THE IMPACT OF PERSONALITY PATHOLOGY ON TREATMENT RESPONSE TO THERAPEUTIC LIFESTYLE CHANGE (TLC) FOR DEPRESSION

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ABSTRACT

The present study investigated the impact of personality pathology on adherence and treatment response to a novel group-based intervention for depression, Therapeutic Lifestyle Change (TLC). Based on existing empirical literature documenting poor adherence and treatment outcome associated with comorbid Axis II pathology, patients with elevated levels of personality pathology were expected to exhibit less adherence and response to TLC for depression. Contrary to expectations, however, no significant association between Axis II pathology and treatment adherence was observed. It is possible that certain features of TLC, such as its highly structured approach, may facilitate adherence among personality-disordered patients. Nevertheless, despite similar levels of treatment adherence in comparison with the rest of the study sample, patients with elevated levels of personality pathology on either Cluster A or Cluster B responded significantly less well to TLC, even when controlling for initial depression severity. Failure of TLC elements to adequately target important features of Cluster A and Cluster B pathology, such as affective dysregulations and substantial interpersonal difficulties, may be responsible, in large part, for the observed differential treatment response.

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The Impact of Personality Pathology on Treatment Response to Therapeutic Lifestyle Change (TLC) for Depression

Depression is a burgeoning public health problem (Keller & Boland, 1998; NIMH, 1999), with an estimated lifetime prevalence rate in the U.S. of approximately 25% (Kessler et al., 2005). In fact, more than 19 million adult Americans now experience depressive illness each year (Ingram, Scott, & Siegle, 1999; National Institute of Mental Health, 1999). Depression is increasingly viewed as a chronic, lifelong condition marked by recurring cycles of recovery and relapse (Frank et al, 1991; Grilo et al., 2005; Mueller et al., 1999), with each successive episode of illness characterized by symptomatology of increasing severity (Thase & Howland, 1994). Accordingly, the disorder is now projected to become the second overall cause of disability worldwide by the year 2020 (Murray & Lopez, 1996).

A large proportion of depressed individuals suffer from co-occurring personality disorders (PD) (Ilardi & Craighead, 1995), which adversely affect cognition and occupational and social functioning (Post, 1994; Young, Weinberger, & Beck, 2001). Personality disorders have been conceptualized as the presence of enduring patterns of perception, cognition, and behavior that are generally inflexible and maladaptive and cause either significant functional impairment or subjective distress (American Psychiatric Association, 1987). The *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV; American Psychiatric Association, 1994) groups Axis II disorders into three clusters according to shared characteristics or features. Cluster A, characterized by "odd" and "eccentric" cognition and behavior, is comprised of schizotypal PD, schizoid PD, and paranoid PD. Cluster B, labeled as the "dramatic" and

"erratic" cluster, encompasses borderline PD, antisocial PD, narcissistic PD, and histrionic PD. Finally, cluster C, characterized by "anxious" and "fearful" thinking and behavior, includes avoidant PD, dependent PD, and obsessive-compulsive PD.

The high degree of comorbidity between depression and PD has been documented by a number of researchers, with observed co-occurrence rates generally ranging from 35% to 87% (Charney et al., 1981; Friedman et al., 1983; Hardy et al., 1995; Shea et al., 1987). This high prevalence of PD in depressed populations has generated considerable interest in the relationship between depression and personality pathology, and several hypotheses have been proposed. Due to its potential clinical utility, the *pathoplasty* model (Klein, Wonderlich, & Shea, 1993; Shea & Yen, 2005) has informed the majority of research in this area. This model assumes distinct etiology, but emphasizes the influence of each co-occurring condition on the presentation and clinical course of the other. In other words, personality pathology may influence the way in which depression is experienced and expressed, and depression may in turn exacerbate the manifestation of the personality pathology.

ACUTE TREATMENT OUTCOME FOR DEPRESSION

The introduction of a separate axis for the diagnosis of personality disorders in the DSM-III (American Psychiatric Association, 1980) brought about a widely accepted conceptualization of PDs and depression as distinct disorders. Consequently, there has been a steady increase in interest in the influence of PD's on the general course of depression, with a particular focus upon treatment response (Shea et al., 1992). The majority of researchers and clinicians have shared the assumption that the co-occurrence

of Axis II disorders and depression may have a deleterious effect on treatment outcome. Given the definition of PDs as enduring, inflexible maladaptive patterns of thinking, behaving, and feeling, it would appear logical to infer that individuals with such characterological dysfunction could prove more difficult to treat, and less responsive to clinical interventions (Saulsman, Coall, & Nathan, 2006).

Early studies in this area primarily focused on somatic treatments for depression (e.g., antidepressant medications), and these studies generally confirmed the hypothesis of PD's exerting a negative impact on the treatment outcome (Farmer & Nelson-Gray, 1990; Kuyken, 2001; Perry, Banon, & Ianni, 1999; Persons & Burns, 1985; Persons, Burns, & Perloff, 1988; Pilkonis & Frank, 1988; Reich & Green, 1991; Reich & Vasile, 1993; Whisman, 1993). Shea and colleagues (1992) noted in their review that the majority of published findings support the common belief that PD's are associated with poorer response to both pharmacologic and psychotherapeutic treatment of depression. The authors also observed a clinical acceptance of the belief that individuals with PD's are typically unresponsive to pharmacotherapy.

Interestingly, in a study of 160 depressed inpatients, Charney et al. (1981) observed that 71% of non-comorbid depressed patients had been prescribed antidepressants compared with only 28% of comorbid PD and depressed patients. Shea and colleagues (1992) hypothesized this treatment discrepancy is due largely to the common belief among clinicians that depression with comorbid PD represents a distinct, perhaps less biologically-based, disorder (Akisal, Hirschfeld, & Yerevanian, 1983). Thus, individuals with comorbid depression and PD – who typically struggle with considerable interpersonal difficulties along with social and occupational impairment –

have been historically viewed as less amenable to treatment with pharmacotherapies designed to address dysregulations in neurotransmission in the central nervous system (Ilardi & Craighead, 1995). Consequently, many early researchers regarded somatic treatments, relative to psychotherapeutic treatments, as less effective in treating individuals with depression and comorbid PD. Furthermore, Shea and colleagues (1992) suggested that the presence of personality pathology and its consequences (e.g., difficulties with interpersonal relationships, lack of adequate social support, high level of perceived life stressors, etc.) complicates response to treatment, makes afflicted patients more resistant to treatment in general, and leaves them with a persistent vulnerability to future depressive episodes.

Ilardi and Craighead (1995) conducted a review of the relevant literature that corroborated the conclusions of Shea and colleagues (1992). These investigators found robust evidence, based on 14 empirical studies, that Axis II disorders predict relatively poor outcomes with somatic treatments (pharmacotherapy and ECT), both acutely and in the long-term. Moreover, the authors discovered a strong negative correlation between the total number of Axis II criteria met and overall pharmacologic treatment response, and an analysis of 7 additional studies that used non-DSM measures of personality pathology (e.g., neuroticism, dependency, etc.) yielded additional supportive evidence. However, the authors noted that insufficient evidence was available to conclude that specific personality disorders or clusters of disorders were associated with especially negative outcomes.

The contention that comorbid Axis II PD predicts relatively poorer response to treatment for depression has not gone unchallenged. Mulder (2002, 2004; Mulder, Joyce,

& Luty, 2003) has written several reviews asserting that, although clinical intuition and early studies assume a sense of pessimism associated with comorbid PD and depression, empirically the answer is not so clear. He suggests that depressed individuals with comorbid PD "fare little if any worse" compared to depressed individuals without co-occurring PD (Mulder, 2002; 2004), and that significant differences in treatment outcome cited in past reviews are simply attributable to differences in study design. Furthermore, Mulder avers that the best-designed studies (i.e., ones that utilized randomized controlled trials and structured interview for personality pathology assessment) reported the least effect of personality pathology on treatment outcome for depression. Thus, Mulder (2004) concludes that the negative effect of co-occurring PD on treatment outcome for depressed individuals may be less robust than previously believed.

Reich (2003), however, has strongly challenged Mulder's claims. In his review of 11 relevant studies that have used psychometrically sound diagnostic instruments to confirm the presence of Axis II pathology, Reich (2003) found that all but one study observed a significantly poorer response to treatment of depression among individuals with comorbid PD. This phenomenon appears to be driven in part by the tendency of Axis II co-morbid patients to experience higher rates of treatment dropout. Although Reich noted that no PD has been differentially associated with especially poor antidepressant response, the magnitude of overall symptom reduction tends to covary inversely with an additive (dimensional) measurement of PD traits across all Axis II disorders. Notably, a recent meta-analysis of the relevant literature (Newton-Howes, Tyrer, & Johnson, 2006) lends support to Reich's (2003) position. Aggregating across all studies, Newton-Howes and colleagues observed poor treatment response among

approximately 55% of individuals with co-occurring PD, compared with an unsatisfactory response among only 45% of individuals without comorbid PD.

As noted previously, reviews of the effect of personality pathology upon treatment outcome have largely focused on psychopharmacological treatments for depression. Increasingly, though, researchers have turned their attention to the influence of comorbid PD on outcomes in psychotherapy for depression, with a particular focus on cognitive-behavior therapy (CBT). Given the established efficacy (Beck, Rush, Shaw, & Emery, 1979; Dobson, 1989; Hollon, Shelton, & Loosen, 1991) and widespread use (De Rubeis & Crits-Christoph, 1998) of CBT to treat depression, it is no surprise that a growing number of studies exploring the impact of co-occurring PD on the treatment response of CBT for depression are emerging in the empirical literature.

Shea and colleagues (1990) evaluated the relationship between PD and treatment outcome in the NIMH Treatment of Depression Collaborative Research Program (TDCRP). The authors reported that patients without PD responded better to treatment (i.e., had lower depression scores at termination) than patients with comorbid PD in all treatment conditions, except in the CBT treatment condition. That is, in the CBT condition the patients with comorbid PD actually performed slightly better than the patients without PD. This finding was certainly unexpected and stimulated interest in the research field.

Moreover, a recent review (Hirani, 2007) provided substantial evidence that the presence of a co-occurring PD does not appear to have an adverse impact on treatment response to CBT for depression. That is, eight out of the ten studies that have investigated the impact of Axis II PD on the response to CBT for depression reported that

the rates of improvement in depressive symptomatology in patients with and without co-occurring PD were not significantly different (Hardy et al., 1995; Kuyken, Kurzer, DeRubeis, Beck, & Brown, 2001; Patience, McGuire, Scott, Freeman, 1995; Persons, Burns, & Perloff, 1988; Saulsman, Coall, & Nathan, 2006; Shea et al., 1990; Stuart, Simons, Thase, & Pilkonis, 1992; Van den Hout, Brouwers, & Oomen, 2006)

The review (Hirani, 2007) noted that a majority of the studies reported elevated posttreatment depression scores for patients diagnosed with comorbid Axis II disorders compared to patients without an Axis II disorder; however, these studies also generally observed a comparable elevation of pretreatment depression scores for the PD groups. Thus, although the patients with comorbid PD exhibited more residual depressive symptomatology, the rate of improvement was similar. In the end, the reviewed studies provide considerable evidence that the presence of a comorbid Axis II disorder did not significantly impact treatment response to CBT for depression.

The reason for the differential treatment response of CBT in treating depression in patients with co-occurring PD compared to other treatments remains unclear. It is perhaps plausible to infer that CBT is specifically beneficial in the treatment of depression in individuals with comorbid PD largely because of its highly structured, time-limited, and problem-focused nature (Freeman, Pretzer, Flemming, & Simon, 1990; Patience, et al., 1995, Shea, et al., 1990). That is, because patients with co-occurring PD and depression tend to experience affective dysregulation, substantial interpersonal difficulties, and multiple crises (Zaretsky, Rosenbluth, & Silver, 2005), they may be more responsive to a direct approach that facilitates the separation of problems into specific tasks and allows for the addressing of depressive symptomatology before attending to

more complex personality difficulties (Hardy et al., 1995). Finally, Beck and Freeman (1990) have posited that, unlike individuals suffering solely from Axis I MDD, depressed individuals with comorbid Axis II PD experience chronic activation of negative schemas even when they are not severely depressed. If this in indeed the case, then the decrease in depressive symptomatology following CBT for this population may be directly related to the modification of their dysfunctional belief system and the development of new strategies to help them expand their perception of distressing situations and become more flexible in their response to stress.

An examination of the empirical literature reveals that far less attention has been given to the evaluation of the impact of comorbid PD on treatment outcomes in interpersonal psychotherapy (IPT) and psychodynamic psychotherapies for depression. Nevertheless, the majority of relevant studies have identified the presence of co-occurring PD as a negative prognostic indicator in the treatment of depression. The large-scale NIMH TDCRP study (Shea et al., 1990), for example, observed that comorbid PD adversely affected treatment response of IPT for depression. In two other randomized controlled trials, Frank and colleagues treated outpatients diagnosed with recurrent unipolar depression with imipramine and IPT, and they observed that the presence of a comorbid PD was associated with a significantly slower response to treatment (Frank, Kupfer, Jacob, & Jarrett, 1987; Pilkonis & Frank, 1988). In addition, a study of 76 depressed outpatients treated with IPT reported that patients with greater co-occurring personality pathology were significantly less likely to respond to IPT for depression (Bearden, Lavelle, Buysse, Karp, & Frank, 1996).

