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Chapter 4

Repetitive negative thinking as a transdiagnostic factor in depression and anxiety: A conceptual replication

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Under revision Behaviour Therapy and Research
Abstract

**Background:** Comorbidity among affective disorders is high. Rumination has been found to mediate cross-sectional and prospective relations between anxiety and depressive symptoms in adolescents and adults. We examined whether rumination and worry, both forms of repetitive negative thinking, also explain the associations between affective disorders.

**Method:** Prospective cohort study. In a mixed sample of 2,981 adults (persons with a prior history of or a current affective disorder and healthy individuals) we assessed DSM-IV affective disorders (CIDI), rumination (LEIDS-R) and worry (PSWQ). All measures were repeated 2 years and 4 years later.

**Results:** Using structural equation models, we found that baseline rumination and worry partly mediated the association of baseline fear disorders (social anxiety disorder, panic disorder, agoraphobia) with distress disorders (dysthymia, major depressive disorder, generalized anxiety disorder). Moreover, baseline fear disorders predicted changes in distress disorders and changes in worry and rumination mediated these associations. The association between baseline distress disorders and changes in fear disorders was mediated by changes in rumination but not by changes in worry.

**Conclusions:** Repetitive negative thinking is an important transdiagnostic factor. Rumination and worry are partly responsible for the cross-sectional and prospective co-occurrence of affective disorders and may be suitable targets for treatment.
Introduction

Comorbidity of mental disorders is the rule rather than the exception, especially among anxiety and depressive disorders (Brown, Campbell, Lehman, Grisham, & Mancill, 2001). Efforts to unravel why there is such a high comorbidity rate have drastically increased in recent years, as has the call for broad and disorder transcending therapies (e.g. Fairburn, Cooper, & Shafran, 2003; Barlow, Allen, & Choate, 2004). Generally it is assumed that there are certain factors that are shared between multiple disorders, which not only contribute to the occurrence of a specific disorder but are also (in part) responsible for comorbidity among these disorders. Such disorder transcending factors are commonly referred to as transdiagnostic factors (e.g. Harvey, Watkins, Mansell, & Shafran, 2004; Ehring & Watkins, 2008). Better understanding of these shared factors is not only of theoretical importance, but also clinically relevant as it could lead to the development of more effective therapeutic interventions.

One of the main candidate cognitive processes involved in comorbidity among emotional disorders is repetitive negative thinking (RNT: Ehring & Watkins, 2008). In the anxiety literature RNT is referred to as worry, in the depression literature it is referred to as rumination. Both processes are characterized by uncontrolled, excessive and repetitive thinking about current concerns, problems, past experiences or worries about the future (Ehring & Watkins, 2008, p. 192). The main difference between the two is that worry is more future focused and rumination is more past focused (e.g. Smith & Alloy, 2009; Watkins et al., 2005). However, it has to be noted that this is a simplification; both processes contain both future- and past-related aspects (e.g. McLaughlin, Borkovec, & Sibrava, 2007). Furthermore, other differences between worry and rumination have also been reported (see Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008 for an overview). Research has shown that both worry and rumination scores are elevated across the emotional disorders (Chelminsky & Zimmerman; see Ehring & Watkins, 2008 for an overview regarding rumination) and that these scores are higher in individuals with multiple diagnoses than in individuals with a single diagnosis (McEvoy, Watson, Watkins, & Nathan, 2013). Although these findings are in line with a transdiagnostic account, the mere presence of these cognitive processes across disorders does not qualify them as causative factors, let alone causative of comorbidity. Evidence for their causal influence comes from experimental studies showing that RNT exacerbates depressed and anxious mood (e.g. McLaughlin, Borkovec, & Sibrava, 2007; Behar, Zueilig, & Borkovec, 2005) as well as from prospective and longitudinal studies supporting RNT’s involvement in the onset, maintenance and recurrence of both anxiety and depressive disorders (e.g. Just & Alloy, 1997, Nolen-Hoeksema, 2000; see Watkins, 2008 for an overview). However if RNT truly contributes to the high comorbidity rates among emotional disorders it should also mediate the relationship between anxiety and depressive disorders and vice versa. McLaughlin and Nolen-Hoeksema (2011) indeed found that rumination mediated the concurrent relationship of depression with anxiety symptoms in two large samples of adults and adolescents. Moreover, baseline depressive symptoms predicted subsequent
increases in anxiety and rumination fully mediated this association. This innovative study has a few limitations. It investigated anxiety and depression symptoms, not clinical diagnoses; and it only examined rumination and did not include other types of RNT (such as worry).

