The handle http://hdl.handle.net/1887/32032 holds various files of this Leiden University dissertation.

**Author:** Drost, Jolijn
**Title:** Worry and rumination: underlying processes and transdiagnostic characteristics
**Issue Date:** 2014-06-10
Chapter 3

Longitudinal associations between repetitive negative thinking and emotional disorders

Jolijn Drost
Philip Spinhoven
Albert M. van Hemert
Mark de Rooij
Brenda Penninx
Willem van der Does

Submitted for publication
Abstract

**Background:** It is undecided whether worry and rumination are conceptualizations of the same underlying process of repetitive negative thinking, which is hypothesized to be a transdiagnostic factor underlying various emotional disorders. The aim of this study is to investigate stable and time specific aspects of worry and rumination in fear disorders (social anxiety, panic disorder, agoraphobia) and distress disorders (dysthymia, depressive disorder, generalized anxiety disorder).

**Method:** Longitudinal cohort study in 2981 participants (healthy controls, persons with a prior disorder history and persons with a current distress and/or fear disorder). Assessment of DSM-IV diagnoses and worry/rumination took place at baseline (T1), 2-year follow-up (T2), and 4-year follow-up (T3). Latent trait-state models were fitted using structural equation analyses.

**Results:** Results revealed that i) worry and rumination each contain stable trait components, which are strongly correlated; ii) state level fluctuations of worry and rumination are moderately and positively correlated; iii) trait worry and trait rumination are strongly associated with the stable components of both distress and fear disorders; iv) state fluctuations in distress/fear disorder predict state fluctuations in worry/rumination 2 years later and not vice versa.

**Conclusion:** Our results highlight the similarities between worry and rumination and support their conceptualization as transdiagnostic processes.
Introduction

Worry is a central feature of generalized anxiety disorder (GAD) and rumination of major depressive disorder (MDD), although, levels are elevated across anxiety and depressive disorders. Both cognitive processes are characterized by uncontrolled, excessive and repetitive thinking about current concerns, problems, past experiences or worries about the future; so called Repetitive Negative Thinking (RNT; Ehring & Watkins, 2008, p. 192). If a distinction is to be made, worry is more about the future, rumination about the past – although this distinction is a simplification. The question is justified whether worry and rumination are conceptualizations of the same underlying process. Another question is whether these process(es) are transdiagnostic or that their presence across emotional disorders is due to comorbidity with one specific disorder. If they are indeed conceptualizations of the same underlying process we would expect the stable trait components of worry and rumination to be highly related, fluctuations at state level to occur in sync and similar relations of worry and rumination with emotional disorders. If worry and rumination are transdiagnostic processes we would also expect them to be present across emotional disorders independent of comorbidity with a specific disorder. Finally, a third question is whether -in line with cognitive theory- reciprocal lagged relations between worry/rumination and emotional disorders are mutually reinforcing each other, setting off a downward spiral towards enduring psychopathology.

The proposition that worry and rumination are transdiagnostic processes (Harvey et al., 2004; Ehring & Watkins, 2008) is predominantly based on studies revealing elevated levels across disorders. These studies were generally focussed on individual disorders (major depressive disorder (MDD) and generalized anxiety disorder (GAD) in particular) while disregarding comorbidity. Comorbidity is however the rule rather than the exception (e.g. Kessler et al., 1994) especially between MDD/DYS and GAD (e.g. Spinhoven et al., 2009; Kessler et al., 1999) and could be responsible for the similar findings across disorders. A recent cross-sectional study by Lamers and colleagues (2011) reported that among MDD patients the co-occurrence with GAD was 31% and life-time comorbidity was as high as 38%. In GAD patients comorbidity numbers were even higher with up to 78% (lifetime 88%) of the patients reporting to suffer from a depressive disorder. The limitation of studying separate disorders is further highlighted by findings that suggest that MDD and GAD share a genetic based common cause (Gorwood, 2004; Kendler et al., 2007). Given the high comorbidity among emotional disorders, studying the relation of worry and rumination with various emotional disorders simultaneously while accounting for their comorbidity may provide additional insight into their potentially transdiagnostic characteristics. One way to address this issue is to cluster related disorders and study them simultaneously. An example of this would be to examine ‘emotional disorders’ or to use the traditional DSM-IV division of emotional disorders into ‘anxiety’ and ‘depressive’ disorder clusters. Another, increasingly popular categorization of emotional disorders is that into fear and distress disorders. The latter division is supported by recent studies of the structure of psychopathology that show that GAD is better placed with the depressive
disorders than the anxiety disorders (for an overview see Beesdo-Baum et al., 2009). The present study will take comorbidity into account by applying this fear-distress model when investigating worry and rumination.

