# THERAPEUTIC LIFESTYLE CHANGE: A BRIEF PSYCHOEDUCATIONAL INTERVENTION FOR THE PREVENTION OF DEPRESSION

by

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*For my father, Dr. Stephen K. Young, on his 60th birthday.*

*Happy Birthday, Dad!*

*May I make you as proud of me as I am of you.*

Abstract

This study aimed to test the efficacy of a brief intervention for the prevention of depression among at-risk college students. Treatment group participants attended a one-hour psychoeducational session based on an acute group-based intervention for depression, Therapeutic Lifestyle Change (TLC; Ilardi et al., 2009). Participants were informed of a set of six modifiable lifestyle factors (omega-3 supplementation, social support, physical exercise, sunlight exposure, healthy sleep, and anti-rumination strategies) which have shown promise in the treatment and prevention of depression (Karwoski, 2006). Treatment group participants evidenced a significant increase in adherence to three of the six targeted TLC elements: supplementing with fish oil, obtaining adequate sleep, and utilizing social support. However, there were no observed between-group differences in self-reported depression symptoms, as reflected in the BDI-II, at either follow-up assessment. Nevertheless, as hypothesized, study participants who received the TLC-based intervention were significantly less likely than were control participants to report the experience of a depressive episode at two- and six-month follow-ups. The intervention appears, therefore, to have reduced the risk of depression onset in a high-risk population, although some interpretational caution is warranted in light of the study's methodological limitations.

# THERAPEUTIC LIFESTYLE CHANGE: A BRIEF PSYCHOEDUCATIONAL INTERVENTION FOR THE PREVENTION OF DEPRESSION

Major depressive disorder (MDD) is a debilitating clinical syndrome that now afflicts nearly one in four Americans by age 75 (Kessler et al., 2005). The disorder is characterized by severe functional impairment and attenuated quality-of-life (Üstün, 2001), and depressed patients spend more cumulative time in bed than do those with arthritis, diabetes, or even lung disease (Wells, Sturm, Sherbourne, & Meredith, 1996). The total cost of major depression – in the form of both lost productivity and direct treatment – amounts to over $83 billion dollars each year in the U.S. alone (Greenberg et al., 2003). In fact, it is now projected that within a decade MDD will become the second most costly disease worldwide in terms of ill health and premature death (Murray & Lopez, 1998).

Depression is also a highly recurrent illness, with up to 85% of afflicted individuals experiencing a clinical course marked by alternating periods of recovery and remission (Coyne, Pepper, & Flynn, 1999). Over a lifetime, the typical clinically depressed patient will experience four to five major episodes, each lasting an average of 20 weeks (Judd, 1997; Gotlib & Hammen, 1992). And this recurrent pattern entails serious consequences. Each successive depressive episode is commonly characterized by increased duration (Coryell, Endicott, & Keller, 1990) and severity of symptomatology (Maj, Veltro, Pirozzi, Lobrace, & Magliano, 1992).

Although there exists a large body of empirical research on both risk factors and protective factors vis-à-vis depression, much less is known about the manner in

which such knowledge can be translated into effective preventive interventions (Paykel, 2006). Only in recent years have researchers begun to make the prevention of depression a principal focus of investigation, and research in this area still lags far behind that pertaining to acute treatment (Le & Boyd, 2006). Indeed, depression prophylaxis has remained something of an esoteric subfield that has been difficult to integrate into clinical practice (Cardemil & Barber, 2001; Muñoz & Ying, 1993). However, increasing awareness of the cascading effect of recurrent depression has catalyzed an increased effort both to identify effective methods of preventing relapse (Hart, Craighead, & Craighead, 2001) and to help vulnerable individuals avert the downward clinical spiral of successive episodes.

## Importance of Prevention

The amelioration of illness via acute treatment is an important clinical aim, but the prevention of illness by means of effective proactive intervention is arguably even more so. In fact, the focus of 21st century medicine is increasingly shifting to the conservation of health rather than the treatment of disease, per se (Monclús, 2003). Prevention appears to be especially important in the case of depression, inasmuch as people with MDD are among the least likely of those suffering from any form of mental illness to seek and receive treatment for their distress. Even among depressed individuals with severe manifestations of mental illness – i.e., a lifetime history of three or more comorbid disorders – the proportion obtaining adequate treatment for their depression is still below 50% (Kessler et al., 1994). This reluctance to seek

professional help may be due in part to negative cultural or personal attributions (Han, Chen, Hwang, & Wei, 2006).

*Type of Prevention*

Traditional definitions of prevention classify intervention efforts into three distinct groups: (a) *primary* prevention programs that intervene with an entire population to prevent new cases of a disorder, (b) *secondary* prevention programs that focus on groups at risk, and (c) *tertiary* prevention programs that focus on maintenance of health and relapse prevention for individuals who have already developed a disorder (Commission on Chronic Illness, 1957). Secondary prevention is often considered the most feasible and cost-effective approach because the targeted population is circumscribed and easily identified (Murray, 1995). In many cases, the differentiation between secondary and tertiary prevention is more implied than real; secondary prevention can legitimately focus on individuals who have experienced a previous episode but are in full remission (Ingram et al., 2004), given that past depression is the best predictor of future depression (Lewinsohn, Hoberman, & Rosenbaum, 1988).

An alternative, more recent subclassification of prevention efforts distinguishes between: (a) *universal* prevention programs that target the general population without identifying at-risk or vulnerable individuals, (b) *selective* prevention programs that target individuals whose risk for the development of a disorder is significantly higher than average, and (c) *indicated* prevention programs that target individuals who already display low but detectable levels of the symptoms

of a disorder (Mrazek & Haggerty, 1994; Muñoz, Mrazek, & Haggerty, 1996). The majority of depression prevention efforts have focused on selective prevention (e.g., Bockting et al. 2005; Thase et al., 2001), although a few have focused on indicated prevention (e.g., Rapaport & Judd, 1998) or a combination of the two (e.g., Paykel et al., 1999).

*Cost-Effectiveness*

Prevention efforts also represent a particularly cost-effective approach to the treatment of chronic illnesses like depression, which have become the predominant global source of morbidity, death, and disease during the 21st century (McQueen, 2007). Smit and colleagues (2006), for example, analyzed the cost-effectiveness of a successful "minimal-contact" intervention designed to prevent depression among subclinically depressed individuals (a population at very high risk for the onset of major depressive illness; Gotlib, Lewinsohn, & Seeley, 1995; Horwath, Johnson, Klerman, & Weissman, 1992; Pintor, Gastó, Navarro, Torres, & Fañanas, 2001). Patients were given a self-help manual with instructions on mood management, which was augmented by an in-person interview with a specialist and six short telephone calls. The intervention incurred an average cost of approximately $576, but this additional cost was fully compensated by savings elsewhere. Over one year, out-of-pocket health care costs between intervention and control groups yielded an average difference of approximately $90 in favor of the intervention. Reduced productivity losses (e.g., absences from work) resulted in further savings – an average advantage of approximately $2510 in total costs per intervention participant.

Likewise, Lynch et al. (2005) recently examined the cost-effectiveness of a group cognitive-behavioral program designed to prevent depression in the adolescent offspring of depressed parents. During the year following the intervention, those who received the intervention used significantly fewer healthcare services than did the control participants. Such services included a variety of specialty mental health services such as family counseling, individual psychotherapy, and outpatient/inpatient hospital visits. Further, across seven different categories of service use, the control participants used services at a rate 15 times higher than that of intervention participants. These findings suggest that the initial cost of depression prevention programs may be easily offset by the financial savings post-intervention.

## *Traditional Preventive Interventions*

### *Pharmacotherapy*

There exists a large body of empirical research examining the effectiveness of antidepressant medication in the prevention of depression, often as a continuation of the medication regimen used to treat acute symptoms. On occasion, antidepressant medications are also employed prophylactically with individuals who have never experienced clinical depression, inasmuch as such medications carry the potential to help prevent the escalation of mild depressive symptoms into full-blown major depressive disorder (Ingram, Odom, & Mitchusson, 2004).

Several different psychotropic medications have been evaluated in continuation treatment, which can be extended for many months (or even years) beyond a patient's initial recovery from a depressive episode (Segal, Williams, &

Teasdale, 2003). Medications seem to be somewhat useful as preventive measures, though perhaps less effective over the long term. For example, Franchini and colleagues (Franchini, Zanardi, Gasperini, & Smeraldi, 1999) evaluated citalopram (a selective serotonin reuptake inhibitor) as a continuation treatment for 40 recovered patients with recurrent depression. Although there were no relapses among the 36 study patients within the first four months of the study, there was a high rate of recurrence (50%) by the study's two-year follow-up, similar to the rate observed in the absence of continued medication. Robert and Montgomery (1995), however, evaluated citalopram as a preventive strategy over a six-month period and found a somewhat lower relapse rate among patients in the treatment group (13.8%) than among patients receiving only pill placebo (24.3%).

Thase and colleagues (Thase, Nierenberg, Keller, & Panagides, 2001) evaluated mirtazapine (an antidepressant that works on noradrenergic, histaminergic, and serotonergic receptors) as a maintenance strategy for 156 fully remitted patients. Continuing mirtazapine therapy significantly reduced the rate of relapse by a 40-week follow-up (19.7% versus 43.9% in the placebo group). Similar reductions in relapse risk have been reported in numerous other studies with continuation antidepressants among remitted patients (e.g., Feiger et al., 1999; Montgomery, 1999). Accordingly, continuation medication as a standard part of clinical care, especially among patients with recurrent depression, is now recommended by many clinical researchers (e.g., American Psychiatric Association, 2006; Dunner, 2001; Nierenberg, 2001).

There are, however, numerous potential problems with the use of antidepressants as a prevention strategy. First, some patients may not be able to tolerate the ongoing difficulties that often characterize the long-term use of antidepressants, such as financial strain and an onerous side effect profile (Mago, 1999; Overholser, 1998). Up to 60% of patients fail to achieve adequate adherence to their medication regimens (Lingam & Scott, 2002; Byrne, Regan, & Livingston, 2006). Second, a large proportion of recovered patients – over half (Franchini et al., 1999; Solomon et al., 2005) – will relapse despite being maintained on medication. It is hypothesized, in fact, that antidepressant medications often lose their effectiveness with continued use (Byrne & Rothschild, 1998). Third, although the continued use of such medications may suppress symptoms, some believe it fails to address the underlying cause(s) of continued vulnerability to depression (Hollon, Shelton, & Loosen, 1991). Finally, although it reduces the risk of recurrence, prophylactic drug treatment does not attenuate the trend toward increasing severity of subsequent depressive episodes (Maj et al., 1992).

In 2008, Kirsch and colleagues obtained data on all clinical trials with full datasets that have submitted to the US Food and Drug Administration for licensing of new-generation antidepressants. They found virtually no difference between drug and placebo in patients with moderate levels of depression, rising to small difference in patients with very severe depression, and reaching clinical significance only in patients at the upper end of the very severely depressed category. The researchers suggested that there is little reason to prescribe antidepressants to any but the most

severely depressed patients unless alternative treatments have been ineffective. Similarly, Turner and colleagues (2008) examined all published and unpublished data on antidepressant medications between 1987 and 2004. They found that approximately half of the studies produced significant drug-placebo differences, although the published literature – by not reporting on 92% of the nonsignificant trials – misleadingly implied that 94% of the trials were significant.

Despite the aforementioned limitations of antidepressant medication use as a prevention strategy, it still constitutes – by far – the most widely used approach in contemporary clinical practice. Antidepressants were the most prescribed class of drugs in the United States in 2006 (Spielmans, Thiegles, Dent, & Greenberg, 2008), and have garnered widespread acceptance among clinicians, the public, and the media alike (Greenberg & Goldman, 2009). Reasons for the popularity of medications may include the simplicity of taking a pill to alter mood, rather than the more intensive and difficult process of therapy, the low time commitment and ease of use perceived by both patients and practitioners, and financial support provided by insurance and pharmaceutical industries (Greenberg & Fisher, 1997). Other preventive strategies, such as ongoing (maintenance) psychotherapy, are rarely employed after the initial treatment for depression (Jarrett et al., 2001).

*Psychotherapy*

Although the number of published studies examining the effects of preventive psychotherapy interventions for mental disorders is not large, a considerable proportion of such studies have targeted the prevention of depression (see Cardemil &

Barber, 2001 and Cuijpers, Van Straten, & Smit, 2005, for reviews). A related literature has examined a felicitous "byproduct" of some forms of acute psychotherapy for depression – a reduced risk of subsequent relapse. In other words, the beneficial effects of psychotherapy often endure long after treatment discontinuation (Jarrett et al., 2001; Seligman et al., 1999), and this prophylactic advantage seems to be larger than that observed with pharmacotherapy. For example, in a review of eight follow-up studies comparing cognitive therapy (CT) with antidepressants, Gloaguen et al. (1998) observed an average relapse rate twice as high among patients treated acutely with antidepressants (60%) compared with those treated with CT (29.5%). Although therapy is often more costly than pharmacotherapy, it may be regarded as an attractive alternative if it provides protection against recurrence (Hollon, DeRubeis, & Seligman, 1992).

*Prophylactic effects of acute CT.* The existing literature suggests that the enduring prophylactic effects of acute treatment are stronger for cognitive therapy (CT) than for medication. For example, Hollon et al. (2005) conducted a randomized controlled trial examining relapse prevention following CT versus medications. At a one-year follow-up, patients withdrawn from CT were significantly less likely to relapse (31%) than patients withdrawn from medications (76%), and no more likely to relapse than patients who continued taking continuation medication (47%). Almeida and Neto (2003) conducted a review on the risk of relapse and recurrence following similar randomized controlled trials of CT for acute depression. In 12 of the 15 reviewed studies, CT as acute treatment significantly lowered the relapse rate in

comparison with a control condition (typically antidepressant medication). The reviewers concluded that CT is efficacious in preventing relapse largely by virtue of its effect in relieving residual subclinical depressive symptoms.

*CT-based prevention programs.* Essau (2004) noted that most programs designed specifically for the prevention of depression are based upon the principles and techniques of CT, due in part to the aforementioned enduring benefit of acute CT. Preventive CT interventions are typically provided as a continuation treatment following acute therapy, either as a stand-alone intervention or as an adjuvant to medication or other treatments. Evidence to date indicates that the preventive effect may be stronger for continuation CT than for continuation medication (Dobson & Ottenbreit, 2004). For example, Jarrett and colleagues (1998) compared two groups of patients who both received acute CT. The study prevention group received an additional eight months of continuation CT. Relapse rates were significantly lower for the treatment group than for the no-intervention control group at follow-up (20% versus 40% at 6 months; 36% versus 70% at 24 months; Jarrett et al., 2001). In a series of impressive two- and six-year follow-up studies, Fava and colleagues (Fava et al., 2004; Fava, Rafanelli, Grandi, Conti, & Belluardo, 1998b) compared two groups of remitted patients, one of which received CT maintenance treatment while the other received only routine clinical management. Relapse rates were significantly lower for the CT treatment group than for the control group at both follow-ups (25% versus 80% at two years; 40% versus 90% at six years).

Bockting et al. (2005) conducted a randomized controlled trial with a preventive group CT intervention among high-risk patients with recurrent depression. All patients received treatment as usual, including continuation of medications, but the treatment group also received eight weekly two-hour sessions of CT. Among patients with five or more previous episodes, CT reduced the relapse rate from 72% to 46% at a two-year follow-up. Similarly, Paykel et al. (1999) compared two groups of remitted patients that continued to experience subclinical symptoms. Both groups received continuation medication, but one group received an additional CT preventive intervention. At a 17-month follow-up, relapse rates were significantly lower in the CT treatment group (29% versus 47% in the medication-only control group). In the same sample, the effects of CT on reduction of recurrence persisted for some time, although with a gradual weakening. The prophylactic benefit of CT was fully lost within four years (Paykel et al., 2005).

Not all published findings, however, support the efficacy of CT as a prevention strategy. For example, Sheffield and colleagues (2006) evaluated multiple types of CT programs for prevention of both first-onset and relapse of depression among 2,479 adolescents in 34 different schools. They observed that none of the CT programs yielded outcomes that differed significantly from their respective no-intervention conditions. Cole and Dendukuri (2004) likewise conducted a review of brief CT interventions to prevent depression in older adults. Only two of 10 reviewed studies yielded statistically significant differences in depression outcome scores favoring the intervention group. Perlis and colleagues (2002) compared two groups of

patients experiencing subclinical symptoms following an acute treatment with fluoxetine (a selective serotonin reuptake inhibitor). Post-intervention, both groups received continuation fluoxetine, but one group also received CT. At a 28-week follow-up, the addition of CT did not lead to lower rates of relapse or treatment discontinuation. Also problematic is the fact that maintenance sessions of CT have not been found to be more effective than either nonspecific "supportive therapy" sessions or no treatment at all among the subset of patients who have fully recovered by the end of acute treatment (Baker & Wilson, 1985; Kavanagh & Wilson, 1987). In conclusion, CT may be considered the most efficacious preventive therapy for depression to date and reduces the risk of relapse by about half. On the other hand, a considerable percentage of depressed individuals relapse despite CT-based prevention, and some studies directly refute the effectiveness of CT.

*Other therapeutic prevention programs.* A few other forms of psychotherapy have also been developed for the prevention of depression. For example, Teasdale and colleagues (Ma & Teasdale, 2004; Segal, Williams, & Teasdale, 2002; Teasdale et al., 2000) have created "mindfulness based cognitive therapy" (MBCT), a program that augments CT techniques with traditional mindfulness meditation. Patients are taught to disengage from depressive thoughts and to experience thoughts and feelings without responding or catastrophizing (see Waller, Carlson, Englar-Carlson, 2006 for a session-by-session review). These techniques were designed for the specific purpose of preventing recurrent depression (Teasdale, Segal, & Williams, 1995). Initial research reveals somewhat promising, though conflicted, results. Two separate

trials of MBCT (Ma & Teasdale, 2004; Teasdale et al., 2000) have found that patients with three or more previous depressive episodes experience a significantly reduced relapse rate following MBCT in comparison with "treatment as usual." However, MBCT patients with only two previous episodes actually showed a nonsignificant increased rate of relapse compared with treatment as usual. Thus, the prophylactic benefit of MBCT may only apply to patients with more extensive histories of repeated depression (Segal et al., 2002).

Fava and colleagues (Fava, Rafanelli, Cazzaro, Conti, & Grandi, 1998a) have created "Well-Being Therapy," a program designed to help remitted patients self-monitor their symptoms and target six therapeutic goals: environmental mastery, personal growth, purpose in life, autonomy, self-acceptance, and developing positive relationships. Preliminary research indicates that this therapy may be as effective as preventive CT in preventing relapse (Fava et al., 1998a; Rafanelli, Park, & Giovanni, 1999).

Interpersonal psychotherapy (IPT) may also confer protective benefit, inasmuch as it extends the well interval for patients with recurrent depression (Frank & Spanier, 1995). Young and colleagues (Young, Mufson, & Davies, 2006) tested the prophylactic efficacy of a group IPT program for subclinically depressed adolescents. At three- and six-month follow-ups, adolescents who received IPT had significantly fewer symptoms, better overall functioning, and fewer depression diagnoses than adolescents in usual care (school counseling). A recent review of IPT as an acute intervention for depression indicates that relapse is less likely to occur following IPT

than after medication or placebo (de Mello, de Jesus Mari, Bacaltchuk, Verdeli, & Neugebauer, 2005).

On the other hand, several findings have failed to support the preventive benefit of IPT. Reynolds and colleagues (1999) compared IPT to nortriptyline (a second generation tricyclic antidepressant) as maintenance treatments for elderly patients with recurrent depression. Recurrence rates over three years were as follows: nortriptyline and IPT, 20%; nortriptyline and medication visits, 43%; IPT and placebo, 64%; placebo and medication visits, 90%. Although combined treatment with nortriptyline and IPT was most effective, IPT with placebo was not significantly more effective than nortriptyline with medication visits. Similarly, at an 18-month follow-up, Shea et al. (1992) found no significant differences in recovery between four treatments: IPT, imipramine, CT, or placebo. The rate of recovery was low, ranging from 19% to 30%. Frank et al. (1990) used maintenance IPT to significantly extend the duration of time patients stayed well before becoming depressed again. However, even though the time to relapse was lengthened with IPT, relapse still eventually occurred for most study patients, and IPT again conferred no benefit above and beyond medication alone. Short-term psychotherapy, specifically IPT or CT, does not have enduring effects for many individuals, particularly in the long term (Frank & Spanier, 1995).

*Prevention for Youth*

Several studies have also evaluated psychotherapeutic depression prevention programs targeting children and adolescents with various risk factors for depression

onset, with mixed results. Clarke et al. (2001) successfully used a relatively brief (15-session) cognitive therapy program with a group of adolescents at risk for depression due to subclinical symptoms or genetic predisposition. At six- and twelve-month follow-ups, adolescents in the treatment group had fewer depressive symptoms and a lower proportion of diagnosable cases (14.7%, versus 25.7% in the control condition). Jaycox et al. (Jaycox, Reivich, Gillham, & Seligman, 1994) tested a program designed to modify cognition, promote social problem solving, and induce efficient cognitive processing among mildly depressed children ages 10-13. Results indicated an acute decrease in symptoms and a reduction in acting-out behaviors, in addition to a sustained decrease in depressive symptoms at six-month and two-year follow-ups. However, because the study had no control group, it is not possible to definitively attribute the observed improvement to the treatment. Another study using a very similar program yielded no significant effect on depressive symptoms (Roberts, Kane, Thomson, Bishop, & Hart, 2003). Shochet and colleagues (2001) tested two prevention programs for all ninth-year students in a secondary school in Australia. One program focused on the adolescents, using coping, cognitive, and interpersonal skills. The other program focused on parents, with a concentration on similar skills. Both prevention groups showed fewer depressive symptoms than a control group at a 10-month follow-up, though there were no significant differences between the two groups.