Likewise, studies investigating the effect of co-occurring PD on the treatment response of psychodynamic psychotherapy for depression have typically observed significantly poorer outcomes for PD clients than NPD clients. Hoffart and Martinsen (1993), for example, treated 77 depressed inpatients with psychodynamic psychotherapy. The authors reported worse outcome in terms of post-treatment depression severity for dependent PD and paranoid PD. Moreover, depressed patients with comorbid avoidant PD reported significantly more depression symptomatology at 1-year follow-up compared to depressed patients without PD (Hoffart & Martinsen, 1993). Furthermore, Hardy et al. (1995) reported that depressed patients with comorbid Cluster C PD's demonstrated a less favorable response to psychodynamic interpersonal psychotherapy vis-à-vis post-treatment BDI.

The evidence in support of a negative effect of comorbid PD on acute treatment outcome of depression in electroconvulsive therapy (ECT) does not appear to be as robust compared to that found for other treatments for depression. For example, Pfohl, Stangle, & Zimmerman (1984) reported that depressed patients with comorbid PD responded as well as NPD patients to ECT. In their review, Ilardi and Craighead (1995) pointed out that the findings from the (ECT) studies available were less conclusive, but a meta-analysis suggested a relatively poorer outcome for individuals with co-occurring depression and PD. Moreover, DeBattista and Mueller (2001) asserted in their review that the presence of a co-occurring PD, especially borderline PD (BPD) was associated with a less favorable treatment response to ECT. In a more recent review, Mulder (2002) noted that, although there was a trend towards worse outcome in all the studies reviewed, the presence of a comorbid PD did not significantly impact ECT treatment outcome for

depression. Finally, Newton-Howes, Tyrer, and Johnson (2006) conducted a meta-analysis and concluded that comorbid PD was associated with a relatively poorer treatment outcome for all treatments of depression (i.e., psychotherapies and pharmacotherapies) with the exception of ECT.

LONG-TERM TREATMENT OUTCOME FOR DEPRESSION

Several studies have provided support for the hypothesis that comorbid PD has a deleterious effect on not only acute treatment outcome for depression, but also for longer-term treatment outcome. Thompson, Gallagher, and Czirr (1988), for example, treated patients with late-life depression with cognitive therapy (CT), behavior therapy, or brief psychodynamic therapy and followed them for 2 years after treatment completion. The authors (Thompson, Gallagher, & Czirr, 1988) reported that the presence of a co-occurring PD was a risk factor for depression following treatment. In addition, Ilardi & Craighead (1995) reported in their review that the presence of a comorbid Axis II disorder was associated with an increased chance of relapse following pharmacological treatments for depression. Furthermore, in a naturalistic study that followed depressed patients for 2-years, Viinamaki et al. (2003) reported that comorbid Cluster C PD was associated with inferior recovery compared to depressed patients in the absence of any PD. Grilo and colleagues (2005) also conducted a naturalistic 2-year follow-up and concluded that co-occurring PD predicted slowed remission from major depressive disorder (MDD) even when controlling for negative prognostic indicators, such as total number of Axis I disorders, dysthymia, single versus recurrent MDD, and age of onset of MDD. It should also be noted, however, that a recent review reported that co-occurring

PD was not associated with significantly reduced maintenance of treatment gains following CBT for depression (Hirani, 2007).

EFFECT OF PD ON ADHERENCE

Treatment noncompliance is a serious issue across intervention modalities, and it has been asserted that the major reason for the discrepancy between efficacy and effectiveness of treatments for psychological disorders is poor treatment adherence (Sajtovic, Davies, & Hrouda, 2004). Cahalane (1997) noted that as few as 28% of clients requesting services at outpatient mental health clinics actually complete a course of treatment. Also, in a large study of inpatients and outpatients diagnosed with MDD, Melartin et al. (2005) observed that 49% of patients who were prescribed antidepressants terminated their treatment prematurely. Moreover, nonadherence has been closely associated with poorer treatment outcome, relapse, rehospitalization, and suicide among patients with major mental illness (Colom & Vieta, 2002; Delaney, 1998; Muller-Oerlinghausen, Muser-Causemann, & Volk, 1992; Scott, 2000).

A number of studies have documented an adverse impact of PD on adherence to several treatments. Ilardi and Craighead (1995) note that depressed patients with co-occurring PD, especially Cluster B, are more prone to medication noncompliance. They also point out that depressed patients with comorbid PD seem to respond slower to antidepressants, perhaps due to poor adherence to prescribed medication schedules. In a similar vein, Sajtovic et al. (2004) reported in their review that the presence of comorbid PD is associated with less adherence to medication and psychotherapy in patients with bipolar disorder.

A negative effect of comorbid PD on treatment adherence has also been observed in studies evaluating psychotherapeutic treatments for Axis I disorders. Persons, Burns, and Perloff (1988), for example, reported that Axis II pathology predicts premature termination from CT for depression. In addition, Cahalane (1997) reported that co-occurring PD predicted significantly poorer adherence to CBT treatment for anxiety disorders. Furthermore, Andreoli et al. (1989) reported that comorbid PD was associated with significantly poorer working alliance with the therapist and higher incidence of dropout in inpatients treated for Axis I disorders.

The literature on substance use disorders (SUD) provides robust evidence for the negative impact of comorbid PD on treatment adherence. Herbeck and colleagues (2005) investigated treatment adherence in 342 SUD outpatients seen in routine psychiatric practice; the authors reported that co-occurring PD was the strongest predictor of treatment compliance problems. Furthermore, in a review of 22 studies, Havens and Strathdee (2005) concluded that the presence of antisocial PD (ASPD) was associated with significantly poorer treatment adherence to opiod treatment.

Personality pathology has also been shown to have a negative impact on adherence to certain behavioral elements that have exhibited antidepressant effects, such as exercise and sleep (Dunn, Trivedi, Kambert, Clark, & Chambliss, 2005; Kuo, Manber, & Loewy, 2001; Mather et al., 2002; Morawetz, 2003). A meta-analysis of 15 studies, for example, concluded that certain personality traits that are characteristic of individuals with PD (e.g., insecurity, social introversion, psychasthenia, etc.) were associated with less exercise adherence (McDonald & Hodgdon, 1991).

In addition, several published studies have demonstrated a positive association between personality pathology and disturbed sleep patterns. A recent study of sleep patterns among college students reported that people with poor sleep quality are less emotionally stable, less self-assured, less observing of rules and regulations, more skeptical of the motives of others, and have higher trait anxiety (Jenkins, 2005). Again, these traits are often observed in PD individuals who, by definition, struggle with interpersonal relationships and social functioning. In addition, Philipsen and colleagues (2005) reported that individuals with BPD exhibited depression-like REM sleep abnormalities. A similarity in EEG sleep profile between BPD and depressed patients was also observed by Aasad, Okasha, & Okasha (2002) suggesting a common biological origin for both disorders. Finally, Dagan and colleagues (1996) reported that the presence of a PD predicted a higher incidence of circadian rhythm sleep disorder (CRSD) diagnosis compared to a control group. Thus, personality pathology has been shown to predict poor adherence to not only numerous somatic and psychotherapeutic treatments for Axis I disorders, but also for documented antidepressant elements, such as exercise and sleep. Accordingly, it is reasonably hypothesized that this adverse effect of comorbid PD on treatment adherence is directly related to the relatively poorer treatment response observed in this population.

HYPOTHESIZED MECHANISMS

Several theories have been forwarded to explain the adverse impact of Axis II disorders on treatment adherence and outcome. For instance, Persons and Burns (1985) asserted that the higher incidence of dropout among depressed patients with comorbid PD

may be due to the relative lack of emotional improvement experienced during individual CT sessions. Kernberg (1975) posited that higher rates of dropout among comorbid PD and depressed patients can be attributed to transference and countertransference problems encountered in this population. In addition, it has been suggested that elevated levels of defensiveness to problems coupled with less motivation for change results in poorer treatment adherence and outcome in patients with co-occurring Axis II disorders. Furthermore, dropout among patients with PD and depression has been viewed as an expression of resistance to change or to the treatment process (Persons, Burns, & Perloff, 1988).

As noted earlier, it has been proposed that individuals with comorbid PD and depression experience elevated levels of depressotypic cognition even when they are not in a depressive episode (Beck & Freeman, 1990). According to this theory, Axis II disordered patients have depressive schemas that are continually activated causing them to perceive and process daily information in a biased manner. Consequently, this stable cognitive bias results in a propensity to experience dysphoric mood and residual depressive symptoms even in the absence of a full-blown depressive episode. Furthermore, it has been suggested that this depressotypic cognitive bias that is characteristic of patients with PD results, in part, in the perception and experience of more negative life events than NPD individuals. This depressotypic bias and higher prevalence of experienced negative life events may serve as a risk factor for depression onset, maintenance, and relapse in this population.

Hypothesized mechanisms for the adverse Axis II-related outcomes observed in many studies also include the interpersonal difficulties and problems in social functioning

characteristic of individuals with PD (Pfohl, Stangle, & Zimmerman, 1984; Shea et al., 1990). Shapiro (1978), for example, posited that increased observed rates of premature termination among depressed patients with borderline PD (BPD) are due to the characteristic perception among such patients of the therapist being insufficiently responsive to emotional needs. In addition, Cahalane (1997) pointed out that the interpersonal difficulties characteristic of individuals with PD limit the ability of these individuals to develop and maintain a working alliance with the therapist. Moreover, difficulties with trust observed in many patients with avoidant PD, paranoid PD, and BPD, may result in an anticipation of disappointment or rejection from the therapist; thereby, precluding a meaningful connection with the therapist and comparable treatment gains with patients without Axis II disorders. Also, the fear of being controlled by the therapist or the fear of giving up on secondary gains (e.g., the need to be taken care of) may also be related to the higher incidence of noncompliance and poor treatment response in patients with PD (Persons, Burns, & Perloff, 1988). Finally, the difficulties in developing and maintaining healthy interpersonal relationships typically observed in patients with PD results in the experience of less social support, less marital status, and lower quality of family relationships (Ilardi & Craighead, 1995; Shea, Widiger, & Klein, 1992). As social support has been shown to serve as a "buffer" for relapse of depression (George, 1989), it appears that the absence of adequate social support experienced by most individuals with PD leaves these patients vulnerable to future episodes of depression.

THERAPEUTIC LIFESTYLE CHANGE (TLC) FOR DEPRESSION

A clinical research team at the University of Kansas, headed by Dr. Stephen Ilardi, has developed of a novel treatment approach to depression based upon the introduction of six distinct lifestyle elements that have been independently shown to be efficacious in the treatment and prevention of depression – exercise (Blumenthal et al., 1999; Dunn, Trivedi, Kambert, Clark, & Chambliss, 2005; Fremont & Craighead, 1987; Mather et al., 2002), omega-3 fatty acid supplementation (Nemets, Stahl, & Belmaker, 2002; Peet & Horrobin, 2002), bright light exposure (Martiny, Lunde, Unden, Dam, & Bech, 2005), enhanced sleep (Morawetz, 2003; Kuo, Manber, & Loewy, 2001), anti-ruminative activity (Fennell & Teasdale, 1984; Gibbons, et al., 1985), and social support (Ezquiaga, Garcia, Pallares, & Bravo, 1999; George, 1989; Oxman, Berkman, Kasl, Freeman, & Barrett, 1992). The evolutionarily informed conceptual framework and rationale for TLC have been detailed elsewhere (Ilardi et al., 2005; Ilardi, Karwoski, Lehman, Stites, & Steidtmann, 2006; Karwoski, 2006). Nevertheless, a brief overview of the treatment is warranted.

TLC is a 15-week, 12-session group protocol that was developed as a response to the recent epidemic of depression observed in developed nations. The treatment is based on the theory that the significant rise in prevalence of depression (Seligman, 1988) is a result of the fundamental mismatch between the way our bodies are designed and the modern, post-industrial environment in which we live (Bowlby, 1969). That is, inasmuch as the human genus has spent 99% of its existence in a hunter-gatherer context, the human body is best adapted to an environment that differs in numerous key respects with that of the 21st-century developed world.

The incidence of depression among modern hunter-gatherer bands appears to be extremely low (e.g., Schieffelin, 1985), and may have been similarly low in ancestral times as a result of protective elements inherent to that milieu. Members of hunter-gatherer societies lived among small groups of people which afforded them close contact and social support. Also, these individuals engaged in significantly more exercise compared to individuals living in the modern environment as they were required to continually be active in order to obtain food and survive. In addition, their being outside for the majority of each day resulted in a considerable amount of bright light exposure in the form of sunlight. As there was no electricity, our human ancestors often retired to bed when the sun went down – resulting in significantly longer sleep duration in comparison with people living in modern industrialized contexts. Furthermore, the diets of people living in ancestral times were significantly different from diets typical in developed societies, namely in the dramatically higher comsumption of dietary omega-3 fatty acids – much of it derived from wild game and fish. Finally, it is believed that the relative absence of social isolation among hunter-gatherers renders them much less likely to ruminate (i.e., to engage in repetitive negative thinking), a process shown to predict the onset of depressive episodes, as well as the severity and chronicity of depressive symptoms (Just & Alloy, 1997; Lam, Smith, Checkley, Rijsdijk, & Sham, 2003). In short, we as humans have adapted to the hunter-gatherer context in which we have spent nearly all of our existence. Consequently, the significant reduction in elements that are believed to have "naturally" limited the incidence of depression, as clinical depression does not confer any fitness advantages, in our modern, post-industrial environment has led to an epidemic of depression.