So far most studies have focused on cross-sectional or uni-directional relationships of worry and rumination with each other and with particular emotional disorders, and did not examine reciprocal effects nor the temporal character of the effects. Longitudinal studies concerning rumination have shown that rumination predicts the occurrence of both anxiety and depressive symptoms over time, including new onset of depressive disorders (Nolen-Hoeksema, 2000). Likewise, worry has been found to be a vulnerability factor predicting increments of anxiety and depressive symptoms over time (Hong, 2007). Unlike the longitudinal studies examining rumination, studies concerning worry usually covered limited time frames of around one week to two months (e.g. Calmes & Roberts, 2007; Hong, 2007; Segerstrom et al., 2000). The role of worry and rumination is further confirmed by an extensive review of the literature on repetitive (negative) thinking (RNT) revealing that RNT is a vulnerability factor for both anxiety and depressive disorders (Watkins, 2008). A reverse effect of psychopathology predicting the occurrence of RNT has, to the best of our knowledge, not been investigated longitudinally.

The assumption that worry and rumination share the same process is largely based on studies showing substantial correlations between the two constructs (e.g. Segerstrom et al., 2000, $r = .32$ to $r = .46$; Muris et al., 2004, $r = .55$; Watkins, 2004, $r = .51$; Hong, 2007, $r = .42$). By using a latent trait-state model (see Naragon-Gainey, Gallagher, & Brown, 2013, and Ormel & Schaufeli, 1991, for the development of similar models) it is possible to separate stable and state components of worry and rumination. This will provide information on whether these supposedly vulnerability factors are indeed stable over time and how they relate to each other and to the stable components of psychopathology. Worry and rumination state levels are also of interest as levels are known to fluctuate and to be heightened during periods of psychopathology (e.g. Bagby et al., 2004; Kasch, Klein, & Lara, 2001). These fluctuations around a person’s set point may put into motion a downward spiral in which increases in worry/rumination lead to heightened levels of psychopathology which in turn triggers worry/rumination. If reciprocal influences are present this could potentially be an important mechanism underlying enduring psychopathology.

The NESDA study, with presently three-wave data available on worry/rumination and emotional disorders, offers an unique possibility to analyse the temporal and directional character of a reciprocal relationship between worry and rumination as well as between emotional disorders and worry/rumination, on the basis of longitudinal data in a relatively large and representative sample of participants with depressive and/or anxiety disorder from different recruitment settings. Using a latent trait-state model, we expect the trait components of worry and rumination to be highly related and fluctuations at state level to occur in sync. Further, we expect that worry/rumination and emotional disorders are mutually reinforcing each other over time in a downward spiral. Finally, we expect both worry and rumination to show stronger associations with distress disorders than with fear disorders.
Method

Participants and Setting
The Netherlands Study of Depression and Anxiety (NESDA) is an ongoing multi-site longitudinal cohort study including 2981 adult subjects aged 18 through 65 years. The baseline sample consists of 687 (23.0%) healthy controls and 2294 (77.0%) persons with a life-time diagnosis of depression or anxiety disorder of whom 1701 (57.1%) have a current diagnosis (past 6 months). In order to be representative of those with depressive and anxiety disorders respondents in different stages of the developmental history of the disorders (normal, high familial risk, subthreshold disorders, first and recurrent episodes) and from different health care settings (community, primary care and specialized mental health care) were included. A general inclusion criterion was an age of 18 to 65 years. An exclusion criterion was a primary psychotic, obsessive compulsive, bipolar or severe addiction disorder. In addition patients who were not fluent in Dutch were excluded. An extensive description of the rationale, method and recruitment strategy can be found elsewhere (Penninx, et al., 2008).

Procedure
The study protocol was approved centrally by the Ethical Review Board of the VU University Medical Centre and subsequently by local review boards of each participating centre. Participants provided written informed consent. Baseline assessment (T1) took place at one of the seven field centre locations during a 4-hour clinic visit. Assessment included demographic and personal characteristics, medical assessment and the standardized diagnostic psychiatric interview Composite Interview Diagnostic Instrument (CIDI, version 2.1).

A face-to-face follow-up assessment, including the same diagnostic interview, was conducted after 2 years (T2 response: n = 2596, 87.1%) and after 4 years (T3 response: n = 2402, 80.6%). During each assessment presence of DSM-IV (APA, 1994) based depressive [Major Depressive Disorder (MDD), Dysthymia (DYS)] or anxiety [Panic Disorder (PAN), Social Anxiety Disorder (SAD), Generalized Anxiety Disorder (GAD), Agoraphobia without panic (AGO)] disorders was established. Presence of the disorders was defined as the occurrence of the disorder at any time during the six months preceding each assessment (T1, T2, and T3).