Seligman and colleagues (Seligman, Schulman, & DeRubeis, 1999) have tested a prevention program specifically designed for college students. The targeted

students did not have MDD, but displayed a pessimistic explanatory style – an established risk factor for the development of MDD. The preventive program focused on the enhancement of cognitive coping skills, and it was implemented weekly for two hours over a span of eight weeks, with added occasional individual sessions. A three-year follow-up revealed a trend toward fewer depressive symptoms, fewer dysfunctional thoughts, less hopelessness, a more positive explanatory style, and fewer cases of generalized anxiety disorder among treated individuals. However, there was no significant reduction in the likelihood of experiencing a diagnosable depressive episode.

## Lifestyle Factors With Preventive Benefit

In addition to pharmacological and psychotherapeutic interventions, several lifestyle variables have been evaluated with respect to their potential prophylactic benefit vis-à-vis depression onset. Following is a brief review of relevant factors:

### *Dietary Omega-3 Fatty Acids*

Omega-3 and omega-6 fatty acids together constitute over 20% of brain tissue and are necessary for neural structure and efficient function (Uauy & Dangour, 2006). These essential fats cannot be made by the body, but must be obtained from diet. Over the vast majority of time during which the human genome evolved, humans consumed a ratio of approximately 1:1 omega-3 to omega-6 fatty acids, and this remains the optimal ratio for humans today (Simopoulos, 2006). However, there has been a significant decrease in the amount of consumed omega-3 fatty acids. The

current ratio of omega-3 to omega-6 in Western diets is approximately 1:15 (Eaton & Eaton, 2000; Simopoulous, 2003).

Evidence for an important linkage between major chronic diseases of industrialized society and the typical Western diet is steadily rising (Eaton & Konner, 1985). For instance, the incidence of major depression is lower in those countries in which natural sources of omega-3 (such as fish) form a key element of the normal diet than in other countries where omega-3 consumption is relatively low (Hibbeln, 1999). Compared with healthy individuals (Hasegawa, 1985), depressed individuals around the world have lower plasma levels of omega-3 fatty acids (Adams, Lawson, Sanigorski, & Sinclair, 1996; Edwards, Peet, Shay, & Horrobin, 1998; Tanskanen et al., 2001; Tiemeier, van Tuijl, Hofman, Kiliaan, & Breteler, 2003). Several recent studies have also supported the benefit of omega-3 supplementation in treating acute depression (Nemets, Nemets, Apter, Bracha, & Belmaker, 2006; Nemets, Stahl, & Belmaker, 2002; Peet & Horrobin, 2002; Silvers, Woolley, Hamilton, Watts, Watson, 2005; Su, Huang, Chiu, & Shen, 2003) and postpartum depression (Freeman et al, 2006). A deficiency of omega-3s in depressed individuals may be exacerbated by the appetite loss and decreased food intake characteristic of many cases of depression (Adams et al., 1996). The most effective daily dosage for acute treatment of depression seems to be 1 g of the omega-3 fat known as eicosapentaenoic acid (EPA) (Peet & Horrobin, 2002; Silvers et al., 2005).

Although there are no known published studies designed specifically to evaluate the preventive benefits of omega-3 fatty acids for MDD, it is believed that

EPA supplementation helps prevent relapse in patients who suffer from recurrent depression (e.g., Peet & Horrobin, 2002; Nemets et al., 2002). Sublette and colleagues (Sublette, Hibbeln, Galfalvy, Oquendo, & Mann, 2006) have also found that low levels of omega-3 fatty acids and high omega-6:omega-3 ratios are associated with a risk for suicidal behaviors, one of the hallmark features of MDD.

In addition, at least two studies provide indirect evidence to suggest that a preventive omega-3 intervention would be effective. Huang, Yang, Chiu, Pariante, and Su (2006) used an animal model demonstrating that a diet enriched with omega-3 fatty acids provides a prophylactic benefit. In this study, rats were assigned either to a normal diet or to a diet supplemented with omega-3 fats. Both groups were then subjected to a forced swim test, which induces a state of despair similar to depression, and which is often used to evaluate the human antidepressant potential of trial medications. The investigators found that rats treated with an omega-3 diet exhibited significantly less despair; that is, the omega-3-fed rats spent a significantly higher amount of time swimming and climbing (active behavior) and significantly less time remaining stationary (passive behavior).

Stoll et al. (1999) have also found that EPA is useful as a preventive measure for bipolar disorder – with particular efficacy in preventing the onset of depressive symptoms. Stoll and colleagues also noted that participants who received EPA were significantly less likely to drop out of the study. The three who received EPA and did drop out attributed their drop-outs to an increase in suffering due to mania, hypomania, or mixed state. In contrast, of the participants who did not receive EPA

and dropped out, nine out of ten attributed their drop-outs to an increase in depressive symptoms.

*Exercise*

Residents of modern industrialized nations typically exert far less physical energy in the course of daily life than people have over the vast majority of human history (Molnar, 2005). Almost 60% of American adults do not engage in any regular physical exercise at all (Centers for Disease Control and Prevention, 2003). This is a cause for concern because exercise confers protective benefit from a variety of illnesses, both physical and psychological. In fact, since the mid-1980s it has been suspected that aerobic exercise provides antidepressant effects (McNeil, LeBlanc, & Joyner, 1991; Ross & Hayes, 1988; Simons, McGowan, Epstein, Kupfer, & Robertson, 1985; Stephens, 1988).

In more recent years, strong evidence has emerged to support the antidepressant benefit of regular exercise (Beck, Steer, & Garbin, 1988; Babyak et al., 2000; Dunn, Trivedi, Kampert, Clark, & Chambliss, 2005; Mather et al., 2002; see Brosse, Sheets, Lett, & Blumenthal, 2002, for a review). In fact, exercise appears to be equally effective in reducing depressive symptoms as psychotherapy or antidepressant medications (Fremont & Craighead, 1987; Greist et al., 1979; Blumenthal et al., 1999; Blumenthal et al., 2007). Purath and colleagues (Purath, Miller, McCabe, & Wilbur, 2004) implemented a single-session intervention to increase physical activity in sedentary working women. At a six-week follow-up, the

intervention group had significantly improved their physical activity, indicating that even very brief programs may help patients increase their level of exercise.

There is also correlational evidence suggesting that physical activity may be useful in the prevention of depression (Ross & Hayes, 1988). Longitudinal studies have clearly shown that regular exercise habits do predict subsequent freedom from depression (Salmon, 2001; Stephens, 1988). For example, Paffenbarger, Lee, and Leung (1994) found that physical activity was negatively correlated with depression over the course of 25 years. In contrast, Camacho and colleagues (Camacho, Robert, Lazarus, Kaplan, & Cohen, 1991) found that lack of physical activity was associated with a greater likelihood of subsequent depression over 18 years, even after controlling for several relevant demographic and clinical variables. Of course, it may simply be that non-depressed individuals are more likely to exercise than depressed individuals. However, studies have also controlled for preexisting depression and *still* found that regular exercise helps ward off future depression (Farmer et al., 1988; Mobily, Rubenstein, Lemke, O'Hara, & Wallace, 1996; Stewart et al., 1994).

Exercise also appears to be useful in the prevention of depression relapse. Blumenthal et al. (1999) compared aerobic exercise, antidepressants, and combined exercise and medication. Although no significant difference in improvement was found between any of the three groups after four months of treatment, after six months it was found that patients in the exercise-only group were at a significantly lower risk of relapse (Babyak et al., 2000). Patients who continued exercising on their own evidenced an even lower risk. Singh, Clements, and Singh (2001) also found that

exercise may help with relapse prevention. Two years after an acute exercise intervention, participants were still less depressed than those in the no-exercise control group. Furthermore, 94% of study participants continued exercising on their own, without supervision. This is impressive given that feasibility, compliance, and long-term adoption are issues of paramount importance for any effective treatment (Singh et al., 2001). Salmon (2001) suggested that exercise may be a particularly effective treatment for patients for whom more conventional interventions are less acceptable, perhaps due to financial reasons, feasibility, or simple unwillingness.

Researchers have also considered what type of exercise is most efficacious in the amelioration of depression. It appears that relatively intensive, active exercise, such as running or biking, is more effective than exercise that requires less intensity, such as stretching or flexibility training (Dunn et al., 2005). Although some studies comparing aerobic and anaerobic exercise have not found a significant difference in efficacy (e.g., Doyne et al., 1987; see Lawlor & Hopker, 2001, for a review), the majority of published controlled studies have utilized aerobic exercise. With respect to antidepressant exercise "dosage," it seems that the determining factor is total energy expenditure. Dunn et al. (2005) found that there was no difference in benefit between participants who exercised three days a week and those who exercised five days a week, as long as the weekly output of energy was in the active, aerobic range (17.5 kcal/kg/week). No matter what kind of exercise individuals choose, the fact that exercise is a "controllable stressor" may make it of special value. To maximize

clinical benefit, patients should feel in control of their exercise regimen (Salmon, 2001) and personalize it to meet their own preferences and needs.

*Sunlight Exposure*

Residents of industrialized nations typically spend very little time outdoors. Remarkably, direct outdoor sunlight is up to 100 times brighter than the intensity of incandescent or fluorescent indoor lighting (Reynolds et al., 2001). This has important implications, inasmuch as adequate light exposure is necessary for regulation of circadian rhythms, sleep regulation, retino-hypothalamic pathways, cortisol, and melatonin attenuation (Küller, 2002), all of which impact mood regulation and psychological functioning (Neuhaus & Rosenthal, 1997). Unfortunately, in a relevant study of people living in San Diego, a relatively weather-friendly and sunny area, the average resident spent only 58 minutes in daylight each day (Espiritu et al., 1994).

Depressed individuals who are particularly sensitive to seasons with reduced light may be diagnosed with seasonal affective disorder (SAD). SAD occurs in a nontrivial subset of the population like clockwork each year, triggered by the short, cold, and cloudy days of winter and subsequent time spent indoors. Indeed, up to 20% of North Americans suffer from winter-onset depressive symptoms (Miller, 2005). Bright light therapy is the least invasive, most natural, and most researched treatment for seasonal affective disorder (Lam et al., 2006; Miller, 2005). Regular time outdoors during daylight hours is an effective alternative to artificial light therapy in the treatment of SAD (Wirz-Justice et al., 1996).

Bright light exposure appears to be a good adjunct treatment for nonseasonal depression, as well (Martiny, Lunde, Unden, Dam, & Bech, 2005). In fact, it seems that light exposure therapy alone can bring up to 35% symptom relief of nonseasonal MDD (for reviews, see Tuunainen, Kripke, & Endo, 2004; Kripke, 1998). Responses to light therapy are as rapid in nonseasonal patients as in patients with seasonal depression, and light therapy is comparable to antidepressant medications in its effectiveness (Kripke, 1998). Light therapy tends to work more quickly than medications, with some patients experiencing benefit within the first week of treatment, as opposed to waiting several weeks or longer for the medication to take effect (Kripke, 1998; Martiny et al., 2005).

Therefore, light therapy may be an especially effective tool in preventing the onset of a depressive episode, particularly for individuals who are already experiencing subclinical symptoms, i.e., those who could easily lapse into a more serious episode. Artificial bright light therapy can also be used prophylactically when patients are approaching the fall and winter seasons. Espiritu and colleagues (1994) found that individuals who spent more than 30 minutes a day in bright light reported little depressive symptomatology, whereas some of those experiencing less illumination reported substantial symptoms. Because chronic low illumination may cause depressive symptoms in susceptible individuals, it is logical to infer that increased light exposure should help prevent those symptoms.

*Social Support*

Americans and other Westerners typically live in urban areas surrounded by thousands or millions of other unrelated humans with whom they often feel little if any meaningful connection (Buss, 2000). Social isolation and a notable lack of intimate social bonding have increasingly become a normal part of life (Putman, 2000). Modern methods of communication such as e-mail and telephone have made it possible to completely avoid regular face-to-face social contact if one so desires. Research indicates that the number of close "discussion partners" in the typical American's interpersonal environment has decreased by about one person since 1985 (from a mean of 2.94 to a mean of 2.08), and that the modal number of discussion partners has gone from three to zero (McPherson, Smith-Lovin, & Brashears, 2006).

It has been known for decades that social support is an important stress-buffering factor, one that promotes physical (e.g., reduced blood pressure; Gump, Polk, Kamarck, & Shiffman, 2001; Holt-Lunstad, Uchino, Smith, Olson-Cerny, & Nealey-Moore, 2003) and emotional health (Cohen, McGowan, Fooskas, & Rose, 1984; Leavy, 1983; Lin, Dean, & Ensel, 1986; Seeman, 1996; Wilcox, 1981). Lack of social support is considered one of the primary vulnerability factors for the onset of depression (Murray, 1995). Withdrawal from social activities is one of the central symptoms of depression (American Psychiatric Association, 2000), reflecting depressed individuals' social deficits. To that end, interpersonal psychotherapy (IPT) was designed specifically to target depressed patients' immediate social context (see Frank & Spanier, 1995, for a review). The recent interest in IPT as both treatment and

prevention of MDD (e.g., Reynolds et al., 1999; Santor & Kusumakar, 2001; for a review, see de Mello et al., 2005) reflects the importance of social connectedness in mental health. Brown and Lewinsohn (1984) examined the efficacy of an acute 12-week psychoeducational intervention for major depression targeting, among other variables, increasing pleasant activities and positive social interactions. At a six-month follow-up, only 25% of participants met criteria for depression. It seems that the relationship between social support and mental health is bi-directional (Johnson, 1991). In other words, depressed individuals are less likely to attract or maintain relationships. Subsequent isolation causes these individuals to become more depressed. Thus, the relationship between these two variables is self-maintaining and intensifies over time.

Social connection may also have preventive value. It is associated with continuing recovery from depression (Reynolds et al., 1999) and is one of the factors most strongly related to complete remission from depression after acute treatment (Ezquiaga, Garcia, Pallares, & Bravo, 1999). Oxman and colleagues (Oxman, Berkman, Kasl, Freeman, & Barrett, 1992) examined the longitudinal effects of emotional support in an elderly population. They found that the greater the adequacy of social support, the lower was the level of depression three years later. This association was found even when other determinants of depression were controlled for, such as disability and socioeconomic variables. It seems that inadequate social support directly contributes to an increase in depressive symptoms. Likewise, a baseline level of adequate social support helps prevent the initial onset of depression

(Dean, Kolody, & Wood, 1990; Krause, 1987; Seeman et al., 1996; see Broadhead et al., 1983 for a review of older literature) and also reduces the risk of depression recurrence in the post-recovery period (de Mello et al., 2005; George, 1989).

*Anti-Ruminative Activity*

Rumination, or the tendency to engage in repetitive negative thinking, is a central cognitive process in depression (Ingram, 1984; Lam, Smith, Checkley, Rijsdijk, & Sham, 2003; Nolen-Hoeksema, 1991). There are at least three mechanisms by which rumination amplifies depressed mood and by which distraction can relieve it (Morrow & Nolen-Hoeksema, 1990). First, depressed mood increases the likelihood of recalling negative information (Clark & Teasdale, 1982). Individuals who distract themselves from their mood will be less likely to reach negative conclusions (Morrow & Nolen-Hoeksema, 1990). Second, rumination maintains and intensifies depressed mood by evoking maladaptive cognitions more often (Ingram, 1984; Zullow, Oettingen, Peterson, & Seligman, 1988). Third, rumination interferes with attention, concentration, and behavioral initiation (Lewinsohn, Hoberman, Teri, & Hautzinger, 1985), leading to fewer opportunities for positive reinforcement and success.

American life today offers ample opportunity for rumination (Ilardi, Karwoski, Lehman, Stites, & Steidtmann, 2009). Rumination is most likely to happen when people are not involved in another mentally stimulating activity (Morrow & Nolen-Hoeksema, 1990), and Americans spend a great deal of time working in

offices, watching television, driving – often alone, allowing abundant occasions to ruminate (Cropley & Purvis, 2003).

Two therapies have been shown to be effective in reducing rumination: metacognitive therapy (Wells & Papageorgiou, 2004) and behavioral activation (BA; Martell, Addis, & Jacobson, 2001). Metacognitive therapy focuses on teaching patients about rumination, helping them become aware of its real-time occurrence, and implementing anti-rumination strategies. In turn, behavioral activation typically focuses on increasing rewarding physical activities. Increased pleasant activities and increase in general activity level are associated with relief from depression (Rehm, 1982). Both strategies may be valuable approaches to decreasing rumination, especially the notion of engaging in activities that suppress rumination (Dimidjian et al., 2006; Ilardi et al., 2009; Karwoski, 2006).

Dimidjian and colleagues (2006) conducted a randomized trial of behavioral activation, cognitive therapy (CT), and antidepressant medication. In contrast to CT, which directly targets thought content, behavioral activation and metacognitive strategies were used to identify ruminative thinking and to turn their attention away from rumination by engaging in active behaviors as alternatives. Specific activation strategies included self-monitoring and structuring and scheduling daily activities. Remarkably, behavioral activation proved superior to antidepressant medication (paroxetine) among severely depressed study patients and significantly outperformed traditional CT. Other research has supported the hypothesis that behavioral activation works at least as well as a full CT program, and it does not lead to higher risk of

relapse over a two-year period (Gortner, Gollan, Dobson, & Jacobson, 1998; Jacobson et al., 1996). Such anti-ruminative, behavioral activation strategies make intuitive sense: ruminative responses tend to increase the length and severity of depressive thoughts, whereas distracting responses can attenuate the experience of depressive symptoms (Nolen-Hoeksema, 1987).

Anti-rumination strategies may be useful in preventing depressive episodes as well. People who ruminate are at a higher risk for longer-lasting and more severe depression (Lam et al., 2003; Nolen-Hoeksema & Morrow, 1993). Rumination can serve to maintain a depressed mood, and a proneness to ruminate has been identified as a marker of subsequent depression vulnerability in individuals who are not yet depressed (Just & Alloy, 1997).

*Sleep*

Sleep abnormalities are commonly found in individuals suffering from MDD, as evidenced by cardinal diagnostic symptoms that include insomnia, restless sleep, or hypersomnia (APA, 2000). In addition to constituting a key symptom of depression, sleep disturbance also contributes to the onset of a depressive episode. While it has historically been assumed that depression causes sleep problems, the converse also appears to be true: disturbed sleep and the dyschronism of circadian rhythms are risk factors for the development or exacerbation of depression (Katz, Knobler, Laibel, Strauss, & Durst, 2002; Kuo, Manber, & Loewy, 2001; Morawetz, 2003). Insomnia and hypersomnia (Breslau, Roth, Rosenthal, & Andreski, 1996; Perlis et al., 2006; Thase, 2005), sleep problems related to jet lag (Katz et al., 2002),

and general sleep disturbance (Cole & Dendukuri, 2003) all put people at a much higher risk for developing major depression. In a review of eight studies, Reimann and Voderholzer (2003) found that insomnia at baseline significantly predicted an increased depression risk at a follow-up one to three years later. They suggested that insomniac symptoms alone seem to be of predictive value for the development of depression.

Interventions designed to enhance both the quality and quantity of sleep have been found effective in treating depression (Morawetz, 2003; Kuo, Manber, & Loewy, 2001). Likewise, it is clear that maintaining healthy sleep helps prevent the onset of depression. Sleep disturbance is the most common symptom to linger after successful treatment for acute depression, reported by 44% of patients (Nierenberg, Alpert, Pave, Worthington, Rosenbaum, & Fava, 1999). Breslau and colleagues (1996) conducted a longitudinal study examining the predictive value of sleep disturbance. They found that the mental disorder most strongly associated with sleep disturbance was MDD, even when the diagnosis of MDD was made on the basis of symptoms other than sleep disturbance. In particular, insomnia was a better predictor of subsequent depression than any other single depressive symptom. The authors suggested multiple explanations for the insomnia–MDD association. First, insomnia may be an early symptom of MDD, with other symptoms appearing later on. Second, insomnia due to exogenous factors may play a causal role in actually precipitating the onset of MDD in predisposed individuals. Either way, the presence of sleep disturbance should be taken as a serious warning sign for people who have previously

experienced depression. Even mild insomnia has a significant association with depression (Hohagen, Rink, Käppler, & Schramm, 1993). Taking steps to prevent sleep disturbance or to deal with disturbance if it arises should help prevent the onset of a depressive episode.

These six lifestyle factors – omega-3 fatty acids, exercise, sunlight exposure, social support, anti-ruminative activity, and sleep – may all have a common denominator. Each is an element that was once a common, even unavoidable, aspect of daily human life. In the modern Western world, however, these elements are much more limited and elusive, requiring special effort to be regularly integrated into life. What is known about the relative lack of these healthy lifestyle factors, and how does this deficit relate to clinical depression?

## An Evolutionary Model of Depression

It has been estimated that the risk of developing major depression has increased tenfold in the U.S. since World War II (Seligman, 1990). Moreover, individuals who live in developing countries are at a lesser risk of developing MDD than those who live in industrialized nations, such as the United States (Weissman et al., 1996). Even within less modernized countries, depression rates are higher among city dwellers with more modern lifestyles than among rural people (Colla et al., 2006). It seems that the more modernized a country is, the higher the risk is of its residents developing depression (Ilardi et al., 2009). Indeed, the risk of depression varies according to the extent of modernization. Countries with lifestyles most removed from modern standards of living typically have the lowest observed

prevalence rates of depression (Schumaker, 1996). Within the United States, there is an example of a traditional community characterized by a markedly reduced risk of depression: the Old Order Amish of Pennsylvania, known for their separateness from the modern world. Lifetime rates of MDD in the Amish culture are far lower, as much as one-tenth of that of other Americans – which is about the rate for all Americans two generations ago, when life was somewhat less "modernized" (Egeland & Hostetter, 1983). A traditional lifestyle seems to offer protection against the negative effects of modernization (Colla et al., 2006).

Research shows that today's hunter-gatherer societies experience a particularly low risk of depression. For example, the Toraja people of Indonesia (Hollan, 1992; Schumaker, 1996) and Trobriand Islanders of Melanesia (Maier, 1996) were found to be extremely resilient to the onset of depression. Schieffelin (1985) spent years studying the Kaluli people, a traditional hunter-gatherer community in Papua New Guinea. Among the thousands of Kaluli assessed for MDD, Schieffelin was able to find only one individual who was symptomatic, despite his observation that the culture encouraged its members to openly and even dramatically display a range of emotions. In contrast, Americans suffer from a suspiciously high lifetime prevalence of MDD (16.2%; Kessler et al., 2003). It seems that modern culture is lacking in preventive buffers to protect its members from depression. It is possible, then, that major depression is less an issue of individual pathology, but more of a culture-bound disorder (Schumaker, 1996).

