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ORIGINAL ARTICLE

Cognitive Moderation of CBT: Disorder-Specific or Transdiagnostic Predictors of Treatment Response

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Abstract

Cognitive vulnerability research has focused on cognitive variables that are hypothesized to confer risk to specific disorders within the mood and anxiety spectrum, while transdiagnostic research has emphasized common risk factors across disorders. The purpose of the present study was to test specific versus common cognitive predictors of treatment response across three treatment groups. Participants (N = 373) with major depressive disorder (MDD; N = 187, panic disorder with/without agoraphobia (PD/A; N = 85), and obsessive compulsive disorder (OCD; N = 101) completed measures of cognitive vulnerability (performance-oriented dysfunctional attitudes, anxiety sensitivity, and obsessive beliefs) and disorder-specific symptom measures at pre- and post CBT treatment. Based on latent difference score analysis, pre-treatment performance-oriented dysfunctional attitudes alone predicted improvement in depressive symptoms in the MDD group; pre-treatment anxiety sensitivity alone predicted reductions in anxious arousal symptoms in the PD/A group; and pre-treatment obsessive beliefs alone predicted change in OCD symptoms in the OCD group. These findings provide support for disorder-specific cognitive factors in the prediction of CBT treatment outcomes and provide guidance towards ways in which current CBT approaches may benefit from augmentation or adjustment.

Keywords Anxiety-sensitivity · Obsessive beliefs · Dysfunctional attitudes · Depression · Panic disorder · Obsessive–compulsive disorder · Cognitive behavioural therapy

Cognitive behavioural therapy (CBT) is a first-line psychotherapeutic intervention for the range of mood, anxiety, and obsessive–compulsive disorder (OCD) conditions. While firmly established as effective treatments, aspects of the taxonomical models of disorders on which traditional CBT approaches are based remain equivocal. Traditional CBT is based on the premise that different forms of psychopathology are defined primarily by their distinct cognitive risk factors. For example, Beck's cognitive content specificity hypothesis proposes that each mood and anxiety disorder has its own unique underlying cognitive vulnerability factors that lead to the onset and/or maintenance of symptoms (Beck et al. 1987). Initially, Beck described the specific types of beliefs and distortions that are characteristic of depression (Beck 1967). Subsequently, cognitive content was described for all of the mood and anxiety disorders (Beck et al. 1987; Beck and Perkins 2001; Clark et al. 1989). Of the broad range of cognitive vulnerability factors examined within programmatic CBT research, some of the most well-studied variables are dysfunctional attitudes in depression; anxiety sensitivity (AS) in panic disorder; and obsessive beliefs in OCD. Based on the cognitive content specificity model, one might expect these cognitive variables to relate to treatment response within their respective diagnostic groups but less so to other disorders that they have not been hypothesized to be central.

Beck outlined that negative automatic thoughts leading to the maintenance of depressive cycles are influenced by underlying dysfunctional attitudes, particularly attitudes regarding performance and interpersonal dependence (Beck 1983). In clinical and nonclinical populations, the presence of dysfunctional attitudes have been found to be predictive of

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depressive symptoms and/or episodes prospectively (Dunkley et al. 2006; Iacoviello et al. 2006; Zuroff et al. 1990). Dysfunctional attitudes have been found to predict daily depressive symptoms (Hankin et al. 2005) as well as the onset of depressive symptoms following life stressors (Abela and D'Alessandro 2002). Dysfunctional attitudes have also been found to moderate treatment effects for major depressive disorder (MDD): individuals with lower severity of dysfunctional attitudes at baseline have been found to have greater treatment response (Hamilton and Dobson 2002; Hawley et al. 2006; Jarrett et al. 1991; Sotsky et al. 1991).

Dysfunctional attitudes have frequently been measured using the Dysfunctional Attitudes Scale. The scale is often demarcated into two subscales, (a) performance-based dysfunctional attitudes, or attitudes of consistent self-criticism towards one's own performance, and (b) need-for-approval dysfunctional attitudes, or attitudes of interpersonal dependency. Of these two subscales, a seminal study found that performance-oriented dysfunctional attitudes were a stronger predictor of treatment outcome compared to need-for-approval attitudes (Blatt et al. 1995). Several subsequent studies have focused on performance-focused attitudes alone, and have found that higher performance-based attitudes at baseline predicted worse treatment outcome (Hawley et al. 2006; Jacobs et al. 2009).

While dysfunctional attitudes have been posited to be a cognitive vulnerability factor in the onset and maintenance of depression, AS was originally described and identified as a vulnerability factor in panic disorder. AS is the fear of the perceived consequences of anxiety (McNally 2002; Reiss 1991; Reiss et al. 1986). In longitudinal studies, higher AS has been found to predict future panic episodes as well as panic disorder onset, and AS is associated with panic symptom severity (Laposa et al. 2015; Li and Zinbarg 2007; Plehn and Peterson 2002; Schmidt et al. 1999). AS has previously been identified as a mediator of treatment outcome in CBT for panic disorder (Smits et al. 2004). However, the role of AS as a moderating variable of CBT outcomes for panic disorder has been less frequently examined. A meta-analysis of predictors of treatment response to CBT for panic disorder with or without agoraphobia (PD/A) found that anxiety sensitivity at baseline did not moderate treatment response (Porter and Chambless 2015). Of note, only four studies in the meta-analysis included AS as a predictor variable. In addition to being studied as a general factor, AS has been conceptualized as having three subfactors: fear of the physical, social, and cognitive consequences of anxiety. Panic disorder has been frequently associated *specifically* with fear of the physical consequences of anxiety (Kemper et al. 2012; Rector et al. 2007; Taylor et al. 2007). In summary, there appears to be less support for the specificity of AS in moderating outcomes to CBT for PD/A, although few studies have examined this question. No study has tested whether the fear of physical symptoms dimension of AS is specifically related to clinical outcomes.

Obsessive beliefs have been hypothesized to confer specific vulnerability to the onset and maintenance of OCD (Obsessive Compulsive Cognitions Working Group [OCCWG] 1997, 2001, 2003, 2005). Cognitive accounts of OCD posit that maladaptive appraisals of intrusive thoughts lead to increased distress and a greater likelihood of compulsive behaviours (OCCWG 1997, 2001, 2003; Rachman 1997, 1998; Salkovskis 1985). These maladaptive appraisals are informed by obsessive beliefs. Obsessive beliefs are associated with obsessive compulsive (O–C) symptoms cross-sectionally in clinical populations (e.g., Fergus and Carmin 2014; OCCWG 2003, 2005; Sica et al. 2004; Wheaton et al. 2010). In nonclinical populations, obsessive beliefs have been found to predict O–C symptom levels prospectively over several weeks or months (Abramowitz et al. 2006, 2007; Coles and Horng 2006). Within the context of CBT interventions, domains of obsessive beliefs at baseline have been found to moderate CBT outcome and predict treatment adherence (Adams et al. 2012; Dowling et al. 2016; Kyrios et al. 2015; for alternate findings see McLean et al. 2001).

Although the study of dysfunctional attitudes, AS, and obsessive beliefs originated within theoretical CBT models to account for the maintenance of specific disorders, recent research suggests that these cognitive variables may not be disorder-specific, and rather relate to a range of psychiatric conditions. For example, dysfunctional attitudes have been found to be equivalent in individuals presenting with anxiety or depressive disorders (Hill et al. 1989) and change in these dysfunctional attitudes have been found to predict treatment outcome in social anxiety disorder (Nishikawa et al. 2017). Similarly, heightened AS has been observed empirically across depression, anxiety and OCD (Laposa et al. 2015; Naragon-Gainey 2010). Obsessive beliefs have been associated with symptoms of disorders other than OCD, such as social anxiety or depression in nonclinical populations (Tolin et al. 2003), and in individuals meeting criteria for an anxiety disorder diagnosis (Tolin et al. 2006) and generalized anxiety disorder, in particular (Viar et al. 2011). These results are consistent with an emerging literature in which multiple psychological factors have been theorized to be transdiagnostic in nature, contributing to psychopathology across multiple disorders rather than one specific diagnosis (e.g., Baer 2007; Carleton 2016; Ehring and Watkins 2008; Ingram 1990).

