOC, life-events, and symptom severity are interrelated, but our SEM model remains inconclusive as whether LOC is mediator or moderator, or both. Besides, the role of LOC may have been influenced by other exogeneous factors, such as neuroticism, a personality trait linked to the development of anxiety and depression (Jeronimus et al., 2016), and to life-events (Jeronimus et al., 2013; Riese et al., 2014). Future research may explore which factors are involved in the interrelationships between LOC, symptom severity of anxiety and depression, and life-events, and how these processes occur.

Strengths, limitations, and future studies

A strength of the current study was the unique longitudinal design with five repeated measurements of the same model over nine year follow-up time. Therefore we were able to examine in detail the bidirectional relationships between LOC orientation, symptom levels of anxiety and depression, and the number of reported positive and negative life-events. The recurring measurements of the same associations within one model makes it unlikely that the associations found were due to type I errors. Additional strengths are the large sample which assured ample statistical power and the inclusion of subjects from various settings and stages of psychopathology. However, three limitations should be acknowledged as well. First, life-events were assessed with self-report measures, which are subject to some recall bias (Althubaiti, 2016). A current anxiety or depressive disorder might have affected the experience and reporting of life-events (Kessler, 1997). In addition, we only registered the number of life-events, not their experienced severity. Nevertheless, the high reliability and validity of the LTE-Q has been demonstrated before. Second, due to the large proportion of subjects with anxiety and depressive symptoms in the sample, the results may not fully generalize to a general population. Nonetheless, this large longitudinal cohort including both individuals with a clinical diagnosis and healthy controls is also a key strength, as it ensures the clinical relevance and variability of all our variables without suffering from restrictions related to testing our hypothesis in relatively arbitrary groups. Third, the association between external LOC and higher symptom severity may be culture-dependent. In cultures characterized by less individualism and more collectivism, the association external LOC-anxiety symptoms, but not the association external LOC-depression symptoms, was found to be weaker than in Western societies such as the Netherlands were the data of the current study were assessed (Cheng et al., 2013). Cultural differences in the meaning of internal control may underlie these differential associations.

Future studies could improve on our work by analyzing indirect effects, whereby one variable mediates or moderates the association between two other variables, for instance an effect of anxiety severity on LOC via life-events. In addition, since there are indications that subjects with an anxiety disorder, a depressive disorder, both, or no mental disorder respond differently to stressful situations (Spinhoven et al., 2011), researchers could aim to replicate our model in the different disorders groups. In addition, our findings regarding LOC may also be interesting for future research using developmental models of closely related personality dimensions including neuroticism, self-esteem, and self-efficacy (Judge et al., 2002).

Conclusion

The stability of LOC was moderate to strong over nine years and comparable to those of most other personality domains. A more externally oriented LOC predicted higher severity of anxiety and depressive symptoms, but LOC did not predict later life-events. Conversely, changes in LOC were predicted by depression severity and also by life-events, but not by anxiety severity. Assessment of LOC can be of interest in clinical practice, as processes that yield a more internally oriented LOC could possibly alleviate the burden of anxiety and depressive symptoms and may improve the way people cope with stress. An important next step is to establish our results at the individual level to confirm that some individuals benefit from changes in their LOC due to treatment (Fisher et al., 2018). Nevertheless, the results of the current study suggest that assessment of LOC may be of added value for clinicians.

Contributors

Data was collected by the Netherlands Study of Depression and Anxiety (NESDA, www.nesda.nl). Authors JH and BJ undertook the analyses; JH wrote the draft of the manuscript. All authors contributed to and have approved the final manuscript.