Measures
Assessment of psychiatric diagnoses
Diagnostic status (6 month recency diagnosis) was established using the Composite Interview Diagnostic Instrument (CIDI-WHO lifetime version 2.1; Ter Smitten, Smeets, & Van den Brink, 1998). The CIDI is a worldwide used instrument which classifies diagnoses according to DSM-IV criteria (APA, 1994). It has shown high interrater reliability (Wittchen et al., 1991), high test-retest reliability (Wacker, Battegay, Mullejans, & Schlosser, 2006) and high validity for depressive and anxiety disorders (Farmer, Katz, Mcguﬀin, & Bebbington, 1987; Wittchen, 1994; Wittchen et al., 1989). The CIDI was conducted by specially trained clinical research staff.
Questionnaires

Worry was measured with the Penn State Worry Questionnaire (PSWQ; Meyer, et al., 1990). This questionnaire consists of 16 items rated on a 5-point Likert scale ranging from ‘1 = not at all typical of me’ to ‘5 = very typical of me’. The PSWQ consists of two subscales: a ‘General worry’ subscale (11 items) and a ‘Not-worry’ subscale (5 items) (van Rijsoort, Emmelkamp, & Vervaeke, 1999). The ‘General worry’ subscale accounts for most of the variance in PSWQ scores (Brown, Antony, & Barlow, 1992; Meyer, et al., 1990; van Rijsoort, et al., 1999), and only this subscale was administered in the NESDA study. Psychometric properties of this Dutch 11-item version are not available but the original PSWQ has been proven to be a valid measure of trait worrying unaffected by the content of the worry (Davey, 1993; Molina & Borkovec, 1994) with high internal consistency, good test-retest reliability and unaffected by social desirability (Meyer, et al., 1990). The adjustments made to the original PSWQ are not expected to have had a negative effect on these characteristics. Internal consistency in the present study was high, namely α = .96 at T1, T2 and T3.

Rumination was assessed with the subscale Rumination on Sadness of the revised version of the Leiden Index of Depression Sensitivity (LEIDS-R; Van der Does, 2002; Williams, et al., 2008). The LEIDS-R is a self-report instrument which measures cognitive reactivity to sad mood and has been found to reliably discriminate between never-depressed and recovered depressed groups (e.g., Firk & Markus, 2009; Merens, et al., 2005; Moulds, et al., 2008; Van der Does, 2002). LEIDS-R scores also correlate with biological vulnerability markers of depression: response to acute tryptophan depletion (Booij & Van der Does, 2007) and a serotonin transporter gene polymorphism (Antypa, Van der Does, & Penninx, 2010).

The subscale Rumination on Sadness (RUM) consists of 6 items. Participants are asked to indicate whether and how their thinking patterns change when they experience mild dysphoria by scoring each item on a 5-point Likert-scale ranging from 0 ‘not at all’ to 4 ‘very strongly’ applicable to me. In the present sample the internal consistency of the RUM-scale was 0.82 at T1, 0.84 at T2 and 0.85 at T3.

Statistical Analyses

Psychopathology measurement model

We expected the distress-fear model (Distress: MDD, DYS, GAD; Fear: PAN, SAD, AGO) to best represent the latent structure and stability of emotional disorders based on high comorbidity rates between GAD and depressive disorders as well as on previous research supporting the distress-fear measurement model (for an overview see Beesdo-Baum et al., 2009). In order to test this assumption confirmatory factor analyses (CFA) were performed to examine the fit of the distress-fear model, the DSM-IV model and a single-factor model to the longitudinally collected diagnostic CIDI data. In these analyses the T1, T2 and T3 assessments of the 6 diagnostic variables (i.e., MDD, DYS, GAD, SAD, PAN, and AGO) were considered as repeated measures. Factor loadings of the observed disorders on their latent trait factor(s) were constrained to be equal over time. Goodness-of-fit was
assessed using: chi-square test of the model ($p > .05$), Comparative Fit Index (CFI; ≥ .96), Tucker-Lewis Index (TLI; ≥ .95), and Weighted Root-Mean-Square Residual (WRMR; ≤ 1.0). However, we considered the Root-Mean-Square Error of Approximation (RMSEA; ≤ .05), as the main index of model fit as it has been shown to be sensitive to model misspecification and less sensitive than other global fit measures to distribution and sample size in badly fitting covariance structure models (Hu & Bentler, 1998).