The questions of specificity within cognitive vulnerability factors might extend to their ability to moderate treatment response: is there content specificity in the moderation of treatment response or do the same cognitive moderators predict treatment response across multiple disorders? While studies have examined the specific moderation effects of

dysfunctional attitudes, anxiety sensitivity, and obsessive beliefs within depression, panic disorder, and OCD respectively, fewer studies have explored the specificity of these types of cognitive content in moderating CBT treatment outcomes *across* disorders. As one example, recent research has found that AS can moderate response to CBT for OCD, either alone (Blakey et al. 2017) or in interaction with obsessive beliefs (Katz et al. 2018). To our knowledge, no published research has examined these key disorder-specific cognitive predictors across multiple conditions to determine whether the moderating influences of cognitive risk factors are disorder-specific or transdiagnostic in nature.

The purpose of this study was to examine the specificity of moderation effects of cognitive content. Dysfunctional attitudes, AS, and obsessive beliefs were selected as key moderator variables based on their theoretical relevance and previous empirical support. Each cognitive variable was explored as a moderator response to group CBT for the disorder with which it was originally identified as a specific risk factor and treatment target within CBT. Two competing hypotheses were explored: (1) based on the content specificity hypothesis, dysfunctional attitudes alone would moderate treatment response for depression, anxiety sensitivity alone would moderate treatment response for panic disorder, and obsessive beliefs alone would moderate treatment response for OCD, (2) based on the transdiagnostic hypothesis, all three cognitive variables would moderate CBT response across the three disorders.

Methods

Participants

The participants were 373 adults ($age_m = 36.19$, $SD = 11.10$)[1] who presented to a large mood, anxiety and OCD university-affiliated outpatient assessment and treatment clinic. The participant sample was 52.3% female (7.5% missing). All participants had a psychiatric consultation, and received a primary diagnosis of MDD (N = 187), PD/A (N = 85), or OCD (N = 101) based on diagnoses made in a psychiatric consult with an expert clinician. Inclusion criteria consisted of a primary diagnosis of OCD, MDD, or PD/A and being between the ages of 18 and 65. Exclusion criteria consisted of: (1) active psychosis or bipolar disorder; (2) substance use that was deemed to be at a level that would interfere with treatment; (3) recent adequate course of CBT for the disorder in question; and (4) recent suicide attempt/active suicidality, or parasuicidal behaviours. Of the participant sample, 66.2% identified as White, 8.8% identified as Asian, 5.4% identified as Black, 10.5% identified as an ethnicity other than those already mentioned, and ethnicity information was not available for 9.1%. Of the 91.7% of participants for whom marital status information was reported, 55.6% were single, 31.6% were married or co-habiting, 12.6% were separated or divorced, and 0.3% were widowed. Of the 92.0% of participants for whom education level was reported, 94.2% completed high school, 67.3% reported that they completed college or university, and 15.7% reported that they completed graduate school.

Measures

Measures of Cognitive Factors

Anxiety Sensitivity Index-3 (ASI-3; Taylor et al. 2007)

The ASI-3 is an 18-item measure of AS. It is composed of three scales: (1) physical concerns (ASI-P), or the fear the physical consequences of anxiety; (2) cognitive dyscontrol (ASI-C), or the fear a loss of cognitive control due to anxiety; and (3) social concerns (ASI-S), or the fear of potential negative social consequences of anxiety. Participants indicate their agreement with each item on a 5-point Likert scale. The ASI-3 demonstrates strong psychometric properties (Taylor et al. 2007). In the current sample, the internal consistency estimates of the ASI-P, ASI-C, and ASI-S were $\alpha = .89$, $\alpha = .91$, and $\alpha = .82$, respectively. Internal consistency of the total ASI-3 was $\alpha = .92$.

Dysfunctional Attitudes Scale—Perfectionism Subscale (DAS-Pft; Weissman and Beck 1978)

The DAS is a 40-item measure of attitudes thought to be related to depression based on cognitive behavioural theory. Participants indicate on a 7-point Likert scale the degree to which they agree with each item. Although several different factor structures have been suggested, one frequently cited factor structure divides items into two subfactors: perfectionism (which can also be called performance-oriented dysfunctional attitudes) and need for approval by others (Imber et al. 1990). Based on prior research finding that the only the DAS-Pft subscale was a significant moderator of CBT for MDD (Blatt et al. 1995), only the DAS-Pft was used in this study. The DAS-Pft consists of 15 items that measure negative, self-evaluative attitudes about performance. The DAS demonstrates strong psychometric properties (Dobson

[1] An analysis of variance found that age significantly differed according to diagnostic groups, $F(2, 326) = 14.63$ $p < .001$. Post-hoc comparisons using Tukey's HSD test indicated that the mean age for the OCD group was significantly lower than for the PD/A or MDD group, $p < .05$. A Chi-Square test indicated that the diagnostic groups did not differ according to gender, $\chi^2 (2) = 2.57$, $p = 0.28$.

and Breiter 1983; Nelson et al. 1992). In the current sample, internal consistency for the DAS-Pft was α = 0.90.

Obsessive Beliefs Questionnaire-44 (OBQ-44; OCCWG 2001, 2003, 2005)

The OBQ-44 is a 44-item measure of beliefs related to maladaptive appraisals in OCD. The OBQ-44 has demonstrated strong psychometric properties (OCCWG 2005). The current study made use of the total score of the OBQ-44. In the current sample, internal consistency for the total OBQ-44 was α = .96.

Symptom Severity Measures

Quick Inventory of Depressive Symptoms (QIDS; Rush et al. 2003)

The QIDS is a 16-item measure of depression symptoms. Responses to each item are made on a 4-point Likert scale. The QIDS demonstrates good psychometric properties (Rush et al. 2003). Information on internal consistency from the current sample was not available, though previous research has indicated good consistency for the QIDS (Rush et al. 2003).

Yale-Brown Obsessive–Compulsive Scale (YBOCS; Goodman et al. 1989a, b)

The YBOCS is a frequently used measure of O-C symptom severity and impairment. The YBOCS has strong psychometric properties (Goodman et al. 1989a, b). In the current sample, internal consistency was α = .82 in the OCD sample.

Depression Anxiety Stress Scales—Anxiety Scale- 21 (DASS-A; Lovibond and Lovibond 1995)

The DASS-A includes 7 items that primarily measure physical symptoms associated with anxiety (Antony et al. 1998). Participants indicate on a 4-point Likert scale the amount to which each statement applies to them. Although not designed as a measure of panic symptom severity, prior psychometric research found that the DASS-A was significantly higher among individuals with panic disorder compared to those with other mood or anxiety disorders (Antony et al. 1998), which is consistent with the particular focus on physical symptoms. Examples of items in the anxiety subscale include "I felt I was close to panic", "I was aware of the action of my heart in the absence of physical exertion (e.g. sense of heart rate increase, heart missing a beat)" and "I experienced breathing difficulty e.g. excessively rapid breathing, breathlessness in the absence of physical exertion". The DASS has strong psychometric properties (Antony et al. 1998). Internal consistency was not available for the current sample but past research has found strong internal consistency for the DASS-A (α = .87; Antony et al. 1998).