It is thus hypothesized that the recent increase in the prevalence of MDD, particularly in industrialized Western cultures, is at least in part due to modernization itself. There is evidence that there is a fundamental mismatch (Ilardi et al., 2009; Karwoski, 2006) between the modern environment and the environment of evolutionary adaptiveness (EEA; Bowlby, 1969), in which humans have spent the vast majority of their existence. The further a society gets from an EEA-like setting, the more at risk they seem to be for depression. Likewise, the closer a society is to the EEA way of life, the more immune their members are to developing multiple diseases (Cosmides & Tooby, 1999), including depression (Buss, 2000; Prince, 1967). This hypothesis also explains the increasing prevalence of depression and younger age of onset among younger cohorts in America (Kessler & Walters, 1998; Kessler et al., 2003). As younger individuals embrace lifestyles that are increasingly modernized, the risk of depression increases.

At first glance, it seems that the modern, post-industrial lifestyle has delivered innumerable conveniences, medical advances, technological innovations, and comforts. Most people no longer find it necessary to hunt for food, flee from predators, lift heavy objects, or rely on physical prowess for survival. Physicians are able to cure ills that would have sentenced our ancestors to a quick and painful death; women no longer run a high risk of dying during childbirth; very few individuals suffer from hunger, endure prolonged exposure to inclement weather, or face the direct threat of predators or parasites. The average life expectancy of Americans is now double that of people who lived in pre-agrarian times (Eaton, Konner, &

Shostak, 1988). Many people might agree that, overall, life is easier and safer than it once was.

The question arises, then: what is so psychologically toxic about modern life? Why are Americans at such great risk for depression? Evolutionary researchers suggest that modern circumstances may also be *causing* a variety of problems that humans must face. For example, our hunter-gatherer ancestors regularly engaged in vigorous physical activity for a variety of activities, including both work and play (Molnar, 2005). In contrast, a majority of modern American adults do not engage in any physical exercise at all (Centers for Disease Control and Prevention, 2003). In ancestral times, people lived in small groups with other people, many of whom were their close genetic relatives. These groups were characterized by a high level of support and interdependence, and social isolation was virtually absent (Gat, 1999). Not only did group activities enhance survival (Molnar, 2005), they offered the chance to socialize a great deal (Deacon, 1999). In comparison, complete social isolation is now a normal part of American life (Putman, 2000). These and other features of ancestral hunter-gatherer life helped combat rumination (Ilardi et al., 2009; Karwoski, 2006), thought to be a central problem in modern-day depression (Ingram, 1984).

In the Western world, the past century has accelerated the differentiation of humans from other mammals (Eaton et al., 1988). While human bodies and mind remain unchanged, shaped by selection pressures in the EEA, the environment has changed drastically in recent times. Culture has been transformed beyond recognition

over the past 10,000 years, especially since the Industrial Revolution, but human genes have hardly changed at all (Eaton et al., 1988). In other words, humans are well adapted to the world of their ancestors – not to the one they live in today. Environmental changes might readily influence the human genome given sufficient time, but such rapid innovation has overreached the capacity of evolution to keep pace (Eaton & Eaton, 2000).

Many mental disorders may be the products of mechanisms functioning normally but in currently maladaptive circumstances (Siegert & Ward, 2002). Depression, especially in its severe form (MDD), is perhaps not "meant to happen," but is a result of modern times. This is not to say that particular individuals are not at higher risk for depression. Certainly, clear genetic risk has been identified (e.g., Kendler, Gatz, Gardner, & Pedersen, 2007), along with individual psychological vulnerability factors (e.g., negative automatic thoughts and tendency toward self-blame; Ingram, Trenary, Odom, Berry, & Nelson, 2007). However, phenotypic expression of genetic vulnerability to clinical depression is highly dependent on the environment (Kendler, 1998; Silberg, Rutter, Neale, Eaves, 2001; Uher, 2008), and the alarmingly high rate of depression in the American population in contrast to less modernized societies suggests that the current way of life may put all individuals, vulnerable or not, at higher risk. To this end, Ilardi and colleagues (2009) argue that the recent epidemic of depression is a result of the gradual disappearance of lifestyle elements that are natural antidepressants. In order to gain relative freedom from chronic illnesses brought on by environmental changes, it is necessary to take a step

backward in time and reintroduce essential lifestyle elements from the Paleolithic world (Eaton et al., 1988). This theoretical backing led to a new treatment for depression based on re-incorporating lost antidepressant elements.

### *Therapeutic Lifestyle Change for Depression*

Ilardi and colleagues (2009) have developed a novel treatment protocol based on an evolutionary model of depression. The treatment rests on the notion that depression is caused by a severe lack of natural lifestyle elements that provide a strong antidepressant benefit. These lifestyle elements include: physical exercise, dietary omega-3 fatty acids, sunlight exposure, sleep, social support, and anti-ruminative activity. Accordingly, a 12-session group therapy program, Treatment Lifestyle Change for Depression (TLC), was developed to help patients implement these various lifestyle elements that are often lacking in our modern world. Preliminary data suggest that among treatment completers, 86% of patients receiving TLC achieved significant symptom relief, defined by a 50% or greater reduction in Beck Depression Inventory (BDI-II, Beck, Steer, & Brown, 1996) scores, compared with 33% of patients in the control group (Karwoski, 2006). To date, however, TLC has not been adapted for use as a preventive intervention.

## The Need for Brief Preventive Interventions

There is a paucity of brief, inexpensive, and practical preventive programs for patients at risk for depression, especially given shrinking health-care budgets (D'Amico & Fromme, 2002; Ludman et al., 2000). Traditional methods of prevention create significant barriers to achieving low financial cost and low time commitment.

Intensive resources are needed to provide both maintenance medication (with monthly follow-up visits) and psychotherapy, even if sessions are relatively few and "brief" (e.g., eight weekly sessions for two hours each, Seligman et al., 1999). Conventional preventive pharmacotherapy and psychotherapy require frequent in-person meetings with specialty providers, making them expensive and logistically impractical for large populations – particularly for schools, which are often overburdened with academic curriculum demands (Essau, 2004). The cost of preventive interventions will always be a factor in terms of feasibility. A costly, time-consuming, comprehensive intervention is unlikely to be widely adopted, not only due to strained resources, but also because recipients do not want to make the necessary commitment (Muñoz & Ying, 1993).

For a subset of vulnerable individuals, acute interventions using therapeutic approaches such as CT, IPT, and mindfulness have proven to confer enduring protection against the future onset of depression. However, these interventions are somewhat lengthy and expensive, and are therefore potentially unrealistic for use with large populations of at-risk individuals (e.g., university students). Moreover, it would be quite challenging to effectively convey the principles of any of these therapy protocols in a *brief* preventive-intervention format. For example, Clarke et al. (Clarke, Hawkins, Murphy, & Sheeber, 1993) attempted to use behavioral strategies using a five-session preventive protocol in an adolescent population. They found no outcome differences between adolescents who received the intervention and those who did not. Additionally, because many similar interventions are still too intensive

and costly to be acceptable in most settings, Clarke and colleagues suggested that the approach with the greatest likelihood of both experimental success and adoption in real-life settings is perhaps the promotion of general healthy living skills. When it comes to the formulation of brief preventive interventions, education regarding basic lifestyle changes would appear to represent a promising, cost-effective approach, especially if non-experts (e.g., educators, registered nurses, etc.) could easily administer such a protocol. This strategy also supports the emerging approach of shifting the focus of intervention for depressed individuals from the prevention of relapse to the maintenance of health (Dobson & Ottenbreit, 2004).

Traditional preventive strategies are often implemented as add-ons to ongoing acute-phase treatments. As such, they occur in the context of existing therapeutic relationships – which generally are not available for high-risk individuals who have not yet experienced a full-blown depressive episode. In addition, some at-risk individuals will lack motivation to complete any preventive intervention that requires a significant time commitment. Someone with a history of depression but no ongoing symptomatology, for example, may not be inspired to seek out extensive treatment. Likewise, an individual who is currently experiencing mild symptoms, but who has never experienced a full episode, may not realize the potentially dangerous territory that could lie ahead. For each of the aforementioned reasons, traditional preventive interventions – which tend to be lengthy and expensive – are generally impractical for individuals who are not in the painful, treatment-seeking state of a major depression episode. The prevention of depression has to be carefully targeted and applied where

it is likely to yield the highest possible health benefit at the lowest possible cost (Smit, Beekman, Cuijpers, de Graaf, & Vollebergh, 2004).

### *Supporting Evidence for Brief Interventions*

Short-term intervention strategies can sometimes be equally effective as longer-term strategies (Miller & Barber, 2002). Specifically, extremely brief (single-session), educational, behaviorally-oriented interventions have successfully modified such difficult-to-change behaviors as adolescent risk-taking (D'Amico & Fromme, 2002) and substance use (Grenard et al., 2007; Martin, Copeland, & Swift, 2005), ineffective contraception use among women (Ingersoll et al., 2005), excessive alcohol use among college students (e.g., Ehrlich, Haque, Swisher-McClure, & Helmkamp, 2006; for a review, see Larimer et al., 2004), smoking among mothers (Yilmaz, Karacan, Yöney, & Yilmaz, 2006), gambling (Hodgins, 2005), infrequent seat belt use (Pastò & Baker, 2001), insufficient dietary intake of fruits and vegetables (Marcus et al., 2001), and physical inactivity among sedentary working women (Purath et al., 2004).

There exist no published data, however, on the use of a single-session psychoeduational intervention to specifically prevent depression. Self-described "brief" interventions targeting depression typically provide between four and fifteen sessions (e.g., Cole & Dendukuri, 2004; Dorwick et al., 2000; Lang, 2003). However, several relevant studies of remarkably simple, pithy interventions provide convincing evidence that a single-session strategy may be helpful in the prevention of depression.

Willemse and colleagues (Willemse, Smit, Cuijpers, & Tiemens, 2004), for example, tested the efficacy of a "minimal-contact" therapeutic intervention for subclinically depressed individuals. The main component of the intervention was a self-help manual with instructions on CBT skills for mood management. The manual also included homework assignments and was augmented by an in-person interview with a specialist and six short telephone calls (maximum 15 minutes each). At a one-year follow-up (Smit et al., 2006), the incidence of major depression among participants who received the intervention was reduced by a third in comparison with the control condition (12% versus 18%).

Ludman and colleagues (2000) conducted what they described as a "low intensity" preventive program targeting patients at high risk of depression relapse. Participants received two thirty-minute videos and a book about recurrent depression, two 60-90 minute sessions with a prevention specialist, an assignment to write a personalized relapse prevention plan, bimonthly meetings with a psychiatrist for one year, follow-up phone consultations, and written personalized feedback. A majority of participants attended both in-person meetings (93%), completed all three telephone calls (80%), returned at least one feedback form (85%; 48% returned all four forms), suggesting that such an intervention is feasible. Participants receiving the preventive program had significantly greater adherence to adequate dosage of antidepressants and were more likely to refill prescriptions. At a one-year follow-up, treatment group participants showed significantly fewer depressive symptoms than the control group, though there was no difference in number of relapse episodes (Katon et al., 2001).

Van Voorhees and colleagues (Van Voorhees, Ellis, Stuart, Fogel, & Ford, 2005) tested an internet-based depression prevention for at-risk late adolescents. Participants had at least one risk factor for developing depression (family history or a past depressive episode) and many were experiencing subclinical depressive symptoms. The intervention included an initial motivational interview, web-based modules based on CBT and IPT concepts, and a follow-up motivational interview. Acceptability of the study was good: 100% of participants completed the initial interview, 13/14 used the website, and 8/14 completed the follow-up interview. Changes in depressive symptoms, dysfunctional thinking, and social support showed favorable, though not statistically significant, trends among completers.

Lang (2003) evaluated the effectiveness of a four-session cognitive-behavioral intervention for co-occurring anxiety and depression. Participants in the treatment condition received four weekly sessions for their symptoms. The intervention led to statistically significant reductions in symptoms across multiple measures of depression and functioning. The relief in depressive symptoms in particular was maintained over a one-month follow-up period, indicating that a relatively brief intervention can successfully treat acute depressive suffering.

Finally, Han et al. (2006) conducted very brief (5-10 minute) single-session interventions with college students. Interventions were designed to enhance either biological knowledge regarding depression, encourage destigmatization of seeking treatment for depression, or a combination of both. All three treatment conditions were associated with significant and beneficial changes in beliefs and attributions

regarding depression. The authors suggested that flyers with similar information could successfully be used in a community setting, at least with individuals with a college-level education.

Although results from these extremely brief interventions are generally not as strong as results from lengthier preventive programs, they do show promise. There exist many large-scale settings where more costly and intensive interventions are simply not feasible, but the risk of depression is very real – such as the college environment. In this sort of location, a single-session intervention may be one of the few realistic ways to inexpensively but effectively reach many at-risk individuals.

## The Present Study

The present study aims to test a novel approach to the prevention of depression: a psychoeducational intervention with a focus upon basic lifestyle changes. This approach was designed in response to the need for brief, inexpensive, acceptable, time-efficient, and easily implemented interventions. It was also designed based on an evolutionary theoretical model of depression that significantly differs from other psychological and medical models. For this reason, other interventions such as cognitive therapy, mindfulness, etc. will not be included. Participants will enroll in a single one-hour group psychoeducational session that covers all six primary TLC elements. In conjunction, they will be given several handouts and a website address, which will provide further information and details about the TLC elements and their utility as a means of preventing depression onset. The six key lifestyle elements of the TLC protocol can all be integrated directly into the fabric of

contemporary life (Ilardi et al., 2009) and appear to be effective as an acute treatment for MDD (Karwoski, 2006). It is hypothesized that they will also be effective at prevention.

The proposed study intervention can be classified both as a secondary and a tertiary prevention effort, and, more specifically, both a selective and an indicated program. In other words, this study will target college students who have suffered at least one major depressive episode in the past *and* are currently suffering from subclinical depression. Both factors are clear risks for the development of a full depressive episode (e.g., Gotlib, Lewinsohn, & Seeley, 1995; Horwath et al. 1992; Lewinsohn et al., 1988; Paykel et al., 1995; Pintor et al., 2001) and thus are appropriate targets of a preventive intervention.

Prevention is particularly relevant for at-risk college students in light of two additional considerations: (a) the increased threat of depression onset faced by younger cohorts, and (b) the high level of depressogenic stress that characterizes the college environment.

*Younger Cohorts at Risk*

As noted previously, the prevalence of depression is increasing over time (Seligman, 1990). There is a higher rate of both major depression and subclinical depression among younger generational cohorts (Kessler & Walters, 1998). Moreover, the age of onset for the initial depressive episode is younger for each successive cohort (Klerman et al., 1985). Colla and colleagues (Colla, Buka, Harrington, & Murphy, 2006) put forth two possible explanations to account for the

relationship between age and depression. First, being younger involves greater conflict over modernization issues, such as figuring out one's role in the family versus in the larger society. Second, when older generations were younger, the upheavals of modernization had not gone forward enough to create much psychological disturbance, and these individuals progressed to older age without many prior depressive episodes. This hypothesis is a promising one and relates to an evolutionary model of depression.

More than half of all individuals who will suffer from depression experience the first onset of the disorder – the initial episode – by age 25 (Sorenson, Rutter, & Aneshensel, 1991). In fact, being in one's teens or twenties is regarded as a primary risk factor for the onset of depression (Lewinsohn, Hoberman, & Rosenbaum, 1988), and one quarter of young adults in their teens and twenties have already suffered an episode of MDD (Kessler & Walters, 1998; Kessler et al., 2003). Preventing depression in young people is extremely important, and effective interventions carry the potential to decrease the chance of premature death due to suicide, reduce future health care costs, and, of course, increase quality of life far into the future (Lynch et al., 2005).

*Stressful College Environment*

Early stress (stress that occurs in early adulthood) is considered a major risk factor for the onset of a depressive episode. A person's first major depressive episode typically occurs in mid- to late adolescence (Hammen, 2001), and early episodes of depression are often preceded by stressful life events (Adams & Adams, 1993). In

fact, according to the diathesis-stress model of depression, without some sort of significant life stressor, even at-risk individuals are not especially likely to become depressed (Beck, 1967; Beck, 1983). For some college students, especially freshmen, the college environment can be extremely stressful. These young adults typically undergo a great deal of adjustment in the transition to college. Some of them encounter many, if not all, of the major stressors believed to precipitate bouts of depression (Vredenburg, Flett, & Krames, 1993): they have recently moved away from home for the first time, left behind their families and many of their closest friends, feel pressure to succeed academically in more difficult coursework, find themselves surrounded by thousands of other students, and may wind up feeling socially isolated and lonely (Petersen, Compas, & Brooks-Gunn, 1992).

A recent study of 1,455 college students from four universities found that 53% of participants experienced what they would label as depression during their time at college (Furr, Westefeld, McConnell, & Jenkins, 2001). Significant costs for institutions of higher education are incurred through untreated depression in students (Levine & Cureton, 1998). The National Mental Health Association suggested that colleges and universities would benefit from programs for prevention and early detection of depression (Field, Elliott, & Korn, 2006). To that end, researchers have created community-based interventions for the prevention of depression designed to reach broad groups of students (Field et al., 2006). Schools are an overlooked resource in combating the depression epidemic, but are important, especially because the school environment may contribute to risk (Parks & Herman, 2003). College

campuses offer abundant opportunities to implement both screenings and interventions (Larimer, Cronce, Lee, & Kilmer, 2004).

Although some researchers have argued against the use of college students in depression research (e.g., Coyne & Gotlib, 1983; Gotlib, 1984), Vredenburg and colleagues (1993) reviewed the existing literature and concluded that studies with college samples are worthwhile. The frequent stressors encountered in the college environment make students an important target for intervention, and there is no compelling evidence that depression in college students is significantly different from depression in psychiatric populations. In fact, college populations are particularly appropriate for investigations into the early development of depression and an important target for intervention efforts (Hart et al., 2001) for several reasons. First, the college environment is relatively homogeneous, and students have a high likelihood of encountering multiple stressful life events that tend to predict the onset of depression. It is important to target populations that are specifically at high risk for the development of depression at the time of the intervention or within the proximal future (Cardemil & Barber, 2001). Second, there is a reduced risk of other psychiatric disorders in college students that could interfere with or account for research findings. Third, depression has a profound impact on disinterest in school and can lead to impaired academic performance (Heiligenstein, Guenther, Hsu, & Herman, 1996). Fourth, the danger of suicide is 50% higher among college students than among non-students of the same age (Beck & Young, 1978). Fifth, because college students

typically do not seek help for depression (Vredenburg, O'Brian, & Krames, 1988), they are in need of intervention.

*Summary of Study Hypotheses*

Past research points to a high risk of MDD onset among individuals who are subclinically depressed or who have experienced a past depressive episode (e.g., Coyne et al., 1999; Judd et al., 1998). There is some evidence, however, that this risk can be attenuated on the basis of preventive psychosocial interventions (Bockting et al. 2005; de Mello et al., 2005; Ma & Teasdale, 2004; Rafanelli, Park, & Giovanni, 1999) as well as adherence to protective lifestyle factors such as exercise (Paffenbarger et al., 1994) and dietary omega-3 consumption (Stoll et al., 1999). Although there has not yet appeared a published evaluation of a single-session preventive intervention for depression, single-session interventions have already proven successful in inducing behavior change and thereby reducing the risk for other adverse clinical outcomes such as excessive alcohol use (Larimer et al., 2004) and risk-taking behaviors (D'Amico & Fromme, 2002). Accordingly, the principal hypotheses of the present study were as follows:

(1) a single one-hour psychoeducational TLC intervention will be sufficient to induce significantly greater adoption of protective lifestyle factors than those observed among a no-intervention control condition over a six-month follow-up period;

(2) participants who receive the preventive TLC intervention will be less likely to experience the onset of a major depressive episode than will controls at both the two-month and six-month follow-up evaluations; and

(3) participants who report greater adoption of protective lifestyle elements should be less likely to experience a major depressive episode than participants who report lower adherence.

## Method

### *Participants*

The study included 107 partial completers over the course of two years (four academic semesters). Participants were undergraduate students enrolled in an introductory psychology course at the University of Kansas. On the basis of information gathered in two self-report measures and a clinical interview, all participants (a) had experienced a past depressive episode of MDD, according to *DSM-IV-TR* criteria (APA, 2000) and/or (b) met criteria for current minor depressive disorder. Individuals who met current criteria for major depressive disorder were not included.

### *Measures*

*BDI-II.* The Beck Depression Inventory (BDI, Beck et al., 1988), and its successor, the BDI-II (Beck et al., 1996), are popular self-report measures designed to gauge the severity of depressive symptoms. The BDI-II is a 21-item questionnaire designed to measure the presence and severity of symptoms over the past two weeks. The Beck measures have been among the most widely used self-report questionnaires for depression, and they exhibit high internal consistency, test-retest reliability, and construct validity (Whisman, Perez, & Ramel, 2000; Beck et al., 1996; 1988). Some prevention researchers have even recommended that the BDI-II be used in all studies in depression, as it has become one of the benchmarks against which other outcomes can be compared (Dobson & Ottenbreit, 2004).

Possible scores on the BDI-II lie between 0 and 63. Typically, scores between 0 and 9 are viewed as normal, while scores between 10 and 20 are associated with mild or subclinical depression. Scores of 17 or higher are considered to be possible candidates for a diagnosis of MDD (Kendall, Hollon, Beck, Hammen, & Ingram, 1987). A cutoff score of 15 ensures a high probability that participants not be diagnosable with MDD. Thus, participants who scored between 10 and 15 on the BDI-II were recruited.