The aim of the present study is to test whether RNT also accounts for the comorbidity among emotional disorders, both cross-sectionally and longitudinally. In other words, we carried out a conceptual replication of McLaughlin and Nolen-Hoeksema (2011), by focusing on clinical diagnoses instead of symptoms and by examining two types of RNT—worry and rumination. We expected that both rumination and worry would account for the cross-sectional overlap of emotional disorders at baseline and would mediate the prospective cross-disorder relations among emotional disorders.

Methods

Participants and design
The Netherlands Study of Depression and Anxiety (NESDA) is an ongoing cohort study designed to investigate determinants, course and consequences of depressive and anxiety disorders. A sample of 2981 persons aged 18 through 65 years, consisting of healthy controls, persons with a prior history of depressive and anxiety disorders, and persons with a current depressive and/or anxiety disorder were included in the study. Respondents were recruited in the general population, through a screening procedure in general practice, or when newly enrolled in specialized health care in order to represent different health care settings and different developmental stages of psychopathology. General exclusion criteria were a primary diagnosis of other severe psychiatric disorders (e.g. psychotic, obsessive compulsive, bipolar or severe addiction disorder) and not being fluent in Dutch.

A detailed description of the NESDA design and sampling procedures is given elsewhere (Penninx et al., 2008). The baseline assessment included assessment of demographic and personal characteristics, a standardized diagnostic psychiatric interview and a medical assessment. This study reports on the baseline and the 2-yr and 4-yr follow-up assessments. The research protocol was approved by the Ethical Committees of participating universities and all respondents provided written informed consent.

Measures

Assessment of psychiatric diagnoses
The diagnostic status (6-month prevalence) of depressive [Major Depressive Disorder (MDD), Dysthymia (DYS)] or anxiety [Panic Disorder with or without Agoraphobia (PAN), Social Anxiety Disorder (SOC), Generalized Anxiety Disorder (GAD), Agoraphobia without panic (AGO)] disorders was established at T0, T2 and T4 using the Composite Interview Diagnostic Instrument (CIDI-WHO lifetime version 2.1; Ter Smitten, Smeets, & Van den Brink, 1998). The CIDI classifies diagnoses according to DSM-IV criteria (APA, 1994).
Trained interviewers can reach high interrater reliability, high test-retest reliability (Wacker, Battegay, Mullejans, & Schlosser, 2006) and high validity for depressive and anxiety disorders (Wittchen, 1994). The CIDI was conducted by 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 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 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 is the strongest of the two (Meyer, et al., 1990; van Rijsoort, et al., 1999) and only this subscale was administered in the NESDA study. Internal consistency in the present study was high, namely α = .96 at T0, T2 and T4.

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 measures cognitive reactivity to sad mood and is a self-report instrument, which has been found to reliably discriminate between never-depressed and recovered depressed groups (e.g., Moulds, et al., 2008; Van der Does, 2002). 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 T0, 0.84 at T2 and 0.85 at T4.

**Statistical analyses**

Differences in rumination (LEIDS-R) and worry (PSWQ) baseline scores between psychopathology groups were analyzed with separate ANOVA’s using five multiple imputed datasets to account for missing data on LEIDS-R and PSWQ. Significant main effects were followed-up by Bonferroni adjusted multiple comparisons. Next, structural equation models were fit using WLSMV parameter estimates in Mplus (version 7.0). Participants who did not participate in the 2-yr and/or 4-yr follow-up assessment were included in the analyses using all available pairwise present information. Standardized estimates, or path coefficients, with a theoretical range from zero (no effect) to + 1 (maximum positive or negative effect) are provided. Model fit was evaluated using the Tucker-Lewis Index (TLI), the Comparative Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA). For the TLI and CFI, values between 0.90 and 0.95 are considered acceptable, and ≥ 0.95 as good. For the RMSEA, acceptable models have values of ≤ 0.10, and good models of ≤ 0.05.