Panic Disorder Severity Scale—Self-Report (PDSS-SR; Houck et al. 2002; Shear et al. 1997)

The PDSS-SR is a 7-item measure of the panic disorder symptom severity. Participants respond to items on a 4-point Likert scale. The PDSS-SR has strong psychometric properties (Houck et al. 2002). In the current PD/A sample, internal consistency was α = 0.90.

Procedures

Informed consent was obtained from all participants. Participants were enrolled in cognitive behavioural group therapy (CBGT) for their primary diagnosis (PD/A, OCD, or MDD). CBGT for MDD consisted of 14 weekly sessions, while CBGT for PD/A and OCD each consisted of 12 weekly sessions. All groups met for 2 h/week, and typically consisted of 8–10 participants per group. All treatments were based on well-validated CBT manuals (Abramowitz 2009; Craske and Barlow 2006; Greenberger and Padesky 1995; Wilhelm and Steketee 2006). Treatment was facilitated by psychologists and other members of allied mental health. Treatment for depression included behavioural activation and cognitive restructuring. Treatment for panic disorder included interoceptive exposure, in-vivo exposure, and cognitive restructuring. Treatment for OCD included exposure and response prevention as well as cognitive techniques to reduce unhelpful appraisals. Participants completed all three measures of cognitive factors (ASI-3, OBQ-44, and DAS) at pre-treatment and post-treatment. Participants also completed the symptom severity measure that corresponded with their primary diagnosis at pre-treatment and post-treatment (the QIDS in the MDD group, the DASS-A and PDSS-SR in the PD/A group, and the Y-BOCS in the OCD group). In the case of the PD/A group, two outcome measures were used: the PDSS-SR and the DASS-A. These two measures were chosen in order to capture two different ways of measuring outcome for PD/A. The PDSS-SR primarily measures frequency and severity of panic attacks as well as change in behaviour and functioning due to attacks. While these items capture primary symptoms of PD/A, it has been suggested that changes in more general symptoms of anxiety are also significant predictors of impairment in panic disorder and therefore outcome (Michelson et al. 1998). Thus, the DASS-A was added as a broader measure of anxiety symptoms in individuals with panic disorder.

Data Analysis

A form of Structural Equation Modelling (SEM) was used, termed "Latent Difference Score" analysis (LDS; see McArdle 2001; McArdle and Hamagami 2001). LDS models integrate features of latent growth curve models (Meredith and Tisak 1990) and cross-lagged regression models (Jöreskog and Sörbom 1979). LDS analysis considers longitudinal growth within a time series while also examining multivariate relationships and determinants. Within each longitudinal series, the latent rate of change is used as the outcome variable. There are several steps involved with any LDS analysis. First, longitudinal measurement invariance was evaluated.[2] Next, a univariate model was established, examining how each variable changes before and after CBT treatment (Hamagami and McArdle 2001; McArdle 2001; McArdle and Hamagami 2001; McArdle and Nesselroade 2002). Although there are several ways to model longitudinal change over time, since each of our time series involve only two time points, we used a "Proportional Change Model" in which latent change is proportional to the latent score from the previous time point.[3] Next, we examined temporal relationships between series by considering cross-lagged or *coupling* regressions. Bivariate coupling occurs if two univariate processes demonstrate a temporal relationship in which one univariate process predicts the subsequent rate of change in the other. Although there are several possible bivariate LDS analyses (see Hamagami and McArdle 2001) we were interested in examining a "reciprocal" model to determine whether pre-treatment symptoms (i.e., QIDS, YBOCS, DASS-A, PDSS-SR) predict subsequent changes in a cognitive variable (e.g., DAS-Pft, OBQ, ASI), and/or if the reverse was true i.e., whether pre-treatment DAS-Pft, OBQ, ASI predict subsequent changes in symptom measures (i.e., QIDS, YBOCS, DASS-A, PDSS-SR) for each diagnostic population. All path model equations can be found in the Appendix 1.

The AMOS 20.0 program (Arbuckle 2011) was used to evaluate all LDS models. Maximum likelihood estimation was used. AMOS provides a variety of measures for assessing absolute and relative model fit. The Chi square index is considered a measure of absolute model fit, and a heuristic is typically used in which Chi square to degrees of freedom ratios (χ^2/df) near two represent acceptable model fit (Byrne 2004). The root mean square error of approximation is provided as a measure of absolute model fit (RMSEA; Steiger and Lind 1980). RMSEA indicates "model discrepancy per degree of freedom," with values less than .05 indicating a "close fit," whereas RMSEA values larger than .10 suggest a "poor fit" (Browne and Cudeck 1993). Further, we consider the *p*-value for testing the null hypothesis that the population RMSEA is no greater than 0.05 (MacCallum et al. 1996), reported as "*p* close fit." The Comparative Fit Index (CFI) indicates the relative reduction in model misfit when comparing the target model relative to a baseline (independence) model. CFI values greater than .90 indicate a good fit of the model to the observed data (CFI; Bentler 1990). Further, the relative fit of competing models is compared using the Akaike Information Criterion (AIC; Akaike 1973), which considers model complexity in relationship to the number of parameters. The model with smaller AIC is preferred. Finally, certain key parameter estimates are considered, although they are not measures of overall model fit. To evaluate the theoretical cogency of competing models, the bivariate LDS models can be discriminated based on whether the coupling parameter (γ) is significant. If the coupling is not significant, the model postulating that effect may not be supported.

Results

Tables 1, 2 and 3 present means, standard deviations, and correlations among study variables at pre-treatment, and post-treatment, for each diagnostic population. Notably, each of the symptom measures (i.e., QIDS, YBOCS, DASS-A) decreased over time. As expected, measures from consecutive assessments were positively correlated for each measure over time (see Tables 1, 2, 3). Of note, while each symptom measure was significantly correlated at baseline with its corresponding "disorder-specific" cognitive vulnerability measure, the cognitive vulnerability measures did not account for all symptom variance. Furthermore, the QIDS was significantly correlated with all three cognitive vulnerability measures at T1 and T2, the DASS-A was also significantly correlated with DAS-Pft at T2, and the YBOCS was significantly correlated with ASI at T2.

Using Bonferroni-adjusted t-tests, all cognitive variables decreased significantly within all groups, all p's < 0.05, except for DAS-Pft in the OCD group, $t\,(66) = 1.65$, $p = .10$.

First, we established an LDS univariate proportional change model for each symptom variable (i.e., QIDS, YBOCS, DASS-A) and each cognitive variable (i.e., DAS-Pft, OBQ, ASI) separately, comparing two time points (i.e.,

[2] Each variable was evaluated for longitudinal measurement invariance. A confirmatory factor analysis (CFA) was first conducted for each measure, and all items were retained. Measurement invariance was evaluated before proceeding with the LDS analysis, testing the null hypothesis of weak (i.e., equal factor loadings over time) and strong (i.e., equal measurement intercepts over time) longitudinal measurement invariance.

[3] Proportional Change Model: Latent change is proportional to the latent score from the previous time point. $E[\Delta \text{Variable}(t)_n] = \beta \text{ x } E[\text{Variable } (t-1)_n]$; $\alpha_s \text{ x } E[s_{s,n}] = 0$.