*IDDL.* The Inventory to Diagnose Depression, Lifetime Version (IDDL; Zimmerman & Coryell, 1987) is a 22-item self-report measure designed to diagnose lifetime history of MDD. It covers the entire range of symptoms used to diagnose current or past clinical depression. The IDDL has good internal consistency (Cronbach's $\alpha = 0.92$), split-half reliability, and test-retest reliability ($\kappa$=0.77; Zimmerman & Coryell), in addition to concordance with the frequently used Structured Clinical Interview for DSM-IV Disorders ($\kappa$=0.75 for the first trial of the IDDL and 0.68 for the second trial; Sato et al., 1996), discriminant validity ($\kappa$ =.59 for diagnosing lifetime history of major depression), sensitivity (83%), and specificity (79%; Sakado, Sato, Uehara, Sato, & Kameda, 1996) in clinical samples.

There is no specific cut-off score determining the presence of a current or past depressive episode on the IDDL. Rather, diagnosis is determined using a series of items that match the *DSM-IV-TR* (APA, 2000) symptom criteria for MDD. Following these criteria, to qualify for a likely diagnosis of MDD, individuals must report five or more depressive symptoms that are present during the same two-week period, at least

one of the symptoms being (a) depressed mood or (b) loss of interest or pleasure. The IDDL also asks for the start and end dates of the episode during which individuals felt the most depressed, and how many other similar episodes they have experienced. Participants who were unlikely to meet criteria for a current depressive episode but who were likely to meet criteria for a minor depressive episode (based on BDI-II responses) and are likely to have had one or more past major depressive episodes (based on IDDL responses) were recruited. The IDDL was used for the first and second semesters of recruitment.

*SCID.* The Structured Clinical Interview for *DSM-IV* Disorders mood module (SCID; First, Spitzer, Gibbon, & Willams, 1997) is a widely used structured clinical interview used to diagnose current and lifetime Axis I mental disorders, including MDD. This is a highly reliable tool and is known as the best diagnostic classification instrument to date. Interrater agreement kappas for diagnosing major depression using SCID range from .64 to .93 in both community and clinical samples (Segal, Hersen, & Van Hasselt, 1994).

Participants who screened into the study based on BDI-II and IDDL scores were invited to come in and undergo only the depression mood module of the SCID. They were considered eligible for the study if they met criteria for a past depressive episode and current minor depressive disorder. Defining subclinical depression can be tricky and is difficult to pin down operationally (Ingram et al., 2004). For the purposes of this study, participants were included if they met criteria for minor depressive disorder, which according to the *DSM-IV-TR* requires at least two but

fewer than five symptoms of a major depressive episode, at least one of the symptoms being either (a) depressed mood or (b) loss of interest or pleasure (APA, 2000). Participants who met criteria for current major depressive disorder were excluded.

Participants were not screened specifically for anxiety disorders; most interventions for depression do not use comorbid anxiety as an exclusionary criterion (e.g., DeRubeis et al., 2005). As Kendall and colleagues (1987) point out, consistent agreement scores across multiple measurement methods are necessary to secure a diagnosis, or lack thereof, of major depression. Thus, although participants were screened into the study on the basis of BDI-II and IDDL scores, the SCID determined whether they were eligible to continue. If, at the conclusion of the SCID interview, participants were not eligible for the intervention, they were not offered the chance to participate in the intervention. However, referral sources as described in the below section were provided to them upon conclusion of the interview.

Training for the four SCID interviewers was done by Chantal Young, M.A., who was trained by watching the standard SCID training tapes prepared at the New York State Psychiatric Institute by First and Gibbon (1996) for *DSM-IV*, and by receiving private training by Dr. Juliet Nelson and performing approximately ten interviews under the supervision of Nelson. The four interviewers for this study were second or third year graduate students who underwent private training by Young. They performed several practice interviews, and then performed approximately 10 interviews on actual participants with Young in the room. Both Young and the interviewers scored the SCID depression module separately, and then met afterward

to examine any discrepancies. When discrepancies did occasionally arise, they were discussed in detail. Interviewers continued to perform interviews under supervision until inter-rater concordance with Young was near 100%.

*TLC Adherence Questionnaire.* An adherence questionnaire (see Appendix A) measured compliance with TLC lifestyle elements by asking participants about the degree to which they are following each element. Both treatment and control group participants completed this questionnaire, because it is feasible that those in the control group may have implemented TLC elements on their own. Mixed in with questions about TLC elements was a set of "distractor" questions about other lifestyle domains unrelated to TLC, included in order to prevent the control group from realizing the nature of study hypotheses.

*Social Support Measure.* Included in the TLC Adherence Questionnaire was a 20-item measure assessing participants' available social support resources and openness to receiving social support, based on prior questionnaires assessing social support (Folkman & Lazarus, 1980; McGrath, Gutierrez, & Valadez, 2000; Sarason, Levine, Basham, & Sarason, 1983; Sarason, Sarason, Shearin, & Pierce, 1987; Vaux, 1985; Vaux, A., Riedel, S., & Stewart, D., 1987). The Social Support Measure included two categories of social support: emotional and instrumental (Carver, Scheier, & Weintraub, 1989; Cutrona, 1990; Florian, Mikulincer, & Bucholtz, 1995; Slavin, 1991). Within these categories, three types of support were included: perceived, actively sought, and passively accepted, derived from past research (Cornman, Goldman, Glei, Weinstein, Chang, 2003; Hawthorne, 2006; Herrero &

Gracia, 2007; Srivastava & Singh, 2006; Vitaliano, Russo, Carr, Maiuro, & Becker, 1985; Zimet, Dahlem, Zimet, & Farley, 1988). Sample items included: *I seek sympathy and understanding from my friends and family* (classified as emotional and actively sought); *I am open to receiving help from other people with day-to-day tasks* (classified as instrumental and passively accepted). Participants rated each item on a 7-point scale, ranging from 1 (*disagree strongly)* to 7 (*agree strongly*). A total score ranging from 0 to 140 was calculated for each participant.

*Adherence Metric*. Six adherence variables, one for each TLC element, were created. Adherence variables reflected participants' proportional adherence (percentage) with respect to the target goal for each element. Target goals as presented in the psychoeducational presentation were as follows: for fish oil, the recommended dosage was at least 6 pills per day (at a per-pill dose of 180mg EPA); for cardiovascular exercise, at least 1.5 hours per week of aerobic activity; for sunlight exposure, at least 60 minutes per day; for social support, as little time as possible spent alone (operationalized as at most one non-sleeping hour alone per day); for anti-rumination, as little time as possible spent ruminating (operationalized as zero hours ruminating per day).

Adherence for fish oil supplementation was calculated as the mean number of pills per day divided by six. For exercise, it was the mean number of hours of cardiovascular exercise per week divided by 1.5. To derive a measure of adherence to sunlight exposure, the mean minutes of exposure to sunlight or artificial bright light was divided by 60. For social support adherence, mean waking hours spent in the

company of others[1] was divided by the number of waking hours. For anti-rumination, the mean number of hours spent ruminating per day was subtracted from the number of waking hours per day, and the difference was divided by the number of waking hours. All adherence percentages were capped at 100%, and participants were not penalized for exceeding recommended dosages.

## *Procedure*

### *Recruitment*

Introductory psychology students were recruited to take part in the study as partial fulfillment of course requirements. They completed an online mass screening questionnaire that asks about current and past depressive symptoms, using questions adapted from the BDI-II and IDDL. Based on these self-report measures, selected participants were unlikely to meet criteria for MDD, but were likely to be in the midst of a subclinical depressive episode and/or have a history of past major depressive disorder. Prospective participants were contacted via a method they indicate as most convenient (e-mail or telephone) and invited to enlist in the study.

Participants were then evaluated in person to verify eligibility by Chantal Young, M.A. or another trained graduate-level research assistant. Eligibility was verified through verbal completion of the portion of the SCID depression module that assesses current and past depression. Participants were selected if they met criteria for current minor depressive disorder and/or had a history of major depressive episodes,

[1] The social support adherence variable was adjusted to allow for one hour of alone time per day.

but did not meet current criteria for MDD. If participants were eligible, they filled out the TLC adherence questionnaire and BDI-II at that time.

Upon conclusion of the in-person evaluation, participants were randomly assigned to either the treatment or control group. Participants assigned to the treatment condition were invited to fulfill further course credit by participating in a one-hour educational session and a subsequent two-month follow-up session. Participants assigned to the control condition were invited to participate in a two-month follow-up session, also for course credit.

*Treatment Group*

Sixty participants were randomly assigned to the treatment group. Random assignment was achieved by using a random number generator to assign each participant a number between 1 and 100. Those who received numbers between 1 and 50 were assigned to the treatment group, and those with numbers between 51 and 100 were assigned to the control group. Those assigned to the treatment group attended a one-hour psychoeducational session on the antidepressant value of TLC lifestyle elements. This session was a highly condensed, streamlined version (see Appendix B) of the original 12-week protocol for treating acute depression (Ilardi et al., 2009). The sessions were run in groups of four to eight participants and were led by Chantal Young, M.A. or another trained graduate-level research assistant. Details of the session are reviewed in this section.

The session began with an introduction. Participants were informed that their only task during the session is to listen attentively and take notes if they wish. They

were assured that they would not be required to share any personal information with the group, but if they chose to do so, everything would be kept confidential. All group members were asked not to share any personal information with anyone outside of the group.

The session moved on to a brief overview of the symptoms of depression, with an emphasis on the recurrent nature of depression, danger of relapse, and subclinical symptoms as a precursor to full-blown MDD (approximately 15 minutes). This information was included because depression is a long-term problem, and researchers argue that it is important to teach individuals how to recognize the early symptoms and encourage them to help themselves at the earliest signs of clinical change (Franchini et al., 1999). Although low mood in and of itself is not necessarily maladaptive (Keller & Nesse, 2005), in at-risk individuals it can lead to major depression, which is an enormously debilitating problem. Participants were advised to watch for signs of extreme decline in mood. They were then informed of the purpose of the session, which was to teach skills that have been shown to help prevent the onset of major depression.

The session continued to an explanation of the evolutionary "mismatch" hypothesis (Ilardi et al., 2009). Participants were asked to consider the vast differences between modern and ancestral lifestyles, and were provided a summary of typical American lifestyle habits that are emotionally toxic (approximately 15 minutes).

Next, participants were given an outline of the six TLC lifestyle elements that are believed to be antidepressant in nature: physical exercise, dietary omega-3 fatty acids, sunlight exposure, sleep, social support, and anti-ruminative activity (remaining 30 minutes). Participants were given specifics for each TLC element, including a brief theoretical explanation of their functions in mental health, followed by precise "dosage" information. They were given supplementary handouts with replication of the same material that was given verbally (see Appendix C). They were also encouraged to visit the TLC website for further information. Finally, they were asked to fill out a goal sheet with their current usage of TLC elements and their personal goals for the semester, following the presentation (see Appendix D). They were instructed to keep these goal sheets for themselves.

Questions and clarifications were welcomed and answered as thoroughly as possible, given time constraints. Upon conclusion of the hour, participants were invited to stay longer on their own time if they had further questions or would like to hear more about the theoretical background of the materials presented. They were provided with and encouraged to use additional referral sources, including contact information for the KU Psychological Clinic, a 24-hour suicide hotline, and multiple private psychotherapists in the Lawrence area. Before leaving, participants signed up for two- and six-month follow-up meetings.

*Website.* Treatment group participants were given the address to the TLC website. This website provides detailed information about major depressive disorder and an outline of each of the TLC elements and how it is relevant to preventing

depression. Participants had access to the website on their own time, at any hour of the day, so that the intervention could be reinforced through this ongoing material. Conveying information passively through written and online materials is not only less expensive, but also increases the feasibility of the intervention because greater contact is maintained with clients in their natural environments (Muñoz, 1993). There is evidence that internet-based preventive measures for depression are effective, especially when paired with in-person elements (Van Voorhees et al., 2005).

*Control Group*

Forty-one participants were randomly assigned to an assessment-only control group. They were provided with and encouraged to use the same referral sources given to the treatment group participants. They signed up for a two-month follow-up upon conclusion of the initial interview, and a six-month follow-up upon conclusion of the two-month follow-up. At the six-month follow-up, control group participants were fully informed of the potential benefit of TLC elements. They were also given the handouts on TLC that were given to the treatment group participants, and encouraged to visit the TLC website for further information.

*Follow-Up Assessments*

All participants were invited to participate in two-month and six-month follow-up meetings. Because the two-month follow-up fell before the end of the semester, partial course credit was given in exchange for completion. However, because the six-month follow-up was beyond the end of the semester, participants were offered participation in a raffle in exchange for their participation. Five prizes of

$100 were given away at the end of the study. At both follow-ups, participants were re-administered the module of the SCID assessing current MDD, the BDI-II, and the adherence questionnaire. If for any reason participants were unable to come in for the six-month follow-up in person, they were mailed the BDI-II and adherence questionnaire and asked to return them in a prepaid envelope. They were also re-administered the module of the SCID assessing current MDD over the phone. Past research shows that telephone assessments of major depression using the SCID have good agreement with in-person interviews ($\kappa$=.64, Cacciola, Alterman, Rutherford, McKay, & May, 1999; $\kappa$=.73, Simon, Revicki, & VonKorff, 1993) and are acceptable to interviewees (Allen, Cull, & Sharpe, 2003). The follow-ups took approximately 20 minutes per person.

*Consent and Confidentiality*

All participants gave informed consent for each step in the study. They were informed that they were free to withdraw from the study without penalty and that they would be given credit for every half hour they spent on the study (excluding the six-month follow-up, for which they received enrollment in the raffle). They were assured that all information they shared, both verbally and on questionnaires, would be kept confidential. Participants were given debriefing forms at the conclusion of the study with information about the purpose of the study.

## Results

*Characteristics*

An initial sample of 406 prospective participants was screened to take part in the study, of which 173 were deemed eligible on the basis of meeting *DSM-IV-TR* (APA, 2000) diagnostic criteria for either a past major depressive episode or a current minor depressive episode (or both). All eligible participants were randomized into one of two conditions: 97 (56%) were assigned to the study's treatment group, and 76 were assigned (44%) to the control group. Among these randomized participants, 107 (aged 18-37 years, mean = 19.5; 67 women, 40 men) attended at least one follow-up session, and were therefore included in the primary study analyses. Of the 107 individuals included in these principal analyses, 60 had been assigned to the treatment condition, and 47 to the control condition. These two resulting groups did not differ significantly[2] (all $p>.05$) with respect to sex, age, year in school, race, baseline BDI-II scores, experience of a current minor depressive episode or a past major depressive episode, current use of psychotherapy or psychotropic medications, or pre-study adherence to TLC healthy lifestyle factors (see Table 1).

*Retention*

As noted, 107 (62%) of 173 eligible participants returned for at least one follow-up. Fisher's exact test revealed no significant difference in retention between

[2]Categorical variables were analyzed using either a chi-square analysis with Yates' correction (Preacher, 2001) or a Fisher's exact test (Preacher & Briggs, 2001). Dimensional variables were analyzed using appropriate t-tests.

Table 1

*Baseline comparison of treatment and control groups*

| | Treatment | Control | Total | Fisher's exact test *p* |
|---|---|---|---|---|
| Sex | | | | |
| Male | 43.3% (26) | 29.8% (14) | 37.4% (40) | .16 |
| Female | 56.7% (34) | 70.2% (33) | 62.6% (67) | |
| Presence of current minor depressive episode | | | | |
| Yes | 46.7% (28) | 63.8% (30) | 54.2% (58) | .08 |
| No | 53.3% (32) | 36.2% (17) | 45.8% (49) | |
| Experience of past major depressive episode | | | | |
| Yes | 73.3% (44) | 68.1% (32) | 71.0% (76) | .67 |
| No | 26.7% (16) | 31.9% (15) | 29.0% (31) | |
| Currently in psychotherapy | | | | |
| Yes | 10.0% (6) | 4.3% (2) | 7.5% (8) | .46 |
| No | 90.0% (54) | 95.7% (45) | 92.5% (99) | |
| Currently using psychotropic medications | | | | |
| Yes | 13.3% (8) | 10.6% (5) | 12.1% (13) | .77 |
| No | 86.7% (52) | 89.4% (42) | 87.9% (94) | |

Table 1, cont.

| | Treatment | Control | Total | $\chi^2$ | df | p |
|---|---|---|---|---|---|---|
| Year in school | | | | | | |
| Freshman | 63.3% (38) | 57.4% (27) | 60.7% (65) | .14 | 3 | .99 |
| Sophomore | 25.0% (15) | 25.5% (12) | 25.2% (27) | | | |
| Junior | 11.7% (7) | 14.9% (7) | 13.1% (14) | | | |
| Senior | 0.0% (0) | 2.1% (1) | 0.9% (1) | | | |
| Race/ethnicity | | | | | | |
| Caucasian | 81.7% (49) | 87.2% (41) | 84.1% (90) | .23 | 7 | 1.00 |
| Latino-Am. | 6.7% (4) | 4.3% (2) | 5.6% (6) | | | |
| Asian-Am. | 3.3% (2) | 4.3% (2) | 3.7% (4) | | | |
| African-Am. | 1.7% (1) | 0.0% (0) | 0.9% (1) | | | |
| Native Am. | 1.7% (1) | 0.0% (0) | 0.9% (1) | | | |
| Bi-/Multicult. | 1.7% (1) | 0.0% (0) | 0.9% (1) | | | |
| International | 1.7% (1) | 4.3% (2) | 2.8% (3) | | | |
| Other | 1.7% (1) | 0.0% (0) | 0.9% (1) | | | |

Table 1, cont.

| | Treatment | Control | Total | F | t | df | p |
|---|---|---|---|---|---|---|---|
| Age | | | | | | | |
| Mean | 19.16 | 19.80 | 19.45 | 5.56 | -1.19 | 99 | .24 |
| Range | 18-29 | 18-37 | 18-37 | | | | |
| SD | 1.74 | 3.54 | 2.70 | | | | |
| Baseline BDI-II score | | | | | | | |
| Mean | 11.65 | 11.38 | 11.53 | 1.37 | 0.17 | 105 | .87 |
| Range | 0-33 | 1-36 | 0-36 | | | | |
| SD | 8.46 | 7.87 | 8.17 | | | | |
| Baseline sleep adherence | | | | | | | |
| Mean | 87.03% | 83.51% | 85.48% | 2.52 | 1.22 | 105 | .23 |
| Range | 56-100% | 0-100% | 0-100% | | | | |
| SD | 11.87 | 17.89 | 14.85 | | | | |
| Baseline social support adherence | | | | | | | |
| Mean | 81.12% | 81.65% | 81.35% | .28 | -.16 | 103 | .88 |
| Range | 17-100% | 0-100% | 0-100% | | | | |
| SD | 15.70 | 18.86 | 17.07 | | | | |

Table 1, cont.

| | Treatment | Control | Total | F | t | df | p |
|---|---|---|---|---|---|---|---|
| Baseline fish oil adherence | | | | | | | |
| Mean | 5.14% | 5.67% | 5.37% | .28 | -.17 | 105 | .86 |
| Range | 0-67% | 0-100% | 0-100% | | | | |
| SD | 13.12 | 18.80 | 17.07 | | | | |
| Baseline sunlight exposure adherence | | | | | | | |
| Mean | 74.93% | 87.01% | 79.98% | 9.71 | -1.87 | 101 | .07 |
| Range | 0-100% | 0-100% | 0-100% | | | | |
| SD | 35.75 | 26.92 | 32.76 | | | | |
| Baseline exercise adherence | | | | | | | |
| Mean | 76.84% | 75.89% | 76.42% | .00 | -.12 | 104 | .90 |
| Range | 0-100% | 0-100% | 0-100% | | | | |
| SD | 39.76 | 39.75 | 39.57 | | | | |
| Baseline anti-rumination adherence | | | | | | | |
| Mean | 91.97% | 91.19% | 91.64% | .03 | .42 | 102 | .68 |
| Range | 42-100% | 61-100% | 42-100% | | | | |
| SD | 9.78 | 8.76 | 9.34 | | | | |

*Note*. None of the differences was statistically significant at the $\alpha$=.05 level.

treatment groups: 60 participants (61%) were retained in the treatment group and 47 participants (62%) in the control group ($p$>.05).

Within both the treatment and control groups, comparisons showed no significant differences between participants who returned and those who did not on the basis of sex, age, year in school, race, baseline BDI-II scores, presence of a current minor depressive episode or a past major depressive episode, psychotherapy participation, psychotropic medication use, or pre-study adherence to TLC healthy lifestyle factors (see Tables 2 and 3).

Among the 107 participants included in main analyses, 42 (39%) were considered partial completers because they attended only the two-month follow-up (41) or only the six-month follow-up (1), while 65 (61%) were considered full completers because they attended both follow-up sessions. Of participants in the treatment group, 60 (100%) completed the two-month follow-up, and 42 (70%) completed the six-month follow-up. Of participants in the control group, 46 (98%) completed the two-month follow-up, and 24 (51%) completed the six-month follow-up.