Hence, the promotion of the six lifestyle elements in TLC might serve to significantly reduce depressive symptomatology. Indeed, initial evaluation of the TLC protocol has documented impressive results (Karwoski, Stites, Lehman, & Ilardi, 2007), with 86% of depressed individuals treated with TLC experiencing a favorable treatment response, in comparison with a response rate of 22% among waitlist controls who received treatment-as-usual in the community.

PRESENT STUDY

Numerous reviews have identified the presence of co-occurring Axis II pathology as a marker of poor depression treatment response and increased probability of relapse following treatment (e.g., Ilardi & Craighead, 1995; Reich & Green, 1991; Shea et al., 1992). Some more recent reviews, however, have brought this long-standing belief into question (Hirani, 2007; Mulder, 2002). In an attempt to facilitate a better understanding of the impact of comorbid Axis II disorders on the response to treatment for depression, the present study examined the effect of co-occurring personality disorders on the adherence and treatment outcome of a promising, novel treatment for depression, TLC (Ilardi et al., 2005; Ilardi et al., 2007).

It is the responsibility of researchers in this field to determine what treatment works, for whom, and under what circumstances. This quest encompasses sound investigations aimed at uncovering factors that are (and are not) responsible for heterogeneity in treatment response. Thus, the impact of comorbid personality disorders on TLC treatment adherence and outcome for depression constitutes an important scientific question with significant clinical implications. In short, the presence of an Axis

II disorder may have considerable consequences vis-à-vis treatment planning. It is possible that typical TLC alone may not be sufficient for the adequate and effective treatment of many depressed patients with co-occurring PD.

The present study addressed several methodological issues that have been encountered in this research domain (Mulder, 2002). First, it statistically controlled for important clinical characteristics of participants, such as initial severity of depression. A reason for the aforementioned discrepancies in the published Axis II-depression literature may be due in part to the use of posttreatment depression scores alone (i.e., end-state functioning) to determine treatment outcome. As noted earlier, these studies often failed to take into account that, although PD groups had significantly more depressive symptomatology at the end of treatment compared to depressed patients without PD, the rate of improvement was often equivalent due to the higher initial depression scores of the PD groups. Moreover, an evaluation of more recent studies demonstrates an increasing acceptance of treatment outcome conceptualized as rate of improvement. Thus, this study statistically controlled for initial depression severity and defined treatment outcome as rate of improvement.

Second, as relevant information is often lost with the exclusive employment of categorical variables, the proposed study utilized continuous personality pathology and outcome data in their analyses (Ilardi, Craighead, & Evans, 1997; Mulder, 2002). Third, a structured interview (i.e., SCID-II) with documented validity and reliability (First, Spitzer, Gibbon, & Williams, 1995) was used to assess personality pathology. The use of a structured interview, rather than a self-report measure or clinician's chart review, is recommended for the assessment of Axis II PD as it is more valid, more reliable, and less

sensitive to the depressotypic cognitive bias that leads to overreporting of personality pathology associated with depressed mood (Mulder, 2002; Zimmerman, 1994).

Fourth, in an effort to promote generalizability, depressed participants in the study were allowed to continue adjuvant pharmacotherapy for depression. Fifth, data from participants who dropped out of treatment were included in the analyses. It has been argued that eliminating dropout data ignores an important outcome variable (Reich, 2003). That is, if individuals with co-occurring PD and depression do worse in treatment because they drop out, it is still indicative of a difference in treatment response between depressed individuals with and without comorbid PD. Finally, a series of multiple regression analyses and a macro (Preacher & Hayes, 2007) were employed to enable detection of a potential mediational role of TLC treatment adherence to treatment response. That is, the present study informs the question of whether treatment adherence is the mechanism through which co-occurring PD has an effect on TLC treatment outcome.

The pessimism generated by past reviews regarding the treatment response of patients with co-occurring depression and PD appears to be largely warranted. Although recent reviews (e.g., Hirani, 2007; Mulder, 2002) report that the presence of co-occurring Axis II disorder does not significantly impact outcome for every treatment for depression, the empirical literature provides evidence that comorbid PD is a negative prognostic indicator for most treatments for depression. Moreover, as noted previously, several studies have reported an adverse effect of PD on treatment adherence to pharmacotherapy, sleep, exercise, and various psychotherapies. Thus, the hypotheses of the proposed study are enumerated as follows:

(1) the presence of comorbid personality pathology will be associated with less treatment adherence to TLC for depression;

(2) the presence of comorbid personality pathology will be associated with poorer treatment response to TLC for depression;

(3) treatment adherence to TLC will be associated with treatment response vis-à-vis reduction in depressive symptomatology; and

(4) the effect of comorbid personality pathology on treatment response will be mediated by treatment adherence.

METHOD

The study methodology is identical in most respects to that detailed previously by Karwoski (2006). However, unlike the previous investigation, the primary goal of this study is the evaluation of the effect of comorbid PD on TLC treatment adherence and outcome. Thus, the key differences, primarily in the assessment of personality pathology, are clarified below.

Participants

As described in Karwoski (2006), participants were recruited through fliers, community referrals, in-class announcements, and newspaper, magazine, and television coverage. Interested individuals were called and screened by telephone. In this initial screening, they were asked the first nine questions from the Structured Clinical Interview for DSM-IV Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1997) mood module to determine the likelihood of their receiving a diagnosis of major depressive disorder. Likely candidate participants were invited to be evaluated in person by a trained graduate

level research assistant to determine eligibility. This evaluation included completion of the SCID mood disorders, substance abuse, and psychotic disorders modules, the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) and Hamilton Rating Scale for Depression (HRSD, Hamilton, 1960) to determine baseline ratings of depressive symptomatology. Subjects were eligible to participate if they received a current SCID-based diagnosis of major depressive disorder according to the DSM-IV diagnostic criteria, were between the ages of 18 and 65, and did not have psychotic symptoms, a substance abuse diagnosis, a history of self-harm behavior during the past two years, or active suicidal ideation.

Measures

Participants were assessed by trained graduate students on the mood, substance abuse, and psychotic modules of the SCID. The SCID is a structured interview which uses standardized clinician-directed queries designed to assess Axis I conditions including depression. Research has provided evidence of the reliability of Axis I diagnoses assigned on the basis of the SCID, with inter-rater agreement kappas ranging from .70 to 1.00 in community and clinical samples (Segal, Hersen, & Van Hasselt, 1994). In fact, the SCID is considered the "gold standard" of diagnostic classification in clinical research settings due to this high level of inter-rater reliability.

Participants were also given the SCID-II (Spitzer, Williams, Gibbon, & First, 1990) within the first 2 weeks of treatment. The SCID-II is a clinician-administered structured interview for diagnosing the 11 Axis II personality disorders of the DSM-III with documented reliability and validity (First, Spitzer, Gibbon, & Williams, 1995). It should be noted that depressive PD was not assessed as it is listed as a "candidate" Axis

II diagnosis in the DSM-IV appendix. The SCID-II was given by a trained graduate student to diagnose PD in each participant. In addition, dimensional scores of personality pathology for each individual cluster as well as overall personality pathology were computed from data obtained from the SCID-II assessment (First, et al., 1995). These scores were computed by summing the number of personality disorder criterions rated as positive. Specifically, 0 points were scored for a personality disorder criterion rated as "absent", 1 point was scored for a criterion rated as "subthreshold", and 2 points were scored for a criterion rated as "threshold". In general there are two or three interview questions for each of the 79 personality disorder criterions and raters are instructed to assign a "threshold" rating if there is sufficient evidence that the characteristic described in the criterion is pathological, persistent, and pervasive (First, et al., 1995). The dimensional PD score can range from 0 to 158. The Cluster A dimensional score ranges from 0 to 46; Cluster B has a range of 0 to 66; and, Cluster C can range from 0 to 46.

Moreover, the SCID-II questionnaire was given to each participant at the screening and at the end of treatment. The SCID-II questionnaire is a 119-item, self-report personality assessment that can be used in conjunction with the SCID-II and provides some insight into the influence of depressive episode on over-reporting of personality pathology (First, et al., 1995). The SCID-II questionnaire score can range from 0-119 as 1 point is scored for each endorsed item. Elevated scores are indicative of higher levels of personality pathology. Furthermore, 8 SCID-II assessments were videotaped and scored by trained graduate students to evaluate inter-rater reliability.

The primary outcome measure for the proposed study is the BDI-II (Beck, Steer, & Brown, 1996), a widely used self-report measure for depression that includes 21 items,

each of which is scored from 0 to 3 to reflect the intensity of corresponding depressive symptoms. The respondent's score is the sum of these item scores and can range from 0 to 63. The BDI–II and its predecessor, the BDI, have been the most widely used self-report measures of outcome in research on cognitive therapy for depression (Beck, Steer, & Garbin, 1988). Psychometric studies indicate that the BDI-II has high internal reliability, with an estimated coefficient alpha of .92 for psychiatric patients (Beck, Steer, & Brown, 1996).

The 17-item HRSD is a widely used clinician-rated scale that covers a set of affective, behavioral, and biological symptoms of depression, with scores ranging from 0 to 52. The HRSD has been found to have acceptable psychometric properties, with inter-rater reliability coefficients ranging from .83 to .94 across different studies (Rabkin & Klein, 1987). Moreover, the HRSD correlates relatively highly with the BDI-II, with studies showing a range of correlations from .68 to .72 (Beck, Steer, & Brown, 1996). The HRSD was given using the interview format developed by Williams (1988) and is included as a secondary outcome measure due to its widespread usage in the psychiatric literature.

As detailed in Karwoski (2006), adherence to TLC homework assignments were measured by asking the patients to record on Weekly Record Forms (Appendix A) each day the degree to which they are following the recommended lifestyle changes and a rating of their mood that day. The Weekly Record Forms were developed by our research team and were modeled after the Diary Cards widely used in Dialectical Behavioral Therapy (DBT; Linehan, 1993). The Weekly Record Forms begin by asking the patient to record whether they have taken the Omega-3 fish oil supplements, a

baseline measurement of sleep and exercise, and a rating of their mood on a Likert scale from 1 to 10. As successive lifestyle changes were introduced, patients were asked to record their adherence to corresponding homework assignments.

For hours of sleep, patients were given a score for compliance calibrated as the percentage of the targeted 8 hours of sleep obtained each night. Hypersomnolent patients had their compliance rating reduced by a commensurate percentage for every hour *above* 9 hours per night. For bright light exposure, a patient was considered 100% compliant if he or she got 30 or more minutes per day. Otherwise, compliance was rated as a percentage of the targeted 30 minutes each day and averaged across the weeks for which data is collected. Likewise, patients were considered 100% compliant with the exercise requirement if they got 35 or more minutes of exercise three times a week. Anything less was rated as a percentage of this target, and a weekly average was calculated. Patients reported whether they had taken their omega-3s for the day, so adherence was measured by averaging the number of days that patients reported taking the full proscribed dosage. Pleasant activities, which are considered anti-ruminative, were measured as the percentage of days per week that patients reported engaging in at least one of them. Patients were asked to engage in at least one social activity each day. Adherence to this TLC element was measured as the percentage of days per week that patients reported engaging in at least one social activity. It should be noted that the ratings of patients who adhered to the TLC elements more than the amount proscribed by the protocol do not reflect the "extra" adherence. That is, a patient that gets 30 minutes of bright light each day and a patient that gets 60 minutes of bright light each day are both considered 100% compliant.

Weekly Record Forms were analyzed to determine the degree to which patients in TLC actually made the major lifestyle changes. It should be noted that upon closer examination, the variable used to measure the sleep component in TLC was judged to be problematic. The data collected for sleep adherence (i.e., the percentage of the targeted 8 hours of sleep obtained each night) is closely aligned with depressive symptomatology. As both assessments of treatment response (BDI-II and HRSD) include items directly related to sleep, the amount of sleep a patient reports may be more of an artifact of their depression than an estimate of compliance to the proscribed 8 hours of sleep. That is, it would be hard to make the theoretical argument that a patient who gets less sleep due to the severity of his or her depression is less "adherent" to the TLC protocol. Consequently, due to this significant conflation of the sleep adherence variable and depressive symptomatology, it was determined that it would be best to leave this variable out of the analyses involving adherence.

Thus, estimates of adherence for 5 of the 6 components of TLC (i.e., omega-3 consumption, bright light exposure, exercise, pleasant activities, and social support) were obtained by computing a mean for each of the 5 variables of the corresponding adherence data collected during the weeks that patients were asked to record them. In addition, a weekly composite adherence score was computed for each participant by averaging the 5 mean z-normed weekly adherence scores for each TLC element. Moreover, a global adherence score was computed for each participant by averaging the mean, z-normed composite adherence scores for each of the 5 TLC elements. Furthermore, although their data was included in all analyses, patients were considered "dropouts" if they missed more than 3 sessions or stated that they would no longer like to attend group. If no

observation was documented at the end of treatment, data were carried forward from the last observation to serve as the outcome variable.

Procedure

Participants were invited to join the first available group, typically beginning within a few weeks of the intake screen. Each participant completed the BDI-II during the first five minutes of group each week. Additionally, each participant was re-assessed on the HRSD by a graduate student rater at the fifth session, the ninth session, and the final twelfth session. Raters were not explicitly told what treatment condition the patients were in.

