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## Behavioral avoidance mediates the relationship between anxiety and depressive symptoms among social anxiety disorder patients $\stackrel{\stackrel{}_{\leftarrow}}{\sim}$

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## Abstract

This study investigated the relationship between social anxiety, depressive symptoms, and behavioral avoidance among adult patients with Social Anxiety Disorder (SAD). Epidemiological literature shows SAD is the most common comorbid disorder associated with Major Depressive Disorder (MDD), though the relationship between these disorders has not been investigated. In most cases, SAD onset precedes MDD, suggesting symptoms associated with SAD might lead to depression in some people. The present study addressed this question by investigating the mediational role of behavioral avoidance in this clinical phenomenon, using self-report data from treatment-seeking socially anxious adults. Mediational analyses were performed on a baseline sample of 190 individuals and on temporal data from a subset of this group. Results revealed behavioral avoidance mediated this relationship, and supported the importance of addressing such avoidance in the therapeutic setting, via exposure and other methods, as a possible means of preventing depressive symptom onset in socially anxious individuals.

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The lifetime prevalence of Social Anxiety Disorder (SAD) in Western societies is quite high, ranging from 7% to 13% (Furmark, 2002). In fact, SAD is the most common anxiety disorder in the U.S. and the third most common psychiatric disorder, exceeded only by alcohol dependence and Major Depressive Disorder (MDD; Kessler et al., 1994). SAD is a disabling condition; compared to people without psychiatric morbidity, adults with SAD report lower employment rates, lower

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income, and lower socio-economic status (Patel, Knapp, Henderson, & Baldwin, 2002).

## 1. SAD and depression

SAD is also the most common comorbid anxiety disorder with MDD, with estimates of SAD ranging from 15% to 37% of depressed patients (Belzer & Schneier, 2004; Fava et al., 2000; Kessler et al., 1994). Comorbid SAD and MDD has been associated with an earlier onset of MDD, more depressive episodes, longer duration of episodes, a two-fold increased risk of alcohol dependence, and an increased risk of suicide attempts leading to hospitalization (Dalrymple & Zimmerman, 2007; Nelson et al., 2000; Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992; Stein et al., 2001). Comorbid MDD and SAD are also

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associated with greater functional impairment, number of disability days, and number of annual mental health visits (Belzer & Schneier, 2004). According to Essau, Condradt, and Petermann (1999), approximately 25– 31% of adolescents and young adults with SAD have a comorbid depressive disorder. Additionally, the onset of SAD preceded the onset of MDD in approximately 70% of comorbid cases in the National Comorbidity Study (NCS) and in the Epidemiological Catchment Area (ECA) study (Kessler, Stang, & Wittchen, 1999; Schneier et al., 1992).

Despite high rates of comorbidity, most socially anxious individuals with depression are excluded from treatment outcome studies (Huppert, Franklin, Foa, & Davidson, 2003), which may limit generalizability of findings. The rationale is that these participants' depressive symptoms would confound interventions designed to treat social anxiety. Therefore, although the epidemiology of these comorbid disorders has been elucidated, this population remains relatively ignored. Some data show reductions in social anxiety symptoms mediating depressive symptoms, but more precise details of this mechanism are unknown. For instance, Moscovitch, Hofmann, Suvak, and In-Albon (2005) demonstrated that reductions in social anxiety fully mediated reductions in depressive symptoms among a sample of 66 socially anxious adults enrolled in weekly cognitive-behavioral group therapy. According to their results, reductions in social anxiety mediated 91% of the improvements in depression over time. However, their study failed to provide evidence as to how reductions in social anxiety lead to reductions in depressive symptoms. Specifically, they did not elucidate which dimensions of SAD in particular were related to depressive symptom reduction.

Lastly, theoretical explanations for the relationship between SAD and depression are scarce, and there are few empirical data addressing this common comorbidity beyond descriptive epidemiological studies. In particular, it is poorly understood why a large proportion, but by no means all, of those with SAD exhibit comorbid depression.