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Conflict of interest

There are no conflicts of interest

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Table 1. Baseline characteristics of the study sample

Baseline characteristics (N=2052)	Mean (SD) / n (%)
<i>Sociodemographics</i>	
Age in years, mean (SD)	41.64 (13.1)
Female gender, n (%)	1330 (64.8)
Education in years, mean (SD)	12.00 (3.3)
<i>Care setting, n (%)</i>	
General population	251 (12.2)
Primary care	1132 (55.2)
Specialized mental health care	669 (32.6)
<i>Diagnosis at baseline, n (%)</i>	
Anxiety disorder	558 (27.2)
Depressive disorder	307 (15.0)
Comorbid anxiety-depressive disorder	535 (26.1)
No diagnosis (healthy controls)	652 (31.8)

Table 2. Descriptives of the variables across the five waves

	N	Mean (SD)	Range	Skewness (SE)	Kurtosis (SE)
Locus of control					
Baseline	1784	16.83 (4.83)	5-25	-0.12 (0.06)	-0.77 (0.12)
2-year wave	1564	18.03 (4.73)	5-25	-0.36 (0.06)	-0.54 (0.12)
4-year wave	1573	18.56 (4.87)	5-25	-0.47 (0.06)	-0.49 (0.12)
6-year wave	1443	18.60 (4.81)	5-25	-0.36 (0.06)	-0.79 (0.13)
9-year wave	1317	18.75 (4.86)	5-25	-0.44 (0.07)	-0.62 (0.14)
Anxiety severity					
Baseline	2041	13.88 (11.52)	0-62	0.93 (0.05)	0.64 (0.11)
2-year wave	1704	9.61 (9.23)	0-60	1.27 (0.06)	1.68 (0.12)
4-year wave	1575	8.89 (9.06)	0-55	1.46 (0.06)	2.22 (0.12)
6-year wave	1441	9.01 (9.03)	0-52	1.37 (0.06)	1.69 (0.13)
9-year wave	1316	8.21 (8.73)	0-54	1.52 (0.07)	2.47 (0.14)
Depression severity					
Baseline	2038	23.82 (15.14)	0-69	0.26 (0.05)	-0.82 (0.11)
2-year wave	1704	16.94 (12.76)	0-65	0.81 (0.06)	0.00 (0.12)
4-year wave	1575	16.44 (12.82)	0-76	0.93 (0.06)	0.35 (0.12)
6-year wave	1443	15.89 (12.56)	0-65	0.89 (0.06)	0.23 (0.13)
9-year wave	1319	15.53 (12.28)	0-69	1.00 (0.07)	0.63 (0.14)
Positive life-events					
Baseline	2052	n.a.	-	-	-
2-year wave	1768	2.47 (1.34)	0-8	0.45 (0.06)	-0.07 (0.12)
4-year wave	1638	2.35 (1.35)	0-7	0.50 (0.06)	-0.11 (0.12)
6-year wave	1527	2.22 (1.26)	0-7	0.60 (0.06)	0.22 (0.13)
9-year wave	685	2.28 (1.29)	0-7	0.65 (0.09)	0.44 (0.19)
Negative life-events					
Baseline	2052	n.a.	-	-	-
2-year wave	1768	1.72 (1.47)	0-10	0.94 (0.06)	1.06 (0.12)
4-year wave	1638	1.55 (1.39)	0-8	1.06 (0.06)	1.30 (0.12)
6-year wave	1527	1.47 (1.34)	0-8	1.03 (0.06)	1.16 (0.13)
9-year wave	685	1.75 (1.40)	0-7	0.82 (0.09)	0.65 (0.19)