**Trait and State Models (T&S)**

Next, we analysed five structural models ((1) Rumination and Worry; (2) Distress Disorders and Worry; (3) Distress Disorders and Rumination; (4) Fear Disorders and Worry; and (5) Fear Disorders and Rumination). See Figures 1-5 for an overview of these models. In order to clarify these models we will describe the four Emotional Disorder-RNT models together (model 2-5). The Rumination-Worry model (model 1) has a similar structure. The models consist of three parts: two identical trait and state (T&S) models for three time points, one addressing psychopathology (Distress or Fear Disorders, top half of the model) and one repetitive negative thinking (Worry or Rumination, bottom half of the model), four correlations (Paths c, d, e, and f) and four regression effects (Paths a1, a2, b1, b2) linking the T&S models. The T&S Disorders model assumes that psychopathology (Distress or Fear disorders) at each time point is the function of two latent (unobserved) variables: a trait component (common factor) and a state component. The state component represents the variance not accounted for by the common factor and consequently reflects time-variant fluctuations within-subject over the 2-yr study period including measurement error. The same assumptions are made for the T&S models of repetitive negative thinking (worry and rumination).

The across-time structure of the latent state psychopathology variables (State 1, State 2, State 3) in the T&S model was modeled as a first-order auto-regressive model. Hence, State 2 and 3 variances consist of variance transmitted from an earlier time point (paths p and q) and new variance resulting from the effects of unobserved variables active during the interval between the measurement points as well as measurement error. The across-time structure of the latent repetitive negative thinking variables was modeled in a similar way (paths r and s).

By combining the T&S models for psychopathology and repetitive negative thinking an integrated model is obtained in which the latent state variables of psychopathology can act as a change agent of repetitive negative thinking and, vice versa, the cross-variable effects. The model allows correlation between the common trait factors (correlation f) and between the state components of psychopathology and repetitive negative thinking (correlations c, d, e). Finally, the effects of the latent state variables can be lagged (Paths a1, a2, b1, b2).

**Model Specification and Identification**

To solve the structural equations of the full model, the following assumptions for both the psychopathology and repetitive negative thinking T&S parts of the models were made:
(a) the regressions of the observed RNT scores and psychopathology factors scores (as derived from the CFA modeling) on their respective latent trait factor are equal over time (equality constraints $x_1 = x_2 = x_3; y_1 = y_2 = y_3$); (b) the lagged cross-variable effects at Time 2 equal those at Time 3 ($a_1 = a_2, b_1 = b_2$) (c.f., Duncan-Jones et al., 1990; Ormel & Schaufeli, 1991); and (c) the residual variances of the observed variables equal 0.

Descriptive statistics as well as model fitting were obtained using the MPlus computer program (version 7; Muthén & Muthén, 1998-2012). Participants who did not participate in the T2 and T3 assessment were included in the analyses by using Full Information Maximum Likelihood (FIML) estimation for missing data. The full model, depicted in Figures 1-5, necessitates the estimation of 18 parameters (variance of the six latent state variables was fixed at unity). Consequently the full model has 3 degrees of freedom left. Standardized estimates, or path coefficients, with a theoretical range from zero (no effect) to ±1 (maximum positive or negative effect) are provided. Path coefficients of < .10 were considered to be negligible, of ≥ .10 and < .30 to be small, of ≥ .30 and < .50 to be moderate and ≥ .50 to be large. As estimation method we used MLR - maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality and non-independence of observations.

Results

Participant Characteristics
The NESDA sample (T1) consists of 2981 adult participants of whom 66.4% is female and the mean age is 41.9 years (SD = 13.1). At T1 1701 participants had a current diagnosis: MDD = 37.4%, DYS = 10.2%, GAD = 15.6%, SAD = 22.3%, PAN = 22.5%, and AGO = 6.3%. As expected comorbidity rates were high with 57.5% of the participants with a current (6-month recency) diagnosis meeting criteria for two or more disorders (see Table 1 for an overview of comorbidity rates). After 2 years (T2) the sample consisted of 2596 (87.1%) participants and after 4 years (T3) of 2402 (80.6%) participants. Potential bias due to selective attrition was checked. Compared to completers, dropouts at T2 and T3 were less educated, younger, had higher latent factor scores for fear and distress disorders, and reported higher worry levels (all $p < .05$). Gender and rumination scores were not associated with attrition.

The relationship of worry with rumination
The rumination-worry model (Figure 1) with cross-variable lagged effects had a good model fit, RMSEA = 0.031. The estimated trait variance of worry scores varied from 59% (.77²) at T1 to 67% (.82²) at T3 and for rumination between 55% (.74²) and 66% (.81²) indicating that up to two thirds of the between subject differences in worry and rumination scores are stable over time. The remaining variance, i.e. state variance, consequently varied between 34% and 45%. See Figure 1 for an overview of the results.
Longitudinal associations between repetitive negative thinking and emotional disorders

Worry and rumination were strongly related at trait level (.76) underlining the interrelatedness of the two forms of RNT. At state level the cross-variable concurrent relationships (c, d, e) were moderate in strength (.44, .39, .45), indicating that participants who increased their engagement in worry also started to ruminate more and vice versa. The cross-variable lagged effects of rumination on worry (a1, a2) were significant but small (.13 and .15). The opposite effects of worry on rumination (b1, b2) were non-significant. Overall it seems that state fluctuations in worry/rumination levels present themselves in sync, while only state fluctuations in rumination have a small effect on state fluctuations in worry 2 years later.