In accordance with previous studies (for a review of the literature see Beesdo-Baum et al., 2009) and a previous confirmatory factor analysis (CFA) of the T0, T2 and T4
assessments of the six diagnostic variables (i.e., MDD, DYS, GAD, SAD, PAN, and AGO) as repeated measures in NESDA (Spinhoven et al., 2014), we choose the distress-fear model (Distress: MDD, DYS, GAD; Fear: PAN, SAD, AGO) to best represent the latent structure and stability of emotional disorders. Latent factor scores for distress and for fear disorders were used as dependent variables in further statistical analyses.

Next, we determined the role of rumination and worry as putative mediators of the cross-sectional relations of distress with fear disorders. More specifically, we determined whether the association of distress with fear disorders was attenuated after including both rumination and worry as independent variables into the prediction model.

Finally, we examined two longitudinal mediation models: one examining the role of rumination as a putative mediator of the longitudinal association of distress with fear disorders (and vice versa) and one examining the role of worry in these longitudinal relations. More specifically, we determined: 1) the association of T0 distress with T4 fear disorders (and vice versa); 2) the association of T0 distress/fear with T2 rumination/worry, controlling for T0 rumination/worry; 3) the association of T2 rumination/worry with T4 distress/fear, controlling for T0 distress/fear; and 4) the attenuation of the association of T0 distress with T4 fear after accounting for changes in rumination/worry from T0 to T2 (and vice versa). In this way we could analyze whether baseline distress disorders predicted changes in rumination/worry and whether these changes predicted subsequent changes in fear disorders (and vice versa). The significance of the indirect effect of fear disorders on distress disorders through changes in rumination/worry (and vice versa) was determined using a bootstrap approximation with 1000 iterations to obtain biased-controlled confidence intervals.

Results

Descriptive statistics
At baseline, we included 2,981 participants with a mean age of 41.9 years (SD = 13.1), a mean duration of education of 12.1 years (SD = 3.3); 66.4% was female. Face-to-face follow-up assessments were conducted with a response of 87.1% (n = 2,596) at 2-yr follow-up and 80.6% (n = 2,402) at 4-yr follow-up. At baseline, 1,701 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 57.0% (n = 725) of the 1,273 participants with a 6-month recency distress diagnosis (MDD, DYS, GAD) meeting criteria for a fear disorder (SAD, PAN, AGO) and 62.9% of the 1,153 participants with a fear disorder having a comorbid distress disorder. Comorbidity rates at T4 were as follows: 43.6% (n = 225) of the 516 participants with a 6-month recency distress diagnosis fulfilled criteria for a fear disorder and 47.4% of the 475 participants with a fear disorder had a comorbid distress disorder.

We examined whether sample attrition had introduced response bias. Compared with completers, dropouts at 2-yr follow-up as well as at 4-yr follow-up were younger, less
educated, more often had an anxiety or depressive disorder at baseline, and also manifested higher levels of worry (PSWQ). There was no significant association of gender with attrition.

Table 1 presents descriptive statistics on rumination and worry in participants with no 6-month recency diagnosis (n = 1280), one or more fear disorders (n = 428), one or more distress disorders (n = 548), and comorbid fear and distress disorders (n = 725). Separate ANOVA’s yielded a significant main effect for group on rumination (F(3, 2977) = 335.49, p < .001) and worry (F(3, 2977) = 527.33, p < .001). Subsequent Bonferroni adjusted comparisons showed that all groups differed significantly from each other (all p < .001) regards rumination and worry. In accordance with expectations the lowest scores were found in participants with no disorder and the highest scores in participants with comorbid disorders. Moreover, participants with distress disorders obtained higher scores than participants with fear disorders.

Table 2 presents the zero-order correlations of rumination and worry scores with factor scores for distress and fear disorders (see below). As hypothesized both rumination and worry were positively associated with each other and with distress and fear disorders, which were also positively associated with each other (all p < .001).

**Measurement model**

In line with expectations, CFA showed that a Distress-Fear (MDD, DYS, GAD vs. SAD, PAN, AGO) model — χ²(110) = 201.1; TLI = .99; CFI = .99; RMSEA = .02 – showed a good fit to the data (see Table 3 for factor loadings on the latent variables).