*Adherence variables*

Six adherence variables, one for each TLC element, were created for the purpose of study analyses. These variables reflected participants' proportional adherence (on a percentage basis) with respect to the target behavioral goal for each respective element. For example, for fish oil, the target dosage (as suggested in the psychoeducational presentation) was six supplement capsules ingested per day. Thus,

Table 2

*Baseline comparison of treatment group participants who returned and those who did not return*

| | Returned | Did not return | Total | Fisher's exact test *p* |
|---|---|---|---|---|
| Sex | | | | |
| Male | 43.3% (26) | 35.1% (13) | 40.2% (39) | .52 |
| Female | 56.7% (34) | 64.9% (24) | 59.8% (58) | |
| Presence of current minor depressive episode | | | | |
| Yes | 46.7% (28) | 59.5% (22) | 51.5% (50) | .30 |
| No | 53.3% (32) | 40.5% (15) | 48.5% (47) | |
| Experience of past major depressive episode | | | | |
| Yes | 73.3% (44) | 62.2% (23) | 69.1% (67 ) | .27 |
| No | 26.7% (16) | 37.8% (14) | 30.9% (30) | |
| Currently in psychotherapy | | | | |
| Yes | 10.0% (6) | 12.1% (4) | 10.8% (10) | .74 |
| No | 90.0% (54) | 87.9% (29) | 89.2% (83) | |
| Currently using psychotropic medications | | | | |
| Yes | 13.3% (8) | 9.1% (3) | 11.8% (11) | .74 |
| No | 86.7% (52) | 90.9% (30) | 88.29% (82) | |

Table 2, cont.

| | Returned | Did not return | Total | $\chi^2$ | df | p |
|---|---|---|---|---|---|---|
| Year in school | | | | | | |
| Freshman | 63.3% (38) | 69.7% (23) | 65.6% (61) | .40 | 3 | .94 |
| Sophomore | 25.0% (15) | 21.2% (7) | 23.7% (22) | | | |
| Junior | 11.7% (7) | 6.1% (2) | 9.7% (9) | | | |
| Senior | 0.0% (0) | 3.0% (1) | 1.1% (1) | | | |
| Race/ethnicity | | | | | | |
| Caucasian | 81.7% (49) | 84.8% (28) | 82.8% (77) | .85 | 7 | 1.00 |
| Latino-Am. | 6.7% (4) | 6.1% (2) | 6.5% (6) | | | |
| Asian-Am. | 3.3% (2) | 3.0% (1) | 3.2% (3) | | | |
| African-Am. | 1.7% (1) | 0.0% (0) | 1.2% (1) | | | |
| Native Am. | 1.7% (1) | 3.0% (1) | 2.2% (2) | | | |
| Bi-/Multicult. | 1.7% (1) | 0.0% (0) | 1.2% (1) | | | |
| International | 1.7% (1) | 3.0% (1) | 2.2% (2) | | | |
| Other | 1.7% (1) | 0.0% (0) | 1.2% (1) | | | |

Table 2, cont.

| | Returned | Did not return | Total | F | t | df | p |
|---|---|---|---|---|---|---|---|
| Age | | | | | | | |
| Mean | 19.16 | 19.61 | 19.32 | 2.61 | -.77 | 85 | .44 |
| Range | 18-29 | 18-38 | 18-38 | | | | |
| SD | 1.74 | 3.71 | 2.60 | | | | |
| Baseline BDI-II score | | | | | | | |
| Mean | 11.65 | 14.54 | 12.72 | .02 | -1.59 | 93 | .12 |
| Range | 0-33 | 0-34 | 0-34 | | | | |
| SD | 8.46 | 8.69 | 8.62 | | | | |
| Baseline sleep adherence | | | | | | | |
| Mean | 87.03% | 87.88% | 87.33% | .02 | -.33 | 91 | .75 |
| Range | 56-100% | 0-100% | 0-100% | | | | |
| SD | 11.87 | 12.30 | 11.97 | | | | |
| Baseline social support adherence | | | | | | | |
| Mean | 81.12% | 75.17% | 79.07% | 2.83 | 1.48 | 88 | .14 |
| Range | 17-100% | 0-100% | 0-100% | | | | |
| SD | 15.70 | 22.26 | 18.32 | | | | |

Table 2, cont.

| | Returned | Did not return | Total | F | t | df | p |
|---|---|---|---|---|---|---|---|
| Baseline fish oil adherence | | | | | | | |
| Mean | 5.14% | 5.67% | 5.29% | .34 | -.13 | 91 | .90 |
| Range | 0-67% | 0-100% | 0-100% | | | | |
| SD | 13.12 | 18.48 | 15.14 | | | | |
| Baseline sunlight exposure adherence | | | | | | | |
| Mean | 74.93% | 85.78% | 78.55% | 6.72 | -1.48 | 88 | .14 |
| Range | 0-100% | 17-100% | 0-100% | | | | |
| SD | 35.75 | 25.67 | 32.99 | | | | |
| Baseline exercise adherence | | | | | | | |
| Mean | 76.84% | 70.71% | 74.64% | 1.71 | .68 | 90 | .50 |
| Range | 0-100% | 0-100% | 0-100% | | | | |
| SD | 39.76 | 44.69 | 41.45 | | | | |
| Baseline anti-rumination adherence | | | | | | | |
| Mean | 91.97% | 87.82% | 90.56% | .62 | 1.92 | 89 | .06 |
| Range | 42-100% | 63-100% | 42-100% | | | | |
| SD | 9.78 | 9.76 | 9.92 | | | | |

*Note*. None of the differences was statistically significant at the $\alpha$=.05 level.

Table 3

*Baseline comparison of control group participants who returned and those who did not return*

| | Returned | Did not return | Total | Fisher's exact test *p* |
|---|---|---|---|---|
| Sex | | | | |
| Male | 30.4% (14) | 31.0% (9) | 30.3% (23) | 1.00 |
| Female | 69.6% (33) | 69.0% (20) | 69.7% (53) | |
| Presence of current minor depressive episode | | | | |
| Yes | 63.8% (30) | 62.2% (18) | 63.2% (48) | 1.00 |
| No | 36.2% (17) | 37.9% (11) | 36.8% (28) | |
| Experience of past major depressive episode | | | | |
| Yes | 68.1% (32) | 75.9% (22) | 70.1% (54) | .60 |
| No | 31.9% (15) | 24.1% (7) | 29.9% (23) | |
| Currently in psychotherapy | | | | |
| Yes | 33.3% (2) | 65.2% (45) | 8.0% (6) | .19 |
| No | 66.7% (4) | 87.9% (29) | 92.0% (69) | |
| Currently using psychotropic medications | | | | |
| Yes | 10.6% (5) | 28.6% (8) | 17.3% (13) | .06 |
| No | 89.4% (42) | 71.4% (20) | 82.7% (62) | |

Table 3, cont.

| | Returned | Did not return | Total | $\chi^2$ | df | p |
|---|---|---|---|---|---|---|
| Year in school | | | | | | |
| Freshman | 57.4% (27) | 71.4% (20) | 62.7% (47) | 3.14 | 3 | .37 |
| Sophomore | 25.5% (12) | 28.6% (8) | 26.7% (20) | | | |
| Junior | 14.9% (7) | 0.0% (0) | 9.3% (7) | | | |
| Senior | 2.1% (1) | 0.0% (0) | 1.3% (1) | | | |
| Race/ethnicity | | | | | | |
| Caucasian | 85.4% (41) | 85.74% (24) | 85.5% (65) | .68 | 5 | .98 |
| Latino-Am. | 4.2% (2) | 3.6% (1) | 3.9% (3) | | | |
| Asian-Am. | 4.2% (2) | 0.0% (0) | 2.6% (2) | | | |
| African-Am. | 0.0% (0) | 3.6% (1) | 1.3% (1) | | | |
| Native Am. | 0.0% (0) | 0.0% (0) | 0.0% (0) | | | |
| Bi-/Multi-cult. | 0.0% (0) | 0.0% (0) | 0.0% (0) | | | |
| International | 4.2% (2) | 3.6% (1) | 3.9% (3) | | | |
| Other | 2.1% (1) | 3.6% (1) | 2.6% (2) | | | |

Table 3, cont.

| | Returned | Did not return | Total | F | t | df | p |
|---|---|---|---|---|---|---|---|
| Age | | | | | | | |
| Mean | 19.80 | 18.69 | 19.39 | 5.16 | 1.55 | 69 | .13 |
| Range | 18-37 | 18-23 | 18-37 | | | | |
| SD | 3.54 | 1.09 | 2.93 | | | | |
| Baseline BDI-II score | | | | | | | |
| Mean | 11.38 | 11.12 | 11.28 | 2.13 | .13 | 74 | .90 |
| Range | 1-36 | 0-37 | 0-37 | | | | |
| SD | 7.87 | 9.45 | 8.45 | | | | |
| Baseline sleep adherence | | | | | | | |
| Mean | 83.51% | 90.85% | 86.25% | 2.36 | -1.96 | 7 | .054 |
| Range | 0-100% | 63-100% | 0-100% | | | | |
| SD | 17.89 | 11.09 | 16.02 | | | | |
| Baseline social support adherence | | | | | | | |
| Mean | 81.65% | 80.95% | 81.38% | 5.16 | 1.55 | 69 | .13 |
| Range | 0-100% | 25-97% | 0-100% | | | | |
| SD | 18.86 | 14.81 | 17.34 | | | | |

Table 3, cont.

| | Returned | Did not return | Total | F | t | df | p |
|---|---|---|---|---|---|---|---|
| Baseline fish oil adherence | | | | | | | |
| Mean | 5.67% | 9.23% | 7.00% | 1.43 | -.74 | 73 | .46 |
| Range | 0-100% | 0-100% | 0-100% | | | | |
| SD | 18.80 | 22.15 | 20.04 | | | | |
| Baseline sunlight exposure adherence | | | | | | | |
| Mean | 87.02% | 84.12% | 85.87% | .28 | .45 | 69 | .66 |
| Range | 0-100% | 7-100% | 0-100% | | | | |
| SD | 26.92 | 26.83 | 26.73 | | | | |
| Baseline exercise adherence | | | | | | | |
| Mean | 75.89% | 88.10% | 80.44% | 9.88 | -1.47 | 73 | .15 |
| Range | 0-100% | 0-100% | 0-100% | | | | |
| SD | 39.75 | 24.37 | 35.13 | | | | |
| Baseline anti-rumination adherence | | | | | | | |
| Mean | 91.19% | 89.78% | 90.65% | 3.09 | .55 | 69 | .59 |
| Range | 61-100% | 45-100% | 45-100% | | | | |
| SD | 8.76 | 12.95 | 10.48 | | | | |

*Note*. None of the differences was statistically significant at the $\alpha$=.05 level.

a participant who reported taking six pills per day would be 100% adherent; a participant who reported taking three pills per day would be 50% adherent. Target goals for the other TLC elements were as follows: exercise, 1.5 hours per week; sunlight exposure, 60 minutes per day; social support, one hour alone per day (at most); anti-rumination, zero hours ruminating per day; sleep, eight hours per night.

*Effect of TLC treatment on TLC adherence*

The first study hypothesis was that the one-hour psychoeducational TLC intervention would be sufficient to induce significantly greater adoption of protective lifestyle factors than those observed among the no-intervention control condition. To test this hypothesis, six 2x2 repeated measures ANOVAs were conducted to compare initial adherence to TLC elements to adherence at the two-month follow-up (see Table 4A). There was a significant main effect of treatment group on adherence to fish oil supplementation, $F(1,103) = 8.05$, $p<.01$, and a significant time-by-treatment interaction effect for fish oil, as well, $F(1,103) = 16.11$, $p<.001$. In addition, there were observed trends for greater sleep adherence in the treatment group, $F(1,104) = 3.52$, $p=.06$, and for a time-by-interaction effect for social support, $F(1, 101) = 3.38$, $p=.069$. There was no significant effect of treatment group on adherence to the remaining TLC elements (exercise, sunlight exposure, and anti-rumination), nor were any other significant time-by-treatment interaction effects observed.

Follow-up *t*-tests were conducted to evaluate mean differences in adherence to fish oil, sleep, and social support (see Table 4B for means and standard deviations).

Table 4A

*Effect of treatment condition on TLC adherence at two- and six-month follow-ups*

| Two-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Sleep | | | | |
| Treatment group | 1366.34 | 3.52 | .06 | 104 |
| Time | 113.17 | 1.33 | .25 | |
| Time * Treatment group | 166.24 | 1.95 | .16 | |
| Social support | | | | |
| Treatment group | 121.85 | .28 | .60 | 101 |
| Time | 3.68 | .03 | .88 | |
| Time * Treatment group | 504.28 | 3.38 | .069 | |
| Fish oil | | | | |
| Treatment group | 5383.71 | 8.05** | .005 | 103 |
| Time | 4677.69 | 12.07** | .001 | |
| Time * Treatment group | 6243.83 | 16.11** | .000 | |
| Sunlight exposure | | | | |
| Treatment group | 5760.38 | 4.30* | .04 | 98 |
| Time | 149.13 | .19 | .67 | |
| Time * Treatment group | 14.16 | .02 | .89 | |

Table 4A, cont.

| Two-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Exercise | | | | |
| Treatment group | 569.60 | .23 | .63 | 101 |
| Time | 1775.86 | 2.01 | .16 | |
| Time * Treatment group | 28.29 | .03 | .86 | |
| Anti-rumination | | | | |
| Treatment Group | 35.25 | .29 | .59 | 100 |
| Time | 164.80 | 3.84 | .053 | |
| Time * Treatment group | .43 | .01 | .92 | |

| Six-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Sleep | | | | |
| Treatment group | 1096.58 | 3.64 | .06 | 63 |
| Time | 360.81 | 3.44 | .07 | |
| Time * Treatment group | 194.95 | 1.86 | .18 | |
| Social | | | | |
| Treatment group | 5.12 | .02 | .88 | 63 |
| Time | 506.92 | 2.96 | .09 | |
| Time * Treatment group | 33.96 | .20 | .66 | |

Table 4A, cont.

| Six-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Fish oil | | | | |
| Treatment group | 3819.87 | 4.88* | .03 | 61 |
| Time | 3554.69 | 7.35** | .009 | |
| Time * Treatment group | 3783.96 | 7.82** | .007 | |
| Sunlight exposure | | | | |
| Treatment group | 3067.26 | 2.04 | .16 | 61 |
| Time | 419.62 | .53 | .47 | |
| Time * Treatment group | .64 | .001 | .98 | |
| Exercise | | | | |
| Treatment group | 306.13 | .14 | .71 | 62 |
| Time | 630.21 | .63 | .43 | |
| Time * Treatment group | 421.88 | .42 | .52 | |
| Anti-rumination | | | | |
| Treatment group | .12 | .001 | .98 | 60 |
| Time | .30 | .002 | .97 | |
| Time * Treatment group | 5.78 | .04 | .85 | |

*$p$<.05
**$p$<.01

Table 4B

*TLC adherence among treatment groups at two- and six-month follow-ups*

| | Two months | | Six months | |
|---|---|---|---|---|
| | Treatment | Control | Treatment | Control |
| Sleep | | | | |
| Mean | 87.34%* | 80.43%* | 89.94%* | 86.46%* |
| SD | 13.00 | 19.03 | 12.09 | 12.18 |
| Social support | | | | |
| Mean | 84.37 | 79.85% | 86.58% | 85.11% |
| SD | 10.90 | 23.06 | 11.84 | 12.59 |
| Fish oil | | | | |
| Mean | 25.69%* | 4.44%* | 27.14%* | 4.51%* |
| SD | 34.73 | 16.05 | 37.35 | 20.41 |
| Sunlight exposure | | | | |
| Mean | 73.71% | 84.03% | 70.67% | 81.70% |
| SD | 32.64 | 31.62 | 33.61 | 31.76 |
| Exercise | | | | |
| Mean | 72.31% | 68.89% | 74.39% | 76.39% |
| SD | 41.59 | 44.04 | 35.37 | 41.10 |

Table 4B, cont.

| | Two months | | Six months | |
|---|---|---|---|---|
| | Treatment | Control | Treatment | Control |
| Anti-rumination | | | | |
| Mean | 93.70% | 91.37% | 92.55% | 91.97% |
| SD | 6.95 | 13.23 | 15.77 | 11.98 |

**T*-tests showed a significant difference between treatment groups at α=.05.

As expected, there was a significant between-group difference at the two-month follow-up regarding adherence to fish oil, $t(103) = 35.96$, $p<.001$, but not at the initial screening, $t(105) = .86$, $p=.86$. Participants in the treatment group had an average fish oil adherence of 25.69% ($SD$=34.73) at two months, while participants in the control group had an adherence of 4.44% ($SD$=16.05). Also as expected, there was a significant difference between the treatment and control groups at the two-month follow-up in adherence to sleep, $t(104) = 2.23$, $p<.05$, but not at the initial screening, $t(104) = 1.15$, $p=.26$. Participants in the treatment group had an average sleep adherence of 87.34% ($SD$=13.00) at two months, while participants in the control group had an adherence of 80.43% ($SD$=19.03). For social support adherence, no significant difference was found between groups at either the initial screening, $t(102) = -.16$, $p=.87$, or two month follow-up, $t(103) = 1.13$, $p=.19$.

A second set of six 2x2 repeated measures ANOVAs was conducted to compare initial adherence to reported adherence at the six-month follow-up (see Table 4A). There was again a significant main effect of treatment group on fish oil supplementation, $F(1,61) = 4.88$, $p<.05$, as well as a significant time-by-treatment interaction effect for fish oil, $F(1,61) = 7.82$, $p<.01$. In addition, there was a trend for greater sleep adherence in the treatment group, $F(1,63) = 3.64$, $p=.06$. No significant main effect of treatment group was observed regarding adherence to the remaining TLC elements (sunlight exposure, exercise, social support, and anti-rumination), nor were any other significant time-by-treatment interaction effects observed.

Follow-up *t*-tests tests were conducted to evaluate mean differences in adherence to fish oil and sleep (see Table 4B for means and standard deviations). As expected, there was a significant difference between the treatment and control groups at the six-month follow-up in adherence to fish oil, $t(63) = 2.87$, $p<.01$, but not at the initial screening, $t(64) = -.08$, $p=.94$. Participants in the treatment group had an average fish oil adherence of 31.55% (*SD*=38.72) at six months, while participants in the control group had an adherence of 6.52% (*SD*=21.17). Also as expected, there was a significant difference in adherence to sleep, $t(63) = 2.76$, $p<.01$. Participants in the treatment group had an average sleep adherence of 88.62% (*SD*=11.59) at six months, while participants in the control group had an adherence of 77.17% (*SD*=21.95).

*Effect of TLC treatment on depression risk*

The second study hypothesis was that participants who received the preventive TLC intervention would be less likely to experience the onset of clinically significant depression. Participants were evaluated for major and minor depressive episodes at two- and six-month follow-ups. Fisher's exact tests were conducted to determine whether the percentage of participants who experienced the onset of depression differed at the time of follow-up. At two months, the onset of both major and minor depression differed significantly by treatment group (see Table 5). In the control group, 17.8% (n=8) of participants met criteria for major depression, versus only 1.7% (n=1) in the treatment group ($p<.01$). In the control group, 57.8% (n=26)

Table 5

*Risk of depression in treatment vs. control groups at two- and six-month follow-ups*

| | Treatment | Control | Fisher's exact test *p* |
|---|---|---|---|
| Two-month follow-up | | | |
| Major depression | 1.7% (1) | 17.4% (8) | .005 |
| No major depression | 98.3% (59) | 82.2% (37) | |
| Minor depression | 28.3% (17) | 56.5% (26) | .005 |
| No minor depression | 72.7% (43) | 43.5% (20) | |
| Six-month follow-up | | | |
| Depression-spectrum disorder | 12.2% (5) | 33.3% (8) | .056 |
| No depression-spectrum disorder | 87.8% (36) | 66.7% (16) | |

*Note.* Major and minor depression were combined for the six-month follow-up because of small numbers in each cell.

met criteria for minor depression, versus only 28.3% (n=17) of the treatment group ($p<.01$).

Due to the relatively small number of participants available for the six-month analyses, those with either major or minor depression were combined into a single "depression spectrum" group for analytic purposes. At six months, there was a trend for the experience the onset of a depression-spectrum disorder significantly differing by treatment group. In the control group, 33.3% (n=8) of participants met criteria for either major or minor depression, versus only 12.2% (n=5) in the treatment group ($p=.056$).

Based on these significant findings, logistic regressions were conducted to test whether the TLC intervention decreased the risk of depression at the time of the follow-ups, even when controlling statistically for the effect of clinical status at the pre-intervention baseline (BDI-II scores, presence of current minor depression, and experience of past major depression). Treatment group significantly predicted the presence of depression at both two and six months (see Table 6). At two months, participants in the control group were 13.23 times more likely than participants in the control group to have experienced the onset of major depression ($p<.05$), and 3.15 times more likely to have experienced the onset of minor depression ($p<.01$). At six months, participants in the control group were 4.74 times more likely than participants in the treatment group to have experienced the onset of a depression-spectrum disorder ($p<.05$).

Table 6

*Comparison of depression risk in treatment and control groups, controlling for baseline depression variables*

| | β | *SE* β | Wald's $\chi^2$ | p | Exp(β) |
|---|---|---|---|---|---|
| *Two-month follow-up: Major depression* | | | | | |
| Treatment group | 2.58 | 1.12 | 5.31** | .02 | 13.23 |
| Baseline BDI-II | .07 | .05 | 1.99 | .16 | 1.07 |
| Baseline current minor depression | .62 | .96 | .41 | .52 | 1.86 |
| Baseline past major depression | 1.51 | 1.13 | 1.79 | .18 | 4.52 |
| *Two-month follow-up: Minor depression* | | | | | |
| Treatment group | 1.15 | .43 | 7.04** | .008 | 3.15 |
| Baseline BDI-II | .03 | .03 | 1.19 | .27 | 1.03 |
| Baseline current minor depression | .84 | .49 | 2.86 | .09 | 2.31 |
| Baseline past major depression | .06 | .49 | .02 | .90 | 1.06 |

Table 6, cont.

| | β | *SE* β | Wald's $\chi^2$ | p | Exp(β) |
|---|---|---|---|---|---|
| *Six-month follow-up: Depression spectrum* | | | | | |
| Treatment group | 1.56 | .72 | 4.70* | .03 | 4.74 |
| Baseline BDI-II | .10 | .05 | 3.8 | .051 | 1.11 |
| Baseline current minor depression | -.78 | .81 | .94 | .33 | .46 |
| Baseline past major depression | .14 | .78 | .03 | .86 | 1.15 |

*$p$<.05
**$p$<.01

In order to test for between-group differences in *severity* of depressive symptoms, mean BDI-II scores were evaluated for the treatment and control groups at two months (treatment $M$=9.55, $SD$=8.94 and control $M$=10.76, $SD$=8.59) and six months (treatment $M$=8.22, $SD$=7.30 and control $M$=7.98, $SD$=5.76). Unexpectedly, t-tests revealed no significant differences in BDI-II scores between treatment groups at either two months, $t(104) = -.70$, $p$=.48, or six months, $t(63) = .14$, $p$=.89.