Psychopathology Measurement Model Selection

In line with expectations CFA showed the best goodness-of-fit for the Distress-Fear (MDD, DYS, GAD vs. SAD, PAN, AGO) model — $\chi^2(110) = 201.140; \text{TLI} = .988; \text{CFI} = .991; \text{RMSEA} = .017; \text{WRMR} = 1.056$. Followed by the DSM-IV (MDD and DYS versus SAD, PAN, AGO and GAD) model $\chi^2(124) = 275.464; \text{TLI} = .978; \text{CFI} = .984; \text{RMSEA} = .022; \text{WRMR} = 1.258$ and finally the single-factor model $\chi^2(124) = 374.281; \text{TLI} = .971; \text{CFI} = .976; \text{RMSEA} = .026; \text{WRMR} = 1.491$. Based on these results and on previous literature supporting a Distress-Fear two factor solution (for a review see Beesdo-Baum et al., 2009) this model was chosen for further statistical analyses. Latent factor scores for Distress and Fear disorders were used as input variables in subsequent Trait&State models (see Table 2 for factor loadings on the latent variables).

The relation of rumination with psychopathology

Rumination’s estimated trait variance within the Fear-model (Figure 2) varied from 48% ($0.69^2$) at T1 and T2 to 58% ($0.76^2$) at T3 indicating that about half of the between-subject differences in rumination scores are stable over time. The other half of the variance consists of state variance and subsequently varied between 52% ($0.72^2$) at T1 and T2 to 42% ($0.65^2$) at

---

**Table 1:** Comorbidity at T1 for the current (6 month recency) sample

<table>
<thead>
<tr>
<th></th>
<th>MDD</th>
<th>DYS</th>
<th>GAD</th>
<th>SAD</th>
<th>PAN</th>
<th>AGO</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDD (n = 1115)</td>
<td>-</td>
<td>23.5%</td>
<td>30.5%</td>
<td>34.7%</td>
<td>35.9%</td>
<td>8.2%</td>
</tr>
<tr>
<td>DYS (n = 305)</td>
<td>85.9</td>
<td>-</td>
<td>45.6</td>
<td>43.6</td>
<td>39.7</td>
<td>10.5</td>
</tr>
<tr>
<td>GAD (n = 464)</td>
<td>73.3</td>
<td>30.0</td>
<td>-</td>
<td>44.0</td>
<td>42.5</td>
<td>10.3</td>
</tr>
<tr>
<td>SAD (n = 665)</td>
<td>58.2</td>
<td>20.0</td>
<td>30.7</td>
<td>-</td>
<td>45.9</td>
<td>9.6</td>
</tr>
<tr>
<td>PAN (n = 670)</td>
<td>59.7</td>
<td>18.1</td>
<td>29.4</td>
<td>45.5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>AGO (n = 187)</td>
<td>48.7</td>
<td>17.1</td>
<td>25.7</td>
<td>34.2</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. MDD = Major Depressive Disorder; DYS = Dysthymia; GAD = Generalized Anxiety Disorder; SAD = Social Anxiety Disorder; PAN = Panic Disorder; AGO = Agoraphobia w/o panic.
Figure 1: Rumination and Worry T&S models linked at trait level via correlations between trait factors (f) and at state level through contemporaneous correlations (c,d,e,) and cross-variable 2-year lagged effects (a,b).

Note. Equality constraints applied to identify model equations were: x1 = x2 = x3; y1 = y2 = y3; a1 = a2; b1 = b2. Significant (p < .05) correlations and regression coefficients are depicted in bold.
Longitudinal associations between repetitive negative thinking and emotional disorders

T3. An almost identical pattern of results was found in the Distress model (Figure 3) where estimated trait variance of rumination ranged from 50% (T1 and T2) to 62% (T3) and state variance ranged from 50% (T1 and T2) to 38% (T3).

The trait fear factor accounted for 67% (.82^2) of the variance in fear scores at T1, 61% (.78^2) at T2 and 76% (.87^2) at T3. These scores are slightly higher than those of the trait factor distress with variances of 52% (T1), 50% (T2) and 59% (T3), suggesting that individual differences in fear scores are slightly more stable over time.