**Mediation analysis**

**Cross-sectional analysis**

T0 distress disorders were significantly associated with T0 fear disorders, β = .84, p < .001. The association of distress with fear disorders was attenuated, β = .70 p < .001, but remained significant after including T0 rumination and worry as predictors into the model (rumination: β = .12 p < .001, and worry: β = .14, p < .001).

**Longitudinal analysis**

Next, we analyzed the associations between T0 distress disorders and T4 fear disorders while controlling for T0 fear disorders (and vice versa). T0 fear disorders significantly predicted T4 distress disorders, controlling for T0 distress disorders, β = .21, p < .001. T0 distress disorders were associated significantly with T4 fear disorders, controlling for T0 fear disorders, β = .07, p < .001. T0 fear was associated with T2 rumination, controlling for rumination at T0, β = .15, p < .001. T2 rumination was associated with T4 distress, controlling for T0 distress, β = .14, p < .001. In the final mediation model, T0 fear was no longer a significant predictor of T4 distress, controlling for T0 distress and T0 rumination, when T2 rumination was added to the model, β = .11, p = .22 (see Fig. 1). The covariance between T0 distress and fear and T0 rumination and both T0 distress and fear was accounted for in the final model. Fit indices
Table 1: Descriptive statistics on rumination (LEIDS) and worry (PSWQ) baseline scores in participants with no 6-month recency diagnosis, one or more fear disorders (SAD, PAN, AGO), one or more distress disorder (MDD, DYS, GAD) and comorbid fear and distress disorders (n = 2981)

<table>
<thead>
<tr>
<th>Variables</th>
<th>1. No disorder (n = 1280)</th>
<th>2. Fear disorder (n = 428)</th>
<th>3. Distress disorder (n = 548)</th>
<th>4. Fear + Distress disorder (n = 725)</th>
<th>F-value (3, 2977)</th>
<th>Contrasts</th>
</tr>
</thead>
<tbody>
<tr>
<td>LEIDS: Rumination</td>
<td>M: 6.3 SD: 4.6</td>
<td>M: 9.2 SD: 5.0</td>
<td>M: 11.3 SD: 4.9</td>
<td>M: 12.5 SD: 4.6</td>
<td>335.49</td>
<td>1&lt;2&lt;3&lt;4</td>
</tr>
<tr>
<td>PSWQ: Worry</td>
<td>M: 23.5 SD: 10.6</td>
<td>M: 32.4 SD: 10.5</td>
<td>M: 35.9 SD: 10.7</td>
<td>M: 40.3 SD: 11.2</td>
<td>527.33</td>
<td>1&lt;2&lt;3&lt;4</td>
</tr>
</tbody>
</table>

Note. MDD = Major Depressive Disorder; DYS = Dysthymia; GAD = Generalized Anxiety Disorder; SAD = Social Anxiety Disorder; PAN = Panic Disorder with or without Agoraphobia; AGO = Agoraphobia w/o panic; LEIDS = Leiden Index of Depression Sensitivity-Revised: Rumination subscale; PSWQ = Penn State Worry Questionnaire: General worry subscale; all Bonferroni adjusted comparisons < .001. Because of missing T0 values on LEIDS (n = 368) and PSWQ (n = 363) and assuming data are missing at random (MAR) five multiple imputed datasets were created and Table 2 presents the pooled estimates for M, SD, F-value and Contrasts. A completers analysis yielded identical results.

Table 3: Factor loadings on the latent variables for the distress-fear CFA solution (n = 2981)

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Distress</th>
<th>Fear</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>T1</td>
<td>T2</td>
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<tr>
<td>MDD</td>
<td>.81</td>
<td>.85</td>
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<tr>
<td>DYS</td>
<td>.81</td>
<td>.86</td>
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<tr>
<td>GAD</td>
<td>.69</td>
<td>.73</td>
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</tbody>
</table>

Note. MDD = Major Depressive Disorder; DYS = Dysthymia; GAD = Generalized Anxiety Disorder; SAD = Social Anxiety Disorder; PAN = Panic Disorder with or without Agoraphobia; AGO = Agoraphobia w/o panic.
indicated that the model showed an excellent fit to the data: $\chi^2 (37) = 103.1; \text{TLI} = .99; \text{CFI} = .98; \text{RMSEA} = .02$. Bootstrapping estimates showed that the indirect effect of fear through rumination on distress ($\beta = .03; 99\% \text{BCI} = .01 - .06$) was significant. Repeating this analysis in participants with complete CIDI and LEIDS data at T0 ($n = 2618$) gave similar results.