Description of Protocol

As described in Karwoski (2006), Therapeutic Lifestyle Change groups followed a detailed 12-session protocol, developed by the members of the TLC Research Group under the direction of Dr. Stephen Ilardi at the University of Kansas (Ilardi et al., 2005). Each part of the protocol was written by Dr. Ilardi or a graduate research assistant in the lab. In addition, the TLC lab developed a set of patient handouts corresponding to each session of the group. A brief description of the structure and content of the group sessions is provided in this section.

The first TLC session is the most psychoeducational in nature. The group co-leaders outline the evolutionary rationale behind the program, emphasizing ways in which our modern environment is different from the ancestral environment and why this is important for depression. Co-leaders also conduct an exercise in which they describe the major areas of functioning affected by depression (mood, cognition, behavior, and physiology) and invite group members to share symptoms they have experienced. The

group concludes with a discussion of the role of diet on neurological function and distribution of the Omega-3 fatty acid supplements (with instructions on how to take them). Homework for the first session consists of taking the Omega-3 supplements with a multi-vitamin, beginning to record daily activities and mood on the weekly record form, and becoming aware of and monitoring when and how much they are ruminating.

Sessions 2 through 7 (and session 9) all follow the same basic format. The first 45 minutes of the session are spent reviewing homework from the week before. Homework assignments involve implementing a lifestyle change based on the psychoeducational topic for that week. When patients report problems with adherence, group members and leaders work to find solutions to obstacles. In the second part of each session, new material for that week is introduced. In the second week, members learn how to use behavioral activation strategies to combat rumination. In the third week, the concept of antidepressant exercise is introduced, and exercise consultants are present at the meeting to schedule each member's first workout. Each group member meets with an exercise consultant for three one-hour sessions in the three weeks following the exercise session to develop a workout plan and learn to monitor heart rate (i.e., to ensure that each workout is of aerobic intensity). During the next four group sessions, members are taught to get daily bright light exposure, to enhance social connectedness, develop better sleep hygiene, and to address the corrosive effects of the modern social environment upon self-esteem. Participants are either given a light box to use for the duration of the treatment or encouraged to use a light box that is available in Fraser Hall when the weather is not sunny.

Sessions 8, 10, 11, and 12 are all devoted to review and relapse prevention. Session 11 takes place in the twelfth week of the program, and session 12 takes place the fifteenth week. Spacing the final sessions out gives patients practice maintaining the lifestyle changes without the support of the group and co-therapists. During these final sessions, patients learn the importance of continuing to adhere to the lifestyle changes, troubleshoot obstacles to adherence in advance, and assess which elements were particularly helpful for them in overcoming depression.

As detailed in Karwoski (2006), the introduction of the material follows a specific order. Nutrition is the first major topic introduced, based on the rationale that taking a nutritional supplement and multivitamin is a relatively easy assignment that most patients should achieve success with. Also in the first session patients are asked to notice when they are ruminating but not to initiate any intervention to stop its occurrence. Again, this is a relatively easy assignment, and once patients notice what rumination is, they are often much more motivated to learn and implement behavioral strategies to combat it, which is the assignment for the second session. Exercise is introduced in the third session, with the idea being that this is early enough that the therapists will have time to help patients get in a regular exercise routine, but late enough so that patients will have had several success experiences and feel confident in their ability to make this change. Light exposure is introduced next, in the fourth session, since it is a major change, and also a change that some patients see benefit from relatively quickly. Socialization and sleep strategies are the last two major elements introduced because they are more easily modifiable in patients who have already begun the recovery process. Esteem

maintenance and *flow* are introduced late in the protocol because they can be considered extensions of, respectively, socialization and behavioral activation.

Aside from the group meetings, the co-therapists also contact each member of the group by phone weekly. Phone calls are targeted to last no longer than 15 minutes apiece, although there is not a strict limit. There are several reasons for these phone calls. At the beginning of therapy, the phone calls are an important way to build rapport, address any concerns the patient might have hesitated to bring up in front of the rest of the group, and begin to get to know the patient on an individual basis. As therapy progresses, the phone calls become much less important for the patients who are adhering well and seeing rapid symptom improvement, but for those who are struggling more, they become a way for the therapist to troubleshoot problems more thoroughly outside of the group context.

Data Analyses

Three principal hypotheses of the proposed study will be tested by means of regression analyses – specifically, that: (1) the presence of comorbid personality pathology is associated with less treatment adherence to TLC for depression; (2) the presence of comorbid personality pathology is associated with poorer treatment response to TLC for depression; and (3) treatment *adherence* to TLC is positively associated with treatment response. Moreover, it has been established that pre-treatment depression severity is positively correlated with level of post-treatment depressive symptomatology (e.g., Croughan et al., 1988). Thus, the importance of determining the extent to which TLC treatment response associated with co-occurring PD is due to the correlation between personality pathology and initial depression severity needs to be addressed

(Ilardi & Craighead, 1995). Consequently, initial depression severity will serve as a covariate in each multiple regression analysis.

Furthermore, the fourth hypothesis of the study postulates that adherence serves as a mediator for the effect of personality pathology on treatment response. One can infer the existence of mediation indirectly from the 3 regression analyses conducted for each model (Baron & Kenny, 1986). Nevertheless, even if the first three principal hypotheses are supported, it is possible that the indirect effect is still not significant. Therefore, a macro designed to generate estimates for indirect effects in a mediator model will be utilized to more directly evaluate the mediational role of adherence on treatment response (Preacher & Hayes, 2007). The macro employs a nonparametric bootstrapping procedure to generate a sampling distribution of the indirect effect of personality pathology on treatment outcome through adherence that does not involve making any distributional assumptions – a feat that can not be accomplished in a mediation test solely employing the three regression analyses of each model. Preacher and Hayes (2004) note "the bootstrapping is accomplished by taking a large number of samples of size *n* (where *n* is the original sample size) from the data, *sampling with replacement*, and computing the indirect effect in each sample (p. 722)." It has been suggested that this method effectively eludes the power problem presented by asymmetries and other forms of nonnormality in the sampling distribution of interest (Bollen & Steine, 1990). In addition, Preacher and Hayes (2004) assert that the macro produces a test that is not based on large-samples theory; thus, it can be applied to small samples with more confidence. 95% bias-corrected and accelerated confidence intervals will be generated to

determine if adherence significantly mediates the effect of personality pathology on TLC treatment response.

The four principal hypotheses were tested in each of 20 sets of models. The 20 sets of models were constructed by interchanging the 5 personality pathology variables, interchanging the 6 adherence variables, and interchanging the 2 treatment outcome measures. Specifically, one series of 10 sets of models was formed according to a 5 personality pathology variables (dichotomous PD, dimensional PD, Cluster A dimensional, Cluster B dimensional, and Cluster C dimensional) X 1 adherence variable (global) X 2 treatment outcome variables (posttreatment BDI-II and posttreatment HRSD) framework; and, the second series was constructed in a 1 personality pathology variable (dimensional PD) X 5 adherence variables (TLC components – omega-3, bright light, exercise, pleasant activities, and social support) X 2 treatment outcome variables (posttreatment BDI-II and posttreatment HRSD) framework. The testing of each model consisted of three regression analyses, in which initial depression severity served as the covariate, and a mediation test conducted with the aforementioned macro (Preacher & Hayes, 2007).

The global adherence variable served as the adherence variable in the first 10 sets of models. This variable was utilized as it gives the best sense of overall adherence to TLC treatment. The investigation of effects of adherence to the *components* of TLC was conducted with the testing of the second series of 10 additional sets of models. As relevant information is often lost with the employment of a categorical variable (Ilardi et al., 1997; Mulder, 2002), the dimensional PD score served as the personality pathology

variable in each set of models. This variable was employed because it provides the best sense of overall personality pathology.

Furthermore, a Fisher's Exact Test was conducted to evaluate between-group differences in treatment response, defined as at least a 50% reduction of depressive symptomatology on the BDI-II or HRSD. In addition, inter-rater reliability of the SCID-II personality pathology assessment and internal reliability of the Weekly Record Form were also assessed.

RESULTS

Characteristics of Patients

Sixty-eight patients met study inclusion criteria. Ages of patients ranged from 18-62 years with an average of 43.4. Twenty (29.4%) of the patients that entered TLC were male and thirty-three (48.5%) were married (Table 1). Also, the mean number of years of education was 16.0. In addition, at intake, fifteen patients (22.1%) were in psychotherapy and forty (58.8%) were on medication. Moreover, the mean baseline BDI-II score (i.e., at intake) was 28.9 with a SD of 8.9. Also, the mean baseline HRSD score, the study's secondary depression measure, was 19.3 with a SD of 5.7.

Table 1.
Characteristics of Participants According to Axis II Personality Disorder Diagnosis.

	Age Mean (SD)	**Male** No., (%)	**Married** No., (%)	**Education** Mean, (SD)	**In therapy at intake** No., (%)	**On medication at intake** No., (%)	**Baseline BDI-II** Mean, (SD)	**Baseline HRSD** Mean, (SD)
No PD (N=30)	44.4 (11.2)	8 (26.7)	13 (43.3)	16.4 (2.1)	8 (26.7)	20 (66.7)	25.8** (9.5)	17.9[a] (6.0)
PD	42.7	12	20	15.7[b]	7	20	31.4**	20.4

(N=38)	(12.0)	(31.6)	(54.1)	(2.5)	(18.4)	(52.6)	(7.7)	(5.3)
Total	43.4	20	33	16.0	15	40	28.9	19.3
(N=68)	(11.6)	(29.4)	(48.5)	(2.6)	(22.1)	(58.8)	(8.9)	(5.7)

** $p < .01$
[a] Data for this variable is missing for 1 participant.
[b] Data for this variable is missing for 2 participants.

Fifty-six percent (N=38) of patients were diagnosed with at least one Axis II personality disorder (PD) according to the SCID-II. The mean of the dimensional PD variable for the study sample (N=68), which ranged from 0 to 158, was 29.3 with a standard deviation (SD) of 16.0. Twelve patients in the study (17.6%) met diagnostic criteria for at least one Cluster A personality disorder. The mean of the Cluster A dimensional variable (N=68), which ranged from 0-46, was 7.4 with a SD of 5.6. Also, five patients (7.4%) received a diagnosis of at least one Cluster B PD; and, the mean of the Cluster B continuous variable, which ranged from 0-66, was 7.9 with a SD of 7.1. In addition, thirty-four patients (50%) were diagnosed with at least one Cluster C PD. The mean of the Cluster C dimensional variable, which ranged from 0-46, was 14.3 with a SD of 7.2. Furthermore, twenty-three patients (60.5%) in the PD group (N=38) were diagnosed with one Axis II PD, eight (21.1%) were diagnosed with 2 PD's, and 7 (18.4%) met diagnostic criteria for 3 or more PD's.

The PD group (i.e., patients diagnosed with at least one Axis II PD) (N=38) and the group of patients without an Axis II PD diagnosis (NPD) (N=30) were comparable on demographic variables (Table 1). Fisher's Exact Tests revealed that the PD and NPD groups did not differ in terms of sex ($p = .79$), proportion of patients in therapy at intake ($p = .56$), and proportion of patients on medication at intake ($p = .32$). Moreover, independent samples t-tests revealed that the two groups did not differ in terms of age

(t(66) = .591, p = .56) and level of education (t(64) = 1.21, p = .23). Although the difference between baseline HRSD scores among groups failed to reach the threshold of significance (t(65) = 1.79, p = .08; Axis II mean = 20.42; non-Axis II mean = 17.93), as expected, patients in the PD group scored significantly higher on the study's primary baseline depression measure, BDI-II (t(66) = -2.72, p < .01; Axis II mean = 31.42; non-Axis II mean = 25.77).

Reliability

An internal consistency estimate of reliability of the TLC Weekly Record Form (WRF) was computed on the basis of reported adherence data from session 10, following a week of treatment in which no new TLC element was introduced. The internal reliability test of the WRF yielded a Cronbach's alpha of .52, indicating that the WRF has a moderate amount of internal consistency. That is, there is reason to believe that the items on the WRF tap into an underlying adherence construct.

A test of inter-rater reliability was conducted using data collected from eight videotaped SCID-II assessments that were scored by trained graduate students on the TLC research team. A two-way random effects model yielded an intraclass correlation of .97, indicating very strong inter-rater reliability for the SCID-II personality pathology assessment.

A paired samples t-test was conducted to compare the scores of the self-report SCID-II questionnaire that was given at baseline and at posttreatment. The t-test indicates that patients obtained a significantly higher score on the SCID-II questionnaire given at baseline than the one given post-treatment (t(44) = 4.3, p < .01). Consistent with the literature (Mulder, 2002; Zimmerman, 1994), the t-test result suggests that the

presence of comorbid depression is associated with potential over-reporting of personality pathology on self-report measures – a problem that is largely addressed in the present study by means of employing a semi-structured interview (SCID-II) as the principal Axis II measure (Spitzer, Williams, Gibbon, & First, 1990).