The fear and distress trait factor are both strongly (.54, respectively .66) related to the rumination trait factor. These numbers demonstrate the interconnection between the stable components of the emotional disorders and the stable trait of rumination.

The cross-variable effects revealed a fairly similar pattern for the fear and distress model. Both models show that all concurrent relationships (c, d, e) are significant and small to moderate in strength, indicating that participants who became more fearful or distressed also engaged more in rumination and vice versa. Small to moderate lagged effects (a1, a2) were also found for state level fluctuations of distress or fear disorders on state rumination indicating that more distress or fear resulted in higher levels of rumination 2 years later. Results of lagged effects (b1, b2) of state level rumination on state level psychopathology differed between the distress and fear model. Where the fear model showed a significant positive effect of state fluctuations in rumination on state fluctuations in fear disorder this effect was not observed for distress disorder. Regression coefficients (.09 and .06) were however so small that these effects can be considered negligible. In sum, state fluctuations in rumination seem to be primarily driven by state fluctuations in psychopathology and not vice versa.

The relation of worry with psychopathology

The estimated trait variance of Worry scores within the Fear-model (Figure 4) varied from 55% (.74^2) at T1, to 58% (.76^2) at T2 and 62% (.79^2) at T3 indicating that over half of the between-subject differences in worry scores are stable over time. The remaining variance, i.e. state variance, consequently varied from 45% (.67^2) at T1 to 42% (.65^2) at T2 and 38%
Figure 2: Rumination and Fear T&S models linked at trait level via correlations between trait factors (f) and at state level through contemporaneous correlations (c,d,e) and cross-variable 2-year lagged effects (a,b).

Note. Equality constraints applied to identify model equations were: x1 = x2 = x3; y1 = y2 = y3; a1 = a2; b1 = b2. Significant ($p < .05$) correlations and regression coefficients are depicted in bold.
Figure 3: Rumination and Distress T&S models linked at trait level via correlations between trait factors (f) and at state level through contemporaneous correlations (c,d,e,) and cross-variable 2-year lagged effects (a,b).

Note. Equality constraints applied to identify model equations were: x1 = x2 = x3; y1 = y2 = y3; a1 = a2; b1 = b2. Significant (p < .05) correlations and regression coefficients are depicted in bold.
Figure 4: Worry and Fear T&S models linked at trait level via correlations between trait factors (f) and at state level through contemporaneous correlations (c,d,e,) and cross-variable 2-year lagged effects (a,b).

Note. Equality constraints applied to identify model equations were: x1 = x2 = x3; y1 = y2 = y3; a1 = a2; b1 = b2. Significant (p < .05) correlations and regression coefficients are depicted in bold.
**Figure 5**: Worry and Distress T&S models linked at trait level via correlations between trait factors (f) and at state level through contemporaneous correlations (c,d,e) and cross-variable 2-year lagged effects (a,b).

**Note.** Equality constraints applied to identify model equations were: $x_1 = x_2 = x_3$; $y_1 = y_2 = y_3$; $a_1 = a_2$; $b_1 = b_2$. Significant ($p < .05$) correlations and regression coefficients are depicted in bold.
Chapter 3

(.62) at T3. In the Distress model (Figure 5) about two-thirds to three quarters of the total estimated variance in Worry scores can be accounted for by the trait rumination factor (scores ranging from 61% to 74%) with state variance scores consequently ranging from 26% to 39%.

The psychopathology parts of the T&S models revealed estimated trait variance for fear and for distress disorders scores comparable to those in the rumination models. Trait components of both psychopathology categories were strongly related to the trait component of worry, .62 for fear and .79 for distress disorders, demonstrating that worry is intertwined with these components.

The results of the cross-variable effects of the concurrent relationships (c, d, e) were all significant. In the fear model they were moderate to large in strength whereas in the distress model relationships were small to moderate. This suggests that state fluctuations in worry go more hand in hand with state fluctuations in fearfulness than with distress. Cross-variable lagged effects (a1, a2, b1, b2) in both the distress and the fear model were either non-significant or of negligible strength. The only exception was the effect of state fear on worry (.21 and .36) with more fear resulting in higher levels of worry 2 years later. In sum, state fluctuations in psychopathology are not driven by state fluctuations in worry and only for fear disorders do state fluctuations in psychopathology affect subsequent worry levels.