Subsequently, we analyzed worry as a putative mediator of the longitudinal fear – distress association in a similar way. T0 fear was associated with T2 worry, controlling for worry at T0, $\beta = .15, p < .001$. T2 worry was associated with T4 distress, controlling for T0 distress, $\beta = .17, p < .001$. In the final mediation model, T0 fear was no longer a significant predictor of T4 distress, controlling for T0 distress and T0 worry, when T2 worry was added to the model, $\beta = .04, p = .70$ (see Fig. 2). Fit indices indicated that the model showed an excellent fit to the data: $\chi^2 (37) = 105.80; \text{TLI} = .99; \text{CFI} = .98; \text{RMSEA} = .02$. Bootstrapping estimates showed that the indirect effect of fear through rumination on distress ($\beta = .06; 99\% \text{BCI} = .01 - .11$) was significant. Repeating this analysis in participants with complete CIDI and PSWQ data across waves ($n = 2613$) gave similar results.

Table 2: Zero-order correlations between rumination, worry and distress and fear disorders ($n = 2981$)

<table>
<thead>
<tr>
<th>Variables</th>
<th>1</th>
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<th>8</th>
<th>9</th>
<th>10</th>
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<tbody>
<tr>
<td>1. LEIDS, Rumination T0</td>
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<td></td>
<td></td>
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<tr>
<td>2. PSWQ, Worry T0</td>
<td>.63</td>
<td>-</td>
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<td></td>
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<tr>
<td>3. CIDI, Distress T0</td>
<td>.63</td>
<td>.73</td>
<td>-</td>
<td></td>
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<td></td>
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<td>4. CIDI, Fear T0</td>
<td>.52</td>
<td>.68</td>
<td>.70</td>
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<td>5. LEIDS, Rumination T2</td>
<td>.73</td>
<td>.54</td>
<td>.53</td>
<td>.46</td>
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<td>6. PSWQ, Worry T2</td>
<td>.57</td>
<td>.75</td>
<td>.59</td>
<td>.58</td>
<td>.62</td>
<td>-</td>
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<td>7. CIDI, Distress T2</td>
<td>.53</td>
<td>.57</td>
<td>.69</td>
<td>.48</td>
<td>.60</td>
<td>.67</td>
<td>-</td>
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<td>8. CIDI, Fear T2</td>
<td>.50</td>
<td>.59</td>
<td>.59</td>
<td>.81</td>
<td>.54</td>
<td>.67</td>
<td>.76</td>
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<td>9. LEIDS, Rumination T4</td>
<td>.65</td>
<td>.52</td>
<td>.53</td>
<td>.45</td>
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<td>.56</td>
<td>.52</td>
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<tr>
<td>10. PSWQ, Worry T4</td>
<td>.51</td>
<td>.67</td>
<td>.54</td>
<td>.52</td>
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<td>.75</td>
<td>.56</td>
<td>.53</td>
<td>.66</td>
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<tr>
<td>11. CIDI, Distress T4</td>
<td>.46</td>
<td>.55</td>
<td>.61</td>
<td>.51</td>
<td>.52</td>
<td>.63</td>
<td>.76</td>
<td>.62</td>
<td>.59</td>
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<td>12. CIDI, Fear T4</td>
<td>.49</td>
<td>.56</td>
<td>.59</td>
<td>.80</td>
<td>.53</td>
<td>.64</td>
<td>.65</td>
<td>.86</td>
<td>.58</td>
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<td>.81</td>
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Note: LEIDS = Leiden Index of Depression Sensitivity-Revised; Rumination subscale; PSWQ = Penn State Worry Questionnaire: General worry subscale; CIDI = Composite Interview Diagnostic Instrument, version 2.1; Distress = combined factor scores for Dysthymia, Major Depression, Generalized Anxiety; Fear = combined factor scores for Social Anxiety, Panic, Agoraphobia; T0 = baseline; T2 = 2-yr follow-up; T4 = 4-yr follow-up; all $p < .001$.
Figure 1: Longitudinal mediation model of rumination mediating fear and distress disorders