Table 1 MDD sample: correlations, means and standard deviations for study measures

Variable	1	2	3	4	5	6	7	8
1. $QIDS_{t1}$	1.00	–	–	–	–	–	–	–
2. $QIDS_{t2}$	.41**	1.00	–	–	–	–	–	–
3. DAS-Pft_{t1}	.40**	.27**	1.00	–	–	–	–	–
4. DAS-Pft_{t2}	.31**	.42**	.58*	1.00	–	–	–	–
5. OBQ_{t1}	.40**	.38**	.69**	.42**	1.00	–	–	–
6. OBQ_{t2}	.07	.48**	.17	.78**	.65**	1.00	–	–
7. ASI_{t1}	.39**	.07	.45**	.31**	.54**	.35**	1.00	–
8. ASI_{t2}	.18	. 34**	.34**	.64**	.41**	.65**	.59**	1.00
M	14.59	9.78	4.11	3.43	175.04	156.10	32.33	24.37
SD	4.65	5.39	1.64	1.14	47.18	50.07	15.71	15.13

QIDS = Quick Inventory of Depressive Symptoms; DAS-Pft = Dysfunctional Attitudes Scale, Perfectionism Subscale; OBQ = Obsessive Beliefs Questionnaire, Total Score; ASI = Anxiety Sensitivity Inventory, Total Score; t1 = CBT pre-treatment; t2 = CBT post-treatment; M = Mean; SD = Standard deviation

*$p < .05$

**$p < .01$

Table 2 OCD sample: correlations, means and standard deviations for study measures

Variable	1	2	3	4	5	6	7	8
1. $YBOCS_{t1}$	1.00	–	–	–	–	–	–	–
2. $YBOCS_{t2}$	.38**	1.00	–	–	–	–	–	–
3. DAS-Pft_{t1}	.11	.01	1.00	–	–	–	–	–
4. DAS-Pft_{t2}	.06	.09	.67*	1.00	–	–	–	–
5. OBQ_{t1}	.33**	.28*	.03	.43**	1.00	–	–	–
6. OBQ_{t2}	.41**	.40*	.47**	.15	.61**	1.00	–	–
7. ASI_{t1}	.16	.11	.55**	.27*	.54**	.40**	1.00	–
8. ASI_{t2}	.08	. 24*	.01	.48**	.35*	.51**	.69**	1.00
M	23.63	13.99	3.68	3.43	14.07	12.21	30.24	23.20
SD	5.59	6.08	1.21	1.32	4.01	3.24	15.58	14.26

YBOCS = Yale-Brown Obsessive Compulsive Disorder Scale; DAS-Pft = Dysfunctional Attitudes Scale, Perfectionism Subscale; OBQ = Obsessive Beliefs Questionnaire, Total Score, Square Root Transformed; ASI = Anxiety Sensitivity Inventory, Total Score; t1 = CBT pre-treatment; t2 = CBT post-treatment; M = Mean; SD = Standard deviation

*$p < .05$

**$p < .01$

pre-treatment and post-treatment). Considering parameter significance and goodness of fit indices, each proportional change model provided an adequate fit, and all parameter estimates were statistically significant ($p < .05$) (see Table 4).

When examining the PD/A sample using PDSS-SR as the symptom measure and ASI-Total as the cognitive variable, the model did not converge. We next examined the ASI-P subfactor, which has demonstrated predictive validity in past research (Nowakowski et al. 2016) and has been more closely linked with PD/A (Kemper et al. 2012; Rector et al. 2007; Taylor et al. 2007; Zinbarg et al. 1997). Still, two of the three models did not converge and the third model did not achieve adequate fit, rendering all three models uninterpretable when PDSS-SR was used as the symptom measure. Using our alternate symptom measure (DASS-A) and reverting back to the ASI total score as the cognitive measure, the model again did not converge. However, the model did converge when ASI-P was used as the cognitive measure and the indices of fit of the resulting model were adequate.

Summary results for the bivariate (i.e., utilizing two longitudinal variables) "Reciprocal Model" analyses are presented in Table 5 (MDD sample), 6 (OCD sample) and 7 (PD/A sample). In each case, we examined reciprocal models involving each symptom variable (i.e., Series 1: QIDS, YBOCS, DASS-A) and each cognitive variable (i.e., Series 2: DAS-Pft, OBQ, ASI-P) (Fig. 4 in Appendix 2).

Table 5 presents the findings for the MDD, OCD, and PD/A sample. In the MDD sample, when examining the

Table 3 PD sample: correlations, means and standard deviations for study measures

Variable	1	2	3	4	5	6	7	8
1. DASS-A_{t1}	1.00	–	–	–	–	–	–	–
2. DASS-A_{t2}	.59**	1.00	–	–	–	–	–	–
3. DAS-Pft_{t1}	.21	.18	1.00	–	–	–	–	–
4. DAS-Pft_{t2}	.33**	.45**	.66**	1.00	–	–	–	–
5. OBQ_{t1}	.17	.49*	.67**	.70**	1.00	–	–	–
6. OBQ_{t2}	.43*	.29	.33*	.65**	.66**	1.00	–	–
7. ASI-P_{t1}	.35*	.32*	−.11	.08	.17	.10	1.00	–
8. ASI-P_{t2}	.40*	.69**	−.11	.19	.29	.30	.48**	1.00
M	11.51	7.55	3.36	2.75	171.48	131.14	12.80	8.44
SD	6.12	6.28	1.13	1.18	52.79	50.23	6.49	5.86

DASS-A = Depression Anxiety Stress Scales, Anxiety Subscale; DAS-Pft = Dysfunctional Attitudes Scale, Perfectionism Subscale; OBQ = Obsessive Beliefs Questionnaire, Total Score; ASI = Anxiety Sensitivity Inventory, Physical Subscale; t1 = CBT pre-treatment; t2 = CBT post-treatment; M = Mean; SD = Standard deviation

*$p < .05$

**$p < .01$

Table 4 Fit indices of proportional change models

Diagnostic Group	Measure	χ^2	χ^2/df	AIC	CFI	RMSEA
MDD	QIDS	(1, $N = 187$) = 0.34	0.34	8.34	.99	.01
	DAS-Pft	(1, $N = 187$) = 0.25	0.25	10.16	.98	.01
	ASI	(1, $N = 187$) = 0.96	0.96	3.96	.99	.02
	OBQ	(1, $N = 187$) = 0.26	0.26	4.61	.99	.01
PD/A	DASS-A	(1, $N = 85$) = 0.36	0.36	8.03	.95	.01
	DAS-Pft	(1, $N = 85$) = 1.43	1.43	26.18	.94	.02
	ASI	(1, $N = 85$) = 1.96	1.96	13.62	.93	.04
	OBQ	(1, $N = 85$) = 1.45	1.45	9.57	.97	.05
OCD	YBOCS	(1, $N = 101$) = 0.26	0.26	4.61	.99	.04
	DAS-Pft	(1, $N = 101$) = 1.41	1.41	9.41	.95	.02
	ASI	(1, $N = 101$) = 0.95	1.95	14.63	.89	.01
	OBQ	(1, $N = 101$) = 0.50	0.50	8.50	.98	.04

QIDS = Quick Inventory of Depression Symptoms; DAS-Pft = Dysfunctional Attitudes Scale, Perfectionism subscale; ASI-P = Anxiety Sensitivity Inventory, Physical subscale; OBQ = Obsessive Beliefs Questionnaire; DASS-A = Depression Anxiety and Stress Scales, Anxiety Scale; YBOCS = Yale-Brown Obsessive Compulsive Scale

three reciprocal models involving the relationship between QIDS symptom change and changes in DAS-Pft, OBQ, or ASI (total score), the overall goodness of fit and parameter estimates were adequate, which allows for further interpretation of the results of these models. Considering the relationship between QIDS and DAS-Pft, the coupling coefficient (γ) in which baseline QIDS predicts subsequent change in DAS-Pft (γ) was significant, with the unstandardized estimate being γ = .12, and the coupling coefficient in which baseline DAS-Pft predicts subsequent change in QIDS scores was significant, with the unstandardized estimate being γ = 5.03. Therefore, for every 1 unit increase in DAS-Pft at time *t*, there is an additional 5.03 unit increase in QIDS from time *t* to time *t* + *1*. All remaining parameter estimates were statistically significant (all *p*s < .05). For the reciprocal coupling model involving QIDS and OBQ, the coupling coefficients were non-significant. Further, the coupling coefficients were also non-significant for the reciprocal coupling model involving QIDS and ASI.