# **2.** Possible explanations for the relationship between SAD and depression

A common biochemical pathogenesis may be shared between SAD and depression. For instance, some research into shared biological substrates between anxiety and depression has suggested mediation by serotonergic pathways due to their effects on the amygdala, the locus ceruleus, and the raphe nuclei (Pohl, Wolkow, & Clary, 1998). From a cognitive– behavioral perspective, SAD and MDD could derive from shared negative cognitive processes. For instance, distorted cognitions associated with depression (Beck & Rush, 1978), such as a fear of being negatively evaluated by others, could extend to social domains. However, this would not account for why only a subset of individuals with SAD exhibit comorbid depression. In addition, the causal status of negative cognitions with respect to psychopathology is controversial. For example, such cognitions may be concomitants, rather than causes, of psychopathology. In the present investigation, we suggest that behavioral avoidance present in some socially anxious individuals may cause depressive symptoms.

## 3. Behavioral avoidance as a potential mediator

The primary symptom of SAD, as defined by the current edition of the Diagnostic and Statistical Manual of Mental Disorders – IV Text Revision (DSM-IV-TR; American Psychiatric Association, 2000), is a marked and persistent fear of one or more social situations. Another significant criterion of SAD is that social situations are avoided or endured with marked distress. Having criteria specifying avoidance or endurance of the feared situation distinguishes SAD from other Axis I diagnoses, including most anxiety disorders. To meet criteria for SAD, an adult must experience clinically significant distress or interference due to the disorder. This dichotomy implies that all people with SAD experience subjective fear, distress, and some functional impairment, but that not everyone with SAD necessarily avoids anxiety-provoking situations. Indeed, willingness to confront anxiety-provoking social situations varies among socially anxious individuals. Some socially anxious adults avoid as many social situations as possible, and are more likely to be unemployed, work out of their home, and have few friends or social contacts (Beidel & Turner, 2007). According to a recent meta-analysis of coping strategies and their effects on distress, Littleton, Horsley, John, and Nelson (2007) found avoidance-focused coping strategies are significantly correlated with increased psychological distress. Additionally, higher levels of social anxiety are associated with less assertive behavior, more conflict avoidance, and greater interpersonal dependency (Davila & Beck, 2002).

In a study of patients in a psychiatric facility which forbade friends or family to maintain regular contact, Overholser (1990) found that recently admitted psychiatric inpatients high in emotional reliance (a social anxiety trait) but experiencing no social loss (presumably because they had very little social contacts in the first place) displayed higher levels of depression than low emotionally reliant subjects who had undergone a social loss. Although a formal diagnosis of social anxiety was not conferred, these data suggest those with socially anxious traits such as high emotional reliance and a dearth of social contact (which is implied by the absence of social loss and may be the result of elevated behavioral avoidance) had higher levels of depression. In fact, lack of social support and level of depressive symptomatology are highly correlated (Booth et al., 1992; Russell & Cutrona, 1991). The literature also suggests interpersonal inhibition (i.e., shyness) is a vulnerability factor for depression (Joiner, 1997). Although most people with SAD are socially inhibited, this symptom may be at its most severe in those who consistently avoid social situations. Thus, it appears that the isolation component of social anxiety may be particularly salient in the genesis of depressive symptoms.

It is theoretically plausible that behavioral avoidance among socially anxious persons may contribute towards the development of depressive symptoms because avoidance can lead to isolation and loneliness. However, behavioral avoidance has not been formally investigated for its role in influencing comorbid disturbance. Although a link between SAD and depressive symptoms has been clearly established in the epidemiological literature, as has the fact that the onset of SAD typically precedes the onset of depressive symptoms, the mechanisms by which the two are linked has not been examined.

## 4. Aim and hypotheses

The goal of the current project was to further examine the relationship between SAD and depression. Specifically, we sought to investigate the role that behavioral avoidance may play in mediating the relationship between social anxiety and depression in a sample of adults seeking outpatient treatment for SAD. We predicted that behavioral avoidance would partially mediate the relationship between social anxiety and depressive symptoms. In particular, we hypothesized that anxiety would be associated with depression and behavioral avoidance, that avoidance would be associated with depression, and that the association between anxiety and depression would be attenuated once the effect of avoidance on depression was partialled out. Moreover, we hypothesized that, among a subsample of SAD patients who had

completed treatment, initial improvements in behavioral avoidance would predict later decreases in level of depression.