Regarding whether baseline variables predicted depression onset, results revealed a trend for BDI-II scores at the initial screening significantly predicting a depression-spectrum disorder at the six-month follow-up ($p$=.051; see Table 6). None of the other baseline variables predicted either minor or major depression risk at either the two- or six-month follow-ups.

*Effect of TLC adherence on depression risk*

The third study hypothesis was that participants who adhered to TLC elements (regardless of their assigned treatment condition) would be less likely to experience an onset of depression. At two months, participants who did not meet criteria for major depression were observed to be more likely to adhere to both anti-rumination, $t(101) = 56.95$, $p$<.001, and exercise goals, $t(101) = .70$, $p$<.05, at the time of follow-up. There was also a trend for greater adherence to social support goals, $t(102) = .23$, $p$=.057. Participants who did not meet criteria for minor depression were more likely to be adhering to anti-rumination, $t(102) = 5.01$, $p$<.05, and sleep goals, $t(104) = 2.89$, $p$<.05, at the time of follow-up. There was also a trend for greater adherence to exercise, $t(102) = 5.07$, $p$=.056. No other adherence variables were significantly

related to presence of major or minor depression at either two or six months (see Table 7).

Associations between adherence variables and BDI-II scores at two- and six-month follow-ups were also evaluated. At two months, BDI-II scores were significantly negatively correlated with adherence to sleep, $r(106) = -.27$, $p < .01$, exercise, $r(104) = -.22$, $p < .05$, and anti-rumination, $r(104) = -.52$, $p < .05$. At six months, BDI-II scores were significantly negatively correlated with adherence to sleep, $r(65) = -.26$, $p < .05$, and anti-rumination, $r(63) = -.37$, $p < .01$ (see Table 8).

*Exploratory analyses of adherence*

*TLC adherence.* In addition to the primary items used to assess adherence to TLC elements, eleven other questions were asked of participants as secondary measures of adherence (see Table 9). Accordingly, 2x2 repeated measures ANOVAs were conducted to compare the treatment and control groups on initial adherence to the exploratory variables versus adherence at the two-month follow-up. There was a significant main effect of treatment group on the number of fish oil pills taken daily, $F(1,103) = 8.34$, $p < .01$. In addition, a significant time-by-treatment interaction effect was found for number of fish oil pills, $F(1,103) = 14.91$, $p < .001$. As expected, a follow-up *t*-test showed a significant difference between the treatment and control groups at the two-month follow-up in number of pills taken, $t(103) = 3.69$, $p < .001$, but not at the initial screening, $t(104) = -.211$, $p = .83$. Participants in the treatment group took an average of 1.66 pills per day ($SD = 2.38$) at two months, while participants in the control group took an average of .27 pills per day ($SD = .96$).

Table 7

*Comparison of mean TLC adherence in participants with and without minor and major depression at two- and six-month follow-ups*

| | Two-month follow-up Major depression | | | | | | |
|---|---|---|---|---|---|---|---|
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Sleep | 76.39% | 21.14 | 84.99% | 15.64 | -1.53 | 103 | .13 |
| Social support | 71.61% | 17.49 | 83.60% | 16.90 | -1.92 | 102 | .057 |
| Fish oil | 8.33% | 17.82 | 17.45% | 30.95 | -.82 | 102 | .41 |
| Sunlight exposure | 75.28% | 34.70 | 78.16% | 32.51 | -.25 | 100 | .80 |
| Exercise | 37.04% | 45.47 | 73.76% | 41.15 | -2.54* | 101 | .02 |
| Anti-rumination | 74.62% | 24.25 | 94.23% | 6.16 | -6.12** | 101 | .000 |

| | Two-month follow-up Minor depression | | | | | | |
|---|---|---|---|---|---|---|---|
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Sleep | 80.38% | 19.27 | 87.05% | 13.19 | -2.12* | 104 | .04 |
| Social support | 79.88% | 22.97 | 84.14% | 11.95 | -1.24 | 103 | .22 |
| Fish oil | 13.49% | 27.85 | 18.65% | 31.53 | -.86 | 103 | .39 |
| Sunlight exposure | 83.19% | 29.61 | 74.77% | 29.61 | 1.29 | 101 | .20 |
| Exercise | 60.98% | 45.89 | 77.25% | 39.18 | -1.93 | 102 | .056 |
| Anti-rumination | 89.56% | 11.60 | 94.77% | 8.48 | -2.64* | 102 | .01 |

Table 7, cont.

| | Six-month follow-up Major depression | | | | | | |
|---|---|---|---|---|---|---|---|
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Sleep | 81.25% | 6.25 | 89.01% | 12.28 | -1.08 | 63 | .28 |
| Social support | 89.49% | 5.97 | 83.60% | 16.90 | .51 | 63 | .62 |
| Fish oil | 5.56% | 9.62 | 19.17% | 34.32 | -.68 | 61 | .50 |
| Sunlight exposure | 75.00% | 25.00 | 74.62% | 33.64 | .02 | 62 | .99 |
| Exercise | 55.56% | 76.08 | 50.92% | 36.79 | - .93 | 63 | .36 |
| Anti-rumination | 90.44% | 6.08 | 92.43% | 14.72 | -.23 | 61 | .82 |

| | Six-month follow-up Minor depression | | | | | | |
|---|---|---|---|---|---|---|---|
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Sleep | 83.17% | 13.35 | 90.02% | 11.56 | -1.85 | 63 | .07 |
| Social support | 89.12% | 12.50 | 85.27% | 11.93 | 1.03 | 63 | .31 |
| Fish oil | 17.31% | 37.03 | 18.83% | 33.11 | -.14 | 61 | .89 |
| Sunlight exposure | 68.65% | 37.92 | 76.16% | 32.04 | -.73 | 62 | .47 |
| Exercise | 69.23% | 41.86 | 76.60% | 36.34 | -.64 | 63 | .53 |
| Anti-rumination | 89.51% | 6.97 | 93.07% | 15.74 | -.79 | 61 | .43 |

*$p$<.05
**$p$<.001

Table 8

*Correlations between TLC adherence and BDI-II scores at two- and six-month follow-ups*

| | Pearson correlation with BDI-II | p | N |
|---|---|---|---|
| Sleep adherence | | | |
| Two months | -.27** | .005 | 106 |
| Six months | -.26* | .04 | 65 |
| Social support adherence | | | |
| Two months | -.15 | .12 | 105 |
| Six months | -.10 | .46 | 65 |
| Fish oil adherence | | | |
| Two months | -.04 | .66 | 106 |
| Six months | -.08 | .50 | 66 |
| Sunlight exposure adherence | | | |
| Two months | .02 | .84 | 103 |
| Six months | -.16 | .22 | 64 |
| Exercise adherence | | | |
| Two months | -.22* | .03 | 104 |
| Six months | .05 | .63 | 65 |

Table 8, cont.

| | Pearson correlation with BDI-II | p | N |
|---|---|---|---|
| Anti-rumination adherence | | | |
| Two months | -.52** | .000 | 104 |
| Six months | -.37* | .003 | 63 |

*$p$<.05
**$p$<.01

Table 9

*Effect of treatment condition on exploratory TLC adherence variables at two- and six-month follow-ups*

| Two-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Hours per day spent in company of others | | | | |
| Treatment group | 6.75 | .14 | .71 | 102 |
| Time | 38.71 | 2.93 | .09 | |
| Time * Treatment group | 21.89 | 1.66 | .20 | |
| Number of social activities per week | | | | |
| Treatment group | 42.34 | 1.18 | .28 | 101 |
| Time | 22.90 | 1.11 | .29 | |
| Time * Treatment group | 16.48 | .80 | .37 | |
| Number of phone calls to people you care about per week | | | | |
| Treatment group | 92.20 | .40 | .53 | 102 |
| Time | 142.32 | 1.28 | .26 | |
| Time * Treatment group | .03 | .000 | .99 | |
| Social Support Measure | | | | |
| Treatment group | 1446.05 | 1.97 | .16 | 94 |
| Time | 1012.69 | 12.05** | .001 | |
| Time * Treatment group | 255.19 | 3.04 | .09 | |

Table 9, cont.

| | Two-month follow-up | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Minutes per day spent outside during daytime hours | | | | |
| Treatment group | 10316.72 | 2.16 | .15 | 102 |
| Time | 540.49 | .17 | .68 | |
| Time * Treatment group | 4599.10 | 1.43 | .24 | |
| Hours per week spent on weight training/strengthening exercises | | | | |
| Treatment group | .13 | .02 | .90 | 102 |
| Time | .05 | .01 | .91 | |
| Time * Treatment group | .30 | .08 | .78 | |
| Number of pleasurable, fun activities per week | | | | |
| Treatment group | 127.14 | 3.13 | .08 | 97 |
| Time | .98 | .05 | .83 | |
| Time * Treatment group | .28 | .01 | .91 | |
| Hours per week spent on pleasurable, fun activities | | | | |
| Treatment group | 33.66 | .25 | .62 | 102 |
| Time | 27.49 | .62 | .43 | |
| Time * Treatment group | 36.56 | .83 | .37 | |

Table 9, cont.

| Two-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Minutes needed to fall asleep | | | | |
| Treatment group | 177.85 | .16 | .69 | 103 |
| Time | 301.79 | .69 | .41 | |
| Time * Treatment group | 1385.53 | 3.16 | .08 | |
| Number of times waking up during the night | | | | |
| Treatment group | 3.58 | .53 | .47 | 103 |
| Time | 12.01 | 2.52 | .12 | |
| Time * Treatment group | 15.56 | 3.26 | .07 | |
| Number of fish oil pills taken per day | | | | |
| Treatment group | 23.24 | 8.34** | .005 | 103 |
| Time | 20.45 | 11.45** | .001 | |
| Time * Treatment group | 26.62 | 14.91** | .000 | |

Table 9, cont.

| Six-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Hours per day spent in company of others | | | | |
| Treatment group | 1.77 | .04 | .84 | 63 |
| Time | 1.39 | .13 | .73 | |
| Time * Treatment group | 1.24 | .11 | .74 | |
| Number of social activities per week | | | | |
| Treatment group | 19.78 | .43 | .52 | 61 |
| Time | 21.18 | .76 | .39 | |
| Time * Treatment group | 41.30 | 1.49 | .23 | |
| Number of phone calls to people you care about per week | | | | |
| Treatment group | 45.94 | .16 | .69 | 63 |
| Time | 262.09 | 2.03 | .16 | |
| Time * Treatment group | 109.78 | .85 | .36 | |
| Social Support Measure | | | | |
| Treatment group | 3235.97 | 6.16* | .02 | 57 |
| Time | 1680.83 | 12.01** | .001 | |
| Time *Treatment group | 33.58 | .24 | .63 | |

Table 9, cont.

| Six-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Minutes per day spent outside during daytime hours | | | | |
| Treatment group | 26098.57 | 3.93 | .052 | 63 |
| Time | 541.92 | .09 | .76 | |
| Time * Treatment group | 299.92 | .05 | .82 | |
| Hours per week spent on weight training/strengthening exercises | | | | |
| Treatment group | .01 | .002 | .96 | 63 |
| Time | 1.22 | .49 | .49 | |
| Time * Treatment group | .01 | .002 | .96 | |
| Number of pleasurable, fun activities per week | | | | |
| Treatment group | 43.90 | .67 | .42 | 59 |
| Time | 4.03 | .20 | .66 | |
| Time * Treatment group | .45 | .02 | .88 | |
| Hours per week spent on pleasurable, fun activities | | | | |
| Treatment group | 80.00 | .70 | .41 | 63 |
| Time | 124.64 | 1.31 | .26 | |
| Time * Treatment group | 245.24 | 2.58 | .11 | |

Table 9, cont.

| Six-month follow-up | | | | |
|---|---|---|---|---|
| Adherence variables | MS | F | p | df |
| Minutes needed to fall asleep | | | | |
| Treatment group | 1630.48 | 2.10 | .15 | 63 |
| Time | 19.54 | .12 | .73 | |
| Time * Treatment group | 355.86 | 2.25 | .14 | |
| Number of times waking up during the night | | | | |
| Treatment group | 3.09 | .34 | .57 | 62 |
| Time | 14.18 | 2.10 | .15 | |
| Time * Treatment group | 14.18 | 2.10 | .15 | |
| Number of fish oil pills taken per day | | | | |
| Treatment group | 14.54 | 4.96* | .03 | 61 |
| Time | 13.56 | 7.27** | .009 | |
| Time * Treatment group | 14.41 | 7.72** | .007 | |

*$p<.05$
**$p<.01$

At two months, there was a significant main effect of *time* on the Social Support Measure, a 20-item measure (possible scores from 0-140) designed to evaluate participants' amount of, and willingness to utilize, social support resources, $F(1,94) = 12.05$, $p<.01$. At the initial meeting, participants had an average Social Support Measure score of 99.48 ($SD$=20.35), whereas at the two-month follow-up, participants had an average score of 105.05 ($SD$=21.32). There was no significant main effect of *treatment group* on the Social Support Measure or any of the remaining exploratory variables, nor were any other significant time-by-treatment interaction effects observed.

A second set of 2x2 repeated measures ANOVAs was conducted comparing the treatment and control groups on initial adherence to the exploratory variables versus adherence at the six-month follow-up. There was a significant main effect of treatment group on the number of fish oil pills taken daily, $F(1,61) = 4.96$, $p<.05$. In addition, a significant interaction effect was found for number of fish oil pills, $F(1,61) = 7.72$, $p<.001$. As expected, a follow-up $t$-test showed a significant difference between the treatment and control groups at the six-month follow-up in number of pills taken, $t(61) = 3.11$, $p<.01$, but not at the initial screening, $t(64) = -.08$, $p=.94$. Participants in the treatment group took an average of 1.67 pills per day ($SD$=.66) at six months, while participants in the control group took an average of .27 pills per day ($SD$=1.23).

At six months, there was also a significant main effect of treatment group on the Social Support Measure, $F(1,57) = 6.16$, $p<.05$. Participants in the treatment

group had an average Social Support Measure score of 110.60 (*SD*=17.29) at six months, while participants in the control group had an average score of 101.27 (*SD*=13.27).There was no significant effect of treatment group on adherence to any of the remaining exploratory variables, nor were any other significant time-by-treatment interaction effects observed.

In addition to the primary exploratory variables described above, which were continuous in nature, eight categorical exploratory variables were examined for their relationship to treatment group, using Fisher's exact tests. At two months, the following items were significantly related to treatment group, in the direction of the treatment group having greater adherence: *Do you take an omega-3 (fish oil) supplement?* ($p<.001$), *Do you feel sleepy during the day?* ($p<.05$). At six months, the following items were significantly related to treatment group, in the direction of the treatment group having greater adherence: *Do you take an omega-3 (fish oil) supplement?* ($p<.001$), *Do you often wake up in the middle of the night?*, ($p<.05$).

*Depression risk.* Exploratory TLC adherence variables were also analyzed for their relationship to depression risk (see Table 10). At two months, participants who met criteria for major depression were more likely to report needing more time to fall asleep at night, $t(102) = 2.08$, $p<.05$, and engaging in fewer social activities per week, $t(101) = -2.14$, $p<.05$, at the time of follow-up. Participants who met criteria for minor depression at the two-month follow-up were more likely to report waking up more times during the night, $t(104) = 2.95$, $p<.01$, and to score lower on the Social Support

Table 10

*Comparison of exploratory TLC adherence variables in participants with and without minor and major depression at two- and six-month follow-ups*

| | Two-month follow-up | | | | | | |
|---|---|---|---|---|---|---|---|
| | Major depression | | | | | | |
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Hours per day spent in company of others | | | | | | | |
| | 5.06 | 4.32 | 7.42 | 5.44 | -1.27 | 103 | .21 |
| Number of social activities per week | | | | | | | |
| | 2.33 | 2.01 | 4.94 | 3.59 | -2.14* | 101 | .04 |
| Number of phone calls to people you care about per week | | | | | | | |
| | 6.67 | 7.65 | 10.66 | 14.08 | -.84 | 103 | .41 |
| Social Support Measure | | | | | | | |
| | 96.33 | 17.46 | 106.08 | 20.63 | -1.37 | 97 | .18 |
| Minutes per day spent outside during daytime hours | | | | | | | |
| | 68.89 | 53.08 | 73.72 | 61.06 | -.23 | 102 | .82 |
| Hours per week spent on weight training/strengthening exercises | | | | | | | |
| | 1.39 | 2.32 | 1.45 | 2.76 | -.07 | 101 | .95 |
| Number of pleasurable, fun activities per week | | | | | | | |
| | 6.33 | 7.24 | 6.97 | 5.13 | -.34 | 100 | .73 |

Table 10, cont.

| | Two-month follow-up Major depression | | | | | | |
|---|---|---|---|---|---|---|---|
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Hours per week spent on pleasurable, fun activities | | | | | | | |
| | 9.61 | 8.30 | 10.68 | 9.83 | -.32 | 102 | .75 |
| Minutes needed to fall asleep | | | | | | | |
| | 44.11 | 35.91 | 25.17 | 24.86 | 2.08* | 102 | .04 |
| Number of times waking up during the night | | | | | | | |
| | 1.78 | 1.28 | 1.24 | 1.32 | 1.16 | 103 | .25 |
| Number of fish oil pills taken per day | | | | | | | |
| | .50 | 1.07 | 1.12 | 2.08 | -.83 | 102 | .41 |

| | Two-month follow-up Minor depression | | | | | | |
|---|---|---|---|---|---|---|---|
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Hours per day spent in company of others | | | | | | | |
| | 7.28 | 6.39 | 7.19 | 4.57 | .83 | 104 | .93 |
| Number of social activities per week | | | | | | | |
| | 4.31 | 3.62 | 4.97 | 3.50 | -.93 | 102 | .36 |

Table 10, cont.

| | Two-month follow-up | | | | | | |
|---|---|---|---|---|---|---|---|
| | Minor depression | | | | | | |
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Number of phone calls to people you care about per week | | | | | | | |
| | 9.16 | 9.31 | 11.17 | 15.92 | -.74 | 104 | .46 |
| Social Support Measure | | | | | | | |
| | 99.71 | 24.19 | 108.86 | 16.47 | -2.25* | 98 | .03 |
| Minutes per day spent outside during daytime hours | | | | | | | |
| | 73.04 | 55.87 | 73.50 | 62.89 | -.04 | 103 | .97 |
| Hours per week spent on weight training/strengthening exercises | | | | | | | |
| | 1.49 | 2.63 | 1.46 | 2.79 | .05 | 102 | .96 |
| Number of pleasurable, fun activities per week | | | | | | | |
| | 6.67 | 6.36 | 7.21 | 4.55 | -.51 | 101 | .61 |
| Hours per week spent on pleasurable, fun activities | | | | | | | |
| | 9.41 | 7.05 | 11.34 | 11.08 | -1.01 | 103 | .32 |
| Minutes needed to fall asleep | | | | | | | |
| | 32.35 | 27.57 | 23.25 | 24.81 | 1.76 | 103 | .08 |
| Number of times waking up during the night | | | | | | | |
| | 1.73 | 1.19 | .99 | 1.32 | 2.95** | 104 | .004 |
| Number of fish oil pills taken per day | | | | | | | |
| | .85 | 1.78 | 1.21 | 2.16 | -.90 | 103 | .37 |

Table 10, cont.

| | Six-month follow-up Depression spectrum | | | | | | |
|---|---|---|---|---|---|---|---|
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Hours per day spent in company of others | | | | | | | |
| | 7.50 | 4.93 | 8.63 | 4.82 | .75 | 63 | .46 |
| Number of social activities per week | | | | | | | |
| | 5.08 | 1.98 | 4.82 | 3.67 | -.24 | 61 | .81 |
| Number of phone calls to people you care about per week | | | | | | | |
| | 6.42 | 5.77 | 9.73 | 9.93 | 1.15 | 62 | .26 |
| Social Support Measure | | | | | | | |
| | 5.06 | 4.32 | 7.42 | 5.44 | 2.24* | 60 | .03 |
| Minutes per day spent outside during daytime hours | | | | | | | |
| | 98.00 | 15.21 | 109.52 | 16.14 | .45 | 63 | .66 |
| Hours per week spent on weight training/strengthening exercises | | | | | | | |
| | .77 | .95 | 1.22 | 1.60 | .96 | 63 | .34 |
| Number of pleasurable, fun activities per week | | | | | | | |
| | 10.38 | 9.74 | 7.06 | 5.22 | -1.68 | 61 | .10 |
| Hours per week spent on pleasurable, fun activities | | | | | | | |
| | 18.42 | 17.89 | 12.08 | 7.92 | -1.94 | 63 | .057 |

Table 10, cont.

| | Six-month follow-up | | | | | | |
|---|---|---|---|---|---|---|---|
| | Depression spectrum | | | | | | |
| | Yes | | No | | | | |
| Adherence variables | Mean | SD | Mean | SD | t | df | p |
| Minutes needed to fall asleep | | | | | | | |
| | 34.85 | 24.19 | 23.97 | 18.49 | -1.78 | 63 | .08 |
| Number of times waking up during the night | | | | | | | |
| | 1.58 | .89 | 1.32 | 1.22 | -.70 | 62 | .49 |
| Number of fish oil pills taken per day | | | | | | | |
| | 1.04 | 2.22 | 1.16 | 2.07 | .87 | 61 | .85 |

*$p$<.05
**$p$<.01

Measure, $t(98) = -2.25$, $p<.05$. Due to the relatively small number of participants available for the six-month analyses, those with either major or minor depression were combined into a single "depression spectrum" group for analytic purposes. At six months, participants who did not meet criteria for a depression-spectrum disorder were more likely to score higher on the Social Support Measure, $t(60) = -2.24$, $p<.05$.

Associations between exploratory TLC adherence variables and BDI-II scores at two- and six-month follow-ups were also evaluated (see Table 11). At two months, BDI-II scores were significantly negatively correlated with hours per day spent in the company of others, $r(104) = -.21$, $p< .05$, and the Social Support Measure, $r(101) = -.29$, $p< .01$, and significantly positively correlated with minutes needed to fall asleep, $r(106) = -.20$, $p< .05$. At six months, BDI-II scores were significantly positively correlated with number of times waking up during the night, $r(64) = .27$, $p< .05$.

