COGNITIVE VULNERABILITY AND THE ACTUARIAL PREDICTION OF DEPRESSIVE COURSE

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ABSTRACT

A wealth of research indicates that depression is a serious global health issue, and that it is often characterized by a complicated and varied course. The ability to predict depressive course would be tremendously valuable for clinicians. However, the extant literature has not yet produced an accurate and efficient means by which to predict the course of depression. Research also indicates that cognitive variables – and cognitive vulnerability factors in particular – are related to the course of depression. In examining data provided by participants in the Temple-Wisconsin Cognitive Vulnerability to Depression Project (N = 345), the current study aimed to elucidate the relationship between cognitive vulnerability and depressive course using an actuarial statistical method. Results indicated that several cognitive measures predicted aspects of the onset and course of depression at rates significantly better than chance; foremost among these was the Cognitive Style Questionnaire (CSQ; Alloy et al., 2000). The CSQ was found to be the variable that best differentiated between participants who developed an episode of depression and those who did not. Furthermore, in comparison to participants who did not develop an episode of depression, the CSQ was found to differentiate between participants who recovered from a given depressive episode and those who did not, as well as between participants who experienced a single episode and those experiencing a recurrent course of the disorder across the prospective phase of the study. Conceptual and clinical implications of these results are discussed, as are directions for future research.

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DEDICATION

For Wanda, whose love, patience, and constant support have helped me through more than anything else.

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CHAPTER 1
INTRODUCTION

A large and ever-expanding body of literature strongly suggests that depression is a highly prevalent and debilitating disorder, affecting more North Americans than any other psychiatric condition (e.g., Murray & Lopez, 1997a; 1997b). Apart from having a direct negative impact on those attempting to cope with the disorder, depression is a serious burden on the health care system and ultimately on society as a whole (e.g., Hersh & Lazar, 1999; Wang et al., 2004; Wang & Kessler, 2005). Depression can take many forms with respect to symptomatology, course, and contributing environmental factors. Indeed, current nosological and diagnostic systems (e.g., DSM-IV-TR; American Psychiatric Association, 2000; ICD-10; World Health Organization, 1992) explicitly acknowledge this variability both across and within the various unipolar mood disorders, suggesting that what is often termed "depression" is indeed a complex and varied construct. Nevertheless, many psychological formulations agree that cognitive variables are extremely important in the onset, maintenance, and treatment of depression (e.g., Abramson et al., 1999; Beck, 1967; 1987; Clark, Beck, & Alford, 1999; Ingram, Miranda, & Segal, 1998; Teasdale, 1988). As such, cognitive variables are valuable not only in terms of understanding the onset and maintenance of depression, but also as targets in models of its treatment (e.g., Cognitive-Behavior Therapy of Depression; Beck, Rush, Shaw, & Emery, 1979) and prevention (e.g., Bieling & Grant, 2007).

Thus, the extant intervention and prevention literatures long have acknowledged the importance of cognitive variables and of cognitive vulnerability to depression. As such, there exists a tremendous wealth of research concerning the nature and predictive

power of these cognitive variables vis-à-vis their contribution to the onset and course of depression; in addition, theoretically-driven measures have been developed specifically to ascertain an individual's relative level of cognitive vulnerability to depression (e.g., Dysfunctional Attitudes Scale; DAS; Weissman & Beck, 1978; Cognitive Style Questionnaire; CSQ; Alloy et al., 2000). However, there exist few if any efficient and accurate measures with which to predictively assess the probability that a given individual will develop depression, that they will experience a particular symptomatic manifestation and/or particular long-term pattern or course of the disorder once it has manifested, and that they will ultimately achieve remission and/or recovery from depression. Such a measure, if it were to be developed, tested, and proven useful from a clinical standpoint, would not only have the potential to inform intervention efforts for individuals already experiencing depressive symptomatology, but also to allow for increasingly targeted prevention efforts – a task that has been tremendously difficult to accomplish for a variety of reasons (e.g., Bieling & Grant, 2007), discussed in greater detail below. As such, the availability of a reliable and accurate measure to predict the course of depression would ultimately help to ease the global burden of depression.

Thus, the present review will briefly summarize the available literature concerning depressive course and cognitive vulnerability to depression. This summary will then be integrated with a discussion of prevention and intervention. Here, a wealth of research demonstrates that the prevalence of depression rises sharply during the teenage years (e.g., Essau, 2004; Hankin et al., 1998; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993), making adolescence highly pertinent to prevention efforts. Furthermore, a review of actuarial prediction and its utility in clinical contexts will serve

to undergird the integration of these content areas. In presenting this synthesis, a case will be made for the potential utility of a statistically-driven measure that can be used to predict depressive course in order to bolster targeted intervention and prevention efforts.

Depressive Course and Cognitive Vulnerability

As noted above, depression is remarkably variable in terms of its course across individuals. For some, depression is a single, episodic event; however, this is a relatively rare phenomenon (e.g., Depue & Monroe, 1986; Judd & Akiskal, 2000). Many individuals experience a long-term course of the disorder, with some experiencing multiple episodes with recovery from symptoms in between, others experiencing only partial remission in between fully symptomatic episodes (i.e., these individuals do not achieve inter-episode recovery), and still others experiencing chronic unremitting symptoms over the course of many years (Depue & Monroe, 1986; Judd & Akiskal, 2000). Depressive course is generally defined in terms of the pattern of symptoms that constitute episodes, and the pattern and timing of depressive symptoms (or lack of symptoms) between episodes. The severity of episodes is also an important consideration when discussing course, both in terms of the number and magnitude of depressive symptoms, as well as the degree of functional impairment experienced by the depressed individual. All of these factors contribute to the diagnosis of depression assigned to a given individual's pattern of symptomatology.

With respect to defining depressive course vis-à-vis these symptom patterns, the extant literature provides general, empirically-supported guidelines (e.g., Frank et al., 1991; Rush et al., 2006). Once a given individual's depressive symptomatology is judged to be clinically significant for a sufficient period of time, a diagnosis of a depressive

episode is warranted. If, with time, the severity of this symptomatology decreases sufficiently so as to no longer be clinically significant, and remains at this level for a sufficient period of time (at least three weeks; Rush et al., 2006), the individual is said to have attained *remission*. If this remission lasts for an extended period of time (at least four months; Rush et al., 2006), the individual is said to have achieved *recovery* from the depressive episode. If, however, the individual's symptoms return to threshold level after remission has been achieved, but before the individual has reached the recovery stage, this is generally considered to be a continuation of the depressive episode, and is, thus, termed a *relapse* (Frank et al., 1991; Rush et al., 2006). If the individual, once recovered, experiences a return of clinically significant depressive symptoms for a sufficient period of time to warrant a diagnosis, this is conceptualized as an entirely new episode, and is, therefore, termed a *recurrence* (Frank et al., 1991; Rush et al., 2006).