For the reciprocal model involving QIDS and DAS-Pft, the magnitude of these coefficients can be interpreted as following: for every one unit increase in QIDS at time *t*, there is a subsequent additional .12 unit increase in DAS-Pft between time *t* and time *t* + *1*. Further, for every one unit increase in DAS-Pft at time *t*, there is a subsequent additional 5.03 unit increase in QIDS between time *t* and time *t* + *1*. Figure 1 illustrates how DAS-Pft and QIDS relate, based on varying levels of initial DAS-Pft scores (i.e., using

Table 5 Parameters and fit indices involving symptom change as related to cognitive variables (DAS, ASI, OBQ)

MDD sample		QIDS	DAS-Pft	QIDS	ASI	QIDS	OBQ
	Proportional coefficient β	−1.75*	−0.51*	−0.27*	0.39*	0.96*	−0.45*
	Cross-lag coefficient γ	0.12*	5.03*	−0.21^{ns}	−0.23^{ns}	0.35^{ns}	−1.41^{ns}
	Parameters	12		12		12	
	Degrees of freedom	2		2		2	
	RMSEA (p close fit)	.02 (.48)		.05 (.16)		.04 (.21)	
	CFI	.99		.94		.95	
	AIC	26.14		38.32		28.78	
	χ^2	2.14		3.67		4.78	
	χ^2/df	1.07		1.83		2.39	
OCD sample		YBOCS	DAS-Pft	YBOCS	ASI	YBOCS	OBQ
	Proportional coefficient β	−0.49*	−0.15*	−0.22*	−0.51*	−0.52*	−0.13*
	Cross-lag coefficient γ	0.12^{ns}	0.52^{ns}	.07^{ns}	0.11^{ns}	0.01^{ns}	0.37*
	Parameters	12		12		12	
	Degrees of freedom	5		5		5	
	RMSEA (p close fit)	.01 (.61)		.05(.43)		.04(.29)	
	CFI	.99		.97		.89	
	AIC	22.53		24.12		27.84	
	χ^2	4.53		6.12		9.28	
	χ^2/df	.91		1.22		1.86	
PD/A sample		DASS-A	DAS-Pft	DASS-A	ASI-P	DASS-A	OBQ
	Proportional coefficient β	−0.37*	−0.18*	−0.39*	−0.24*	−0.52*	−0.23*
	Cross-lag coefficient γ	0.18^{ns}	0.16^{ns}	0.07^{ns}	0.35**	0.02^{ns}	0.06^{ns}
	Parameters	10		10		10	
	Degrees of freedom	4		4		4	
	RMSEA (p close fit)	.05 (.24)		.04 (.27)		.04 (.26)	
	CFI	.94		.90		.94	
	AIC	26.19		26.13		26.18	
	χ^2	6.20		6.13		6.18	
	χ^2/df	1.55		1.53		1.55	

0 (=) indicates parameter is not estimated

QIDS = Quick Inventory of Depression Symptoms; YBOCS = Yale-Brown Obsessive Compulsive Scale; DASS-A = Depression Anxiety Stress Scales, Anxiety Scale-21; DAS-Pft = Dysfunctional Attitudes Scale, Perfectionism subscale; ASI = Anxiety Sensitivity Inventory, Total Score; ASI-P = Anxiety Sensitivity Inventory, Physical Concerns; OBQ = Obsessive Beliefs Questionnaire

ns non-significant

*$p < .05$

**$p < .01$

***$p < .001$

the mean score, and scores that are one standard deviation above or below the mean).

In the OCD sample (see Table 5), when examining the three reciprocal models involving the relationship between YBOCS symptom change and changes in DAS-Pft, OBQ, or ASI (total score), the overall goodness of fit and parameter estimates were adequate, which allows for further interpretation of the results of these models. Considering the relationship between YBOCS and DAS-Pft, the coupling coefficients were non-significant. Considering the YBOCS and ASI reciprocal model, the coupling coefficients were non-significant. Considering the relationship between YBOCS and OBQ, the coupling coefficient (γ) in which baseline OBQ predicts subsequent change in YBOCS was significant, with the unstandardized estimate being γ = .37. All remaining parameter estimates were statistically significant (all *ps* < .05).

For the reciprocal model involving YBOCS and OBQ, the magnitude of these coefficients can be interpreted as following: for every one unit increase in OBQ at time *t*, there is a

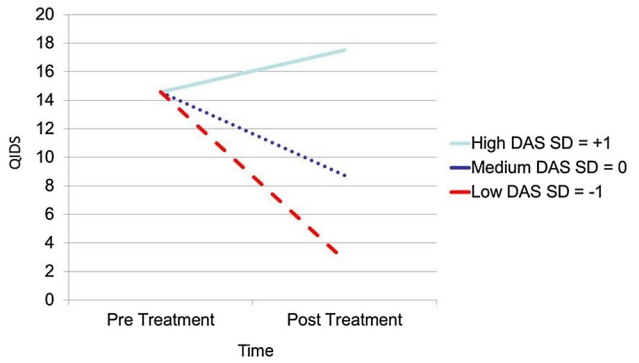


Fig. 1 Estimated change trajectories involving change in QIDS (Quick Inventory of Depression Symptomatology) scores comparing pre-treatment to post treatment based on varying levels of DAS-Pft (Dysfunctional Attitudes Scale, Perfectionism Subscale) for the MDD group. SD = 0 refers to an estimated QIDS trajectory using the initial mean DAS-Pft score for the MDD group. SD = + 1 refers to an estimated QIDS trajectory using an initial DAS-Pft score that is one standard deviation higher than the sample mean. SD = − 1 refers to an estimated QIDS trajectory using an initial DAS-Pft score that is one standard deviation lower than the sample mean

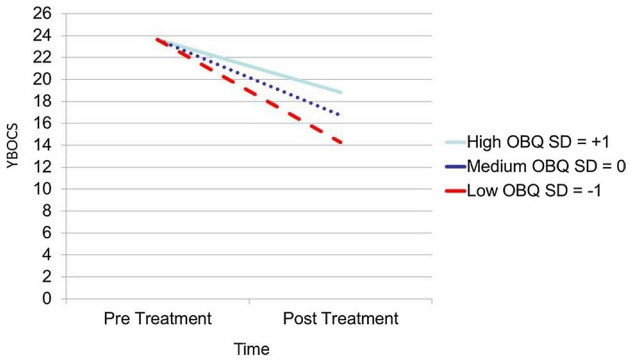


Fig. 2 Estimated change trajectories involving change in YBOCS (Yale-Brown Obsessive Compulsive Scale) scores comparing pre-treatment to post treatment based on varying levels of OBQ (Obsessive Beliefs Questionnaire) for the OCD group. SD = 0 refers to an estimated YBOCS trajectory using the initial mean OBQ score for the OCD group. SD = + 1 refers to an estimated YBOCS trajectory using an initial OBQ score that is one standard deviation higher than the sample mean. SD = − 1 refers to an estimated QIDS trajectory using an initial DAS-P score that is one standard deviation lower than the sample mean

subsequent additional .37 unit increase in YBOCS between time *t* and time *t* + *1*. Figure 2 illustrates how YBOCS and OBQ relate, based on varying levels of initial OBQ scores (i.e., using the mean score, and scores that are one standard deviation above or below the mean).