In addition to the primary exploratory variables, which were continuous in nature, eight categorical exploratory variables were examined for their relationship to depression risk, using Yates' corrected chi-square or Fisher's exact tests (see Table 12). At two months, the following items were significantly related to depression risk, in the direction of the non-depressed participants having greater adherence: *Is your sleep restful?*, $\chi^2(2) = 8.74$, $p<.05$, *Do you often wake up in the middle of the night?*, $\chi^2(2) = 12.00$, $p<.01$, *Do you exercise on a regular basis?*, $\chi^2(2) = 6.41$ $p<.05$, *Do you find yourself ruminating often?*, $\chi^2(2) = 8.75$, $p<.05$, *Is it easy for you to stop ruminating?*, $\chi^2(2) = 8.58$, $p<.01$. Due to the relatively small number of participants available for the six-month analyses, those with either major or minor depression

Table 11

*Correlations between exploratory TLC adherence variables and BDI-II scores at two- and six-month follow-ups*

| | Pearson correlation with BDI-II | *p* | N |
|---|---|---|---|
| Hours per day spent in company of others | | | |
| Two months | -.21* | .03 | 104 |
| Six months | -.08 | .55 | 65 |
| Number of social activities per week | | | |
| Two months | -.12 | .21 | 104 |
| Six months | -.14 | .29 | 63 |
| Number of phone calls to people you care about per week | | | |
| Two months | -.09 | .34 | 104 |
| Six months | .16 | .20 | 64 |
| Social Support Measure | | | |
| Two months | -.29** | .003 | 101 |
| Six months | -.20 | .12 | 62 |
| Minutes per day spent outside during daytime hours | | | |
| Two months | -.09 | .34 | 101 |
| Six months | .05 | .63 | 65 |
| Hours per week spent on weight training/strengthening exercises | | | |
| Two months | .03 | .75 | 105 |
| Six months | -.05 | .69 | 65 |

Table 11, cont.

| | Pearson correlation with BDI-II | *p* | N |
|---|---|---|---|
| Number of pleasurable, fun activities per week | | | |
| Two months | -.18 | .08 | 100 |
| Six months | .03 | .79 | 63 |
| Hours per week spent on pleasurable, fun activities | | | |
| Two months | -.17 | .08 | 104 |
| Six months | .04 | .74 | 65 |
| Minutes needed to fall asleep | | | |
| Two months | .20* | .04 | 106 |
| Six months | .12 | .35 | 65 |
| Number of times waking up during the night | | | |
| Two months | -.02 | .82 | 105 |
| Six months | .27* | .03 | 64 |
| Number of fish oil pills taken per day | | | |
| Two months | .03 | .77 | 106 |
| Six months | -.11 | .38 | 63 |

*$p<.05$
**$p<.01$

Table 12

*Comparison of exploratory TLC adherence variables in participants with no depression, minor depression, and major depression at two- and six-month follow-ups*

| | Two-month follow-up | | | | |
|---|---|---|---|---|---|
| Adherence variables | No depr. | Minor depr. | Major depr. | $\chi^2$ | p |
| Do you take an omega-3 (fish oil) supplement? | | | | | |
| Yes/Sometimes | 31.7% (19) | 28.6% (10) | 7.1% (2) | .02 | .99 |
| No | 68.3% (41) | 71.4% (41) | 92.9% (26) | | |
| Is your sleep restful? | | | | | |
| Yes/Sometimes | 96.61% (57) | 74.29% (26) | 87.5% (7) | 8.74* | .01 |
| No | 3.39% (2) | 25.71% (9) | 12.5% (1) | | |
| Do you feel sleepy during the day? | | | | | |
| Yes/Sometimes | 89.8% (53) | 97.1% (34) | 100.0% (9) | .96 | .62 |
| No | 10.2% (6) | 2.9% (1) | 0.0% (0) | | |
| Do you often wake up in the middle of the night? | | | | | |
| Yes/Sometimes | 45.0% (27) | 82.9% (29) | 75.0% (6) | 12.00** | .002 |
| No | 55.0% (33) | 17.1% (6) | 25.0% (2) | | |
| If so, are you able to fall asleep again quickly? | | | | | |
| Yes/Sometimes | 91.2% (31) | 85.2% (23) | 83.3% (5) | .19 | .91 |
| No | 8.8% (3) | 14.8% (4) | 16.7% (1) | | |

Table 12, cont.

| Two-month follow-up | | | | | |
|---|---|---|---|---|---|
| Adherence variables | No depr. | Minor depr. | Major depr. | $\chi^2$ | p |
| Do you exercise on a regular basis? | | | | | |
| Yes/Sometimes | 76.7% (46) | 57.1% (20) | 33.3% (3) | 6.41* | .04 |
| No | 23.3% (14) | 42.9% (15) | 66.7% (6) | | |
| Do you find yourself "ruminating" (thinking negatively) often? | | | | | |
| Yes/Sometimes | 49.2% (29) | 74.3% (26) | 100.0% (8) | 8.75* | .01 |
| No | 50.8% (30) | 25.7% (9) | 0.0% (0) | | |
| If so, is it easy for you to stop ruminating? | | | | | |
| Yes/Sometimes | 82.4% (28) | 63.0% (17) | 33.3% (3) | 8.58* | .04 |
| No | 17.6% (6) | 37.0% (10) | 66.7% (6) | | |

| Six-month follow-up | | | |
|---|---|---|---|
| Adherence variables | No depression | Depression spectrum | Fisher's exact test p |
| Do you take an omega-3 (fish oil) supplement? | | | |
| Yes/Sometimes | 30.8% (16) | 30.8% (4) | 1.00 |
| No | 69.2% (36) | 69.2% (9) | |
| Is your sleep restful? | | | |
| Yes/Sometimes | 88.5% (46) | 84.6% (11) | .66 |
| No | 11.5% (6) | 15.4% (2) | |

Table 12, cont.

| | Six-month follow-up | | |
|---|---|---|---|
| Adherence variables | No depression | Depression spectrum | Fisher's exact test p |
| Do you feel sleepy during the day? | | | |
| Yes/Sometimes | 90.4% (47) | 91.7% (11) | .73 |
| No | 9.6% (5) | 8.3% (1) | |
| Do you often wake up in the middle of the night? | | | |
| Yes/Sometimes | 57.7% (30) | 76.9% (10) | .34 |
| No | 42.3% (22) | 23.1% (3) | |
| If so, are you able to fall asleep again quickly? | | | |
| Yes/Sometimes | 97.0% (32) | 77.8% (7) | .11 |
| No | 30.0% (1) | 22.2% (2) | |
| Do you exercise on a regular basis? | | | |
| Yes/Sometimes | 73.1% (38) | 91.7% (11) | .49 |
| No | 26.9% (14) | 8.3% (2) | |
| Do you find yourself "ruminating" (thinking negatively) often? | | | |
| Yes/Sometimes | 51.9% (27) | 100.0% (12) | .002** |
| No | 48.1% (25) | 0.0% (0) | |
| If so, is it easy for you to stop ruminating? | | | |
| Yes/Sometimes | 84.8% (28) | 76.9% (10) | .67 |
| No | 15.2% (5) | 23.1% (3) | |

*$p<.05$
** $p<.01$

were combined into a single "depression spectrum" group for analytic purposes. At six months, the following item was significantly related to depression risk, in the direction of the non-depressed participants having greater adherence: *Do you find yourself ruminating often?*, $\chi^2$ (2) = 6.29, $p$<.01.

*Self-report measure of adherence.* Finally, treatment group participants were asked to provide a set of global ratings of how well they believed they adhered to each of the TLC elements suggested in the psychoeducational presentation (rating themselves from 1 to 5; 1 being no adherence, 5 being perfect adherence). These global adherence scores were analyzed to determine whether participants' self-reported adherence was correlated with risk for onset of depression. None of the adherence scores were significantly associated with onset of either major or minor depression, at either two or six months. As a more subtle measure of depressive symptoms, BDI-II scores at two- and six-month follow-ups were evaluated using correlations. None of the self-report global adherence scores were significantly correlated with BDI-II scores at either two or six months.

## Discussion

This study aimed to test the efficacy of a brief intervention for the prevention of depression among college students at high risk for depression by virtue of a prior history of the illness or elevated (sub-clinical) symptomatology. Treatment group participants attended a one-hour psychoeducational session based on an acute group-based intervention for depression, Therapeutic Lifestyle Change (TLC; Ilardi et al., 2009), that targets six modifiable lifestyle factors (omega-3 supplementation, social support, physical exercise, sunlight exposure, healthy sleep, and anti-rumination strategies) useful in the treatment and prevention of depression (Ilardi, 2009; Karwoski, 2006). As hypothesized, study participants who received the TLC-based intervention were significantly less likely than were control participants to report the experience of clinically significant depression at either the two-month or six-month follow-up assessments.

In logistic regression models that controlled statistically for the effect of depressive symptoms and clinical status at the pre-intervention baseline, participants who received the study intervention were found substantially less likely than study controls to report the experience of either a major or minor depressive episode. At the two-month follow-up, those in the intervention group had an estimated 13-fold reduction in the risk of major depression diagnosis, and a 3-fold reduction in the risk of minor depression. At the six-month follow-up, their estimated odds of experiencing a depressive episode (either major or minor) were 4.74 times lower than those of control group participants.

*Adherence to TLC elements*

What mediated the apparent reduction in depression vulnerability associated with the TLC intervention? In order to help address this question, post-intervention adherence to each TLC element was examined to determine the degree to which participants incorporated the recommended preventive lifestyle strategies. At both two- and six-month follow-ups, treatment group participants were found more likely than control group participants to report adherence to two of the six core TLC elements: taking fish oil supplements and obtaining adequate sleep. Further, at the six-month follow-up, treatment group participants had a higher mean score on the Social Support Measure, which reflects utilization of social support resources. It is notable that the study's brief, low-cost preventive intervention – lasting only one hour and presented in group format – was capable of inducing enduring behavioral changes in this manner.

Participant adherence to each TLC element was also examined vis-à-vis its potential to confer protection from depression risk. At the two-month follow-up, participants who adhered to the protocol's prescribed sleep, exercise, and anti-rumination goals (regardless of their assigned treatment condition) experienced a significant reduction in the risk of both major depression and minor depression. At six months, participants who adhered to sleep and anti-rumination goals were again less likely to report the experience of depression. Likewise, BDI-II scores were significantly negatively correlated with sleep and anti-rumination adherence at both two- and six-month follow-ups, and with exercise adherence at two months.

Exploratory analyses also revealed several more specific facets of TLC protocol adherence that were related to lower depression risk at both follow-ups. At two months, these facets included engaging in more social activities, spending more time in the company of others, scoring higher on the Social Support Measure, needing less time to fall asleep at night, and waking up fewer times during the night. At six months, lowered depression risk was related to scoring higher on the Social Support Measure. Similarly, BDI-II scores at two months were inversely correlated with spending time in the company of others, needing more time to fall asleep, and social support. At six months, BDI-II scores were significantly positively correlated only with wakeful episodes during the night.

It is important to note that the observed associations between adherence to TLC-based strategies and reduced depression risk could have several possible explanations. For example, participants who were more severely depressed over the study follow-up period may simply have been less motivated or less energized to engage in healthy behaviors. In fact, the absence of some such salubrious behaviors – e.g., social contact – may be regarded as a direct manifestation of depressive symptomatology. Similarly, it is possible that one or more unmeasured constructs – e.g., trait neuroticism, alcohol use, "willpower," etc. – could have influenced both adherence to the targeted lifestyle strategies and to depression risk, thereby yielding an artifactual association between the two. Nevertheless, it is certainly possible that higher adherence to TLC lifestyle factors directly reduced participants' risk of depression. This may be especially likely in the case of adherence to targeted sleep

goals (i.e., obtaining at least 8 hours of sleep per night): treatment group participants were not only more likely to obtain adequate sleep over the follow-up period, but sub-optimal sleep was also significantly related to depression risk at both follow-up time points. The study also provided evidence consistent with a protective effect of social support. Treatment group participants scored higher on the Social Support Measure at the six-month follow-up, and scores on this measure were inversely associated with depression vulnerability. Fish oil supplementation, however, was not found to decrease depression risk, despite the fact that participants who received the intervention clearly consumed more fish oil at both follow-ups.

*Advantages of the TLC Approach*

TLC-based prevention strategies are, at heart, simple behavioral interventions. Thus, the clinician's ability to effectively conduct the psychoeducational TLC group session is unlikely to be dependent on a high level of therapeutic skill or experience. All four group leaders in the present study were graduate students, three of whom had less than one year of psychotherapy experience, and one of whom had no previous treatment experience at all. Because the intervention is straightforward, with a clear outline and easy-to-follow handouts, it appears amenable to effective implementation even by clinicians of modest experience and skill levels.

It is also notable that the intervention was administered in a group format – a relatively low-cost mode of service delivery. Because the psychoeducational session was set up like a class, with little, if any, interaction between participants, it should prove similarly useful even with much larger groups of participants (e.g., as part of

freshman orientation at a college or university). And given the paucity of truly brief and efficacious preventive programs for individuals at risk for depression, in tandem with shrinking health-care budgets (D'Amico & Fromme, 2002; Ludman et al., 2000), the inherent cost effectiveness of the study's TLC-based intervention could render it a particularly attractive treatment option. Further, unlike some traditional strategies for depression prevention – such as cognitive therapy (CT) or mindfulness training – which are challenging to present effectively in a single one-hour class, TLC strategies lend themselves well to coverage within the constraints of a brief presentation format. Thus, the study's TLC-based intervention may represent a promising alternative to existing protocols for the prevention of depression.

*Questions and Concerns*

The most puzzling finding in the present investigation was the absence of a statistically significant difference in BDI-II scores between the treatment and control groups at either the two-month or six-month follow-ups, despite a significant between-group difference in the occurrence of major and minor depression according to SCID interviews. Ironically, it was anticipated that BDI scores would provide a *more* sensitive measure of between-group differences in depression symptoms over time than would SCID-based diagnoses, since the former are dimensional, and the latter categorical. Moreover, BDI-II scores are reasonably strongly correlated with the number of depressive symptoms endorsed on the SCID by college students (Sprinkle et al., 2002).

However, there are some respects in which the BDI may be regarded as inferior to the SCID – or to any structured diagnostic interview – in the assessment of clinical depression (Kendall et al., 1987). First, the BDI was developed as a measure of depressive symptom severity, not diagnostic status (Beck et al., 1988; O'Hara, Stuart, Gorman, & Wenzel, 2000), and the measure has only modest diagnostic specificity (Sloan et al., 2002; Kendall et al., 1987). In fact, it has been argued that the BDI is most strongly associated with self-reported *distress* and *malaise*, which are quite distinct from depression (Coyne, 1994; Pérez & López-Durán, 2005). Steer et al. (1998) found that general distress explains approximately 50% of the common variance between the Beck self-report anxiety and depression measures, whereas unique depression factors only explain approximately 20%. Both high- and low-end specificity are of concern, in that high scores can represent constructs other than depression, such as other psychological illness (Kendall et al., 1987; Sloan et al., 2002), while low scores represent underreporting of symptomatology for a significant subset of individuals (Joiner, Schmidt, & Metalsky, 1994). It has been recommended, therefore, that the BDI serve only as an initial screening tool, rather than a diagnostic instrument (Shean & Baldwin, 2008).

Additionally, several BDI items are not strongly or uniquely related to depression (Lovibond & Lovibond, 1995), and BDI scores have been found to be strongly correlated with constructs other than depression, such as anxiety, low self-esteem, situational stress, and general intensity of affect (Andrade, Gorenstein, Vieira Filho, Tung, & Artes, 2001; Jacobs & Boze, 1993; Osman, Downs, Barrios, Kopper,

Gutierrez, & Chiros, 1997; Rudd & Rajab, 1995; Sloan et al., 2002). In general, it is considered poor practice to use any self-report measure to diagnose depression, or any other psychological disorder (Rudd & Rajab, 1995; Sheeran & Zimmerman, 2002), even though it is common practice in psychological research (Sloan, 2002).

It is possible, therefore, that the study's TLC-based intervention yielded genuine benefit with respect to preventing episodes of major and minor depression, despite bringing about no significant reduction in observed scores on the BDI-II. While SCID interviews explicitly ask about the simultaneous experience of all relevant symptoms over a continuous two-week period during the most recent month, BDI scores tend to be based in part on one's *current* emotional state (Richter, Werner, Heerlein, Kraus, & Sauer, 1998), despite the measure's purported aim of measuring across the most recent two-week interval. Thus, it is feasible that treatment group participants successfully used TLC-based strategies to fend off the *continuous* experience of symptoms for two weeks or more, as required for a SCID diagnosis – despite being about as likely as control participants to experience temporary distress, as indicated by the BDI. Likewise, some treatment group participants may have allowed themselves to become temporarily distressed, as indicated by the BDI, after which they used TLC strategies to prevent themselves from lapsing into a full-blown depressive episode (as indicated by the SCID).

On the other hand, the aforementioned discrepancy in SCID-based diagnoses and BDI-II scores may have arisen from between-group differences in perceived experimental demand characteristics. It is likely that treatment group participants

were able to determine the study's aim of preventing the emergence of depression symptoms, given that the intervention centered on the utilization of antidepressant strategies. Thus, treatment group participants may have felt that they *should* report less depression at the follow-ups, a reporting bias which could have been more of a factor during face-to-face (SCID) versus paper-and-pencil (BDI) assessments. Control group participants – who received no active intervention – had no such direct exposure to the study's aims, and therefore faced much less in the way of a potential biasing influence of perceived social demand characteristics.

It is also worth noting that the study researchers, due to limited resources, were not always effectively blinded to each participant's assigned treatment group when performing follow-up SCID interviews (e.g., because the same researcher often led the participant's group-based intervention and then later conducted the SCID). Thus, it is possible that inadvertent experimenter bias in conducting or interpreting the SCID may have played some role in influencing the study interview-based outcomes.

Ongoing questions remain regarding the fact that the TLC intervention appeared to be moderately effective in changing some behaviors (fish oil intake, sleep habits, utilization of social support) but not others (sunlight exposure, anti-rumination, exercise). With such a brief intervention, it is encouraging that *any* behavioral changes took place in an at-risk population. Future research, however, might look at what is needed to elicit significant, enduring change across all six targeted domains within the context of a single hour-long intervention – if indeed

such a treatment goal is even attainable. Perhaps, for example, it would prove useful to provide participants with high-quality written take-home material – or internet-based resources – to provide further explication and review of the intervention's content.

It is also unclear why some targeted preventive strategies were found to be unrelated to depression risk, despite previously published evidence of their prophylactic benefit. Sunlight exposure and fish oil supplementation, for example, did not emerge as significant protective factors in *any* of the risk analyses performed – despite the fact that treatment group participants were significantly more adherent to fish oil than were control group participants. The lack of significant findings for sunlight exposure could conceivably be due to the fact that many of the participants in this study already get a sufficient amount of sunlight as college students on a largely outdoor campus. (At baseline, the mean adherence to sunlight exposure targets was 79.98%.) Therefore, the truncated range of reported sunlight adherence in this population may not have allowed enough variation to detect a significant impact on depression risk. Similarly, the range for fish oil supplementation was also truncated, albeit at the low end of the continuum. The mean fish oil adherence in the treatment group was only 25.69% at two months and 27.14% at six months. While this was significantly greater adherence than that observed in the control group, it represents only about 1.5 fish oil pills per day, whereas the most widely accepted therapeutic dose is approximately 6 pills per day (Ilardi et al., 2009) required to meet the 1,000 mg target EPA dosage (Nemets et al., 2002; Peet & Horrobin, 2002; etc.).

*Study Limitations*

This study followed participants for six months. However, it remains unknown how effectively patients might maintain behavioral changes over even longer periods of time, or to what extent the implementation of TLC strategies would provide preventive benefit over time among high-risk individuals if they were maintained.

Another key limitation of this study is the fact that the control condition included only an initial assessment – it did not include an intervention of any kind (e.g., an attentional control or a supportive group session). It is possible that this between-group procedural discrepancy introduced important participant differences in perceived demand characteristics of the study, inasmuch as treatment group participants likely ascertained the aim of the study based on the anti-depressant slant of the intervention, whereas control group participants had no such access to information about what the study was about.

It should also be noted that while TLC is conceptually distinct from existing psychotherapies for depression, there are also areas of overlap (Karwoski, 2006). For example, TLC's approach to stopping rumination – a behavior clearly related to depression risk, as seen in this study's results – is based to a large extent on behavioral activation, which is included in the CT protocol (Beck et al., 1979). However, the behavioral activation strategies of TLC address the *process* of negative or ruminative thinking, whereas CT maintains a primary focus on the content of the rumination (Ilardi, 2009; Karwoski, 2006). Similarly, TLC's approach to seeking out

social support is related to IPT's central focus on the immediate social context (Frank & Spanier, 1995). However, social support strategies of TLC focus principally on enhancing the frequency and quality of social interactions, rather than the more detailed and nuanced IPT approach to interpersonal dynamics. TLC is primarily a behavioral therapy that is unique for its explicit integration of multiple biological approaches to depression prevention.

*Future Directions*

This study represents an initial look at TLC as prevention for depression. Future research should focus on clarifying the efficacy of each TLC element, and disentangling the aforementioned cause-and-effect issues between adherence to TLC and the experience of depression symptomatology. Also, it would be useful to determine why the study intervention was capable of inducing at least some increased adherence to target goals regarding fish oil supplementation, healthy sleep, and utilization of social support, but not to TLC elements such as exercise, sunlight exposure, and anti-ruminative activity.

Perhaps the most exciting potential future direction is the utilization of a similar psychoeducational program with considerably larger at-risk populations. Preventive interventions are almost nonexistent in most communities (Muñoz, 1998), despite the fact that the prevention of depression provides much greater benefits to society and to individuals than does the treatment of one depressed individual at a time (Ingram et al., 2004). It is crucial, therefore, for clinicians to identify groups at the highest risk for depression and to intervene early (Le & Boyd, 2006). One such

target population, for example, would be individuals terminating psychotherapy or pharmacotherapy due to the experience of depression remission. Further, because college students, as a group, face a high level of depression vulnerability (Furr, Westefeld, McConnell, & Jenkins, 2001; Vredenburg, Flett, & Krames, 1993), and given the recommendation of the National Mental Health Association that colleges and universities would benefit from programs for prevention and early detection of depression (Field, Elliott, & Korn, 2006), a brief intervention such as TLC might be appropriate at a freshman orientation meeting or resident hall presentation.