However one chooses to examine depressive course, the available research suggests that cognitive variables – and cognitive vulnerability in particular – are in many cases related to the onset and course of the disorder. In one conceptualization, cognitive vulnerability to depression is manifested in a maladaptive cognitive style that directly influences a given individual's inferences for environmental events. In this view, high-risk individuals view negative events as arising from stable, internal and global causes (as contrasted with unstable, external, and specific causes), as likely to lead to negative consequences, and as indicating that they are flawed or unworthy. Due to these inferences, negative events are viewed as highly pertinent to the individual both in terms of immediate impact and in terms of future consequences, such as a high likelihood that such negative outcomes will persist or recur (e.g., Abramson, Metalsky, & Alloy, 1989;

Alloy et al., 1999). To the extent that one makes such depressogenic inferences, he or she is said to be cognitively high-risk. An alternate conceptualization holds that cognitive vulnerability to depression is represented in dysfunctional attitudes that are usually latent, but when activated are associated with negative affect (e.g., Segal, Gemar, & Williams, 1999; Segal et al., 2006; Taylor & Ingram, 1999). Such attitudes promote maladaptive and negative thinking about experiences in an individual's life, which, in turn, leads to negative beliefs about the self, the world, and the future (i.e., a negative cognitive triad; Beck, 1967). If an individual engages in this type of maladaptive thinking in the face of life stress, he or she is said to be relatively high-risk from a cognitive standpoint. Importantly, regardless of which conceptualization of cognitive vulnerability one chooses to adopt, high-risk individuals are likely to display evidence of their vulnerability even in the absence of overt signs or symptoms of depression; this cognitive diathesis, in turn, ultimately predisposes high-risk individuals to a higher likelihood of manifesting depressive symptomatology in the face of life stress relative to individuals at low cognitive risk for developing depression. A wealth of research (e.g., Alloy et al., 2000; 2006; Bos et al., 2005; Brown, Hammen, Craske, & Wickens, 1995; Dykman & Johll, 1998; Hankin, Abramson, Miller, & Haeffel, 2004; Joiner, Metalsky, Lew, & Klocek, 1999; Klocek, Oliver, & Ross, 1997; Kwon & Oei, 1992; Lewinsohn, Allen, Seeley, & Gotlib, 1999; Lewinsohn, Hoberman, & Rosenbaum, 1988; Lewinsohn, Joiner, & Rhode, 2001; Mongrain & Blackburn, 2005; Olinger, Kuiper, & Shaw, 1987; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999) provides strong support for the notion that cognitively high-risk individuals are more prone to developing depressive symptoms, an initial episode of depression, a relapse of symptoms following remission,

or a recurrence of the disorder following recovery relative to those who are cognitively low-risk.

Results from several studies suggest a link between cognitive vulnerability factors and depressive course vis-à-vis relapse-recurrence of depression (e.g., Burcusa & Iacono, 2007). For example, in an examination of a large sample of adolescents (n = 1709), Lewinsohn et al. (1999) demonstrated that cognitive vulnerability to depression (i.e., dysfunctional thinking as assessed with the DAS) was more highly correlated with dysphoric mood in individuals with a history of depression than in individuals without such a history. Furthermore, these authors found that, in conjunction with dysphoric mood, dysfunctional thinking was predictive of depressive recurrence, but not of first-onset episodes of depression. These results are consistent with additional research linking cognitive vulnerability to the recurrence of depression. In examining a sample of graduate students with at least one prior episode of depression (n = 97), Mongrain and Blackburn (2006) reported that cognitive vulnerability (here, a negative attributional style as assessed with a precursor to the CSQ) significantly predicted recurrences of depression across a 16-month follow-up period. In examining participants from the Temple-Wisconsin Cognitive Vulnerability to Depression Project (CVD Project; Alloy & Abramson, 1999; Alloy et al., 2000; 2006), the authors offered further evidence for a relationship between markers of cognitive vulnerability and the course of depression. Specifically, Alloy and colleagues (2006) followed a sample of initially non-depressed study participants (n = 347), and found that cognitive vulnerability (negative cognitive style as assessed with both the CSQ and DAS) is similarly predictive of both first onsets and of the subsequent recurrence of depression (in contrast to results of the Lewinsohn et

al., 1999 study). Finally, additional evidence of a different kind is seen in studies targeting manifestations of cognitive vulnerability through CBT-based prevention and intervention efforts, which may be an effective way to reduce the likelihood of relapse-recurrence in depression (e.g., Bockting et al., 2005; Vittengl, Clark, Dunn, & Jarrett, 2007).

Thus, results of a wealth of research support the existence of a relationship between cognitive vulnerability and recurrence of depression. With respect to relationships between cognitive vulnerability and other course variables, however, relatively less research has been done. Nevertheless, several studies in adult samples suggest that cognitive vulnerability and other cognitive risk factors are related to other aspects of the course of depression. For example, in an examination of symptom severity in depressed individuals (n = 20), Scott, Harrington, House, and Ferrier (1996) reported that higher initial scores on measures of dysfunctional attitudes (as assessed using the DAS) were associated with significantly greater symptom severity at three months following the onset of a depressive episode, as well as with non-recovery from an episode after six months. Riso and colleagues (2003) reported that in their sample (n = 93), relative to individuals without a chronic depressive course, those with chronic major depression scored significantly higher on a measure of dysfunctional attitudes (i.e., the DAS). Additionally, Dent and Teasdale (1988) reported that in their sample (n = 57), negative self-appraisal, as assessed by the tendency of participants to endorse negative trait words as self-descriptive, was associated with both the severity of depressive episodes and with a more persistent course of the disorder over a 5-month follow-up.

Additional evidence of a relationship between cognitive variables and depressive

course apart from relapse-recurrence comes from studies examining participants in the CVD Project. For example, using retrospective data in examining the diagnostic history of the study sample (n = 349), Alloy et al. (2000) found that CVD participants in the high cognitive risk group experienced a greater number of previous depressive episodes than did low-risk participants; furthermore, these prior episodes were marked by greater symptomatic severity. Of course, in using retrospective data, the directionality of the relationship between cognitive variables and depressive course cannot be elucidated. Iacoviello, Alloy, Abramson, Whitehouse, and Hogan (2006) found that across a prospective 2.5-year follow-up of a subsample of initially nondepressed CVD Project participants (n = 159), high, relative to low, cognitive risk was associated with a greater number of episodes of depression, more severe episodes, and a more chronic course of the disorder. Risk group differences were not found for duration of episodes. In another prospective study using a subsample of CVD Project participants, Grant, Alloy, Abramson, and Iacoviello (2009) found that among a subsample of CVD participants (n = 177) who experienced at least one episode of depression across the study follow-up period of 2.5 years, high-risk participants were less likely to recover from a given episode of depression than were low-risk participants. Furthermore, a higher probability of recovery from a given episode of depression during the prospective follow-up phase of the study was associated with more positive self-referent information processing (SRIP; a putative cognitive vulnerability marker assessed behaviorally in the CVD Project; Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997) at the outset of the study (Grant et al., 2009).