Discussion

The aim of the present study was to investigate how trait and state components of worry and rumination relate to each other and to emotional disorders (fear and distress). The 3-wave setup of our study design enabled us to separate trait and state effects and to examine the temporal and directional character of reciprocal effects. Results confirmed our expectation that trait components of worry and rumination are highly related and that fluctuations at state level occur in sync. Regarding repetitive negative thinking (RNT; worry & rumination) and emotional disorders we predicted that they would mutually reinforce each other over time, setting off a downward spiral. This hypothesis was not confirmed by the present study: fluctuations in psychopathology predicted state levels of worry/rumination 2 years later but not vice versa. Finally, we expected both worry and rumination to show stronger associations with distress disorders than with fear disorders. Results showed strong associations with both fear and distress disorders but as expected those with the distress disorders were the strongest. Overall, these results support the idea that worry and rumination contain a shared underlying process as well as that they both have transdiagnostic characteristics.

The relationship between worry and rumination was assessed in one comprehensive design using trait-state models which enabled us to operationalize each cognitive process as the function of a trait component, stable across time, and a state component reflecting fluctuations over the 4 year period. The trait variance was substantial across
waves (explained variance for worry: 59% - 67%; and for rumination 55% - 66%) suggesting the presence of a stable underlying trait. Moreover, the trait components of worry and rumination were highly correlated (.76) and at state level correlations between concurrently measured worry and rumination were of medium strength (.39 - .45). These findings support the idea that worry and rumination have a solid, shared, base.

The similarities between worry and rumination were further explored by examining whether worry and rumination differed in their relationships with psychopathology. The latent structure and stability of emotional disorders was as expected – and in accordance with previous studies (for a review see: Beesdo-Baum et al., 2009) – best represented by the distress (GAD, DYS, MDD) - fear (PAN, SAD, AGO) model allowing for analyses to be performed while taking comorbidity among these disorders into account. The latent factor scores derived for fear and distress disorders permitted us to examine reciprocal and temporal relationships of repetitive negative thinking (worry and rumination) with psychopathology. Traditionally worry is linked to GAD and rumination to MDD. Considering that both disorders are now placed within the distress disorders category it was expected that RNT would show stronger associations with the distress than the fear disorders. This was indeed the case although differences were modest and correlations between the trait components of RNT and psychopathology were strong in all four T&S models investigated. These results suggest that trait worry and trait rumination show similar relationships to both trait distress and trait fear disorders, which has several implications. Firstly, there is no differential effect of worry and rumination regarding fear and distress disorders, thus underlining the similarities of the two constructs. Secondly, repetitive negative thinking is involved in both distress and fear disorders even when GAD is grouped with the depressive disorders, hence supporting the notion of a transdiagnostic process (Harvey et al., 2004; Ehring & Watkins, 2008). It should be noted however, that the direction of the relationships between the trait components cannot be determined within our study design. It could reflect the influence of a third variable in line with the common cause model (e.g. genetic vulnerability) (for an explanation of the model see Klein, Kotov, & Bufferd, 2011) or for instance a directional relationship, c.q. predisposition model. There are studies supporting the predisposition model showing a causal relationship with repetitive negative thinking preceding changes in psychopathology (for an overview see Watkins, 2008; and Ehring & Watkins, 2008). Rumination has received more attention in this regard than worry (Topper, Emmelkamp, & Ehring, 2010). Our results suggest that it would be informative to conduct prospective studies on the nature and direction of the relationship of trait RNT (as also measured with generic measures for RNT, such as the Repetitive Thinking Questionnaire (RTQ; McEvoy, Mahoney, & Moulds, 2010) or the Perseverative Thinking Questionnaire (PTQ; Ehring et al., 2011)), with various emotional disorders while taking comorbidity into account.

By combining T&S models for psychopathology and repetitive negative thinking we were also able to examine whether state level fluctuations on either one led to state level fluctuations on the other. The 3-wave set up of our study provided a unique opportunity to examine both concurrent relations and lagged cross-variable effects. As is to be
expected the concurrent relations showed that state fluctuations in psychopathology were accompanied by state fluctuations in repetitive negative thinking in the same direction (positive correlations d and e). This is in line with experimental studies which have repeatedly shown that experimentally induced worry or rumination directly and negatively affects anxious and depressed mood states (e.g. Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; McLaughlin, Mennin, & Farach, 2007). Similarly, in accordance with the ‘differential activation’ hypothesis of Teasdale (1988) and the ‘mood state’ hypothesis of Miranda and Persons (1988) latent dysfunctional attitudes have been found to become activated during sad mood.

Contrary to our expectations, analyses concerning cross-variable lagged (2-yr follow-up) effects at state level did not support our mutual reinforcement hypothesis. State fluctuations in worry and rumination were not predictive of state fluctuations in fear or distress levels two years later. However the opposite cross-variable lagged effects – state fluctuations in psychopathology on state fluctuations in repetitive negative thinking – were significant albeit small to moderate in strength.