Effect of Personality Pathology on Adherence to TLC Treatment

PD and NPD groups were comparable on adherence to components of TLC (Table 2). Independent samples t-tests indicate that the two groups do not significantly differ in terms of adherence to omega-3 ($t(66) = -.64$, $p = .52$), bright light exposure ($t(63) = .338$, $p = .74$), exercise ($t(66) = .565$, $p = .57$), pleasant activities ($t(65) = .797$, $p = .43$), and social activity ($t(48) = 1.33$, $p = .19$). In addition, a Fisher's Exact Test ($p = .49$) indicates that the rate of dropout among the PD and NPD groups is not significantly different. Moreover, Figure 1 shows that the PD and NPD groups exhibit a similar pattern of adherence throughout treatment. An independent samples t-test also indicated that there was no significant difference among the PD and NPD groups with regard to their global adherence to the TLC treatment ($t(66) = .80$, $p = .43$).

Table 2.

Degree of Adherence to TLC Components.

	Omega 3 %, (SD)	**Bright Light** %, (SD)	**Exercise** %, (SD)	**Pleasant Activity** %, (SD)	**Social Support** %, (SD)	**Global**[a] (overall) Mean, (SD)	**Dropout**[b] No., %
No PD (N=30)	88.8 (17.7)	66.0[c] (17.3)	66.0 (22.7)	69.6[d] (20.0)	60.7[e] (22.4)	.060 (.59)	3 (10)
PD (N=38)	90.1 (8.8)	64.3[f] (22.1)	62.9 (23.0)	65.9 (17.7)	51.7[g] (24.1)	-.05 (.52)	7 (18.4)
Total (N=68)	90.0 (13.4)	65.0 (20.0)	64.2 (22.8)	67.5 (18.7)	55.3 (23.6)	0.0 (.55)	10 (14.7)

[a] Calculated as the mean of the z-scored component adherence variables.
[b] Missed more than 3 sessions.
[c] Data for this variable is missing for 2 participants.
[d] Data for this variable is missing for 1 participant.
[e] Data for this variable was not collected for 10 patients (i.e., the revised Weekly Record Form had not been introduced during treatment for these participants).
[f] Data for this variable is missing for 1 participant.
[g] Data for this variable was not collected for 8 patients (i.e., the revised Weekly Record Form had not been introduced during treatment for these participants).

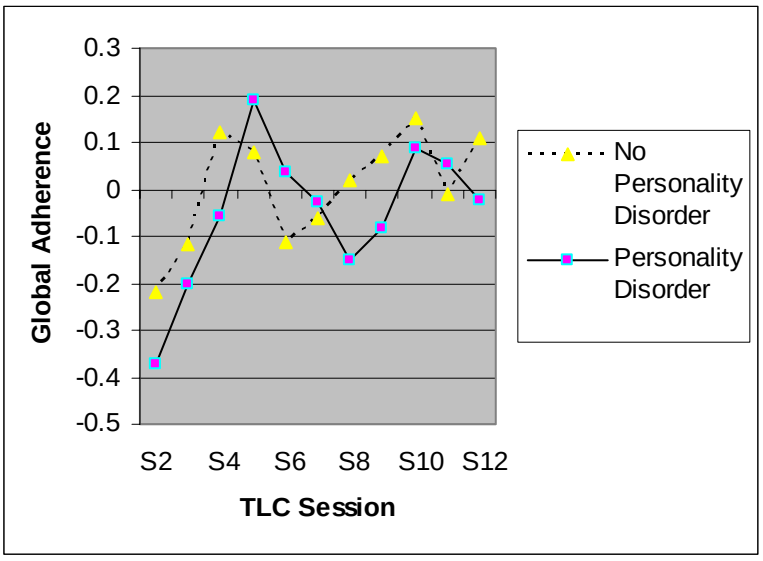

Figure 1. Global adherence by session.

Linear regression analyses were conducted to predict global adherence to TLC treatment from the dichotomous PD variable with pretreatment BDI-II and pretreatment HRSD (Table 3) serving as covariates. The regression results ($p = .36$ and $p = .17$, respectively) suggest that a diagnosis of at least one Axis II PD does not significantly predict overall adherence to TLC treatment after controlling for initial depression severity.

Table 3.

Summary of the Linear Regression Analyses for Personality Pathology predicting Global Adherence with Initial Depression Severity serving as a Covariate.

Variable	**B**	**SE B**	**β**
Pretreatment BDI-II	.004	.008	.07
Dichotomous PD	-.13	.14	-.12
Pretreatment HRSD	.03	.01	.30
Dichotomous PD	-.19	.13	-.171
Pretreatment BDI-II	.002	.008	.031
Dimensional PD	.000	.005	-.002
Pretreatment HRSD	.03	.01	.28
Dimensional PD	-.003	.004	-.08
Pretreatment BDI-II	.002	.008	.03
Cluster A	.001	.01	.006
Pretreatment HRSD	.03	.01	.26
Cluster A	-.002	.01	-.021
Pretreatment BDI-II	.002	.008	.04
Cluster B	-.001	.01	-.02
Pretreatment HRSD	.03	.01	.30
Cluster B	-.008	.01	-.106
Pretreatment BDI-II	.002	.008	.028
Cluster C	.001	.01	.01
Pretreatment HRSD	.03	.01	.28
Cluster C	-.005	.01	-.06

The hypotheses that dimensional PD predicts global adherence to TLC treatment with pretreatment BDI-II and pretreatment HRSD (Table 3) serving as covariates were also evaluated. The results of the regression analyses ($p = .99$ and $p = .53$, respectively) suggest that dimensional PD score does not significantly predict overall adherence to TLC after controlling for initial depression severity.

Linear regression analyses were also carried out to predict global adherence to TLC treatment from Cluster dimensional scores with pretreatment BDI-II and pretreatment HRSD serving as covariates. The results of the regression analyses for Cluster A ($p = .96$ and $p = .86$, with pretreatment BDI-II and pretreatment HRSD serving

as covariates, respectively), Cluster B (p = .89 and p = .41), and Cluster C (p = .94 and p = .62) (Table 3) suggest that one's Axis II cluster pathology does not significantly predict his or her overall adherence to TLC after controlling for initial depression severity.

Linear regression analyses were also conducted to predict adherence to TLC components from dimensional PD with pretreatment BDI-II and pretreatment HRSD serving as covariates. The regression results for omega-3 consumption (p = .87 and p = .76, with pretreatment BDI-II and pretreatment HRSD serving as covariates, respectively), bright light (p = .55 and p =.91), exercise (p = .86 and p = .68), pleasant activities (p = .58 and p = .50), and social support (p = .81 and p = .57) (Table 4) suggest that dimensional PD score does not significantly predict adherence to TLC components after controlling for initial depression severity.

Table 4.

Summary of the Linear Regression Analyses for Dimensional PD predicting Adherence to TLC Components with Initial Depression Severity serving as a Covariate.

TLC Component	**Variable**	**B**	**SE B**	**β**
Omega-3	Pretreatment BDI-II	.01	.02	.09
	Dimensional PD	.001	.008	.02
	Pretreatment HRSD	.01	.02	.06
	Dimensional PD	.003	.008	.04
Bright Light	Pretreatment BDI-II	-.008	.02	-.07
	Dimensional PD	.005	.01	.09
	Pretreatment HRSD	.04	.02	.21
	Dimensional PD	-.001	.008	-.01
Exercise	Pretreatment BDI-II	-.002	.02	-.02
	Dimensional PD	-.002	.008	-.02
	Pretreatment HRSD	.02	.02	.11
	Dimensional PD	-.003	.008	-.05
Pleasant Activity	Pretreatment BDI-II	.01	.02	.11
	Dimensional PD	-.005	.008	-.08

	Pretreatment HRSD	.02	.02	.13
	Dimensional PD	-.005	.008	-.088
Social Activity	Pretreatment BDI-II	-.01	.02	-.13
	Dimensional PD	.002	.01	.04
	Pretreatment HRSD	.03	.03	.16
	Dimensional PD	-.006	.01	-.09

Effect of Personality Pathology on TLC Treatment Outcome

Depressive symptomatology was greatly reduced for both the PD and NPD groups (Figure 2; Table 5). 23 patients out of 30 (77%) in the NPD group achieved a clinically significant treatment response, defined as a reduction of at least 50% in depressive symptomatology from baseline, vis-à-vis the BDI-II. 26 patients out of 38 (68%) in the PD group achieved a clinically significant treatment response. A Fisher's Exact Test was conducted to compare the rate of recovery among the two groups. The results of the test indicate that the PD and NPD groups do not differ significantly on rate of recovery ($p = .59$). Furthermore, 19 patients out of 29 (66%) in the NPD group achieved at least a 50% reduction in depressive symptomatology according to the HRSD compared to 19 patients out of 38 (50%) in the PD group. A Fisher's Exact Test indicates that this difference is also not significant ($p = .33$).

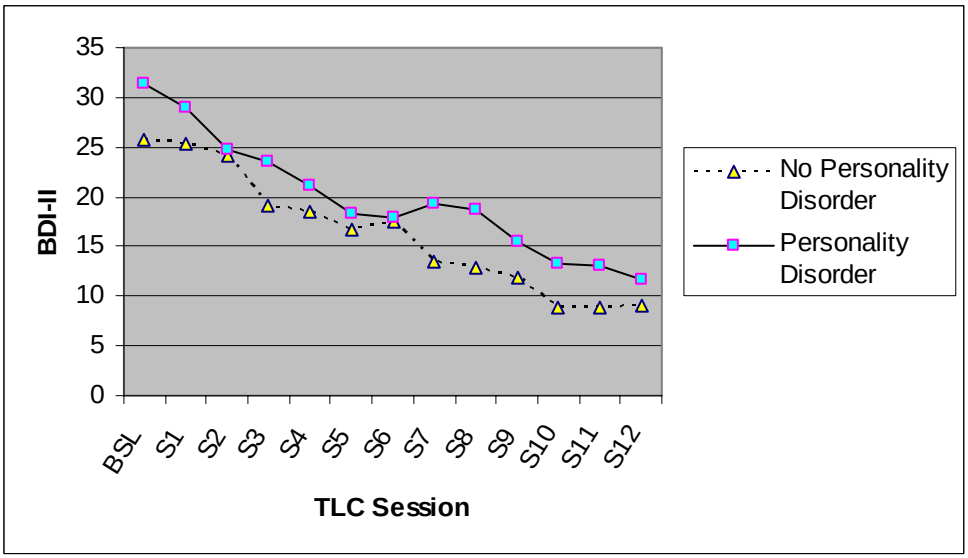

Figure 2. BDI-II scores by session for PD and NPD groups.

Table 5.

Mean Baseline and Post-Treatment BDI-II and HRSD Scores.

	No PD (N=30)	**PD** (N=38)
	MEAN (SD)	MEAN (SD)
BDI-II		
Baseline	25.8 (9.5)	31.4 (7.7)
Post-Treatment	9.0 (7.2)	11.6 (11.1)
HRSD		
Baseline	17.9 (6.0)[a]	20.4 (5.3)
Post-Treatment	8.0 (6.7)	10.8 (7.8)

[a] Data for this variable is missing for 1 participant.

Linear regression analyses were conducted to predict treatment response from the dichotomous PD variable with pretreatment BDI-II (Table 6) and pretreatment HRSD (Table 7) serving as corresponding covariates. The regression results ($p = .50$ and $p =$

.15, respectively) suggest that a diagnosis of at least one Axis II PD does not significantly predict treatment outcome after controlling for initial depression severity.

Table 6.

Summary of the Linear Regression Analysis for Dichotomous PD predicting Posttreatment BDI-II with Pretreatment BDI-II serving as a Covariate.

Variable	B	SE B	β
Pretreatment BDI-II	.16	.14	.15
Dichotomous PD	1.7	2.5	.09

Table 7.

Summary of the Linear Regression Analysis for Dichotomous PD predicting Posttreatment HRSD with Pretreatment HRSD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.13	.16	-.11
Dichotomous PD	2.7	1.8	.20

The hypotheses that dimensional PD is predictive of treatment response, with pretreatment BDI-II (Table 8) and pretreatment HRSD (Table 9) serving as corresponding covariates, were examined in linear regression frameworks. The results of the regression analyses ($p = .18$ and $p = .13$ respectively) suggest that dimensional PD score does not significantly predict treatment outcome after controlling for initial depression severity.

Table 8.

Summary of the Linear Regression Analysis for Dimensional PD predicting Posttreatment BDI-II with Pretreatment BDI-II serving as a Covariate.

Variable	B	SE B	β
Pretreatment BDI-II	.11	.14	.10
Dimensional PD	.11	.08	.18

Table 9.

Summary of the Linear Regression Analysis for Dimensional PD predicting Posttreatment HRSD with Pretreatment HRSD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.13	.16	-.11
Dimensional PD	.09	.06	.21

In addition, linear regression analyses were carried out to predict treatment response from the Cluster A variable with pretreatment BDI-II (Table 10) and pretreatment HRSD (Table 11) serving as corresponding covariates. The regression results ($p = .02$ and $p = .01$, respectively) suggest that higher Cluster A pathology predicts poorer treatment outcome after controlling for initial depression severity. Figure 3 illustrates the patterns of response of Cluster A-disordered patients and patients without a PD diagnosis.

Table 10.

Summary of the Linear Regression Analysis for Cluster A predicting Posttreatment BDI-II with Pretreatment BDI-II serving as a Covariate.

Variable	B	SE B	β
Pretreatment BDI-II	.09	.13	.09
Cluster A *	.53	.21	.31

* $p < .05$

Table 11.