However, the literature concerning cognitive variables related to depressive

course is not without limitations. Certainly, methodological issues such as small sample size (e.g., Dent & Teadale, 1988; Scott et al., 1996) are cause for concern in some of these studies. Furthermore, participant selection procedures are another potential issue in terms of limiting the generalizability of results; an example of this is evident in the CVD Project, where only participants in the highest and lowest quartiles on both the DAS and CSQ were included in the study as high-risk and low-risk participants, respectively. In addition, the fact that different indices of cognitive vulnerability (i.e., DAS, CSQ, both, or neither) were used across studies is a potential limitation in terms of making direct comparisons. However, the most striking limitation in this literature is the relatively small number of studies directly examining the role of cognitive vulnerability in the course of depression. This is especially surprising given that many of the course variables discussed above, particularly the recurrent nature of depression, are key factors in the staggering overall societal cost of the disorder (e.g., Bockting et al., 2005; Murray & Lopez, 1997; Vittengl et al., 2007). Additional research concerning the relationship between cognitive vulnerability and depressive course is clearly warranted.

Importantly, in spite of these limitations, the available research strongly suggests a relationship between cognitive variables and depressive course. It is interesting to consider whether this relationship can also inform intervention and prevention research, and whether cognitive predictors of depressive course might allow for targeted prevention and intervention efforts. Indeed, previous research specifically highlights the link between cognitive variables and prevention and intervention of depression (e.g., Bieling & Grant, 2007; Garber, 2006; Merry, McDowell, Wild, Bir, & Cunliffe, 2004; Shochet et al., 2001; Shochet, Holland, & Whitefield, 1997; Spence, Sheffield, Donovan,

& Price, 1997). In addition to this general focus on cognitive variables, elucidation of cognitive predictors related to course specifiers such as remission, recovery, and non-recovery in depression will also be informative to intervention and prevention efforts. Identification of potentially psychologically modifiable variables related to an increased probability of achieving remission or recovery might speak to prime targets to address in the prevention and intervention efforts themselves. How these statistical connections can ultimately aid in the prevention and treatment of depression is outlined further in the next section.

Prevention, Intervention, and the Importance of Adolescence

That cognitive variables may play an important role in the prevention of and intervention for depression is by no means a novel idea. Many existing empirically-supported treatments for depression target manifestations of cognitive vulnerability to depression; for example, cognitive restructuring techniques are an essential component of CBT for depression (e.g., Beck et al., 1979). More recent formulations of CBT for depression, such as Mindfulness-Based Cognitive Therapy (MBCT; Segal, Williams, & Teasdale, 2002), also explicitly acknowledge and target cognitive factors, including a focus on fostering increased awareness of maladaptive thinking patterns and negative belief systems. Several prevention programs also have placed an emphasis on cognitive interventions as a means of prophylaxis against the future development of depressive symptomatology, especially those based on CBT approaches (e.g., Merry, McDowell, Wild, Bir, & Cunliffe, 2004; Shochet et al., 2001; Shochet, Holland, & Whitefield, 1997; Spence, Sheffield, Donovan, & Price, 1997; Stice, Rhode, Seeley, & Gau, 2008). However, treatment targets are only one component of any program of prevention; many

other considerations are of equal importance.

One such crucial question in the development of prevention efforts concerns timing. That is, when during the lifespan is it most desirable to implement such a program if one wishes to prevent the emergence of depressive symptomatology? Certainly, such a program must be implemented in advance of the development of most cases of depression in the general population, and importantly cannot be contraindicated with respect to implementation at that point in the lifespan. For these reasons, adolescence has been suggested as an extremely important juncture with respect to prevention efforts (e.g., Bieling & Grant, 2007; Garber, 2006, 2008; Horowitz & Garber, 2006; Merry, 2007). The prevalence of depression appears to rise dramatically in early- to mid-adolescence (Essau, 2004; Hankin et al., 1998; Lewinsohn, Clarke, Seeley, & Rhode, 1994; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993); as such, a prevention program implemented in early adolescence would have the benefit of reaching most – but admittedly not all – individuals before the onset of an initial clinically-significant depressive episode. There is also nothing to suggest that prevention efforts focusing on cognitive variables would be unsafe for adolescents; furthermore, such a program would in all likelihood not be contraindicated for the relatively small proportion of adolescents who have already experienced one or more episodes of depression (e.g., Bieling & Grant, 2007). Of further relevance to the issue of timing, the fact that the majority of individuals are in school at this point in life makes dissemination of any such program far more efficient and convenient for administrators, and makes the implementation of a standardized program more feasible than at other times in the lifespan (e.g., Shochet, Holland, & Whitefield, 1997; Spence, Sheffield & Donovan,

2003).

Additional factors that contribute to adolescence representing a sensible period during which to introduce prevention efforts are related to various aspects of development at this stage of life. For instance, especially pertinent to the present discussion is the rapid cognitive development taking place during adolescence (e.g., Steinberg, 2008); this, in turn, allows for more reflection on the self, the world, and the future (e.g., Keating, 2004), which may facilitate the expression of cognitive vulnerability to depression vis-à-vis dysfunctional attitudes and/or a maladaptive inferential style as it does in an adult population (Abramson et al., 1989; Alloy et al., 1999; Beck, 1967). Consistent with this position, relative to children, depressed adolescents have been shown to demonstrate more negative cognitions, as reflected by hopelessness, helplessness, etc. (Yorbik et al., 2004). However, this latter point can be contrasted with the suggestion that one's attributional style is often already established by adolescence (e.g., Kaslow, Adamson, & Collins, 2000; Nolen-Hoeksema, 1994); importantly, this suggestion has received some empirical support but has not been shown to apply universally. Even if the majority of individuals have an established cognitive style prior to adolescence, and although these styles are often relatively stable over time, they are by no means immutable (Bieling & Grant, 2007; Just, Abramson, & Alloy, 2001). Furthermore, adolescence is a highly stressful time in general for many individuals. In addition to stresses related to development, this period also heralds a variety of social, familial, academic, and other role transitions; as such, the opportunity to specifically address the role of stressful life events in the onset and maintenance of depression might be especially important at this stage (e.g., Brooks-Gunn, 1991; Ge, Lorenz, Conger, Elder, &

Simons, 1994). Thus, from a cognitive-developmental standpoint, adolescence is a natural choice for the implementation of prevention and/or early intervention efforts, especially with respect to those programs employing cognitive-behavioral components.

Notably, numerous prevention efforts based on CBT models have met with some success when implemented with adolescents in various age groups. For example, in a recent brief CBT-based intervention (Stice, Rhode, Seeley, & Gau, 2008), high-risk adolescent participants (n = 341; mean age = 15.6) with elevated depressive symptoms were randomized to a brief group CBT intervention targeting cognitive restructuring, to a group supportive-expressive intervention, to a bibliotherapy condition, or to an assessment-only control condition. The CBT and supportive-expressive interventions each consisted of six weekly 1-hour group meetings. Participants in the CBT group showed significantly greater reductions in depressive symptoms relative to participants in the supportive-expressive, bibliotherapy, and assessment-only control conditions following the interventions; however, these significant differences were only maintained relative to participants in the control group at 6-month follow-up (i.e., there were no significant between-groups differences for the interventions at follow-up).