## 5. Method

#### 5.1. Participants

The study utilized existing data from a larger outcome trial that provided free treatment for patients with the generalized subtype of SAD at a psychology department clinic. Adults ranging in age from 18 to 60 were eligible for participation. The basal cut-off for inclusion was determined because the present study sought to examine symptoms in adulthood and the ceiling cut-off for inclusion was determined because senior citizens often lose social support (due to deaths, moving to assisted living areas, relocating to retirement areas, etc.) and commonly experience isolation resulting from such environmental changes (Antonucci & Jackson, 1987).

Another inclusion criterion was that SAD be the participants' primary diagnosis. Primacy was determined by initial onset and relative degree of distress associated with each condition. Individuals suffering from the following disorders were excluded from the present study and referred to other treatment providers: schizophrenia, mental retardation, and substance dependence.

The initial sample consisted of 190 adults (51.1% female) ranging in age from 18 to 59 years old. A majority was Caucasian (69.7%), single (63.3%), and employed full-time (53.5%). This sample's educational attainment was relatively high: 34.2% attended some college, 34.8% had a college degree, and 21.9% attended graduate/professional school (see Table 1 for demographics information divided by analytic sub-sample).

## 5.2. Procedure

Participants were recruited through community advertisements in local media, as well as referrals from health care providers. Interested potential participants made initial contact by telephoning the clinic. Research staff conducted a brief, semi-structured telephone screening to determine a caller's likely eligibility for the study. If a potential participant "passed" the phone screen, he or she was invited to be interviewed in person in the clinic. Diagnostic interviews were conducted using the Structured Clinical Interview for the DSM-IV (SCID-IV; First, Spitzer,

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Table 1 Demographic data for baseline and temporal analyses samples

	Baseline $(n = 190)$	Temporal $(n = 86)$
Gender		
Male	50% (95)	48% (41)
Female	50% (95)	52% (45)
Race		
African American	20% (37)	11% (10)
Asian	4% (7)	7% (6)
Caucasian	70% (131)	56% (48)
Latino	4% (7)	7% (6)
Other	2% (3)	19% (16)
Education		
Some high school	1% (1)	1% (1)
High school diploma	9% (16)	20% (20)
Some college	34% (64)	19% (16)
College degree	35% (65)	33% (38)
Graduate degree	21% (41)	27% (22)
Employment		
Unemployed	14% (27)	14% (12)
Part-time	14% (27)	19% (16)
Full-time	54% (99)	56% (48)
Student	17% (31)	11% (10)
Retired	1% (1)	0% (0)
Marital status		
Single	63% (119)	59% (51)
Married	24% (45)	13% (11)
Divorced	7% (12)	4% (3)
Separated	4% (8)	2% (2)
Widowed	1% (1)	2% (2)
Other	1% (1)	20% (20)

Gibbon, & Williams, 1996). The SCID-IV is a commonly used and psychometrically sound instrument that assesses the full range of psychopathology outlined in Axis I of the *DSM-IV*, including past and current symptoms. Several diagnosticians conducted interviews in order to increase external validity. All interviews were audiotaped, and inter-rater reliability on a random sample of 30% of the interviews was high (intraclass correlation  $\alpha = .96$ ). Participants who met study criteria based on this interview were invited to join the study. If they agreed, they were given a baseline series of self-rating questionnaires that served as the primary source of data in the present investigation.

## 5.3. Measures

For the purposes of the treatment outcome study, questionnaires were administered at baseline, 6 weeks into treatment (i.e., mid-treatment), and at the conclusion of treatment (12 weeks). The following measures were used:

Beck Depression Inventory-II (BDI-II; Beck, Steer, Ball, & Ranieri, 1996). The BDI-II is an extensively used and studied inventory designed to assess current severity of depression, which was developed from clinical observations of depressed and non-depressed psychiatric patients. Attitudes and symptoms consistent with depression are represented in a 21-item questionnaire, and patients are asked to rate the severity of each on an ordinal scale from 0 to 3 (Katz, Katz, & Shaw, 1999). The BDI-II is scored by summing the ratings, and cut-scores may be used to classify patients according to depression severity. The BDI-II is based largely on the first edition of the BDI, which has indicated good test-retest (r = .86 for psychiatric patients, .81 for non-psychiatric subjects) and strong content, concurrent, and discriminant validity in clinical and non-clinical samples (see Beck, Steer, & Garbin, 1988 for a review).