Summary of the Linear Regression Analysis for Cluster A predicting Posttreatment HRSD with Pretreatment HRSD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.10	.15	-.08
Cluster A**	.41	.15	.35

** $p \leq .01$

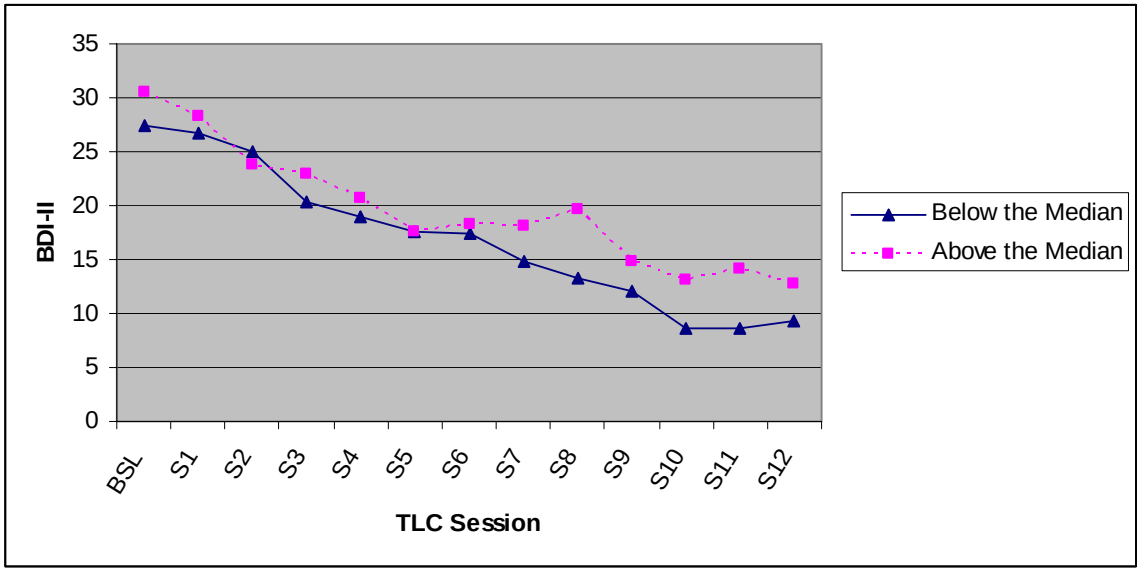

Figure 3. BDI-II scores by session for patients above and below the median on dimensional Cluster A score.

Linear regression analyses were also performed to predict treatment response from the Cluster B variable with pretreatment BDI-II (Table 12) and pretreatment HRSD (Table 13) serving as corresponding covariates. The regression results ($p = .01$ and $p = .19$, respectively) suggest that higher Cluster B pathology predicts poorer treatment

response on the primary treatment outcome measure, BDI-II, after controlling for baseline BDI-II score. Moreover, Figure 4 shows that Cluster B-disordered patients and the NPD group exhibit contrasting patterns of response throughout treatment.

Table 12.

Summary of the Linear Regression Analysis for Cluster B predicting Posttreatment BDI-II with Pretreatment BDI-II serving as a Covariate.

Variable	B	SE B	β
Pretreatment BDI-II	.05	.14	.05
Cluster B**	.43	.17	.32

** $p \leq .01$

Table 13.

Summary of the Linear Regression Analysis for Cluster B predicting Posttreatment HRSD with Pretreatment HRSD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.13	.17	-.11
Cluster B	.22	.17	.18

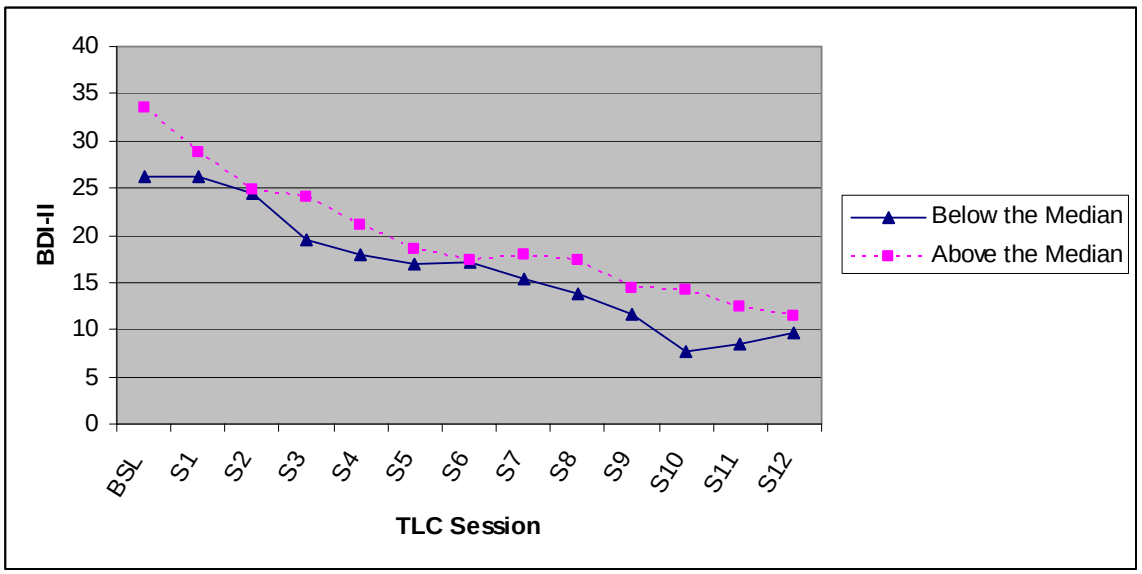

Figure 4. BDI-II scores by session for patients above and below the median on dimensional Cluster B score.

The hypotheses that the Cluster C variable is predictive of treatment response with pretreatment BDI-II (Table 14) and pretreatment HRSD (Table 15) serving as corresponding covariates were tested in linear regression analyses. The regression results ($p = .22$ and $p = .72$, respectively) suggest that Cluster C pathology does not significantly predict treatment outcome after controlling for initial depression severity. It is evident from Figure 5 that Cluster C-disordered patients respond similarly compared to patients without an Axis II PD diagnosis.

Table 14.

Summary of the Linear Regression Analysis for Cluster C predicting Posttreatment BDI-II with Pretreatment BDI-II serving as a Covariate.

Variable	B	SE B	β
Pretreatment BDI-II	.24	.14	.22
Cluster C	-.21	.17	-.16

Table 15.

Summary of the Linear Regression Analysis for Cluster C predicting Posttreatment HRSD with Pretreatment HRSD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.09	.17	-.08
Cluster C	.05	.13	.05

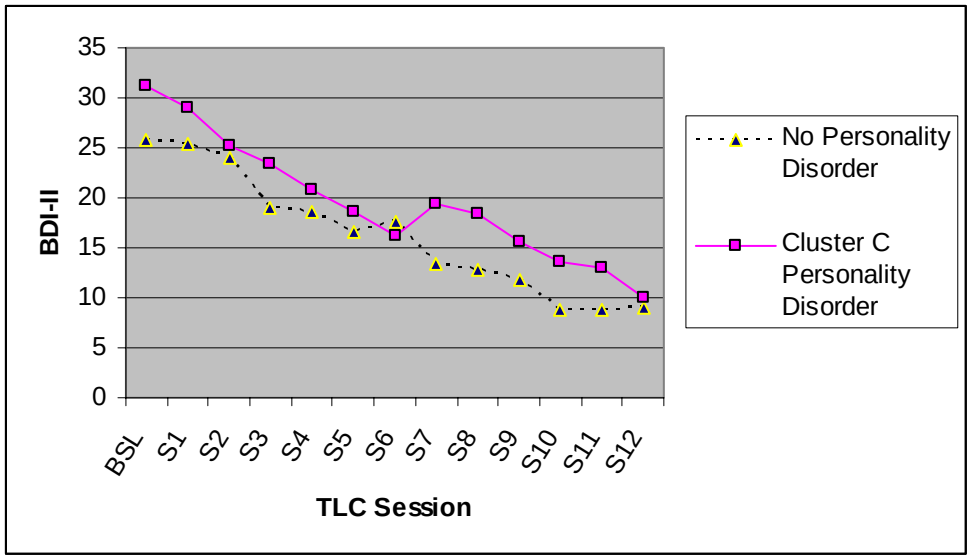

Figure 5. BDI-II scores by session for Cluster C-disordered patients and patients without an Axis II PD.

Effect of Adherence on TLC Treatment Outcome

The third principal hypothesis – that adherence to TLC predicts TLC treatment response – was also tested with a number of linear regression analyses, with initial depression severity and personality pathology variables serving as covariates. Linear regression analyses were conducted to predict treatment response from the global adherence variable with dichotomous PD serving as a covariate and pretreatment BDI-II (Table 16) and pretreatment HRSD (Table 17) serving as corresponding covariates. The regression results ($p = .047$ and $p = .59$, respectively) suggest that higher overall adherence to TLC predicts better treatment response on the primary treatment outcome measure, BDI-II, after controlling for initial depression severity and the dichotomous personality disorder variable.

Table 16.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment BDI-II with Pretreatment BDI-II and Dichotomous PD serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.18	.14	.16
Dichotomous PD	1.1	2.4	.06
Global Adherence*	-4.2	2.10	-.24

* $p < .05$

Table 17.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment HRSD with Pretreatment HRSD and Dichotomous PD serving as Covariates.

Variable	B	SE B	β
Pretreatment HRSD	-.11	.17	-.09
Dichotomous PD	2.5	1.9	.19
Global Adherence	-.93	1.7	-.07

The hypotheses that global adherence is predictive of TLC treatment outcome were evaluated in linear regression frameworks with dimensional PD serving as a covariate and pretreatment BDI-II (Table 18) and pretreatment HRSD (Table 19) serving as corresponding covariates. The results of the regression analyses ($p = .04$ and $p = .15$, respectively) suggest that higher overall adherence to TLC predicts enhanced treatment response vis-à-vis the primary treatment outcome measure, BDI-II, after controlling for initial depression severity and dimensional PD.

Table 18.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment BDI-II with Pretreatment BDI-II and Dimensional PD serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.12	.14	.11
Dimensional PD	.11	.08	.18
Global Adherence*	-4.4	2.1	-.25

* $p < .05$

Table 19.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment HRSD with Pretreatment HRSD and Dimensional PD serving as Covariates.

Variable	B	SE B	β
Pretreatment HRSD	-.12	.17	-.10
Dimensional PD	.09	.06	.20
Global Adherence	-1.14	1.7	-.09

Linear regression analyses were also carried out to predict treatment response from the global adherence variable with each Axis II cluster serving as a covariate and pretreatment BDI-II and pretreatment HRSD serving as corresponding covariates. The regression results controlling for Cluster A ($p = .03$ and $p = .49$, with pretreatment BDI-II and pretreatment HRSD serving as covariates, respectively) (Table 20 & 21), Cluster B ($p = .04$ and $p = .47$) (Table 22 & 23), and Cluster C ($p = .04$ and $p = .48$) (Table 24 & 25) suggest that higher overall adherence to TLC predicts better treatment response on the primary treatment outcome measure, BDI-II, after controlling for initial depression severity and Axis II cluster pathology.

Table 20.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment BDI-II with Pretreatment BDI-II and Cluster A serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.10	.13	.09
Cluster A	.53	.20	.31
Global Adherence*	-4.4	2.0	-.25

* $p < .05$

Table 21.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment HRSD with Pretreatment HRSD and Cluster A serving as Covariates.

Variable	B	SE B	β
Pretreatment HRSD	-.08	.16	-.07
Cluster A	.41	.15	.35
Global Adherence	-1.13	1.63	-.09

Table 22.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment BDI-II with Pretreatment BDI-II and Cluster B serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.06	.13	.06
Cluster B	.43	.17	.32
Global Adherence*	-4.3	2.0	-.24

*$p < .05$

Table 23.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment HRSD with Pretreatment HRSD and Cluster B serving as Covariates.

Variable	B	SE B	β
Pretreatment HRSD	-.11	.17	-.09

Cluster B	.22	.17	.18
Global Adherence	-1.2	1.7	-.10

Table 24.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment BDI-II with Pretreatment BDI-II and Cluster C serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.25	.13	.23
Cluster C	-.21	.17	-.16
Global Adherence*	-4.34	2.06	-.25

$p < .05$

Table 25.

Summary of the Linear Regression Analysis for Global Adherence predicting Posttreatment HRSD with Pretreatment HRSD and Cluster C serving as Covariates.

Variable	B	SE B	β
Pretreatment HRSD	-.07	.17	-.06
Cluster C	.04	.13	.04
Global Adherence	-1.24	1.74	-.10

Also, the hypotheses that adherence to each TLC component is predictive of TLC treatment outcome were evaluated in linear regression frameworks with dimensional PD serving as a covariate and pretreatment BDI-II and pretreatment HRSD serving as corresponding covariates. The results of the regression analyses for Omega-3 consumption ($p = .68$ and $p = .61$, with pretreatment BDI-II and pretreatment HRSD serving as covariates, respectively) (Table 26 & 27), bright light ($p = .34$ and $p = .78$) (Table 28 & 29), pleasant activities ($p = .76$ and $p = .65$) (Table 30 & 31), and social support ($p = .53$. and $p = .55$) (Table 32 & 33) suggest that specific adherence to these four components of TLC does not significantly predict treatment outcome after

controlling for initial depression severity and dimensional PD. However, the regression results for the exercise component (p = .003 and p = .38, respectively) (Table 34 & 35) suggest that higher adherence to the exercise component of TLC predicts significantly better treatment response on the primary treatment outcome measure, BDI-II, after controlling for initial depression severity and dimensional PD.