The Penn Prevention Program (PPP; later the Penn Resiliency Program; PRP; e.g., Gillham, Hamilton, Freres, Patton, & Gallop, 2006; Jaycox, Reivich, Gillham, & Seligman, 1994) represents another CBT-based program targeting adolescents at high risk for developing depression based on the presence of subthreshold depressive symptoms. In a study conducted shortly after inception of the PRP (Jaycox et al., 1994), participants (n = 143; mean age = 11.4) were randomized to a cognitive intervention group examining the link between thoughts and feelings, a social problem-solving group,

a combined cognitive and social-problem solving group, a wait-list control group, or to a no-participation control group. Participants in the active interventions met for 12 weekly 90-minute group sessions. The authors chose to report results of their analyses in terms of treatment versus control groups rather than differentiating between the various participant groups in each of these categories, making differences between active intervention groups difficult to elucidate. However, relative to the controls, participants in the treatment groups displayed a significantly greater reduction in depressive symptoms from pre- to post-intervention; furthermore, these group differences were maintained at a 6-month follow-up, as well as at a later 2-year follow-up (Gillham, Reivich, Jaycox, & Seligman, 1995). Subsequent studies on the PRP utilizing different samples have largely echoed these initial successes; for example, authors have reported large preventive effects of the program relative to controls at 6-month follow-up assessments (Cardemil, Reivich, & Seligman, 2002).

Recently, in implementing the Penn Resiliency Program (PRP) in a primary care setting, Gillham and colleagues (2006) targeted early adolescents at risk for depression by virtue of elevated scores on measures of depressive symptomatology. Participants (n = 271) were 11- to 12-year-old HMO plan members, randomized to either the PRP condition or to a "usual care" condition. The PRP intervention was delivered in 12 weekly 90-minute group meetings by a therapist in the HMO setting. Results of this intervention were relatively small and inconsistent; for example, the intervention was associated with a significantly greater reduction in depressive symptoms (relative to the controls) from pre- to post-test for girls, but not for boys. Furthermore, in examining the sample as a whole, the authors noted that the PRP intervention was not effective in terms

of preventing depressive disorders. However, in examining participants with the greatest initial severity of depressive symptoms, a significant preventive effect was noted for future depressive, anxiety, and adjustment disorders (when examined in aggregate) relative to control participants across a 12-month follow-up.

Further examples of the implementation of targeted prevention efforts can be seen in studies conducted by Clarke and colleagues. In one such study (Clarke et al., 1995), investigators targeted high-risk participants exhibiting elevated (subthreshold) depressive symptomatology. One hundred fifty such participants (mean age = 15.3) were assigned to either a CBT intervention focusing on cognitive restructuring and the development of effective coping strategies, or to a "usual care" control condition in which participants were free to continue with community-based interventions or to seek additional care for depressive symptomatology. The study intervention was delivered over five weeks in the form of 45-minute group sessions held three times per week. Relative to control participants, those in the cognitive intervention demonstrated a significantly greater reduction in depressive symptomatology from the initial assessment point to the post-intervention follow-up; however, these group differences did not extend to a 12-month follow-up. Survival analyses indicated a distinct advantage for the intervention in terms of development of clinically-significant affective disorders over the year following the intervention, with significantly fewer participants in the intervention group developing a diagnosable depressive disorder in comparison to the controls.

In a subsequent study (Clarke et al., 2001), the authors targeted adolescents (n = 94, mean age = 14.6) at high risk for developing depression by virtue of having parents with affective disorders. Here, the at-risk adolescents also had elevated, but

subthreshold, depressive symptomatology. Participants were randomized to either a CBT intervention focused on cognitive restructuring techniques, or to a "usual care" condition similar to that employed in the Clarke and colleagues (1995) study. The study intervention was delivered in 15 1-hour group sessions. Participants in the CBT condition demonstrated a significantly greater decline in depressive symptoms from pre- to post-intervention in comparison to the controls; furthermore, these group differences persisted at 12-months post-intervention. As in the Clark et al. (1995) study, survival analyses suggested a preventive advantage for the study intervention across the first year of follow-up following the intervention, with significantly fewer participants in the CBT group manifesting diagnosable depression than those in the "usual care" group. This advantage also persisted at 18- and 24-month follow-up points, but to a greatly attenuated degree, leading the authors to conclude that the preventive effects of the CBT program diminished over time.

In terms of targeting a slightly older adolescent sample, Seligman, Schulman, and Tryon (2007) implemented a brief preventive program to at-risk college students (i.e., those with mild to moderate depressive symptomatology). Participants were 240 first-year undergraduates (mean age not reported) randomized to either a brief CBT-based program focused on many of the interventions contained in traditional CBT (e.g., the relationship between thoughts, feelings, and behaviors, identifying negative automatic thoughts, cognitive restructuring), or to an assessment-only control group. The CBT-based program was delivered over 8 weeks in weekly 2-hour group meetings. In addition, participants in the intervention condition had access to a "web-based supplement" following completion of the program that served to reinforce principles and

skills articulated during the intervention. Following the intervention and at 6-month follow-up, participants in the CBT group demonstrated significantly fewer depressive symptoms than did participants in the control condition. However, there were no significant between-groups differences in terms of the number of clinically-significant depressive episodes at either post-intervention time point.

In another recent study, Garber and colleagues (2009) targeted at-risk adolescents of depressed parents in a trial examining the effects of a CBT-based prevention program. Participants (n = 316, mean age = 14.8) had a past history of depression, current elevated (but subthreshold) depressive symptomatology, or both. Participants were randomized to either a CBT-based intervention focused on cognitive restructuring and other CBT staples, or to a "usual care" control condition. Participants in the CBT-based intervention group received 8 weekly 90-minute group sessions, followed by six monthly continuation sessions. Relative to participants in the control condition, those in the CBT group evidenced a significantly greater reduction in depressive symptoms from pre- to post-intervention; these gains were also maintained at the 6-month post-continuation follow-up. Furthermore, participants in the CBT condition experienced significantly fewer depressive episodes over the continuation phase than did control participants.

Interestingly, prevention programs also have been integrated into school curricula, demonstrating the potential feasibility of such a model. Horowitz, Garber, and Ciesla (2007) compared a CBT-based prevention program to an Interpersonal Psychotherapy (IPT)-based program. In this trial, participants were not selected based on risk for depression. Participants were 380 high school students (mean age = 14.4) enrolled in wellness classes as part of the regular school curriculum. Participants were randomized

to a CBT-based prevention program examining such issues as mood monitoring, cognitive restructuring, and challenging negative automatic thoughts; to an IPT-based condition examining communication and social skills development; or to a no-intervention control condition. The interventions both involved eight 90-minute weekly sessions, all of which were delivered in the high school setting by trained therapists in the context of weekly wellness classes. Following the interventions, participants in both the CBT and IPT conditions reported significantly lower levels of depressive symptoms than did the controls. The two intervention groups were not significantly different from each other in terms of reduction in depressive symptomatology at post-intervention, which speaks to the potential promise of prevention approaches other than those based strictly on CBT. However, any preventive effects of the interventions relative to the control condition were no longer evident at a 6-month follow-up, as there were no significant group differences on severity of depressive symptomatology at that time point.