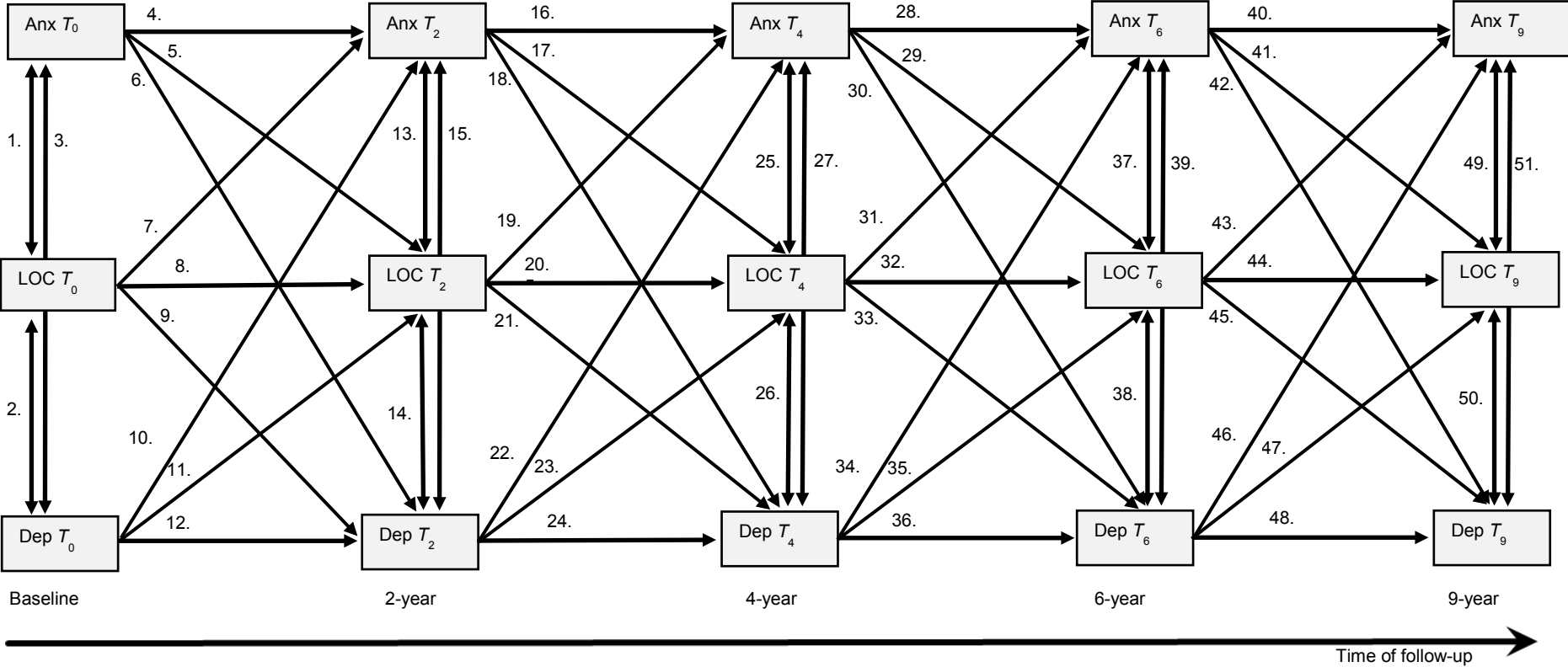
Notes: Anxiety severity = severity of anxiety symptoms; depression severity = severity of depressive symptoms; n.a. = not applicable

Table 3. Characteristics of the paths in the model for locus of control, severity of anxiety and depressive symptoms, and positive and negative life-events

Predictor and effect type	Span	Yrs	Paths #	Fig.	Path numbers	Observations		Beta Effect(s)	
						#	(%)	Min	Max
Locus of control									
Concurrent	T_x	—	10			10	100%	-0.71	-0.27
<i>Anxiety severity</i>		—	5	A	1, 13, 25, 37, 49	5	100%	-0.27	-0.57
<i>Depression severity</i>		—	5	A	2, 14, 26, 38, 50	5	100%	-0.39	-0.71
Stability		2-3	4	A	8, 20, 32, 44	4	100%	0.53	0.58
Direct	T_{x+1}	2-3	16			5	31%	-0.17	0.12
<i>Anxiety severity</i>		2-3	4	A	7, 19, 31, 43	2	50%	-0.08	-0.11
<i>Depression severity</i>		2-3	4	A	9, 21, 33, 45	2	50%	-0.13	-0.17
<i>PLEs</i>		1-1.5	4	B	54, 71, 88, 105	1	25%		0.12
<i>NLEs</i>		1-1.5	4	B	55, 72, 89, 106	0	25%		-0.09
Delayed	$T_{x+2}/T_{x+3}/T_{x+4}$	3-9	24	—		2	8%	-0.06	0.10
<i>Anxiety severity</i>		4-9	6	—	122, 124, 126, 138, 140, 148	0	0%		
<i>Depression severity</i>		4-9	6	—	123, 125 , 127, 139, 141, 149	1	17%	-0.06	
<i>PLEs</i>		3-7	6	—	158, 160, 162, 192, 194 , 214	1	17%		0.10
<i>NLEs</i>		3-7	6	—	159, 161, 163, 193, 195, 215	0	0%		
Anxiety severity									
Concurrent	T_x	—	10	A	1, 3, 13, 15, 25, 27, 37, 39, 49, 51	10	100%	-0.57	0.78
Stability	T_{x+1}	2-3	4	A	4, 16, 28, 40	4	100%	0.53	0.59
Direct	T_{x+1}	1-3	16			2	13%	0.09	0.10
		1-3	8	A	5, 6, 17, 18, 29, 30, 41, 42	2	25%	0.09	0.10
		1-3	8	B	52, 53, 69, 70, 86, 87, 103, 104	0	0%		
Delayed	$T_{x+2}/T_{x+3}/T_{x+4}$	3-9	24	—		0	0%		
		3-9	12	—	116-121, 134-137, 46, 47	0	0%		
		3-9	12	—	152-157, 188-191, 212-213	0	0%		
Depression severity									
Concurrent	T_x	—	10	A	2, 3, 14, 15, 26, 27, 38, 39, 50, 51	10	100%	-0.71	0.78
Stability	T_{x+1}	2-3	4	A	12, 24, 36, 48	4	100%	0.58	0.69
Direct	T_{x+1}	1-3	16			11	69%	-0.24	0.22
			8	A	10, 11, 22, 23, 34, 35, 46, 47	8	100%	-0.24	0.21

