Cognitive predictors and moderators of winter depression treatment outcomes in cognitive-behavioral therapy vs. light therapy

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There is no empirical basis for determining which seasonal affective disorder (SAD) patients are best suited for what type of treatment. Using data from a parent clinical trial comparing light therapy (LT), cognitive-behavioral therapy (CBT), and their combination (CBT + LT) for SAD, we constructed hierarchical linear regression models to explore baseline cognitive vulnerability constructs (i.e., dysfunctional attitudes, negative automatic thoughts, response styles) as prognostic and prescriptive factors of acute and next winter depression outcomes. Cognitive constructs did not predict or moderate acute treatment outcomes. Baseline dysfunctional attitudes and negative automatic thoughts were prescriptive of next winter treatment outcomes. Participants with higher baseline levels of dysfunctional attitudes and negative automatic thoughts had less severe depression the next winter if treated with CBT than if treated with LT. In addition, participants randomized to solo LT who scored at or above the sample mean on these cognitive measures at baseline had more severe depressive symptoms the next winter relative to those who scored below the mean. Baseline dysfunctional attitudes and negative automatic thoughts did not predict treatment outcomes in participants assigned to solo CBT or CBT + LT. Therefore, SAD patients with extremely rigid cognitions did not fare as well in the subsequent winter if treated initially with solo LT. Such patients may be better suited for initial treatment with CBT, which directly targets cognitive vulnerability processes.

Winter seasonal affective disorder (SAD) is characterized by recurrent Major Depressive Episodes that begin in the fall or winter and remit in the spring (APA, 2000). Untreated and annually recurring SAD episodes lead to impairments in activities of living, emotional well-being, and overall health in the winter (Sclager, Froom, & Jaffe, 1995). Given that 10%–20% of all cases of recurrent depression follow a seasonal pattern (Blazer, Kessler, & Schwartz, 1998; Magnusson, 2000), it is a public health priority to develop interventions that prevent recurrence of depressive episodes over subsequent winter seasons.

Bright light therapy (LT) and SAD-tailored cognitive-behavioral therapy (CBT) have been shown to be efficacious in the acute treatment of SAD (Golden et al., 2005; Rohan et al., 2007). In an uncontrolled feasibility study comparing LT, CBT, and their combination in the treatment of adult SAD patients, CBT (alone or combined with LT) was comparably efficacious to LT alone in reducing acute SAD symptoms (Rohan, Tierney Lindsey, Roecklein, & Lacy, 2004). A subsequent controlled, randomized clinical trial found that participants randomized to CBT, LT, or combination treatment evidenced significant and comparable reductions in depressive symptoms at post-treatment relative to a concurrent wait-list control group (Rohan et al., 2007). Although there were no statistically significant differences between treatments in full remission status at post-treatment, the combined CBT + LT condition had the largest proportion of participants classified as remitted (73–79%). In a naturalistic follow-up during the next winter season, the CBT (70%) and CBT + LT treatments (55%) had significantly smaller proportions of winter depression recurrences than the solo LT treatment (36.7%; Rohan, Roecklein, Lacy, & Vacek, 2009). In addition, solo CBT, but not combination treatment, had less severe blind interviewer- and self-rated depressive symptoms the next winter than solo LT. Therefore, although daily LT use has significant antidepressant effects during the initial winter of treatment (Golden et al., 2005), results of recent clinical trials suggest that CBT may be an effective alternative treatment to LT in treating acute SAD (Rohan et al., 2004; 2007) and may have more enduring effects than LT in preventing SAD episode recurrence and reducing symptom severity during the subsequent winter (Rohan, Roecklein, & Haaga, 2009; Rohan, Roecklein, Lacy, et al., 2009).
Despite empirical support for LT and CBT as efficacious acute SAD treatments, approximately 43–60% of patients randomized to either solo CBT or LT do not meet remission criteria at post-treatment (Rohan et al., 2007; Terman, Terman, & Quittkin, 1989). Moreover, many formerly treated SAD patients continue to experience clinically significant depressive symptoms in the winter subsequent to study treatment (proportions of participants in remission the next winter: 30% LT, 37% CBT + LT, 58% CBT; Rohan et al., 2009; Rohan, Roecklein, Lacy, et al., 2009). Yet, little is known about factors that predict favorable or unfavorable acute or next winter treatment outcomes in SAD. More work is needed to inform an empirical basis for determining which SAD patients are best suited for what type of treatment. To our knowledge, only one study has examined a cognitive predictor of LT response (Levitan, Rector, & Bagby, 1998) and no studies have evaluated cognitive moderators of CBT outcomes among SAD patients. Levitan et al. (1998) reported that baseline negative attributional style (i.e., the tendency to make stable and global attributions about life events) did not predict response to LT among SAD patients. However, this one study does not inform whether this or other putative cognitive vulnerability constructs might be prognostic of treatment outcomes across treatment modalities or prescriptively impact the relative efficacy of CBT, LT, or their combination.