Another school-based universal prevention program that has met with some success is the *Resourceful Adolescent Program* (RAP; Shochet, Holland, & Whitefield, 1997; Shochet, Dadds, Holland, Whitefield, Harnett, & Osgarby, 2001), a CBT-based prevention program that focuses on such issues as cognitive restructuring, problem-solving, and stress management. The RAP program consists of 11 weekly one-hour sessions. In one evaluative study, a group of 9^{th} grade Australian adolescents (n = 260; mean age = 13.5) were randomized to either the RAP program, the RAP program plus a three session program for parents, or to an assessment-only control condition. Sessions were conducted during regular class time and delivered by university staff trained by the researchers, and were part of the normal school curriculum. The two RAP conditions had

similar effects, and were both superior to the control condition. Upon completion of the program, adolescents with initial subclinical depressive symptomatology demonstrated the greatest benefit – as many as 75% of adolescents who had subclinical depression scores before the RAP program were nondepressed following the program, in comparison to approximately 35% of participants in the control condition (Shochet et al., 2001). Gains associated with involvement in the RAP program also extended to a 10-month follow-up. A subsequent study concerning RAP involved a group of 9th and 10th grade students in New Zealand (n = 392; mean age = 14.2) randomized either to RAP or to a placebo program. Both conditions were delivered by teachers in the New Zealand public school system. As in the first study, involvement in RAP was associated with significantly greater reductions in depression scores relative to the placebo condition; additionally, depression scores over an 18-month follow-up were significantly lower in the RAP group (Merry, McDowell, Wild, Bir, & Cunliffe, 2004).

Additionally, the *Problem Solving for Life Program* (Spence, Sheffield, Donovan, & Price, 1997; Spence, Sheffield, & Donovan, 2003) is another school-based, teacher-implemented CBT prevention program that has been evaluated in Australia. This program has at its focus the development of problem-solving skills, as well as an understanding of the connections between thoughts, feelings, and behaviors, and is delivered in eight 40-50 minute sessions. In a study evaluating this program, participants were a group of 8th grade adolescents (n = 1500; mean age = 12.9), and schools involved were randomized to deliver either the prevention program or an assessment-only control condition. The authors also classified participants as high- or low-risk based on their initial levels of depressive symptomatology. Outcomes were assessed immediately

following the intervention, as well as 12 months later. High-risk students demonstrated a significantly greater reduction in symptoms immediately following the intervention relative to the controls. However, these gains failed to extend to the 12-month follow-up, at which point there were no discernable group differences with respect to the percentage of high risk students who developed a clinically-significant episode of depression (Spence et al., 2003).

Thus, there is some cross-national evidence for the efficacy and effectiveness of CBT-based depression prevention programs implemented in adolescence, relative to both control conditions and to alternative prevention efforts. This is true with respect to both universal programs and to those targeted to high-risk youth, as evidenced by the results of many of the studies reviewed above. However, these prior studies are not without limitations. For example, it is tremendously difficult to compare the studies as there is little in the way of a common metric with which to do so. Although many of these programs utilize CBT-based prevention strategies, and although many also use the well-established indices of self-report depressive symptomatology, this is not universally true, and the measures used by investigators vary widely across studies. Furthermore, there is nothing to ensure that the programs delivered across studies are comparable in terms of content. Large variations across studies in intensity, frequency, and number of sessions, in the mean age of participants, in implementation setting, and in individuals implementing the programs make side-by-side comparison of these studies extremely difficult. These points speak in part to the sheer complexity of the task of prevention itself and to the notion that there is as yet no standard mode of program implementation and delivery; however, they also underscore potentially serious issues in the literature.

Reports of differential effects of a given program when implemented in different settings are also troublesome. As an example, in referencing the accumulated results of studies examining the PRP, Gillham and colleagues (2006) note that the strongest results (i.e., greatest preventive effects of the program) tend to occur when the PRP is delivered by its creators, by members of their research team, or by graduate students closely supervised by the research team. This is interesting, considering the fact that this long-standing prevention program has seen implementation efforts in a number of contexts. The authors point to this as one potential reason why the implementation of the PRP by therapists in a primary care setting (Gillham et al., 2006) did not fare better with respect to results. Furthermore, the authors note that much smaller effects have been obtained in studies where the PRP was implemented by schoolteachers who were nonetheless trained by the research team (Yu & Seligman, 2002). More troubling still are recent results from a study examining the RAP when attempting to train existing school personnel to implement it in a real-life school setting; that is, the delivery of the program did not result in any beneficial preventive effect for participants (Harnett & Dadds, 2004). Previous authors (e.g., Weisz, Donenberg, Han, & Weiss, 1995) suggested that interventions usually have stronger effects when implemented in research settings than in real-world or clinical settings, and several examples suggest that implementing depression prevention programs for adolescents in non-research settings may be similarly difficult (but see Merry et al., 2004; Spence et al., 2003, for evidence supporting the potential feasibility of doing so).

A somewhat related issue (vis-à-vis relevance to implementation in real-world settings) is that of attrition. Although this is an issue in nearly all research studies, it has

the potential to be especially serious in the present context. Any effective program-based model of prevention will by definition have its effect through participants' engagement with the materials and concepts presented in the program, as well as through their active attendance and participation. Stated another way, we will never know the true effects of a given program over time if adolescents do not participate. In some studies, attrition rates have been extremely high (e.g., exceeding one-third by the 24-month follow-up in Gillham et al., 2006). This suggests that brief prevention efforts might have some advantage (e.g., Stice et al., 2008), especially if they can be shown through empirical validation to have comparable effects to programs of longer duration. Furthermore, this issue underscores the need to make the material in the programs engaging and enjoyable – especially if the target population consists of adolescents.

From a methodological standpoint, selection of high-risk participants based on the presence of current subthreshold depressive symptomatology is also potentially problematic. Here, targeted CBT-based prevention trials may benefit from the strengths of previous prospective high-risk research in stratifying samples by risk based not on the presence of the very symptoms one is trying to ultimately prevent, but instead on variables hypothesized to temporally precede the onset of depressive symptoms. One example that has been largely successful in identifying participants at high risk for developing future depression is negative cognitive style, as assessed with measures such as the CSQ and/or DAS (e.g., Alloy et al., 2000, 2006). In assigning risk status in such a manner, one would ultimately have additional evidence consistent with the relevance of a CBT-based approach to prevention by virtue of identifying cognitive variables as risk markers for the onset of depressive symptoms. Researchers adopting this strategy would

also have the option of controlling for initial levels of depressive symptomatology in statistical analyses instead of relying on their presence for the purposes of participant selection; this would certainly speak to the applicability of a given program of prevention beyond adolescents with current depressive symptoms.