For the PD/A sample (see Table 5), PDSS-SR was initially used as an outcome measure but two of the three models did not converge and the third model did not achieve adequate fit, rendering all three models uninterpretable. Therefore, the DASS-A was used as a measure of symptom severity. The DASS-A measures physical symptoms of anxiety and has previously been shown to be at significantly higher levels among individuals with panic disorder compared to those with other mood or anxiety disorders (Antony et al. 1998). When examining the three reciprocal models involving the relationship between DASS-A symptom change and changes in DAS-Pft, OBQ, or ASI-P (physical subscale), the overall goodness of fit and parameter estimates were adequate, which allows for further interpretation of the results of these models. Considering the relationship between DASS-A and DAS-Pft, the reciprocal coupling coefficients were non-significant. Considering the relationship between DASS-A and ASI-P, the coupling coefficient (γ) in which baseline ASI-P predicts subsequent change in DASS-A was significant, with the unstandardized estimate being γ = .35. Notably, the coupling coefficient in which baseline DASS-A predicts subsequent change in ASI-P scores was not significant. All remaining parameter estimates were statistically significant (all *ps* < .05). When considering the DASS-A and OBQ reciprocal model, the reciprocal coupling coefficients were non-significant.

For the reciprocal model involving DASS-A and ASI-P, the magnitude of these coefficients can be interpreted as following: for every one unit increase in ASI-P at time *t*, there is a subsequent additional .35 unit increase in DASS-A between time *t* and time *t* + *1*. Figure 3 illustrates how DASS-A and ASI-P relate, based on varying levels of initial ASI-P scores (i.e., using the mean score, and scores that are one standard deviation above or below the mean).

Discussion

The present study examined the cognitive specificity hypothesis by using methods that may offer a stronger test of specificity than has previously been reported. The current analyses increased our understanding of the relationship between cognitive variables and symptom measures by allowing us to clarify degree and specificity of predicted change, as well as the directionality of change.

By extending the cognitive content specificity hypothesis, it could be hypothesized that each disorder would have its own separate cognitive predictors of treatment response. The results of the present study found that MDD, PD/A, and OCD each had a different cognitive moderator of response to CBT. Within the MDD treatment group, performance-oriented dysfunctional attitudes significantly predicted change in depressive symptoms during therapy, such that higher baseline dysfunctional attitudes predicted a poorer outcome over the course of treatment, while interpretation of the distribution of data based on Fig. 1 suggest that lower baseline dysfunctional attitudes predict better outcome. In contrast,

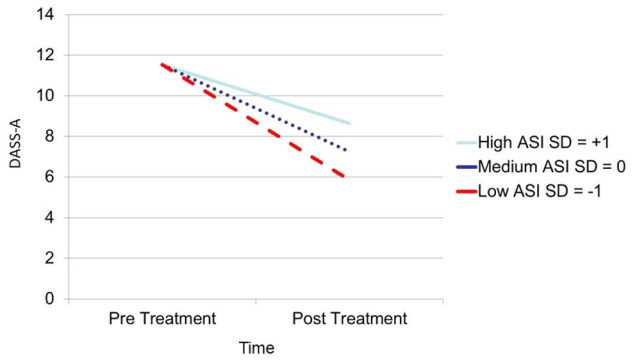


Fig. 3 Estimated change trajectories involving change in DASS-A (Depression Anxiety Stress Scales, Anxiety Subscale) scores comparing pre-treatment to post treatment based on varying levels of ASI-P (Anxiety Sensitivity Inventory, Physical Subscale) for the PD group. SD = 0 refers to an estimated DASS-A trajectory using the initial mean ASI-P score for the PD group. SD = + 1 refers to an estimated DASS-A trajectory using an initial ASI-P score that is one standard deviation higher than the sample mean. SD = -1 refers to an estimated DASS-A trajectory using an initial ASI-P score that is one standard deviation lower than the sample mean

AS and obsessive beliefs at baseline did not predict symptom change over treatment in the MDD group. These results are in line with previous research that found that depressive attitudes predict depressive symptoms longitudinally (Dunkley et al. 2006; Iacoviello et al. 2006; Zuroff et al. 1990) and that high pre-treatment performance-oriented dysfunctional attitudes predict less symptom reduction (Hawley et al. 2006). The reverse relationship was also found to be significant, in that depressive symptoms predicted change in depressive attitudes during treatment. Previous research has also found a bidirectional relationship between cognitive variables and depressive symptoms or onset, particularly in adolescent and young adult samples (Calvete et al. 2013, 2016; Nolen-Hoeksema et al. 2007). A reciprocal relationship may therefore exist, in which depressive attitudes maintain depressive symptoms, while depressive symptoms increase the accessibility of depressive attitudes (Teasdale 1983). While previous research has linked both AS (Naragon-Gainey 2010) and obsessive beliefs (Tolin et al. 2003) to depressive symptoms or MDD, these variables did not predict treatment response to group CBT for depression in the current LDS analysis.

For participants with OCD, obsessive beliefs alone significantly predicted change in O-C symptom severity, such that higher obsessive beliefs at baseline predicted poorer outcome on the Y-BOCS. Interpretation of the distribution of data based on Fig. 2 suggests that lower obsessive beliefs at baseline predicted improved outcome on the Y-BOCS. This result is consistent with past research that demonstrates cross-sectionally (Fergus and Carmin 2014; OCCWG 2003, 2005; Sica et al. 2004; Wheaton et al. 2010), longitudinally (Abramowitz et al. 2006, 2007; Coles and Horng 2006) and within treatment contexts (Dowling et al. 2016; Kyrios et al. 2015), OBQ scores are associated with OCD symptom expression and severity. The lack of moderation by AS is in contrast to the study by Blakey et al. (2017) who found that lower baseline AS predicted better outcome. Of note, participants in the Blakey et al. (2017) study were clients at a residential treatment setting at which they completed on average 28.5 h of exposure and response prevention each week. The treatment was therefore more intensive and it is possible that the participants also had a greater severity of OCD than those in the current sample, as indicated by the need for residential treatment, though average Y-BOCS scores were not reported. It could be that high baseline AS becomes a larger impediment in the treatment of OCD as severity and/or treatment intensity increases.

For participants with PD/A, the findings were somewhat less conclusive, as three sets of models failed to converge before we elected to examine the cognitive measure of ASI-P and symptom measure of DASS-A. Nevertheless, in the final model, baseline ASI-P significantly predicted DASS-A change, such that higher baseline ASI-P predicted reduced treatment response in DASS-A from pre- to post-treatment whereas DAS-Pft and OBQ scores were unrelated to DASS-A change. Interpretation of the distribution of data based on Fig. 3 suggests that lower baseline ASI-P predicted better outcome on the DASS-A. Though the DASS-A does not measure the frequency or severity of panic episodes, it does measure physical symptoms of anxiety and has been shown to be significantly elevated in individuals with PD compared to those with other disorders (Antony et al. 1998); therefore, the DASS-A may be particularly relevant to PD/A symptom severity. As such, our findings suggest that the physical dimension of AS may be a specific predictor of changes in anxiety, consistent with prior research showing that the ASI-P dimension is specifically associated with anxious symptoms in PD/A (Kemper et al. 2012; Rector et al. 2007; Taylor et al. 2007). The results of ASI-P moderating symptom change in PD/A are in contrast to the findings of Porter and Chambless (2015), whose review and meta-analysis failed to find that AS was a significant moderator of CBT for PD/A. Of note, none of the four studies examining AS as a moderator that were reviewed by Porter and Chambless used the ASI-P subscale from the ASI-3. Instead, they used the total ASI score or a combination of other "fear of fear" measures. Thus, the physical concerns dimension of AS may be a specific or more robust moderator of CBT outcome for PD/A, which could explain the previous null findings.