Studies examining predictors of nonseasonal depression treatment outcomes have distinguished between prognostic (i.e., predict efficacy irrespective of the treatment modality) and prescriptive (i.e., account for a different pattern of outcome between treatments — moderators; Kraemer, Wilson, Fairburn, & Agras, 2002) patient baseline characteristics and traits (e.g., Fournier et al., 2009). Identifying prognostic factors has both theoretical and clinical utility. Pre-randomization factors that are associated with superior or refractory treatment response in SAD may help to identify novel etiological risk and protective processes, which could then be incorporated into theory and become novel targets of treatment and prevention. SAD patients who do not respond favorably to available treatments, irrespective of treatment modality, may benefit from a novel, extended, or supplementary treatment. That is, novel interventions could be developed or existing empirically-supported interventions could be revised (e.g., increased intensity, extended length, new components added) to mitigate or explicitly target prognostic factors. Identification of prescriptive factors may enhance clinical practice by providing guidelines for selecting an appropriate and evidence-supported intervention for each presenting SAD patient (i.e., personalized medicine). Comparisons of prescriptive factors for LT, CBT, and their combination would be particularly informative because CBT and LT are designed to target different etiological processes, have different putative mechanisms of action, and are vastly different interventions in terms of what is required of the patient. Baseline cognitive constructs are candidate prescriptive factors, given their explicit focus in CBT for SAD (Rohan, Sigmon, & Dorhofer, 2003) and their accessibility to measurement.

Recent studies on differential response to depression treatments have distinguished between compensation and capitalization approaches for adapting treatments to patients’ needs. Under the compensation model (Cheavens, Strunk, Lazarus, & Goldstein, 2012), an intervention targets disorder-specific etiological processes and vulnerabilities with the aim of addressing or modifying the vulnerability. According to this model, patients who display a certain characteristics should, theoretically, benefit more from an intervention aimed at modifying this disorder-specific vulnerability than patients who receive a treatment that does not directly target said vulnerability. In contrast, under the capitalization model, the most effective intervention option would target an individual’s strengths to bolster them further (Cheavens et al., 2012; Simon & Perlis, 2010). The rationale underlying most, if not all, SAD treatments is consistent with a compensation conceptualization (i.e., the treatment is presumed to remedy vulnerabilities associated with winter depressive symptoms). Therefore, if compensation applies to SAD, SAD patients with a high cognitive vulnerability should benefit from CBT more than those with a lower cognitive vulnerability who receive CBT and more than those with a high cognitive vulnerability treated with LT because only CBT is designed to target maladaptive cognitions. Conversely, according to compensation, those with a high physiological vulnerability (e.g., a circadian phase shift in the winter) should benefit more from LT than those with a lower physiological contribution to their SAD and those with a high physiological vulnerability treated with CBT. It is less straightforward to conceptualize SAD treatments from a capitalization perspective. To match depressed patients to cognitive, behavioral, mindfulness, or interpersonal intervention modules based on capitalization; Cheavens et al. (2012) administered the two treatments that were most consistent with the types of mood-regulation strategies patients were already using at baseline. Applying this type of personalization based on capitalization to SAD, it is possible that patients who cope with their symptoms using behaviors such as adopting a new perspective and pushing themselves to stay active in the winter benefit the most from CBT whereas patients who cope by taking a vacation to a warm, sunny place benefit the most from LT. In keeping with capitalization, it is also possible that a more general construct such as locus of control moderates SAD treatment outcome. For example, perhaps those with a more internal locus of control benefit from CBT whereas those with a more external locus of control benefit from LT.

In nonseasonal depression, higher pre-treatment levels of dysfunctional attitudes (i.e., stable and global beliefs concerning perfectionism, need for approval, inadequacy, and perceived requirements for happiness) “appear to be a prognostic indicator of poor response to CBT” (Hamilton & Dobson, 2002, p. 887). Contrary to the compensation model, across studies, after controlling for pre-treatment depression severity; depressed patients with higher pre-treatment levels of dysfunctional attitudes had poorer outcomes with CBT in terms of post-treatment scores on self-report and interviewer-administered measures of depressive symptoms (Jarrett, Eaves, Brannemann, & Rush, 1991; Keller, 1983; Simons, Gordon, Monroe, & Thase, 1995; Sotsky et al., 1991) and a greater likelihood of relapse over 1-year follow-up (Thase et al., 1992). Therefore, a theoretical match between a patient’s depressogenic vulnerability at baseline (i.e., highly dysfunctional attitudes) and the domain targeted by the applied intervention (i.e., CBT) may not translate into superior therapeutic outcomes. It is possible that extremely dysfunctional attitudes at baseline limits the efficacy of CBT by interfering with a patient’s capacity to identify and effectively challenge unhelpful patterns of thinking as well as with a patient’s ability to plan and engage in distracting and pleasant activities. Preliminary evidence suggests that higher baseline dysfunctional attitudes may be prognostic of worse depression treatment outcomes not only in CBT, but across different treatment modalities, including interpersonal psychotherapy (Carter et al., 2007) and antidepressant medications (Jacobs et al., 2009; Peselow, Robins, Block, Barouch, & Fieve, 1990; Sotsky et al., 1991). Thus, highly rigid depressogenic attitudes may work against beneficial treatment effects, regardless of depression treatment modality.

Studies have also examined the prognostic and prescriptive utility of a rumination response style (i.e., the tendency to respond to dysphoric mood and symptoms of depression by repetitively thinking about their causes and consequences; Nolen-Hoeksema, 1987) in predicting treatment outcomes in nonseasonal depression. In several studies, higher baseline rumination was prognostic.
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