Social Phobia and Anxiety Inventory (SPAI; Turner, Beidel, Dancu, & Stanley, 1989). The SPAI is a selfreport measure with a 32-item social phobia subscale and a 13-item agoraphobia subscale. This measure was designed to produce subscale total scores and a subscale difference score due to speculation that some social anxiety symptoms may be better accounted for by agoraphobia, or vice versa. The respondent rates on a 1-7 Likert scale how often he or she experiences the listed thoughts and feelings in social situations. For the purposes of this study, only the social phobia (SP) subscale was utilized because this subscale has been indicated as a better index of social anxiety symptoms than the difference subscale score and has been used as such (Herbert, Bellack, & Hope, 1991). The SPAI has strong test-retest reliability (r = .86; Turner et al., 1989), internal consistency (Cronbach's alpha = .94 to .96; Osman et al., 1996), and concurrent and external validity (Herbert et al., 1991; Osman et al., 1996; Turner et al., 1989).

Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987). The LSAS is a 24-item measure that consists of a list of 13 items that describe performance situations and 11 items that describe social situations. In the present study, we used the self-report version, which was created from the clinician-administered version. Fresco et al. (2001) showed both versions were equally internally consistent and reliable. Furthermore, Baker, Heinrichs, Kim, and Hofmann (2002) suggested it was a more efficient measure of social anxiety symptoms than the clinician-administered version. The LSAS provides two subscores: fear and avoidance. Each situation is rated on a Likert scale from 0 to 3 for severity of fear and degree of avoidance. According to Gibb, Coles, and

Heimberg (2005), the LSAS is an important assessment tool among socially anxious individuals because it can differentiate social anxiety and depression, rather than simply reflecting one construct of overall distress. Additionally, the LSAS self-report version has strong internal consistency (Cronbach's alpha = .94–.95), and strong convergent and discriminant validity (Fresco et al., 2001).

## 5.4. Data analysis

Due to procedural changes introduced in revised iterations of the treatment study, the available sample size varies between parts one and two of the current study (baseline mediation: n = 190; temporal mediation: n = 86). Levels of social anxiety, depression, and avoidance were measured by the SPAI-SP, BDI-II, and LSAS-Avoidance Subscale, respectively. Single and multiple regressions were used to explore the possible mediational effects of behavioral avoidance at baseline. This mediational model was examined using the Sobel test of mediation (Sobel, 1982) to determine whether indirect paths from each predictor on each dependent variable were significantly different than zero. Hypothesis testing for temporal analyses followed a similar strategy by using correlational analyses. Residualized change scores were calculated to afford the ability to examine data controlling for baseline and mid-treatment changes, per the recommendations of Steketee and Chambless (1992).

#### 6. Results

#### 6.1. Descriptive statistics

Symptom severity is reported for all participants at baseline and at mid- and post-treatment for those included in the temporal mediational analyses (see Table 2 for pre-treatment, mid-treatment, and post-treatment descriptive statistics for the SPAI, LSAS-Avoidance subscale, and BDI-II).

Table 2 Means (S.D.) for temporal participants

	1 1	1	
	Baseline	Mid-treatment	Post-treatment
BDI-II	13.29 (10.89)	9.88 (8.24)	6.82 (8.25)
LSAS-Avoid	35.47 (12.11)	27.60 (11.82)	23.65 (11.18)
SPAI-SP	133.61 (24.37)	112.66 (24.71)	97.21 (30.95)

*Note*: BDI-II, Beck Depression Inventory-II; LSAS-Avoid, Liebowitz Social Anxiety Scale-Avoidance subscale; SPAI-SP, Social Phobia and Anxiety Inventory-Social Phobia subscale.