Delayed	$T_{x+2}/T_{x+3}/T_{x+4}$	3-9	8	B	56, 57 , 73, 74 , 90 , 91, 107, 108	3	38%	-0.07	0.22
		24	—	—		4	17%	-0.10	0.20
		3-9	12	—	128- 131 -133, 142-145, 150, 151	1	8%	-0.10	
		3-9	12	—	164 , 165- 167 , 168, 169 , 196-199, 216, 217	3	25%	-0.06	0.20
Positive life-events									
Concurrent with NLEs	T_x	—	4	B	58, 75, 92, 109	4	100%	0.21	0.25
Stability	T_{x+1}	2-3	3	B	60, 77, 94	3	100%	0.39	0.47
Direct	T_{x+1}	1-1.5	14	B	59, 61-63 , 76, 78, 79, 95-97, 110, 111, 112	6	46%	-0.12	0.10
Delayed	$T_{x+2}/T_{x+3}/T_{x+4}$	6	14	—	170-178, 200, 201, 202 , 203-205	1	7%	-0.04	
Negative life-events									
Stability	T_{x+1}	2-3	3	B	67, 84, 101	3	100%	0.24	0.29
Direct	T_{x+1}	1-1.5	14	B	64-66, 68, 81, 82, 83, 85, 98-100, 102 , 113, 114, 115	15	87%	-0.08	0.13
Delayed	$T_{x+2}/T_{x+3}/T_{x+4}$	6	14	—	179-187, 206-211	0	0%		
Total									
Concurrent	T_x	—	34			34	100%	-0.71	0.78
Stability	T_{x+1}	2-3	18			18	100%	0.24	0.69
Direct	T_{x+1}	1-3	76			38	50%	-0.17	0.22
Delayed	$T_{x+2}/T_{x+3}/T_{x+4}$	3-9	100			7	7%	-0.10	0.20

Notes: The table shows the assessments (Span; T_x = x-year follow-up measurement; T_{x+l} = subsequent measurement wave; T_{x+2} , T_{x+3} , T_{x+4} = two, three, or four waves further in time, respectively) and the average number of years in-between (Yrs), the number (#) of paths in the default model (Figure 1) and their path numbers (with the bold paths significant at $p < .01$). Observations: the number (#) of observed paths in the final model (supplemental materials Figure S2) and the percentage observed paths relative to the default model, and the observed range of beta-coefficients for each path type. Anxiety severity = severity of anxiety symptoms; depression severity = severity of depressive symptoms