Note: Parameter estimates for the longitudinal mediation model of rumination mediating the prospective relations between fear and distress disorders in 2981 participants from the Netherlands Study of Depression and Anxiety (NESDA). Squares represent observed variables and ovals latent variables. Single-headed arrow path coefficients represent standardized partial regression coefficients. Two-headed arrows represent correlation coefficients. Loadings, standardized estimates, or path coefficients, with a theoretical range from zero (no effect) to +1 (maximum positive or negative effect) are provided. MDD = Major Depressive Disorder; DYS = Dysthymia; GAD = Generalized Anxiety Disorder; SAD = Social Anxiety Disorder; PAN = Panic Disorder with or without Agoraphobia; AGO = Agoraphobia w/o panic. T0 = baseline; T2 = 2-yr follow-up; T4 = 4-yr follow-up. All parameter estimates were significant at p < .001, except the association of Fear T0 – Distress T4 (p = .22), DYS T0-DYS T4 (p < .01), MDD T0 – MDD T4 (p < .05) and GAD T0 – GAD T4 (p < .05).
Repetitive negative thinking as a transdiagnostic factor in depression and anxiety

Figure 2: Longitudinal mediation model of worry mediating fear and distress disorders

Note: Parameter estimates for the longitudinal mediation model of worry mediating the prospective relations between fear and distress disorders in 2981 participants from the Netherlands Study of Depression and Anxiety (NESDA). Squares represent observed variables and ovals latent variables. Single-headed arrow path coefficients represent standardized partial regression coefficients. Two-headed arrows represent correlation coefficients. Loadings, standardized estimates, or path coefficients, with a theoretical range from zero (no effect) to +1 (maximum positive or negative effect) are provided. MDD = Major Depressive Disorder; DYS = Dysthymia; GAD = Generalized Anxiety Disorder; SAD = Social Anxiety Disorder; PAN = Panic Disorder with or without Agoraphobia; AGO = Agoraphobia w/o panic. T0 = baseline; T2 = 2-yr follow-up; T4 = 4-yr follow-up. All parameter estimates were significant at p < .001, except the association of Fear T0 – Distress T4 (p = .70), GAD T0 – GAD T4 (p = .13) and DYS T0 – DYS T4 (p < .05).
ruminations or T2 worry ($\beta = -.06$, $p = .63$) into the model (figures not shown). Subsequent bootstrapping indicated a significant indirect effect of distress at T0 through rumination at T2 on fear at T4 ($\beta$ indirect effect = .03; 99% BCI = .01 - .06). The indirect effect through worry at T2, however, was not significant ($\beta$ indirect effect = .03; 95% BCI = -.01 - .06).

**Discussion**

Our findings indicate that 1) repetitive negative thinking in the form of rumination and worry partly accounts for the concurrence of distress and fear disorders; 2) baseline fear disorders predicted changes in distress disorders and vice versa; and 3) changes in rumination mediated these longitudinal associations, and changes in worry mediated the fear $\rightarrow$ distress association but not the distress $\rightarrow$ fear association.

Our baseline findings showed that worry and rumination scores were elevated in both the fear and distress group when compared to participants with no current emotional disorder. Moreover, participants with distress disorders obtained higher scores than participants with fear disorders. This is not surprising as the disorders typified by the presence of worry (GAD) and rumination (MDD) were both represented in the distress disorder group. Critically, worry and rumination levels were particularly high in those with comorbid fear and distress diagnoses. This finding is in line with results from the McEvoy et al. (2013) study and supports the transdiagnostic hypothesis, which assumes that higher levels of RNT are associated with higher levels of comorbidity.