To expand further on this potential limitation, a related issue seen in many of the foregoing studies (e.g., Gillham et al., 2006; Horowitz et al., 2007; Jaycox et al., 1994; Spence et al., 2003) is that of differential outcome based on level of severity of initial depressive symptomatology. Specifically, many of these studies report that participants with greater initial symptom severity tend to respond more favorably to the intervention in question. This highlights questions concerning treatment versus prevention (e.g., Garber, 2008), in that amelioration of current subthreshold depressive symptomatology does not necessarily constitute prevention per se, but may instead represent treatment of subsyndromal depression. However, such a practice may be considered preventative with respect to conferring a diminished probability of developing a future episode of clinical significance (i.e., secondary prevention). This issue is complex for many reasons, chief among them the fuzzy distinction in many cases with respect to prevention and treatment. However, it is unclear at this juncture what, if any, implications this issue will have on the implementation of prevention programs in real-life contexts, as these programs would not necessarily be contraindicated for individuals with current depressive symptomatology, or even with previous episodes of depression (e.g., Bieling & Grant, 2007). In addition, research suggests that subthreshold depressive symptomatology in adolescents may in many cases be a robust predictor of future episodes of depression

(e.g., Seeley, Stice, & Rhode, 2009), serving to bolster the argument in favor of including individuals with elevated depressive symptomatology in prevention trials.

Finally, in examining the extant adolescent prevention literature in aggregate, effect sizes of even the most effective and efficacious existing prevention programs have been small to modest, and the effects of the programs generally have not endured over time (e.g., Garber, 2006, 2008; Horowitz & Garber, 2006). Nonetheless, in taking stock of the extant literature on depression prevention in aggregate, Barrera, Torres, and Muñoz (2007) suggest that although the field is not presently in the position to claim success in the prevention of depression, this goal may indeed be feasible in the not-too-distant future. This certainly echoes the perspective of Garber (2008), who notes that at present we have the "…beginnings of a good road map" (2008; p. 340) to the prevention of depression. It is also consistent with the essence of the foregoing review, which demonstrates some success in terms of preventing depressive symptoms and episodes in adolescents, but which also suggests that a great deal more work must be done in order to arrive at a truly effective real-world program to prevent depression.

As noted above, an additional important issue in the prevention literature that bears mention is whether prevention efforts should be universal or targeted/selective. The latter approach is more attractive on many levels, as only those individuals deemed at high risk would be selected to receive the prevention program. This would result in more efficient delivery of services, and also ultimately in substantial cost savings. Furthermore, as reported by Horowitz and Garber (2006), targeted prevention efforts, on average, are associated with significantly larger effect sizes than are universal programs. In many respects, the same logic can be extended to intervention efforts apart from

prevention, in which specific individuals can be targeted to receive treatment, or in which specific treatment targets are indicated. Of course, there are also potential pitfalls to consider in selective prevention and early intervention efforts, not the least of which may be stigmatization of those selected to participate (Essau, 2004), and potential implications of selecting far more adolescent females than males due to the emerging gender differences in depression during adolescence (e.g., Bieling & Grant, 2007; Garber, 2006). Nevertheless, if these potentially negative aspects could be addressed, targeted prevention potentially would be far more desirable than universal prevention efforts by virtue of its efficiency.

To that end, with respect to both prevention and intervention, the literature concerning markers of cognitive vulnerability to depression may be extremely valuable in allowing for the identification of high-risk individuals to target. However, such a predictive tool with the requisite psychometric properties (e.g., reliability and validity, as well as high sensitivity and specificity) does not yet exist. Furthermore, although present indices of cognitive vulnerability (e.g., CSQ; Alloy et al., 2000; DAS; Weissman & Beck, 1978) have shown promise with respect to their prospective predictive power, no single measure within the relatively young, extant theory-guided research concerning cognitive vulnerability to depression has proven capable of providing both accurate and efficient identification of high-risk individuals for the specific purpose of predicting future depressive onset and course in real-world settings, at least to the extent that something as important as a targeted prevention program might demand. As such, it may make practical sense at this juncture to use the wealth of cognitive research in aggregate, together with statistically-guided variable selection in order to create an actuarial measure

to predict depressive onset and course that might be used in targeted prevention efforts in adolescence and in intervention efforts across the lifespan.

Actuarial Prediction and Clinical Course

Actuarial, or statistical, prediction (e.g., Meehl, 1954; Sarbin, 1944) in essence is a means of coming to a prognostic decision by utilizing a strictly and explicitly mathematical process. Often contrasted with clinical judgment, actuarial prediction has been alternately lauded and vilified in comparison to predictive decisions made by trained clinicians, especially as concerns the prediction of behavior (e.g., Grove & Meehl, 1996). In this longstanding debate, some professionals have described prediction using statistical algorithms in favor of expert judgment in terms such as *mechanical*, *artificial*, *forced*, and *arbitrary* (Meehl, 1954). Proponents of the actuarial process would not necessarily disagree entirely with these descriptors, but would instead contrast them with terms such as *objective*, *reliable*, *rigorous*, and *empirical*; thus, many professionals would not see the actuarial prediction process as negative or lacking in relation to its ability to provide an accurate and efficient basis for prediction (Meehl, 1954).

It is important in presenting a brief overview of this debate to note that Meehl, while often seen as one of the field's strongest proponents of actuarial prediction, did not subjectively endorse these methods of prognostication in favor of clinical decision-making. He chose to define the problem of actuarial versus clinical prediction in relatively objective terms, and chose to allow the empirical evidence to speak for itself. In fact, as a practicing clinician himself, Meehl saw clinical experience as conferring expertise; as such, he saw clinical judgment as quite valuable in many cases (Meehl, 1954; 1992; Westen & Weinberger, 2005). Recent studies also echo this position,

indicating that clinical judgment is indeed valuable in a wide variety of settings, and suggesting that the accuracy of clinical judgment can improve with experience (e.g., Spengler et al., 2009).

Nevertheless, with the publication of *Clinical Versus Statistical Prediction* in 1954, Meehl initially took the position that the available empirical literature strongly favored actuarial as opposed to clinical methods in terms of providing a means by which to accurately, reliably, and efficiently predict behavior. Several studies published soon after Meehl's seminal work also echoed this position, speaking to either the superiority of actuarial methods (e.g., Wirt, 1956) or to their equality with clinical judgments (e.g., Grebstein, 1963). Importantly, in the intervening half-century, Meehl's position has stood the test of time and a great deal of subsequent research extremely well (e.g., Dawes, Faust, & Meehl, 2002; Grove, 2005; Grove & Lloyd, 2006; Grove & Meehl, 1996; Marchese, 1992). For instance, recent meta-analyses support the general superiority of actuarial methods over clinical prediction in a wide variety of cases. Grove, Zald, Lebow, Snitz, and Nelson (2000) examined 136 comparisons between the two methods of prediction, and found that actuarial methods of prediction were equal or superior to clinical judgment in all but eight (i.e., in all but 6% of comparisons) regardless of judgment task, types of judges, judges' levels of experience, or types of data available to the judges. Also of note was the finding that depending on the type of analysis employed by the authors, actuarial prediction outperformed clinical judgment by a wide margin in at least 33% of comparisons. Additionally, in the 6% of cases in which clinical prediction outperformed actuarial methods, the clinician judges had the advantage of more relevant data (Grove et al., 2000).

In another recent meta-analysis, Ægisdóttir and colleagues (2006) examined the predictive accuracy of actuarial methods over and above clinical methods as opposed to the relative accuracy of the two. The authors derived effect sizes by aggregating the results of 67 studies comparing clinical and actuarial prediction, and found a 13% increase in accuracy using actuarial methods. They noted that although this effect was relatively modest in size, it could have considerable implications if translated effectively into the domain of prediction concerning public health matters (Ægisdóttir et al., 2006).