The findings of the present study provide direct support for the differential importance of cognitive risk factors for thematically-related clinical conditions and they may also have relevance to clinical practice. The disorder-specific moderators of treatment response identified in this study are precisely the cognitive variables that are targeted by their

respective disorder-specific treatments: CBT interventions for MDD, PD/A, and OCD were developed to explicitly identify, test, and correct dysfunctional attitudes, anxiety sensitivity, and obsessive beliefs respectively. In each analysis, higher baseline cognitive vulnerabilities were shown to predict worse clinical outcomes. The question for future research remains as follows: how can treatment approaches improve for individuals who otherwise may not be able to obtain optimum outcomes due to high baseline levels of disorder-specific cognitive variabilities? While the findings from the present study cannot answer this question, through providing additional taxonomic information on cognitive vulnerabilities and treatment moderation, the findings can be used to help future intervention research focus on the variables that are most pertinent for each disorder.

The present study has several limitations. None of the participants were diagnosed using structured standardized measures. However, the psychiatrists completing the initial psychiatric consultation were employed in a large university-affiliated centre specializing in OCD, mood, anxiety disorders. The study variables were measured only at pre- and post-treatment. Measurement of the cognitive and symptom variables at multiple points during the course of treatment would permit testing of cognitive predictors of early versus late response in addition to overall treatment response, as well as cognitive mediation of treatment response. Furthermore, all patients did not complete all four of the symptom severity measures. While this was necessary to reduce patient burden, it nevertheless limited the ability to measure whether anxiety symptoms changed in depression or vice versa, and if so whether these secondary symptom changes were associated with the same cognitive mechanism as primary symptom change. In addition, the sample size for the PD/A group was relatively small; to increase generalizability future research can increase the sample size. Future research can expand the number of disorders under study and relevant cognitive mechanisms, for example, by including individuals with generalized anxiety disorder and a measure of metacognitive beliefs surrounding worry. Finally, the current study included only disorder-specific group treatments; future research could also compare specificity of cognitive moderators in disorder-specific versus transdiagnostic treatment approaches.

Despite the limitations, the current study also contains considerable strengths. It remains one of the few studies in which the specificity of multiple cognitive predictors of treatment response were examined across multiple disorders. The overall sample size was relatively large for a treatment study, which permitted the use of LDS analysis to evaluate the hypothesized moderation and reciprocal relationships. In addition, the data are from a real-world outpatient treatment clinic with minimal inclusion/exclusion criteria, thus enhancing the external validity of the study and the applicability of the results to individuals seeking treatment for mood and anxiety disorders. Although there are undoubtedly other cognitive, behavioural, and social variables that contribute to the onset, treatment outcome for and maintenance of each condition, the results provide provisional support for cognitive specificity of change as well as for the CBT framework for the disorders examined within the study.

Compliance with Ethical Standards

Conflict of interest Danielle Katz, Judith Laposa, Lance Hawley, Leanne Quigley, and Neil Rector declare that they have no conflict of interest.

Research Involving Animal Rights No animal studies were carried out by the authors for this article.

Informed consent Informed consent was obtained from all individual participants included in the study.

Appendix 1

LDS Reciprocal Models

Bidirectional relationships between symptom measures and cognitive variables.

MDD Sample

Equation: Reciprocal model examining the relationship of QIDS and DAS-Pft:

$$\mathrm{E}[\Delta \mathrm{QIDS}(\mathrm{t})_{\mathrm{n}}] = \beta_{\mathrm{QIDS}} \times \mathrm{E}\left[\mathrm{QIDS}_{\mathrm{n}}\right] + \gamma \times \mathrm{E}\left[\text{DAS-Pft}(\mathrm{t}-1)_{\mathrm{n}}\right]$$

$$\mathrm{E}[\Delta \text{DAS-Pft}(\mathrm{t})_{\mathrm{n}}] = \beta_{\text{DAS-Pft}} \times \mathrm{E}\left[\text{DAS-Pft}(\mathrm{t}-1)_{\mathrm{n}}\right] + \gamma_{\mathrm{QIDS}} \times \mathrm{E}\left[\mathrm{QIDS}(\mathrm{t}-1)_{\mathrm{n}}\right]$$

Equation: Reciprocal model examining the relationship of QIDS and OBQ:

$$\mathrm{E}[\Delta \mathrm{QIDS}(\mathrm{t})_{\mathrm{n}}] = \beta_{\mathrm{QIDS}} \times \mathrm{E}\left[\mathrm{QIDS}_{\mathrm{n}}\right] + \gamma \times \mathrm{E}\left[\mathrm{OBQ}(\mathrm{t}-1)_{\mathrm{n}}\right]$$

$$\mathrm{E}[\Delta \mathrm{OBQ}(\mathrm{t})_{\mathrm{n}}] = \beta_{\mathrm{OBQ}} \times \mathrm{E}\left[\mathrm{OBQ}\ (\mathrm{t}-1)_{\mathrm{n}}\right] + \gamma_{\mathrm{QIDS}} \times \mathrm{E}\left[\mathrm{QIDS}(\mathrm{t}-1)_{\mathrm{n}}\right]$$

Equation: Reciprocal model examining the relationship of QIDS and ASI total score:

$$\mathrm{E}[\Delta \mathrm{QIDS}(\mathrm{t})_{\mathrm{n}}] = \beta_{\mathrm{QIDS}} \times \mathrm{E}\left[\mathrm{QIDS}_{\mathrm{n}}\right] + \gamma \times \mathrm{E}\left[\mathrm{ASI}(\mathrm{t}-1)_{\mathrm{n}}\right]$$

$$\mathrm{E}[\Delta \mathrm{ASI}(\mathrm{t})_{\mathrm{n}}] = \beta_{\mathrm{ASI}} \times \mathrm{E}\left[\mathrm{ASI}(\mathrm{t}-1)_{\mathrm{n}}\right] + \gamma_{\mathrm{QIDS}} \times \mathrm{E}\left[\mathrm{QIDS}(\mathrm{t}-1)_{\mathrm{n}}\right]$$

OCD Sample

Equation: Reciprocal model examining the relationship of YBOCS and DAS-Pft:

$$E[\Delta YBOCS(t)_n] = \beta_{YBOCS} \times E\left[YBOCS_n\right] + \gamma \times E\left[DAS\text{-}Pft(t-1)_n\right]$$

$$E[\Delta DAS\text{-}Pft(t)_n] = \beta_{DAS\text{-}Pft} \times E\left[DAS\text{-}Pft(t-1)_n\right] + \gamma_{YBOCS} \times E\left[YBOCS(t-1)_n\right]$$

Equation: Reciprocal model examining the relationship of YBOCS and OBQ:

$$E[\Delta YBOCS(t)_n] = \beta_{YBOCS} \times E\left[YBOCS_n\right] + \gamma \times E\left[OBQ(t-1)_n\right]$$

$$E[\Delta OBQ(t)_n] = \beta_{OBQ} \times E\left[OBQ(t-1)_n\right] + \gamma_{YBOCS} \times E\left[YBOCS(t-1)_n\right]$$

Equation: Reciprocal model examining the relationship of YBOCS and ASI total score:

$$E[\Delta YBOCS(t)_n] = \beta_{YBOCS} \times E\left[YBOCS_n\right] + \gamma \times E\left[ASI(t-1)_n\right]$$

$$E[\Delta ASI(t)_n] = \beta_{ASI} \times E\left[ASI(t-1)_n\right] + \gamma_{YBOCS} \times E\left[YBOCS(t-1)_n\right]$$