Cognitive theory posits that change in cognition precedes change in symptoms of psychopathology, a view that has led to the development of therapies aimed at altering cognition (i.e. CBT) and the assessment of cognitive change in establishing treatment progress (e.g. Dozois, Covin & Brinker, 2003). However evidence for this supposition is not unequivocal as some studies have shown that cognitive change is not specific to therapies tackling cognitions and that this even occurs when using pharmacotherapy (e.g. Simons, Garfield, & Murphy, 1984). Moreover, and in line with our state level findings, it has been reported that changes in cognitive content during CBT are not predictive of changes in depressive symptoms (Jarrett et al., 2007). Also, it has been found that changes at cognitive level and changes in symptomatology occur in tandem and a recent review of cognitive mediation in CBT for anxiety disorders concluded that it is too early to conclude that cognitive changes cause improvement and that cognitive change is not a proxy for other third variables (Smits et al., 2012). The state-trait distinction may also be relevant in this context. Changes in state cognitions (such as negative automatic thoughts) may be primarily a reflection of changes at symptom level. However, as has been stressed in the earliest formulations of cognitive theory (Beck, 1967), it may well be that in order to establish long lasting changes in psychopathology it is necessary to alter underlying cognitive vulnerabilities (i.e., cognitive structures and schema’s) which, when activated, give rise to momentary cognitive content c.q. negative automatic thoughts. The therapeutic relevance of altering stable underlying cognitive vulnerabilities instead of more time variant cognitions is in line with the present findings which show that RNT and psychopathology are highly interrelated at the trait level in particular.

Several limitations should be taken into account when interpreting the current results. Firstly, distress and fear disorders had to be analyzed in separate models and as a consequence results may have been confounded by the high comorbidity of these disorders. Secondly, attrition was not completely random. The response rate was 87.1%
at wave 2 and 80.6% at wave 3, and non-response was significantly higher among those with younger age, lower education, higher levels of psychopathology and higher levels of worry consequently somewhat restricting generalizability of study results. Thirdly, the present models did not differentiate between groups of first onset, current psychopathology and remitted anxiety/depression. These groups may show slightly different response patterns for instance due to scarring effects. Fourthly, rumination was assessed using the subscale ‘rumination on sadness’ of the LEIDS-R, an instrument measuring cognitive reactivity. There are several other questionnaires that measure rumination; results may differ depending on the instrument chosen. Finally, the present study did not include a generic RNT instrument. Therefore, our findings may not be representative of all types of repetitive negative thinking such as post-event processing.

The present study also has several strengths: i) a longitudinal design in a representative sample of participants with depressive and/or anxiety disorder from different recruitment settings; ii) use of a structured diagnostic interview to assess presence of depressive and anxiety disorders; iii) examining the structure of anxiety and depressive disorders instead of individual disorders separately; iv) use of trait and state model in analyzing temporal and reciprocal relationships of emotional disorders with RNT.

Conclusions
The present data show that worry and rumination have strong trait-like components, which are also strongly interrelated. Moreover, state fluctuations around set-point in worry level covary with state fluctuations in the level of rumination. These findings combined with the similar relationships of worry and rumination with emotional disorders are in line with the idea that they are conceptualizations of the same underlying process of repetitive negative thinking. Furthermore, the lack of differential relationships of worry and rumination with fear and distress disorders supports the notion that they can be conceptualized as transdiagnostic processes critically involved in emotional disorders.

However, we did not find support for the hypothesis that state fluctuations of RNT offset a downward spiral in which psychopathology and RNT mutually reinforce each other. State fluctuations in worry/rumination are preceded by state fluctuations in psychopathology but not vice versa. This pattern suggests that fluctuations in RNT may merely be epiphenomena of emotional disorders. From a clinical perspective it seems more pertinent to modify the underlying trait component(s) of worry and rumination in order to obtain endurable therapeutic benefits.

Acknowledgements
The infrastructure for the NESDA study (www.nesda.nl) is funded through the Geestkracht program of the Netherlands Organisation for Health Research and Development (Zon-Mw, grant number 10-000-1002) and is supported by participating universities and mental
health care organizations (VU University Medical Center, GGZ inGeest, Arkin, Leiden University Medical Center, GGZ Rivierduinen, University Medical Center Groningen, Lentis, GGZ Friesland, GGZ Drenthe, Scientific Institute for Quality of Healthcare (IQ healthcare), Netherlands Institute for Health Services Research (NIVEL) and Netherlands Institute of Mental Health and Addiction (Trimbos).

The contribution of A.J.W. Van der Does was supported by Netherlands Organisation for Scientific Research (NWO) Vici Grant # 453-06-005. The funding sources had no role in the study design, in the collection, analysis and interpretation of data, in the writing of the report, and in the decision to submit the paper for publication.
References


Longitudinal associations between repetitive negative thinking and emotional disorders