Table 26.

Summary of the Linear Regression Analysis for adherence to Omega-3 component predicting Posttreatment BDI-II with Pretreatment BDI-II and Dimensional PD serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.12	.14	.11
Dimensional PD	.11	.08	.18
Omega-3	-.49	1.17	-.05

Table 27.

Summary of the Linear Regression Analysis for Omega-3 component predicting Posttreatment HRSD with Pretreatment HRSD and Dimensional PD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.13	.17	-.11
Dimensional PD	.09	.06	.22
Omega-3	-.44	.85	-.07

Table 28.

Summary of the Linear Regression Analysis for adherence to Bright Light component predicting Posttreatment BDI-II with Pretreatment BDI-II and Dimensional PD serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.09	.15	.08
Dimensional PD	.14	.08	.23
Bright Light	-1.13	1.19	-.12

Table 29.

Summary of the Linear Regression Analysis for Bright Light component predicting Posttreatment HRSD with Pretreatment HRSD and Dimensional PD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.13	.17	-.11
Dimensional PD	.09	.06	.21
Bright Light	-.26	.91	-.04

Table 30.

Summary of the Linear Regression Analysis for adherence to Pleasant Activities component predicting Posttreatment BDI-II with Pretreatment BDI-II and Dimensional PD serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.13	.14	.12
Dimensional PD	.11	.08	.19
Pleasant Activites	-.37	1.17	-.04

Table 31.

Summary of the Linear Regression Analysis for Pleasant Activites component predicting Posttreatment HRSD with Pretreatment HRSD and Dimensional PD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.14	.17	-.12
Dimensional PD	.09	.06	.21
Pleasant Activites	.41	.91	.06

Table 32.

Summary of the Linear Regression Analysis for adherence to Social Activities component predicting Posttreatment BDI-II with Pretreatment BDI-II and Dimensional PD serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.13	.15	.13
Dimensional PD	.19	.09	.31
Social Activities	-.84	1.32	-.09

Table 33.

Summary of the Linear Regression Analysis for Social Activities component predicting Posttreatment HRSD with Pretreatment HRSD and Dimensional PD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.29	.20	-.22
Dimensional PD	.13	.07	.30
Social Activities	-.60	1.00	-.09

Table 34.

Summary of the Linear Regression Analysis for adherence to Exercise component predicting Posttreatment BDI-II with Pretreatment BDI-II and Dimensional PD serving as Covariates.

Variable	B	SE B	β
Pretreatment BDI-II	.11	.13	.01
Dimensional PD	.10	.07	.17
Exercise**	-3.30	1.09	-.34

** $p < .01$

Table 35.

Summary of the Linear Regression Analysis for Exercise component predicting Posttreatment HRSD with Pretreatment HRSD and Dimensional PD serving as a Covariate.

Variable	B	SE B	β
Pretreatment HRSD	-.14	.17	-.12
Dimensional PD	.09	.06	.20

Exercise	-.85	.97	-.12

Mediation Tests

The linear regression analyses used to test the first three principal hypotheses of the study (i.e., (1) the presence of comorbid personality pathology will be associated with reduced treatment adherence to TLC for depression; (2) the presence of comorbid personality pathology will be associated with poorer treatment response to TLC for depression; and (3) treatment adherence to TLC will be associated with treatment response vis-à-vis reduction in depressive symptomatology) combine to form a type of mediation test (Baron & Kenny, 1986). However, a macro (Preacher & Hayes, 2007) designed to generate estimates for indirect effects in a mediator model was used as a more thorough test of mediation. The macro employed 5,000 bootstrap resamples to generate a sampling distribution of the indirect effect of personality pathology on treatment outcome through adherence that does not involve making any distributional assumptions. Moreover, 95% bias-corrected and accelerated confidence intervals were generated to determine if adherence significantly mediates the effect of personality pathology on TLC treatment response.

A macro was run for each of the 20 sets of models tested. Each resulting confidence interval bracketed the value 0.0 (Table 36), consistent with the hypothesis that adherence does not mediate the effect of personality pathology on treatment outcome.

Table 36.

Macro[a] test of Adherence Mediating the effect of Axis II Personality Pathology on Treatment Outcome.

Mediator (adherence)	**Axis II**	**Treatment Outcome**	**CI[b] – Lower Bound**	**CI[b] – Upper Bound**
Global	Dichot. PD	BDI-II	-.720	2.365
Global	Dichot. PD	HRSD	-.412	1.424
Global	Dimen. PD	BDI-II	-.055	.048
Global	Dimen. PD	HRSD	-.009	.035
Global	Cluster A	BDI-II	-.144	.106
Global	Cluster A	HRSD	-.034	.073
Global	Cluster B	BDI-II	-.099	.136
Global	Cluster B	HRSD	-.019	.087
Global	Cluster C	BDI-II	-.125	.093
Global	Cluster C	HRSD	-.025	.068
Omega-3	Dimen. PD	BDI-II	-.018	.027
Omega-3	Dimen. PD	HRSD	-.016	.011
Bright Light	Dimen. PD	BDI-II	-.066	.012
Bright Light	Dimen. PD	HRSD	-.017	.023
Exercise	Dimen. PD	BDI-II	-.046	.066
Exercise	Dimen. PD	HRSD	-.008	.035
Pleasant Activ.	Dimen. PD	BDI-II	-.012	.038
Pleasant Activ.	Dimen. PD	HRSD	-.039	.008
Social Support	Dimen. PD	BDI-II	-.041	.019
Social Support	Dimen. PD	HRSD	-.009	.037

[a] Designed by Preacher & Hayes (2007) – using 5000 bootstrap resamples.
[b] Confidence Interval – 95% bias-corrected and accelerated.

DISCUSSION

Effect of Personality Pathology on Adherence to TLC Treatment

Based on existing empirical literature documenting poor adherence associated with Axis II pathology (Cahalane, 1997; Herbeck et al., 2005; Sajtovic et al., 2004), patients with elevated levels of personality pathology were expected to exhibit less adherence to the TLC protocol in the present study. Contrary to expectations, however,

no significant association between Axis II pathology and treatment adherence was observed. This finding suggests that the characterological difficulties associated with diagnosed PD (e.g., excessive emotionality, deficits in interpersonal and occupational functioning, etc.) do not substantially impede patient adherence to the TLC protocol.

Although this is a somewhat surprising result, it is possible that certain features of TLC facilitate adherence among personality-disordered patients. For example, there is evidence that personality-disordered individuals respond better to more structured treatments with clearly defined goals in comparison with less-focused, insight-oriented interventions (Hirani, 2007; Saulsman et al., 2006; Van den Hout et al., 2006). Thus, the structured nature of TLC – a protocol that emphasizes the adoption of concrete behavioral lifestyle changes – may serve to enhance compliance in individuals with personality pathology. Also, the group format of TLC, which provides for implicit support and accountability between group members, may serve to facilitate adherence. Furthermore, the protocol's provision of weekly therapist "coaching calls" may also increase patient accountability and provide enhanced motivation for compliance.

In addition, the method of enrollment of study participants may have resulted in a more highly motivated sample of personality-disordered individuals than is typically observed in the published treatment outcome literature. Specifically, the present study was given widespread, favorable coverage by the local news media in the immediate catchment area, and was typically described as a "holistic" approach to the treatment of depression. Moreover, numerous study participants commented (anecdotally) that they were drawn to the evolutionarily informed conceptual framework upon which TLC was developed – typically describing it as very believable and something that "just makes

sense". Consequently, it is possible that the compelling theoretical rationale of TLC, coupled with a pre-existing patient interest in adopting a healthier lifestyle, resulted in a sample of participants with uncharacteristically high motivation for treatment adherence.

Effect of Personality Pathology on TLC Treatment Outcome

It was further hypothesized, based on the existing literature (Frank et al, 1987; Pilkonis & Frank, 1988; Ilardi & Craighead, 1995; Reich & Vasile, 1993; Shea et al., 1992; Whisman, 1993), that the presence of comorbid Axis II personality disorder would have a negative impact on treatment response. This hypothesis found only modest support in the present investigation. Depressed patients with and without a comorbid PD diagnosis experienced a similar rate of improvement in depressive symptomatology over the course of treatment. In addition, no significant covariation was observed between the overall magnitude of personality pathology (as indicated by a dimensional PD score) and treatment outcome. Consistent with the extensive literature on cognitive therapy for depression – in which personality-disordered patients generally respond every bit as favorably as do those without personality pathology (Hirani, 2007; Saulsman et al., 2006; Van den Hout et al., 2006) – it is possible that TLC is specifically beneficial in the treatment of depression among patients with comorbid PD largely because of its highly structured, time-limited, and problem-focused nature.

Although no global effect of comorbid PD on TLC treatment response was observed, the study does provide evidence that TLC treatment outcome may vary as a function of specific Axis II disorders or domains. Specifically, despite similar levels of treatment adherence in comparison with the rest of the study sample, patients with elevated levels of personality pathology on either Cluster A or Cluster B responded

significantly less well to TLC, even when controlling for initial depression severity. This finding may be explained, in part, by the failure of TLC elements to adequately target important features of Cluster A and Cluster B pathology, such as affective dysregulations and substantial interpersonal difficulties. The characterological difficulties associated with Cluster A and Cluster B pathology tend to engender a relatively high number of negative life events and crises, real or perceived, that can trigger a sustained level of distress capable of exacerbating or maintaining depression (Flett, Hewitt, Endler, & Bagby, 1995; Segal, Shaw, Vella, & Katz, 1992; Whisman, 1993). Consequently, it is possible that although such personality-disordered patients are similarly motivated and adherent in comparison with the rest of the study sample, they do not respond as well due to an enduring experience of interpersonal chaos (and associated affective instability) not directly addressed in treatment.

Comorbid Cluster C pathology, which is characterized by high trait anxiety and neuroticism, was not shown to have a significant impact on TLC treatment outcome. Interestingly, a number of TLC elements may also inadvertently target features of Cluster C pathology that could serve as an impediment to treatment adherence and response. For example, it is likely that the anti-ruminative strategies promoted in TLC not only have an effect on depressive symptomatology, but – via their effect in limiting anxiogenic rumination – also serve to reduce anxiety and neuroticism associated with the chronic activation of negative schemas that characterize Cluster C pathology. In addition, it is likely that the exercise component of TLC results in a reduction of anxiety (McDonald & Hodgdon, 1991) in this personality-disordered group of patients. Moreover, the structure of TLC, noted above, likely has a positive effect on Cluster C pathology. Specifically,

the group format may be especially beneficial, since it often serves as a forum for patients to share with fellow group members their anxieties, identify with each other's worries, and receive support and assistance in challenging dysfunctional cognitions related to their anxieties. Thus, the study findings suggest that by concurrently addressing their depressive symptomatology and Cluster C personality pathology, TLC effectively treats depression in this specific comorbid population.

It should also be noted that the failure of overall personality pathology (i.e., aggregate dimensional score) to significantly predict treatment outcome in the present study may be due to the participant sample being fairly "top-heavy" with Cluster C pathology. That is, a significant proportion of patients (50%) in the sample met diagnostic criteria for a Cluster C PD, while only 13 patients (19%) received a diagnosis of either Cluster A or Cluster B PD. The study's explicit exclusion of individuals with psychotic symptoms, substance abuse diagnoses, a history of self-harm behavior during the past two years, or active suicidal ideation likely precluded several depressed patients with Cluster A or Cluster B PD's from participating. Thus, the relative effectiveness of TLC in the treatment of depression in patients with comorbid Cluster C PD, and the high proportion of Cluster C-disordered individuals in the PD group, may have obscured detection of a possible adverse effect of overall personality pathology on treatment outcome.

Effect of Adherence on TLC Treatment Outcome

The hypothesis that adherence to the TLC treatment protocol would predict treatment response was supported. As expected, patients who exhibited higher levels of compliance tended to report significantly lower levels of depressive symptomatology on

the primary outcome measure, the BDI-II, after controlling for initial depression severity and personality pathology. This finding is consistent with the hypothesis that adherence to the TLC protocol, rather than the indirect effect of nonspecific treatment factors like enhanced positive expectancies associated with merely being in treatment (Frank, 1973), is responsible, to some degree, for the observed reduction in depression experienced by the patients in the sample.

It should also be noted that in the study's evaluation of adherence to specific TLC components, only exercise adherence was found to yield significant prediction of treatment outcome, after controlling for initial depression severity (i.e., baseline BDI-II) and overall level of personality pathology (i.e., dimensional PD score). For every additional 27 minutes exercised each week, patients experienced an additional 3.3 point reduction in depressive symptomatology (BDI-II). This study finding suggests that the reduction in depressive symptomatology observed as a result of adherence to the TLC protocol may be driven to a considerable extent by adherence to the exercise component. This finding, in turn, raises the question of whether all six primary components of the TLC protocol are necessary. It is possible that some components of TLC do not contribute significant unique variance in the prediction of treatment outcome sufficient to warrant inclusion in the protocol. Alternatively, it may be that the relatively truncated range of adherence rates to some protocol elements – e.g., the 90% rate of overall adherence to omega-3 supplementation – attenuated the likelihood of detecting adherence effects in this investigation. It is also worth bearing in mind that the present study, with only 68 participants (and 58 treatment completers), was not adequately powered to detect beneficial small-to-medium-sized effects attributable to specific protocol elements.