It would also be quite useful to see a replication of the present study with a non-college participant sample. College students are in some ways an ideal sample for a depression prevention study, given that: (a) more than half of depressed individuals will experience their initial episode by age 25 (Sorenson, Rutter, & Aneshensel, 1991); (b) college students are almost by default under unusual and heightened stress; and (c) being in one's teens or twenties is regarded as a de facto risk factor for the onset of depression (Lewinsohn et al., 1988). However, Coyne (1996) points out potential problems with an excessive reliance on college students in the investigation of depressive illness. He argues that many people will have their initial episode after the typical college age, that college students are often distressed in the absence of clinical depression, and that the gender skew, high intelligence, high socioeconomic status, and underrepresentation of minorities on college campuses are not reflective of the general population.

In conclusion, TLC represents a set of specific lifestyle changes integrated into a novel intervention for depression, predicated on the idea that the higher rates of MDD today are a result of a mismatch between the modern environment and the environment for which humans are best adapted (Ilardi et al., 2009). Each of these lifestyle elements has been independently demonstrated to protect against the onset and/or reoccurrence of MDD, and TLC has already been successfully used as a treatment for acute depression (Ilardi et al., 2007; Karwoski, 2007). In this preliminary study of the use of TLC as prevention, healthy lifestyle factors were presented to participants in a one-hour group psychoeducational session. Participants in the treatment group experienced a significant reduction in the subsequent onset of depression in comparison with a non-treatment control group. They also reported increased adherence to fish oil supplementation and healthy sleep goals. Accordingly, TLC appears to have some promise as a low-cost, time-efficient alternative – or adjuvant – to existing programs for the prevention of depression.

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Appendix A: TLC Adherence Questionnaire

# LIFESTYLE QUESTIONNAIRE

Your name: _____
KU ID: _____
Date: _____

**<u>Instructions</u>: Please think back *over the past three months*. On average, how much have you been doing the following things?**

On average…

…how many hours of sleep have you been getting per night? _____

…how many hours a day have you spent doing homework? _____

…how many academic credits have you been enrolled in? _____

…how many minutes of bright sunlight have you been getting per day? _____

…how many hours have you spent doing cardio exercise per week? _____

…how many pleasurable, fun activities have you engaged in per week? _____

…how many social activities have you engaged in per week? _____

…how many non-academic books have you read per month? _____

…how many times have you been eating out at a restaurant per week? _____

…how many minutes have you spent ruminating (thinking negatively) per day? ____

...how many hours have you spent in the presence of friends or other people you care about per week? _____

**<u>Instructions</u>: Please circle the answer that best corresponds to your current practices.**

Do you take a daily multivitamin? Yes No Sometimes

If so, what is your daily dosage? _____

Do you take a Vitamin E supplement? Yes No Sometimes

If so, what is your daily dosage? _____

Do you take a melatonin supplement? Yes No Sometimes

If so, what is your daily dosage? _____

Do you take a Vitamin A supplement? Yes No Sometimes

If so, what is your daily dosage? _____

Do you take prescription allergy medicine? Yes No Sometimes

If so, what brand(s) do you take? _____

Do you take an omega-3 (fish oil) supplement? Yes No Sometimes

If so, how many pills do you take daily? _____

If so, what is your daily dosage of EPA? _____

If so, what is your daily dosage of DHA? _____

Do you take a birth control pill? Yes No Sometimes

If so, what brand do you take? _____

Is your sleep usually restful? Yes No Sometimes

Appendix B: Psychoeducational group session outline

## Session Outline

### Introduction

- The purpose of this group is to spend one hour discussing depression and new ways that have emerged to prevent depression.
- Rules
    - Your only job during this session is to listen attentively. You may also want to take some notes on the things we discuss.
    - You will not be required to share any personal information with the group, and you can remain completely silent if you like.
    - Everything in this group is completely confidential. I will not share any information shared by anyone with anybody else. I ask the same of each of you: do not share anything you hear with anyone outside of this group. This is important so that we can establish a comfortable environment in which we can discuss some important topics. Please nod now if you agree.
- We will be discussing how to prevent depression from occurring and we will describe specific ways you can prevent the onset of a depressive episode.
    - This will include lifestyle changes that research shows help people ward of depression.

### Risk factors for depression

- There are two important things that put people at risk for lapsing into depression: 1) previous depression; 2) current mild depression.
- Previous depression as a risk factor
    - Depression is a recurrent disorder
    - Most people with depression will experience more than one depressive episode, averaging 4-5 major episodes over a lifetime.
    - Each episode makes it increasingly difficult to recover.
    - Preventing a second episode is especially important. Those who can prevent a second episode after having their first episode have a much better chance of recovering for life.
- Mild depression as a risk factor
    - Severe depression is often preceded by mild depression.
    - Mild depression is painful and debilitating in and of itself.
    - Mild depression predicts the onset of a severe episode.
    - If you find ways to incorporate the lifestyle changes I describe to you today, you have a much better chance of staying away from depression.

**High rates of depression**

- Rates of depression are skyrocketing and becoming quite prevalent in our society. There is almost a 20% lifetime risk: one out of every five people in the U.S. will suffer from depression at some time in their lives.
    - This is true despite the fact that so many people use antidepressant medications.
- It hasn't always been like this. So why are people more depressed now? Why are we seeing an epidemic of depression? We believe that the world we live in has qualities that are toxic to many people's mental health and actually contribute to depression.
    - The modern human's body and mind was not designed for life in 2009, but for an earlier period in human history!
- Consider life in the 1800s. Most people lived on farms, doing lots of physical labor. They had to walk everywhere, spent lots of time outside, lived with many family members for their whole lives.
- Now, think back a few thousand years. People used to live in a world that looks nothing like today's! But this is the world the human body and the human mind are really designed for. The modern American environment may have characteristics that actually increase our risk for depression.
- The huge mismatch between early and modern environments explains the increase in risk for depression.

**Ancestral vs. modern life**

- [Can elicit suggestions from participants if they choose to contribute]
- Physical activity
    - Used to be necessary for survival – hunting, gathering, building shelter. Heavy lifting and fast running were daily activities.
    - Most modern jobs require us to be indoors; college requires you to be in class and inside studying.
    - We don't even need to walk much if we don't want to!
- Nutrition/Diet
- Sunshine
    - Most activities required people to be outdoors. All work had to be done outside.
    - Now, we miss out on a lot of sunlight. We can stay inside all day if we want.

- Social contact
    - Used to be that people were virtually never alone. You were constantly surrounded by people who are related to you.
    - Now, many people live alone or with only one other person.
    - Our typical hobbies include TV, video games, and other non-social activities.
    - People often move away from family and friends (especially college students).
- Regular sleep

    - All dark hours were spent sleeping; if anything, people got *too much* sleep.
    - The average American gets less than 7 hours of sleep
- So, it's obvious that our lifestyles have changed a whole lot. And it's tempting to think that all of our technological and social changes are good – and in many ways, they are! I would rather live in this century than 10,000 years ago! But a lot of the changes make it more likely for us to be depressed.
    - We have to work harder at it these days to stay away from depression.

Today's session

- The strategies we will teach you today focus on changing aspects of our daily routines and lifestyles.
    - Each of these lifestyle changes has been supported by research studies and have been shown to help reduce or eliminate depression.
- There are some modern ways of dealing with depression, such as anti-depressants and traditional talk therapy. These can also be valuable. But we will show you some strategies that have no side effects, often cost nothing, and are good for your overall health.
- If you are interested in doing this, you should have a really good chance of succeeding after this session, and some things you can start doing today!

**Omega-3**

- You probably know that our diets today are not healthy in general. One thing we are going to focus on today is to make sure you are getting the nutrients that are the most important in preventing depression.
- You may or may not have heard about omega-3 fatty acids. This is a substance that is essential in proper brain functioning.
- It comes from natural plants, and humans usually get it through eating animals that feed on these natural plants. People used to get it through eating fish and cattle that eat natural plants. But now, the meat and fish you get at the store are raised using artificial means and don't eat those natural plants anymore. So people don't get anywhere close to the right amount of omega-3 fatty acids in their diet.
- So why are omega-3 fatty acids so important? They have antidepressant properties just like Paxil, Zoloft, and Prozac. This is because they help the brain work more efficiently, and they enhance brain processing in areas that control some aspects of depression. In fact, omega-3s work on the same brain circuits that antidepressants do.
    - Specifically, omega-3s help circuits that function using serotonin and dopamine in the brain. These are two chemicals related to depression. They tend to be low in people who are depressed, so certain brain circuits don't work as well. The omega-3s help those circuits perform their function better.
- Research studies now show that people taking a specific dose of omega-3 had a great improvement and mood and a decrease in depression symptoms. They even beat out antidepressant drugs in some studies.

- Specifically, you will need 1000 mg of a certain version of omega-3: EPA. You can buy omega-3 pills at any pharmacy or drugstore like Walgreens, Target, or the Merc. You need to look on the back of the bottle and find the information on EPA. You should take however many pills are needed to add up to 1000 mg of EPA per day. This is usually about pills per day.
- It is good if you take a multivitamin. This will help your body digest the omega-3 so that your body will get the most out of it. Any normal multivitamin will do.

**Light Exposure**

- We spend very little time outside.
- Have you heard of people who get depressed or moody during the winter, and bounce back once spring comes around?
    - SAD tells us something about the relationship between light and mood, even for people who don't have it.
    - It's not the cold, or the snow. It's actually the level of light: daylight gets shorter, the sun is not as high in the sky, and we get less light.
    - Same kind of feeling when there is really bad weather and you just don't feel like getting out of bed. It's normal to feel down when there is poor light.
    - That's why bright light helps our mood.
- Light is also related to the circadian rhythm, which is a cycle that controls bodily functions like sleeping and waking. It resets our internal clocks so that we follow a 24-hour cycle.
    - When the circadian rhythm gets out of sync, we mess up our sleep cycle and sleep too much or too little. But we can readjust it if we expose ourselves to bright light.
    - This is true even for people whose mood is not affected by light.
- What is bright light?…light is measured in "lux". A sunny day is about 10,000 lux, compared to about 1,000 lux on a cloudy day. Normal indoor lighting is only 500 lux and does not provide an adequate amount of light.
    - Studies show that depressive mood diminishes if participants spend at least one hour per day at 3,000 lux. But 10,000 lux provides the most benefit.
    - It's very important to be consistent and to get light at the same time every day.
    - For people who stay up late and sleep in, morning light is better. For people who go to bed early and get up early, evening light is better.
- A good goal is to get at least 60 minutes of bright light every day.
- Getting light outdoors
    - Can combine with exercise
    - Must be on a moderately bright day, in the middle of the day
- Getting light from a light box
    - If you can't be outdoors, or it's winter and there are few sunny days, you may choose to get light from an artificial source.
    - A light box gives off a very strong light of 10,000 lux

    - Don't look directly at the box, put it to the side, within two feet
    - Can read, eat, or watch TV
- Remember, indoor light doesn't work! However, sitting near a window at home or while driving is better than nothing.

**Exercise**

- One of the big symptoms of depression is a lack of energy.
- Energy level is directly related to how much activity you want to engage in. Engaging in regular activity is like presetting your brain for a higher level of energy. Your brain keeps track of how much energy you use, and then gives you that amount of energy to use the next day.
    - If you don't exercise, you have no energy, which leaves you susceptible for developing depression.
- There is little demand for physical activity in our daily lives. In fact, people don't need to exercise anymore, ever, if they don't want to. So, purposefully engaging in a regular exercise program is the only way to get this antidepressant benefit.
- Research using exercise to treat depression finds that it works just as well, or even better, than antidepressant medications!
    - There is a clinic in California that treats depression with exercise alone, no medications at all, and the rate of recovery is very high.
- So, you probably already knew that exercise is good for you. Not exactly breaking news. But did you know it can prevent depression? And who really gets regular, vigorous exercise, at least 3 times per week? Not many of us.
- The optimal level for mental health is about 35 minutes of aerobic/cardio exercise, 3 times per week.
    - You may not exercise at all right now, which is okay.
    - This is a do-able goal that you can build up to over time.
- Aerobic exercise is anything that keeps your heart rate up to about 120-160 beats per minute. What are some options?
- [Demonstrate how to take heart rate.]

**Anti-rumination Strategies**

- Rumination is worrying or stressing out about something, dwelling on little things, mentally replaying things that didn't go the way you wanted, worrying about the future. This is thinking about the same negative thought over and over without being able to come up with a solution or move on. It's almost like a tape of negative statements playing in your mind.
- You might have something normal but disappointing happen, like getting a bad grade or rejection by a friend…or it might be something that hasn't even happened, like something in the future. But you tend to ruminate over what has gone wrong or what could go wrong.
    - This is not the helpful kind of thinking where you solve problems and make plans. It's just negative, pointless, painful thinking.

- Dwelling on these thoughts has a snowball effect, so that you start feeling more and more bad or guilty – maybe even guilty for spending too much time thinking!
- This style of thinking makes you much more likely to get depressed. It's like wearing dark-colored glasses. And the worse your rumination is, the more likely you are to get depressed and stay depressed.
- The first step is to notice when you are doing it! It's very easy to get caught in the habit of ruminating and not even be aware that you do it. It can be a way of life. So you need to step back and notice when you ruminate. Then you can take steps to stop it.
- How to stop rumination
    - Schedule fun/pleasurable activities. When you notice you are ruminating, think, "I am ruminating and I should immediately get involved in an activity." Pleasurable activities actually light up a part of the brain that counteracts depression (the left frontal cortex). Try to think of some activities right now that you could use. Try to think of at least 3 fun things you could do for yourself this week. Any ideas?
        - Don't feel guilty about doing fun and pleasant things for yourself. They can keep you from getting depressed.
    - Spend time with a friend. It is very difficult to ruminate while having a conversation.

**<u>Social Support</u>**

- One of the most unfortunate side effects of depression is social isolation. Social activities are fun and positive. But when you are depressed, you might not feel like doing much except going to class.
- When do people feel the most depressed? – often, at night, when they are alone.
- Being around other people makes us feel better, even when we are not feeling sociable! When we are depressed, sometimes we worry about being a burden to others, or we prioritize other things (like school) over socializing.
- When you don't have a social support network in place, you are *vulnerable* to getting depressed!
    - Being with other people gives us someone to talk to about our problems – but it also distracts us, and helps us get our mind off our problems.
    - We automatically feel better just being around other people, feeling loved and cared for by others, laughing and being social.
- People are designed to be social.
    - Just look at kids who are raised without social contact!
    - People used to spend virtually every moment with other people. They spent almost zero time alone. They used to live in villages with all their extended family near by. How many of you live in the same town as your parents?
    - Being alone during most of human history was a time to panic, because it was dangerous. You could literally die from being alone. So, depression and anxiety are natural reactions to being alone too much.
    - The idea that we "should" be independent and okay on our own is false.

- So how do you increase your social contacts?
    - Spend as much time with other people as possible – literally, all day.
        - Do it even when you don't feel like it.
    - Think about people that are important to you that are far away. Would it help to reconnect with them over the phone and talk for half an hour or so?
    - Who lives around here that you can spend more time with?
    - If you don't have anyone close to you here, think about ways to enhance your connectedness with people around you. Is there someone living in your hall that you could invite to coffee or a movie, or to study together?

**Sleep Hygiene**

- Lots of research has shown that when we don't get enough sleep, our mood goes down the next day…or that when we are really well-rested, our mood is better.
- 8 hour rule: on average, we really do need at least 8 hours of sleep.
    - The problem is, most of us don't get 8 hours. Our lifestyles have gotten very busy. Many of us stay up late watching TV or surfing the internet.
    - The average number of sleep Americans get has decreased from 9 hours in 1900 to less than 7 today!
        - There was no choice but to sleep, before electricity.
        - Our bodies are designed to get tired when it gets dark, but now we can distract from that with activities.
- How do you know if you're getting enough sleep? → If you are drowsy during the day, especially during boring lectures or other times you are understimulated.
    - This is actually sleep deprivation.
- Other parts of this program that improve sleep
    - Exercise (but be sure not to do it 4 hours before bedtime)
    - Bright light – stabilizes sleep-wake schedule
    - Omega-3s can help
- Get ready for bed.
    - Think about putting a two-year-old to bed. You can't just stop playing and get them straight to sleep. You set up the environment: put on PJs, wash up, turn off the lights, read a quiet story.
    - It's the same for adults. Create a ritual for yourself.
        - Turn down the thermostat. People sleep better when it's colder.
        - Turn off bright indoor lights about an hour before bed.
        - Read a book (either fun or school), take a warm bath, listen to soft music
        - Avoid screens (TV/internet)
- One of the biggest problems people have is getting impatient and upset if they can't fall asleep
    - It's like ruminating about not sleeping
    - An important thing to remember is to not lie down and try to sleep if you're not tired. And if you lie in bed for awhile and can't fall asleep, get up and do something quiet and relaxing until you're tired.
    - But try not to worry and worry about not sleeping.

- Don't do anything in bed except sleep (and maybe have sex)! No reading *in* bed, no phone calls, no TV. This way when you get into bed, your body knows it's time to sleep.
- Try to keep a regular sleep schedule
    - Go to bed at the same time every night and wake up at the same time every morning. No naps during the day.
    - This is really difficult to do in college – but it really makes a difference.
    - It's tough to get up on days that you don't have to. What are some ways to motivate yourself to get up?
- Caffeine: avoid coffee, soda, energy drinks, chocolate, etc. before bedtime
- Food: don't go to bed hungry, but don't go to bed really full; this makes it hard to sleep.
- Alcohol: it's a myth that alcohol helps you sleep. Sometimes it can help you *fall* asleep, but then it makes you restless and causes you to wake up. It also makes you dehydrated or have to pee frequently. Avoid alcohol within 4 hours of bedtime.
- Try to be really tired by the time you go to bed – no naps, exercise, etc.
- Consider wearing earplugs if your roommate or other noises wake you up.
- Sleep deprivation is probably the BIGGEST trigger for the recurrence of depression. Even if everything else is going okay, depression can begin if you are sleep deprived.

Appendix C: Supplementary handout 1

## Antidepressant Strategies

### Exercise

Three times a week, get 35 minutes of aerobic exercise. Aerobic exercise is anything that gets your heart rate elevated to about 120-160 beats per minute. Activities can include things like running, walking fast, biking, or playing basketball.

Anaerobic exercise (like yoga or weightlifting) is better than nothing, but the strongest antidepressant effects have been observed from aerobic exercise. Lots of people report that finding a regular exercise partner and routine helps them stay motivated.

### Omega-3 fatty acid (fish oil) supplements

You can buy these at a grocery store, drugstore, or health food store (like Walgreens, Target, the Merc, etc.). Look for a brand that will give you at least 1000 mg of EPA per day. You will have to take about six pills every day to get this amount, which has been shown in studies to be beneficial to people with depression. Don't forget to take a multivitamin, too.

You can take these even if you are on antidepressant medication; there are no known interactions with drugs. The only side effect people have reported is that they sometimes burp up a fishy taste after taking them. Solutions to this problem are to freeze the pills and take them right before a meal.

### Light Exposure

This strategy is most effective for people who notice that there is a seasonal component to their depression, but it can be helpful for everyone. We recommend that people get at least 60 minutes of bright light exposure per day. The best way to do this is to actually go outside in the sun (take off the sunglasses, but leave on the sunscreen!). This works best on sunny days.

Or, you can get light exposure from a special light box that emits the same amount of light (10,000 lux). You can try www.LightTherapyProducts.com to order a light box; a good one costs around $175.

You should try to get light exposure at the same time every day. And don't miss a day of light exposure if you can help it. This is something that will only work for you cumulatively if you are consistent!

### Anti-rumination Strategies

Rumination is the habit that many depressed people get into of dwelling on their negative thoughts. Rather than coming up with a solution to a problem and acting on it, people with depression often let their negative thoughts spiral out of control. It is important to recognize rumination for what it is and put a stop to it immediately. Rumination only

makes your mood worse and does nothing to actually help you. When you find yourself doing it, do one of these things: call a friend, take a walk outside, exercise, write down the negative thoughts in a journal, or do some other pleasant activity (like knitting, reading, or another hobby). Try to schedule at least one pleasant activity per day.

**Social Support**

You have probably noticed that as you get more depressed, you are less motivated to seek out others for socializing. You must try to spend *as much time as possible* with others, even when you don't feel like it. You should have at least two social contacts per week. This is a powerful way to distract yourself from rumination and get you the support you need.

**Sleep Hygiene**

You need to get at least 7 or 8 hours of sleep per night. One of the biggest risk factors for depression is sleep deprivation. Go to sleep and wake up at the same time every night. Prepare yourself for bed by having a "bedtime ritual." About an hour before bedtime, dim the lights, turn off the TV and computer, put on your PJs, and do a quiet activity like reading. Avoid caffeine and alcohol for several hours before you plan to go to bed.

*Remember: even though these strategies seem like just common sense, each of them has been shown to be effective in rigorous scientific studies. To prevent the onset of depression, try to maintain each of these lifestyle habits as best you can. If you feel yourself starting to become depressed, start implementing as many strategies as possible *right away*.

Appendix D: Supplementary handout 2

## What is my current lifestyle like?

I exercise _____ times per week for _____ minutes.

I take _____ mg of EPA omega-3 per day.

I get _____ minutes of bright sunlight per day.

I spend _____ minutes ruminating per day.

I spend _____ hours around other people per day.

On average, I get _____ hours of sleep per night.

## What are my goals for this semester?

To exercise _____ times per week for _____ minutes.

To take _____ mg of EPA omega-3 per day.

To get _____ minutes of bright sunlight per day.

To spend _____ minutes ruminating per day.

To spend _____ hours around other people per day.

On average, to get _____ hours of sleep per night.