Thus, several recent aggregate studies suggest that actuarial methods of prediction are equal or superior to clinical judgment in a wide variety of cases and settings (Grove et al., 2000), and that in many instances actuarial methods afford greater predictive accuracy over and above clinical judgment (Ægisdóttir et al., 2006). As such, it should be noted that actuarial methods of prediction may be effective and efficient in and of themselves (relative to clinical judgment) in many contexts, but it is also especially important to consider that they are far from infallible; along with clinical judgment, actuarial methods often achieve only modest results (Dawes et al., 2002). Even so, it is virtually indisputable given the sum of the available research that actuarial methods of prediction may serve as valuable supplements to expert human judgment in real-world settings (e.g., Dawes et al., 2002). Actuarial measures indeed have played an important role in several domains of predictive decision-making, including, but certainly not limited to, violent recidivism (e.g., Snowden, Gray, Taylor, & MacCulloch, 2007) and sexual reoffending (e.g., Kingston, Firestone, Wexler, & Bradford, 2008). In addition, a classic example of the utility of actuarial prediction in clinical settings as it pertains to psychological and personality constructs is the Minnesota Multiphasic Personality

Inventory (MMPI; Hathaway & McKinley, 1940) and subsequent MMPI for Adolescents (MMPI-A; Butcher et al., 1992) and MMPI-2 (Butcher, Dahlstrom, Graham, Tellegen, & Kraemmer, 1989). Yet another example is available in the domain of domestic assault. Hilton and colleagues (2004) used logistic regression on a large pool of varied potential predictors to construct a brief (13-item) measure for use in predicting wife assault recidivism, the Ontario Domestic Assault Risk Assessment (ODARA). The authors reported that the statistically-driven measure showed large effect sizes in predicting new assaults.

Given the clear potential for actuarial measures to add predictive accuracy relative to clinical judgment alone, it is somewhat surprising that no such measures are in wide use in the domain of prediction of clinical course of depression. Although there exist a wide variety of empirically-validated measures to assess cognitive vulnerability to depression and other cognitive variables related to the onset and maintenance of the disorder, and although these constructs can be used in some cases to predict certain aspects of depressive course (e.g., Alloy et al., 2000; 2006; Grant et al., 2009; Iacoviello et al., 2006; Iacoviello, Alloy, Abramson, Whitehouse, & Hogan, 2009), the development of measures themselves, and thus the inclusion of the full measures in predictive statistical analyses, is largely a theory-driven enterprise. That is, theoretical hypotheses guide the development of the often face-valid items included in these measures, and their administration as unitary measures – and subsequent analysis as such – by definition also serves to test the underlying hypotheses in their entirety. However, it is virtually certain that some of these measures, and certain items within these measures, provide more predictive power than others; similarly, certain items contained in these measures will

likely fare better statistically when other demographic, life, and personality characteristics are included in a given predictive model. Thus, there is a clear place for the development and validation of an efficient, actuarial approach to predicting the onset and course of depression. I propose utilizing the existing wealth of research that has borne established and validated indices of these aspects of depression – cognitive and otherwise – and using statistical methods in order to create a composite measure that can serve as an efficient and accurate predictive tool in the prevention and treatment of depression. This is discussed in greater detail below.

Toward an Integrated Prediction, Prevention and Intervention Strategy

As noted above, previous research suggests a variety of important relationships between cognitive variables and both the onset and course of depression. Thus, it is likely that if one examines the relative position of an individual on continua representing these variables, these statistical relationships may serve to allow for greater predictive accuracy with respect to who is at higher risk for developing depression, as well as who is at risk for developing a serious course of the disorder. Stated another way, one could conceivably administer a large battery of measures designed to assess cognitive vulnerability to a never-depressed individual. If the individual scored relatively highly on several of these measures, one might consider the individual to be at high risk for depression relative to individuals scoring relatively low. Alternatively, one might also use a similar battery of measures to assess an individual's risk of developing a long-term episodic (e.g., multiple relapses and/or recurrences) or chronic course of the disorder, or to assess the probability that the individual will recover from a given episode. One could thus implement this battery of measures as part of a prevention-targeting process in

adolescence as discussed above, or as part of a treatment-planning process at any stage of the lifespan for an individual already experiencing depressive symptomatology. This process, however, would be highly problematic for several reasons. First, it would be burdensome on the clinician or agency seeking to make prognostic decisions, as well as on the individual in question, to complete such a large battery of even relatively brief measures. Furthermore, this burden is compounded greatly when, for example, one considers the scope of targeting prevention efforts to high-risk adolescents – that is, it would be tremendously costly in terms of resources (financial and otherwise) to administer a large battery of measures to every adolescent in a given school in order to determine who is at the high end of the continuum of risk. In addition, exactly which measures to use, which measures should be more heavily weighted in determining risk given that they are not perfectly correlated and thus will disagree in a large number of cases, determining cut-off scores on various measures and their combinations, and other such considerations would make the implementation of such a predictive strategy prohibitive.

However, many of these issues could be addressed if one were to take data from existing methodologically rigorous longitudinal examinations of the relationships between cognitive variables and depression, and subject these data to statistical analysis in order to determine which individual variables best serve to differentiate between certain groups of participants for the specific purpose of maximizing predictive accuracy and efficiency in administration. For example, in utilizing data from the CVD Project (Alloy & Abramson, 1999; Alloy et al., 2000; 2006), one could operationalize participant groups in terms of the presence versus absence of depression over the course of the study,

in terms of recovery versus non-recovery from a given episode of depression, or in terms of the pattern of depressive course (e.g., number of relapses and/or recurrences of depression). Having grouped participants in such a manner, one could then perform a purely statistical, relatively theory-free procedure (e.g., stepwise discriminant function analysis) on measures of cognitive vulnerability used in the study such as the DAS and CSQ, and from related measures including the Self-Referent Information Processing Task Battery (SRIP Task Battery; Alloy et al., 1997), Self-Consciousness Scale (SCS; Fenigstein, Scheier, & Buss, 1975), Response Style Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991), and Sociotropy-Autonomy Scale (SAS; Beck, Epstein, Harrison, & Emery, 1983) in order to determine which combinations and weightings of variables serve to maximally differentiate between the participant groups. One could also include relevant demographic and diagnostic variables in these analyses. In this way, one would then have a mathematical algorithm by which to classify a given participant according to predefined criteria of interest (e.g., relatively high probability of experiencing depression, probability of recovering from a given episode of depression). One could then create a composite measure containing only the variables of interest, and use this measure as a tool to predict various aspects of depressive course in new samples. In so doing, one would ostensibly have a relatively brief and efficient measure to use for predictive purposes. Of course, such a measure would ultimately require empirical validation, perhaps through prospective tests on independent samples of participants in both research and real-world settings, to assess its ultimate predictive accuracy.