#### 6.2. Test of mediation at baseline

Mediational analyses were conducted according to steps outlined by Baron and Kenny (1986) and Frazier, Tix, and Barron (2004). Steps 1-3 were assessed to establish Paths a, b, c, and c' as described in Baron and Kenny. First, depressive symptoms were regressed on social anxiety symptoms to establish the "Path c" relationship between the predictor (i.e., social anxiety symptoms) and the outcome (i.e., depressive symptoms), indicating a statistically significant relationship. Next, behavioral avoidance was regressed onto social anxiety to establish Path a (Step 2), indicating a statistically significant relationship. Step 3 was then conducted to test whether behavioral avoidance was significantly related to depressive symptoms. We regressed depressive symptoms simultaneously on social anxiety symptoms and behavioral avoidance symptoms to establish Paths b and c'. The coefficient associated with the relation between behavioral avoidance and depressive symptoms (controlling for social anxiety symptoms) was statistically significant. This significant relationship confirms that the condition for Step 3 was met. Lastly, this third regression equation also provided an estimate of Path c', the relation between social anxiety symptoms and depressive symptoms, controlling for behavioral avoidance. Path c' (b = .075, p = .036) was smaller than Path c, indicating partial mediation (see Fig. 1 for summary of path strengths). As path c' was not equal to zero, behavioral avoidance was not a complete mediator. The drop from .129 to .075 (i.e., from c to c') was significant, according to the Sobel test (Sobel, 1982). Lastly, it was calculated that 41.9% of the total effect of social anxiety symptoms on depressive symptoms was mediated by behavioral avoidance, based on the formula recommended by Shrout and Bolger (2002).

## 6.3. Test of mediation over the course of treatment

Although a formal test of mediation was not conducted because of the lack of a control group, the second part of this investigation examined the mediational effects of behavioral avoidance over the course of treatment to examine why decreases in social fear could lead to later changes in depressive symptoms. Because of the potential shortcomings of utilizing simple raw change scores (e.g., over- and underestimation of change), residualized change scores were calculated using recommendations by Steketee and Chambless (1992) and recently applied in two studies of mediating E. Moitra et al. / Journal of Anxiety Disorders 22 (2008) 1205-1213

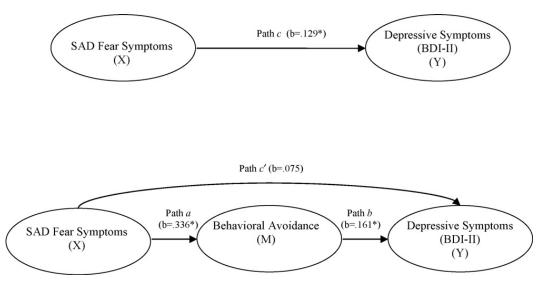


Fig. 1. Results of the hypothesized mediational model with behavioral avoidance as a mediator between social anxiety fear and depressive symptoms (unstandardized regression coefficients presented; \*p < .05; Sobel Test: z = 2.17).

variables in SAD (Dalrymple & Herbert, 2007; Hofmann, 2004). To examine whether pre-mid changes in behavioral avoidance were associated with later changes in depressive symptoms, we correlated residual gain scores of the LSAS (pre- to mid-treatment) with the residual gain scores of the BDI-II from mid-treatment to post-treatment. The results showed that the Pearson correlation coefficient between the two residual gain scores was moderate in strength and statistically significant: r = -.25 (p < .05).

## 7. Discussion

## 7.1. Baseline mediation

Despite high rates of comorbidity, the relationship between SAD and depressive symptoms is not clearly understood. We hypothesized that the behavioral avoidance element of social anxiety partially mediates the role between anxiety and depressive symptoms because this type of avoidance could lead to a dearth of social support and loneliness, two important causal factors in depressive symptom onset. Initially, we tested this hypothesis according to the steps outlined by Baron and Kenny (1986) using baseline data gleaned from individuals enrolled in behaviourally based individual treatment for SAD. Although these analyses were not temporal, they provided initial support for continued testing of the mediational model. Steps 1-3 were met, suggesting behavioral avoidance does indeed mediate the role between social anxiety and depressive symptoms, albeit a partial one. Post hoc examinations of these results revealed that behavioral avoidance accounted for over 40% of the relationship between these affective symptoms and was a statistically significant mediator. Although it appeared behavioral avoidance was a significant variable in this relationship, further investigation was needed, particularly because these findings lacked temporal testing, a critical element in mediational investigations.