Mediation

Study findings suggest that adherence to TLC treatment (or lack thereof) does not mediate the adverse effect of personality pathology (Clusters A and B) on treatment outcome. Instead, it is likely that the characterological issues associated with specific clusters of Axis II PD determine, in large part, the effect of personality pathology on treatment response. For example, it is believed that the unaddressed affective dysregulations and considerable interpersonal difficulties typically observed in patients with Cluster A and Cluster B are responsible, in large part, for the differential treatment outcome observed.

Limitations

The present study is characterized by several important limitations. One notable limitation concerns the incompleteness of study adherence data. As previously noted, sleep adherence data were not included in study analyses, as they were considered problematic due to their significant conflation with depressive symptomatology. (In other words, a patient's failure to obtain the protocol's recommended nightly duration of sleep could reflect the enduring *effects* of depression rather than a genuine lack of adherence.) Consequently, one of the six primary components of the TLC protocol was omitted from study analyses. It is quite possible, however, that the inclusion of sleep adherence data would not have made a significant difference in study results, inasmuch as: (a) sleep enhancement receives considerably less emphasis in the protocol than other TLC elements; (b) it is the last element introduced in treatment (at Session 6); and (c) three other elements of treatment – omega-3, bright light, and exercise – have a documented

potential of enhancing sleep quality and quantity (Fetveit, Skjerve, & Bjorvatn, 2003; Stevens et al., 1996; Trivedi, Greer, Grannemann, Chambliss, & Jordan, 2006)).

Another limitation of the present study was the study's relatively modest sample size (68 participants, and 58 treatment completers), which did not afford adequate power to detect beneficial small-to-medium-sized effects. It is also important to note that the Weekly Record Form (WRF) was revised after the present investigation was already under way – specifically, following completion of the first two treatment groups. Consequently, social support data was not collected for the first 18 study patients. It is assumed that these missing data should not significantly affect the analyses, as they are "missing at random" – i.e., there is no reason to believe that the first two TLC groups significantly differ from the rest of the sample on this variable. Nevertheless, the absence of these data constitutes a study limitation.

Also, the observed internal consistency of the WRF was indicative of an acceptable, but not ideal, level of reliability. It is possible that the WRF's merely moderate internal reliability is not a measurement problem, but rather a result of the phenomenon that people's adherence truly varies across the protocol's different treatment elements. Such idiosyncratic adherence across domains is consistent with the subjective self-report of numerous study patients. For example, several patients have discovered the exercise component of TLC to be the most difficult to adhere to while others have found it to be the easiest. Consequently, there is reason to believe that there is some domain-specific variability involved in the adherence construct in addition to the common variance.

Finally, the methodology for obtaining the study sample – inclusion and exclusion criteria, in addition to recruitment secondary to news media coverage – also introduces some important limitations. It is likely, for example, that the study exclusion criteria resulted in a sample that had a restricted range of personality-disordered patients, with relatively few who met diagnostic criteria for Axis II disorders on Clusters A and B. It is possible that this selectivity of the sample was responsible, in part, for the failure to observe an effect of overall personality pathology on treatment outcome. In addition, the recruitment of volunteers for a well-publicized "holistic" treatment for depression may have resulted in a group of extraordinarily motivated patients. Thus, these sample issues limit the ability to generalize the study's findings to other depressed populations.

Future Directions

Due to the limitations outlined above, a replication that addressed these shortcomings would be beneficial to corroborate the present study's findings. Moreover, the results of this investigation suggest that future studies in the field should avoid "lumping" clusters of Axis II personality disorders together to form a single, monolithic PD group. The differential treatment effects observed for depressed patients with either comorbid Cluster A or Cluster B personality pathology underscores the notion that clusters of Axis II PD's are not homogenous groups that can be combined. It is likely that particular characteristics associated with certain clusters affects treatment response in distinct ways. Consequently, combing PD clusters produces excess noise that limits the detection of differential treatment effects. Furthermore, the combination of PD clusters into a single PD group may explain, in part, the notable inconsistency of reported outcomes in the empirical literature.

In the same vein, future investigations should move toward increased specificity with respect to the effects of personality pathology. That is, evaluation of specific PD's may well uncover that treatment response varies appreciably as a function of each disorder. The information generated from these studies will also have the potential to enhance treatment of comorbid PD populations. The identification of pretreatment clinical characteristics that significantly impact treatment outcome may be used to guide treatment choices and lead to more effective interventions.

The deleterious effects of comorbid Cluster A and Cluster B pathology on TLC treatment outcome observed in the study warrant a discussion of possible modifications to the TLC protocol to render it more efficacious for the this subset of patients. It should be noted that almost 20% of patients in the total study sample met diagnostic criteria for either a Cluster A or a Cluster B PD. Moreover, the general depressed population may be comprised of an even higher proportion of individuals with a comorbid Cluster A or Cluster B PD (Casey et al., 2004). Thus, the study findings suggest that a sizable subset of depressed individuals do not respond as favorably as depressed individuals without comorbid Cluster A or Cluster B PD's to TLC. Consequently, a modification of the TLC protocol would be clinically useful.

It is posited that the characterological issues typically observed in depressed individuals with comorbid Cluster A or Cluster B pathology, namely affective dysregulation, interpersonal chaos, and the experience of numerous negative life events (e.g., failures, crises, etc), have an adverse effect on treatment outcome for TLC for depression. Thus, increased attention devoted to addressing these issues will likely lead to more favorable treatment response for these comorbid populations.

Lengthening the TLC treatment may be needed to adequately address patients' characterological issues and to limit their deleterious influence on patient response to TLC for depression. For example, a session could be included that focused on understanding this comorbid population's vulnerabilities and nurturing their strengths. In addition, it would be beneficial for patients to learn strategies that more directly target their characterological difficulties and to receive assistance in coping with the chronic stressors that they encounter regularly. Furthermore, therapist phone calls to patients could be lengthened or conducted more frequently to provide more opportunities to address and minimize the influence of specific characterological issues on treatment outcome. Finally, treating depressed patients with comorbid Cluster A or Cluster B pathology with concurrent individual therapy may yield a more favorable treatment response. Future studies that evaluate modifications to TLC treatment to more effectively treat these comorbid populations may prove worthwhile; however, the clinical trials to examine a differential effect of a refined treatment would have to be very large as the effect size is likely to be small.

Given the study finding that exercise was the only domain in which adherence significantly predicted treatment outcome, future component analyses of the TLC protocol appear warranted. First, future studies that included less problematic sleep adherence data would be useful. In addition, it is possible that the decision in the present study to cap the adherence data – i.e., to impose an upper bound of 100% on adherence for each element, even if a patient actually *exceeded* protocol's recommended daily "dosage" for any given element – limited the variance in treatment outcome that could be predicted. That is, a patient in this study who received thirty minutes of bright light each

day and a patient that received sixty minutes of bright light were both considered 100% compliant on this component. However, it is plausible that these two patients are experiencing different levels of antidepressant effect from the bright light exposure and utilization of this adherence data will lead to an increase in predicted variance that would provide a clearer picture of which TLC components significantly predict treatment outcome. Furthermore, dismantling studies that more closely evaluated the "need" of each TLC component may lead to a worthwhile conservation of resources.

Finally, the present study did not include follow-up data (although such data are being collected by study researchers for future analysis). It has been documented that differences in treatment outcome according to PD diagnosis tends to be more prominent after long-term follow-up compared to short-term outcome (Cyranowski et al., 2004; Saulsman et al., 2006). Thus, it is important for future studies to assess not only who gets better after treatment, but who maintains treatment gains. It is possible that the enduring characterological issues associated with comorbid Cluster A and Cluster B pathology that influenced TLC treatment response in this study also confer an increased vulnerability to future relapse. In addition, it's possible that although depressed patients with comorbid Cluster C pathology respond to TLC at a comparable rate to patients without comorbid Axis II pathology, the Cluster C-disordered patients exhibit differential relapse rates. These are important questions that need to be answered.

The present study's findings are consistent with an emerging literature on the influence of Axis II personality disorder on depression, which suggests that the presence of a comorbid PD may not have a significant effect on short-term treatment response (Hirani, 2007; Mulder, 2002; Mulder et al., 2003). However, after a closer examination

of personality pathology, the study findings indicate that comorbid Cluster A and Cluster B personality pathology should be viewed as negative treatment indicators for TLC for depression. In the end, this study facilitates understanding of the impact of co-occurring PD on treatment adherence and outcome of TLC for depression. This, in addition to the knowledge generated by future investigations in this field, should lead to more effective treatment of depression in this comorbid population.

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Appendix A Therapeutic Lifestyle Change for Depression Record Form

	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday	Monday
Week 14	**May 16** **Omega-3:** Full Partial None **Multivitamin:** Y N **Bright Light:** # of min: _____ **Sleep:** # of hr: _____ Disturbances: Onset Middle Late Restful: Y N Strategies: Y N Helpful: Y N **Exercise:** Type: _____ # of min: _____ Pulse: _____ **Pleasant Activity:** Y N Expected: _____ Actual: _____ **FLOW Activity:** Y N **Social Activity:** Contact Near: Y N Activity: _____ Contact Far: Y N **Self-Esteem:** 1. _____ 2. _____ 3. _____ **Daily Mood Rating:** 1 2 3 4 5 6 7 8 9 10 Poor - - - - - Good	**May 17** **Omega-3:** Full Partial None **Multivitamin:** Y N **Bright Light:** # of min: _____ **Sleep:** # of hr: _____ Disturbances: Onset Middle Late Restful: Y N Strategies: Y N Helpful: Y N **Exercise:** Type: _____ # of min: _____ Pulse: _____ **Pleasant Activity:** Y N Expected: _____ Actual: _____ **FLOW Activity:** Y N **Social Activity:** Contact Near: Y N Activity: _____ Contact Far: Y N **Self-Esteem:** 1. _____ 2. _____ 3. _____ **Daily Mood Rating:** 1 2 3 4 5 6 7 8 9 10 Poor - - - - - Good	**May 18** **Omega-3:** Full Partial None **Multivitamin:** Y N **Bright Light:** # of min: _____ **Sleep:** # of hr: _____ Disturbances: Onset Middle Late Restful: Y N Strategies: Y N Helpful: Y N **Exercise:** Type: _____ # of min: _____ Pulse: _____ **Pleasant Activity:** Y N Expected: _____ Actual: _____ **FLOW Activity:** Y N **Social Activity:** Contact Near: Y N Activity: _____ Contact Far: Y N **Self-Esteem:** 1. _____ 2. _____ 3. _____ **Daily Mood Rating:** 1 2 3 4 5 6 7 8 9 10 Poor - - - - - Good	**May 19** **Omega-3:** Full Partial None **Multivitamin:** Y N **Bright Light:** # of min: _____ **Sleep:** # of hr: _____ Disturbances: Onset Middle Late Restful: Y N Strategies: Y N Helpful: Y N **Exercise:** Type: _____ # of min: _____ Pulse: _____ **Pleasant Activity:** Y N Expected: _____ Actual: _____ **FLOW Activity:** Y N **Social Activity:** Contact Near: Y N Activity: _____ Contact Far: Y N **Self-Esteem:** 1. _____ 2. _____ 3. _____ **Daily Mood Rating:** 1 2 3 4 5 6 7 8 9 10 Poor - - - - - Good	**May 20** **Omega-3:** Full Partial None **Multivitamin:** Y N **Bright Light:** # of min: _____ **Sleep:** # of hr: _____ Disturbances: Onset Middle Late Restful: Y N Strategies: Y N Helpful: Y N **Exercise:** Type: _____ # of min: _____ Pulse: _____ **Pleasant Activity:** Y N Expected: _____ Actual: _____ **FLOW Activity:** Y N **Social Activity:** Contact Near: Y N Activity: _____ Contact Far: Y N **Self-Esteem:** 1. _____ 2. _____ 3. _____ **Daily Mood Rating:** 1 2 3 4 5 6 7 8 9 10 Poor - - - - - Good	**May 21** **Omega-3:** Full Partial None **Multivitamin:** Y N **Bright Light:** # of min: _____ **Sleep:** # of hr: _____ Disturbances: Onset Middle Late Restful: Y N Strategies: Y N Helpful: Y N **Exercise:** Type: _____ # of min: _____ Pulse: _____ **Pleasant Activity:** Y N Expected: _____ Actual: _____ **FLOW Activity:** Y N **Social Activity:** Contact Near: Y N Activity: _____ Contact Far: Y N **Self-Esteem:** 1. _____ 2. _____ 3. _____ **Daily Mood Rating:** 1 2 3 4 5 6 7 8 9 10 Poor - - - - - Good	**May 22** **Omega-3:** Full Partial None **Multivitamin:** Y N **Bright Light:** # of min: _____ **Sleep:** # of hr: _____ Disturbances: Onset Middle Late Restful: Y N Strategies: Y N Helpful: Y N **Exercise:** Type: _____ # of min: _____ Pulse: _____ **Pleasant Activity:** Y N Expected: _____ Actual: _____ **FLOW Activity:** Y N **Social Activity:** Contact Near: Y N Activity: _____ Contact Far: Y N **Self-Esteem:** 1. _____ 2. _____ 3. _____ **Daily Mood Rating:** 1 2 3 4 5 6 7 8 9 10 Poor - - - - - Good