PD/A Sample

Equation: Reciprocal model examining the relationship of DASS-A and DAS-Pft:

$$E[\Delta DASS\text{-}A(t)_n] = \beta_{DASS\text{-}A} \times E\left[DASS\text{-}A_n\right] + \gamma \times E\left[DAS\text{-}Pft(t-1)_n\right]$$

$$E[\Delta DAS\text{-}Pft(t)_n] = \beta_{DAS\text{-}Pft} \times E\left[DAS\text{-}Pft(t-1)_n\right] + \gamma_{DASS\text{-}A} \times E\left[DASS\text{-}A(t-1)_n\right]$$

Equation: Reciprocal model examining the relationship of DASS-A and OBQ:

$$E[\Delta DASS\text{-}A(t)_n] = \beta_{DASS\text{-}A} \times E\left[DASS\text{-}A_n\right] + \gamma \times E\left[OBQ(t-1)_n\right]$$

$$E[\Delta OBQ(t)_n] = \beta_{OBQ} \times E\left[OBQ(t-1)_n\right] + \gamma_{DASS\text{-}A} \times E\left[DASS\text{-}A(t-1)_n\right]$$

Equation: Reciprocal model examining the relationship of DASS-A and ASI physical subscale:

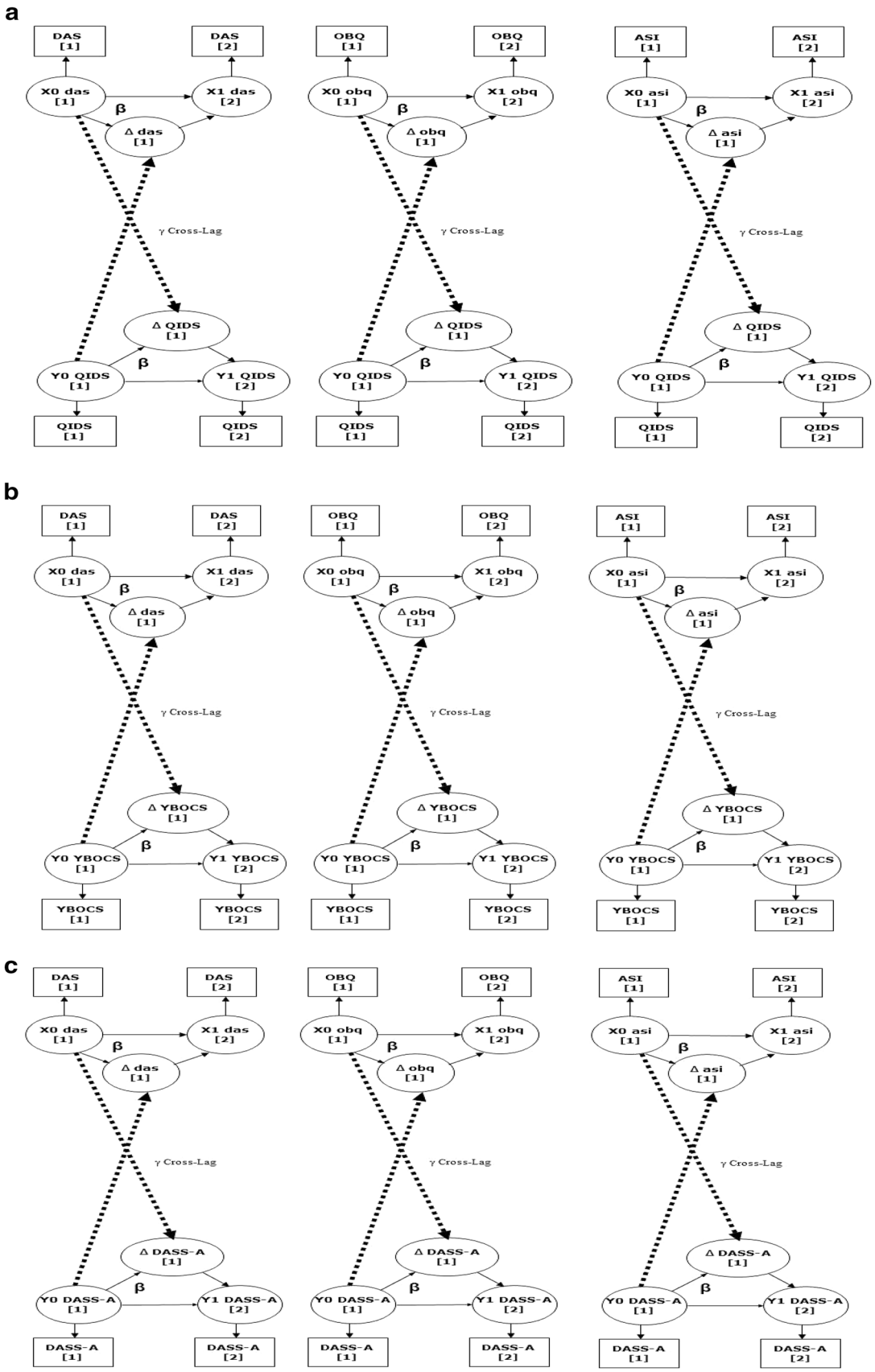
$$E[\Delta DASS\text{-}A(t)_n] = \beta_{DASS\text{-}A} \times E\left[DASS\text{-}A_n\right] + \gamma \times E\left[ASI\text{-}P(t-1)_n\right]$$

$$E[\Delta ASI\text{-}P(t)_n] = \beta_{ASI\text{-}P} \times E\left[ASI\text{-}P(t-1)_n\right] + \gamma_{DASS\text{-}A} \times E\left[DASS\text{-}A(t-1)_n\right]$$

Appendix 2

See Fig. 4.

Fig. 4 **a** SEM pathways for the "Reciprocal Model" for the MDD Sample. **b** SEM pathways for the "Reciprocal Model" for the OCD sample. **c** SEM pathways for the "Reciprocal Model" for the PD/A sample


a
DAS [1]
DAS [2]
X0 das [1]
X1 das [2]
β
Δ das [1]
γ Cross-Lag
Δ QIDS [1]
β
Y0 QIDS [1]
Y1 QIDS [2]
QIDS [1]
QIDS [2]
OBQ [1]
OBQ [2]
X0 obq [1]
X1 obq [2]
β
Δ obq [1]
γ Cross-Lag
Δ QIDS [1]
β
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QIDS [1]
QIDS [2]
ASI [1]
ASI [2]
X0 asi [1]
X1 asi [2]
β
Δ asi [1]
γ Cross-Lag
Δ QIDS [1]
β
Y0 QIDS [1]
Y1 QIDS [2]
QIDS [1]
QIDS [2]
b
DAS [1]
DAS [2]
X0 das [1]
X1 das [2]
β
Δ das [1]
γ Cross-Lag
Δ YBOCS [1]
β
Y0 YBOCS [1]
Y1 YBOCS [2]
YBOCS [1]
YBOCS [2]
OBQ [1]
OBQ [2]
X0 obq [1]
X1 obq [2]
β
Δ obq [1]
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Y1 YBOCS [2]
YBOCS [1]
YBOCS [2]
ASI [1]
ASI [2]
X0 asi [1]
X1 asi [2]
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YBOCS [1]
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γ Cross-Lag
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Y0 DASS-A [1]
Y1 DASS-A [2]
DASS-A [1]
DASS-A [2]
OBQ [1]
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X1 obq [2]
β
Δ obq [1]
γ Cross-Lag
Δ DASS-A [1]
β
Y0 DASS-A [1]
Y1 DASS-A [2]
DASS-A [1]
DASS-A [2]
ASI [1]
ASI [2]
X0 asi [1]
X1 asi [2]
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Δ asi [1]
γ Cross-Lag
Δ DASS-A [1]
β
Y0 DASS-A [1]
Y1 DASS-A [2]
DASS-A [1]
DASS-A [2]

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