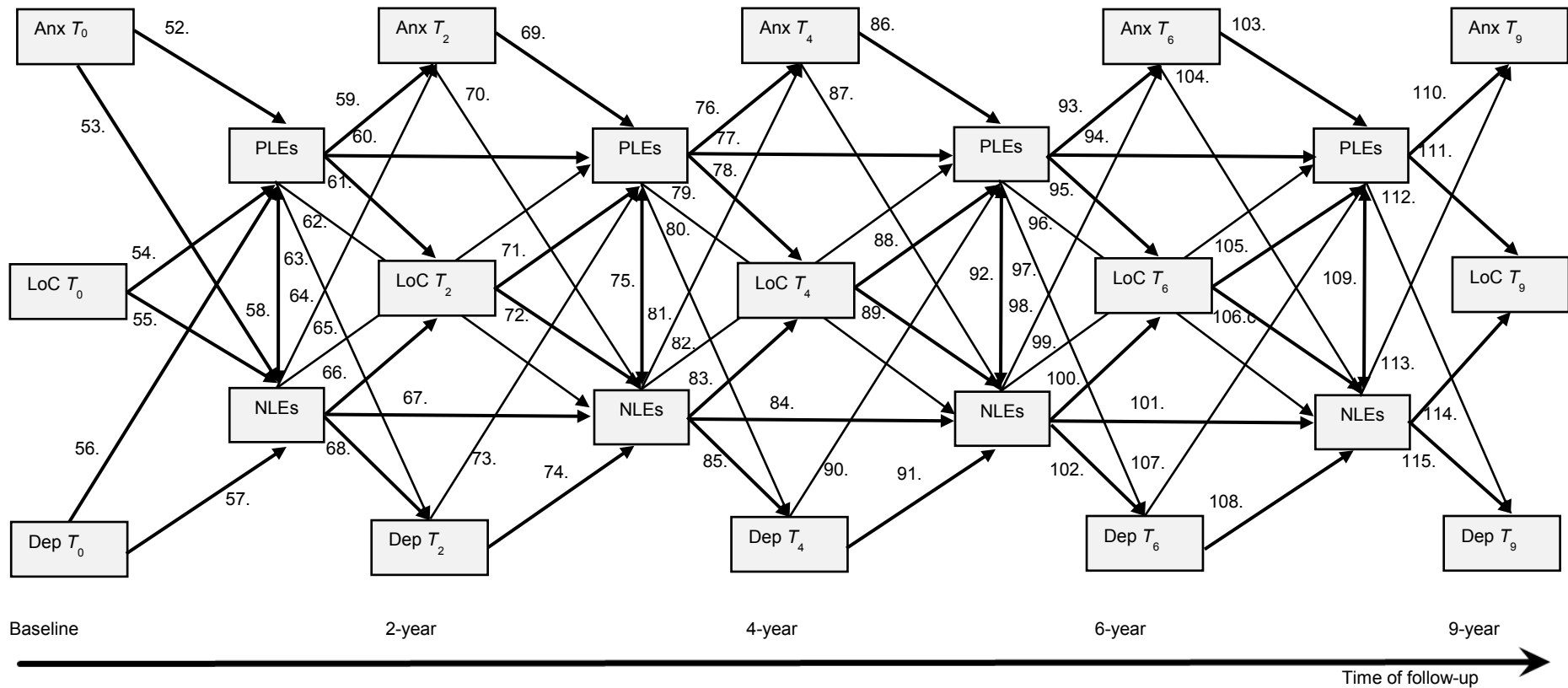


Figure 1. Default model of the associations between locus of control (LOC), severity of anxiety symptoms (Anx), severity of depressive symptoms (Dep), positive life-events (PLEs), and negative life-events (NLEs) over 5 waves and 9 years. For purposes of clarity only, the figure of the default model is split up into two complementary parts, which are supposed to be projected over each other. In the upper part all association paths between LOC, Anx, and Dep are shown; in the lower part all association paths of PLEs and NLEs are shown. T_0 = baseline; T_2 = 2-year follow-up; T_4 = 4-year follow-up; T_6 = 6-year follow-up; T_9 = 9-year follow-up. Numbers plotted at the arrow headed lines indicate the number of the association path (see Table S4). All direct paths are depicted, whereas delayed paths over multiple assessment intervals were included in the model but not shown in the figure. Details about all paths can be found in Table 3 and in Table S4 of the supplemental materials.