## 7.2. Temporal mediation

To test the temporal hypothesis, we examined the effects of changes in behavioral avoidance over the course of individual treatment for SAD; that is, we sought to test the hypothesis that decreases in behavioral avoidance lead to later decreases in depressive symptoms. Pre-mid changes in behavioral avoidance significantly correlated with the mid-post changes in depressive symptoms in this sample, suggesting that early changes in behavioral avoidance are associated with later changes in depressive symptoms among socially anxious adults, supporting the proposed mediational model.

### 7.3. General discussion

Sparse research attention has been devoted to uncovering the associations between depression and social anxiety. As previously noted, epidemiological data support the temporal sequencing of SAD onset followed by the development of MDD (Kessler et al., 1999; Schneier et al., 1992). However, the reasons that only some people with SAD develop MDD have remained unclear. We hypothesized behavioral avoidance, as a result of social anxiety, plays a mediational role in the maintenance of depressive symptoms because it may lead to vulnerabilities such as loneliness and loss of social support that have been linked to depressive symptoms (Booth et al., 1992; Russell & Cutrona, 1991).

The present findings support the hypothesis that behavioral avoidance plays a significant role in mediating the relationship between SAD-related fear and depressive symptoms; albeit a partial one. Moreover, these data suggest decreases in avoidance are associated with later decreases in depressive symptoms. Although we do not directly address etiology, the present study design permitted results consistent with what might precipitate the development of depressive symptoms in adults with SAD. Therefore, the genesis of MDD in individuals with SAD may be explained in part by increases in social anxiety-related avoidance behavior.

The role of behavioral avoidance in social anxietylinked depression raises questions about the nature of the relationship between anxiety and behavioral avoidance. Herbert and Cardaciotto (2005) proposed a model of SAD emphasizing the role of experiential avoidance (attempts to minimize, avoid or suppress distressing private experiences; Hayes, Strosahl, & Wilson, 1999) in driving behavioral avoidance—future research is needed to examine the relationship between experiential avoidance and behavioral avoidance, and the role played by each in mediating the effects of social anxiety on depression.

## 7.4. Clinical implications

These findings suggest novel implications for understanding the nature and maintenance of comorbid social anxiety and depressive symptoms, highlighting the clinical importance for targeting of behavioral avoidances in psychotherapy. Some models of CBT for SAD stress the importance of focusing on behavioral avoidance (Rodebaugh, Holaway, & Heimberg, 2004). Clinical practitioners must continue to be vigilant about minimizing behavioral avoidance. The use of exposure therapy, activity scheduling, and exposure-based homework assignments should be emphasized. Additionally, novel CBT approaches to the treatment of SAD (e.g., Dalrymple & Herbert, 2007), warrant further investigation as they use exposure in addition to techniques to reduce experiential avoidance.

## 7.5. Strengths and weaknesses

Given the limitations of the study, these results should be viewed as preliminary and tentative, and should serve as an impetus for further research into the relationship between SAD and depression. Despite the variety of statistical approaches used in this study, an important limitation is the correlational nature of these results, which precludes definitive causal conclusions. In addition, the analyses focused on avoidance behavior, without incorporating other possible variables such as neurobiological characteristics of individuals that may underscore both social anxiety and depression. Finally, the study design did not fully meet criteria for formal mediation as outlined by Kraemer, Wilson, Fairburn, and Agras (2002) because we did not have data available for more than three time points and we did not have a control condition. A more careful analysis of the specific mediational role of behavioral avoidance secondary to SAD would come from data gleaned at multiple time points, perhaps from sessionby-session measures, which was not possible due to the clinical restrictions of the treatment program.

Despite these shortcomings, there are several noteworthy strengths of the study. The relatively restricted range of avoidance severity may be considered a strength, suggesting that even relatively subtle avoidance significantly exacerbates depressive symptomatology. Perhaps a more severe sample would even more clearly outline similar relationships. Another strength is the longitudinal nature of one set of our findings. Notably, this study is the first to our knowledge to investigate, longitudinally, a phenomenon that has been observed in the epidemiological data for over a decade. Replication is needed, particularly in a sample with a broader range of avoidance and utilizing more frequent symptom assessments.

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