An additional step in exploring whether worry and rumination are transdiagnostic factors was to determine whether both cognitive processes explained the cross-sectional and longitudinal associations between fear and distress disorders. Cross-sectional results revealed that worry and rumination partly mediated the co-occurrence of fear and distress disorders. Although promising, this does not shed light on the direction of the relationship and whether changes in worry or rumination mediate the longitudinal relationship between fear and distress disorders and vice versa. Our longitudinal data provided more insight into this question. Worry and rumination both significantly mediated the longitudinal association between baseline (T0) fear disorders and later (T4) distress disorders. Importantly, the three time-point set up of our study allowed for the mediator to reflect change in worry/rumination scores. So, fear disorders (T0) predicted change (T0-T2) in worry/rumination scores, and this change in worry/rumination scores subsequently predicted changes in distress disorders (T4). These mediation results suggest that the increased risk of a future distress disorder when suffering from a fear disorder is partly attributable to worry and rumination. Partly attributable, as the mediation effects are small. The same applies for the longitudinal distress (T0) $\rightarrow$ fear (T4) association with the exception that only rumination significantly mediated this association and worry did not.

The results from the present study suggest that RNT in the form of worry and rumination is indeed involved in the high comorbidity between fear and distress disorders.
This support for the transdiagnostic account suggests that transdiagnostic treatments could benefit from including interventions aimed at reducing RNT. With regards to treatments specifically targeting worry and rumination, several options are available. Worry has received most attention, probably because it is the key feature of GAD. The most well-known treatment targeting worry is metacognitive therapy (Wells, 1999), which has proven to be reasonably successful (e.g. Wells & King, 2006; Van der Heiden, Muris, Van der Molen, 2012). Rumination on the other hand has only recently started to receive attention as a specific element that needs to be targeted in therapy. Results from rumination focussed cognitive behavioural therapy (Watkins et al., 2007) are promising, and in line with the transdiagnostic hypothesis, also reduced the prevalence of comorbid disorders (Watkins et al., 2011). These positive therapeutic results combined with findings on the role of worry and ruminations in the onset, maintenance and recurrence of emotional disorders indicate that RNT is an important candidate transdiagnostic process, which should be seriously considered when developing transdiagnostic therapies for emotional disorders. As yet it is still unclear where transdiagnostic therapies fit within the present therapeutic landscape and how they relate to the popular evidence based disorder specific interventions. There are several options: they could (i) replace disorder specific therapies, (ii) be used as a pre-therapy preceding the ‘real’ disorder focussed therapy, or (iii) function as the main therapy followed by a few sessions targeting disorder specific elements. Currently, the overall consensus seems to be that they should be complementary as they are very unlikely to outperform disorder specific, tailored, interventions (Clark, 2009).

The present study has several strengths. It has a longitudinal design and a representative sample of participants with depressive and/or anxiety disorder(s) from different recruitment settings. Furthermore, structured diagnostic interviews are used to assess presence of depressive and anxiety disorders instead of using only self-report measures of psychopathology.

Our study also has a few limitations that should be kept in mind when interpreting the results. Most importantly, mediation effects are relatively small. This is in line with findings from the McLaughlin and Nolen-Hoeksema (2011) study where the indirect effect was of a comparable small magnitude. Thus although RNT seems to be a factor involved in explaining comorbidity, it only accounts for a part of it. These results therefore warrant the search for additional factors influencing the occurrence of comorbidity. Secondly, we used a fear-distress measurement model to categorize the disorders as this model showed the best fit to repeated CIDI diagnoses in NESDA, but a DSM-IV based anxiety-depression measurement model might have yielded different results due to the high comorbidity between GAD and MDD (now both subsumed under distress disorders). A third limitation concerns the limited number of clinical diagnoses included in the present study. Emotional disorders such as PTSD and OCD were not represented, neither were other disorders known for elevated levels of RNT among which pain disorders, eating disorders and hypochondria (for an overview of disorders with elevated RNT levels see Ehring & Watkins, 2008). 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. Also, we did not measure other candidate transdiagnostic factors like perfectionism (for an overview see Egan, Wade, & Shafran, 2011) and intolerance of uncertainty (e.g. Mahoney & McEvoy, 2012).

Overall, the findings from the present study suggest that repetitive negative thinking in the form of rumination or worry constitutes an important transdiagnostic factor responsible for the co-occurrence of emotional disorders. In transdiagnostic treatment interventions for emotional disorders it seems warranted to include interventions specifically targeting this transdiagnostic factor.

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