This new composite measure, if it subsequently proved viable in adding even modestly to the accuracy of prognostic decisions, would be a valuable tool in future

efforts to target prevention efforts to high-risk individuals, as well as to assess an individual's risk for experiencing a specific course of the disorder in order to inform intervention efforts. Inasmuch as one might choose to examine variables related to non-occurrence of depression, as well as to remission and recovery from depressive episodes in individuals who manifest the disorder, one would also have at their disposal statistically-selected variables potentially related to resiliency, or to psychologically-modifiable cognitive variables amenable to treatment. As such, these might represent variables to target in intervention efforts, and might also serve to guide the aforementioned targeted prevention efforts.

Thus, although positive results are by no means certain given the complexity of the task at hand, the initial construction of a statistically-guided measure to predict depressive onset and course is certainly a feasible undertaking, especially given the existing precedent for such measures within the clinical decision-making literature.

The Present Study

Given the foregoing, a statistically-driven measure to predict the onset and course of depression would be useful in a variety of clinical contexts, including both intervention and targeted prevention efforts. By including variables related to recovery from depressive episodes, specific cognitive resiliency factors might also be underscored – factors that might be addressed and emphasized in intervention and prevention efforts. Additionally, by also including factors related to non-recovery from depressive episodes, important and specific treatment and prevention targets might be highlighted.

However, it is important to note that such an efficient and accurate predictive measure that is also feasible to develop and validate is strictly hypothetical at present;

furthermore, initially the proposal to create such a measure does seem to oversimplify the nature of the onset, maintenance, and amelioration of depressive symptoms to a great degree. For instance, the foregoing fails to take into account many contextual factors related to depression, such as the influence of family, schools, neighborhoods, peers, etc. Such contextual factors also may be especially important in selecting adolescents at high risk for developing depression (Paunesku et al., 2008; Seeley et al., 2009). This measure also would not initially be capable of addressing the complex role of underlying neurobiology in depression, and even though the bulk of the extant research suggests that prevention efforts are best targeted to adolescents, it could not address the roles of neurobiological and social development during the adolescent years. Additionally, the complicated nature and role of life events in depression could not be given full justice at the outset of the development of this measure; this is especially true concerning the many transitions and stressful life events experienced by almost all adolescents. In brief, however, such a measure would not necessarily be concerned – at least initially – with taking the full spectrum of risk factors for depression into account; rather, it would be concerned solely with affording practicing clinicians and relevant agencies greater predictive accuracy in terms of who is at risk, while maintaining a high degree of simplicity of administration and interpretation. Once developed and validated, it may prove feasible to take additional factors into account to provide even more predictive power without necessarily sacrificing efficiency. In the interim, many of these potential shortcomings could be addressed by the delivery of supplemental measures to those identified by the measure as high risk for developing depression, or for developing a relatively chronic or complicated course of the disorder. In that way, the efficiency of the

screening process itself would not be compromised.

In addition, at times the present discussion admittedly blurs the distinction between purely actuarial measures and theory-driven ones. Although the proposed predictive tool would make use of previously-established measures, as well as cognitive vulnerability theories more broadly, it would do so for the sake of using statistical processes to achieve the highest degree of predictive accuracy possible. That is, although perhaps a "soft" actuarial tool by virtue of its reliance on previously articulated clinical hypotheses and theories, the proposed measure would nonetheless represent a novel combination of cognitive and other risk factors for depression weighted solely by statistical procedures, which is in essence an actuarial application.

Thus, the relationships between cognitive factors and many facets of depressive course, the importance of the adolescent years to cognitive vulnerability and to the onset and prevention of depression, and the importance of actuarial prediction to prevention and intervention all speak to the potential utility of a statistically-driven, predictive tool that can be used in intervention efforts across the lifespan, and in prevention efforts best targeted to the adolescent population. The present dearth of such predictive tools is highly problematic, in that lower ability to make accurate prognostic decisions can seriously hamper potentially useful efforts to prevent the onset of depression, and to predict who might respond best to what type of treatment. As depression is undeniably burdensome on society, heightened ability to make such decisions accurately would not only aid those who will eventually themselves deal with depression, but would also aid their families, the medical and mental health communities, and could ultimately aid in a small way in terms of the mollification of the effects of depression on society.

Thus, the primary aim of this study is to create a tool for the accurate and efficient prediction of depressive course. In order to achieve this aim, the present wealth of research concerning cognitive vulnerability to depression will be integrated with statistically-guided (actuarial) variable selection methods to ultimately produce a measure capable of differentiating individuals based on their prospective course of depression. A great deal of previous research suggests that cognitive variables – and cognitive vulnerability in particular – are related to the course of depression. This is true with respect to a higher rate of relapse and recurrence of depression (e.g., Alloy et al., 2006; Bockting et al., 2005; Burcusa & Iacono, 2007; Iacoviello et al., 2006; Lewinsohn et al., 1999; Mongrain & Blackburn, 2006; Vittengl et al., 2007), and to persistence of depressive symptoms, non-remission, and non-recovery from depressive episodes (e.g., Dent & Teasdale, 1988; Grant et al., 2009; Iacoviello et al., 2006; Riso et al., 2003; Scott, Harrington, House, & Ferrier, 1996). Thus, the use of cognitive and related variables as a starting point in the prediction of depressive course is clearly indicated. The ability to accurately and efficiently predict the course of depression would be invaluable for a number of reasons, chief among them the manner in which such predictive power, in highlighting potentially modifiable variables of interest, would ultimately inform targeted prevention and intervention efforts. Importantly, however, clinicians do not currently have at their disposal a tool with which to make such predictions

As an initial step toward achieving these aims, participants from the Temple-Wisconsin Cognitive Vulnerability to Depression Project (CVD Project; Alloy & Abramson, 1999; Alloy et al., 2000, 2006) were grouped based on numerous variables related to depressive course in order to allow for the generation of statistically-guided

predictive models of course using discriminant function analysis.

Hypotheses

The use of discriminant function analysis for the purposes of maximizing predictive accuracy is not conducive to the generation of a priori hypotheses regarding between-groups differences on specific variables. However, in general, I hypothesize that this method of data reduction will allow for the generation of a relatively brief and efficient means by which to classify participants in the CVD Project according to numerous variables relevant to the course of depression at a rate significantly higher than chance.

CHAPTER 2
METHODS

Participants

Participants in the present study took part in the larger CVD Project, a seminal prospective, behavioral high-risk study of cognitive and psychosocial factors in the development of depression in college freshmen (Alloy et al., 2000; 2006). Participants were recruited at two sites: Temple University (TU), and the University of Wisconsin – Madison (UW). In total, 5378 college freshmen were given Phase I screening measures including the Cognitive Style Questionnaire (CSQ; Alloy et al., 2000) and Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978). Those scoring in the highest quartiles on both the CSQ composite for negative events and the DAS were considered for future participation as members of the high-risk (HR) group; those scoring in the lowest quartiles on both measures were considered for participation as members of the low-risk (LR) group. In Phase II of participant selection, a random subset of eligible participants from Phase I were given a diagnostic interview (expanded Schedule for Affective Disorders and Schizophrenia – Lifetime; SADS-L; Endicott & Spitzer, 1978) by interviewers blind to participants' risk group status. Participants were excluded from the study if they met Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) or DSM-III-R (American Psychiatric Association, 1987) criteria for any current Axis I disorder or if they evidenced significant psychotic symptoms. Participants were also excluded if they reported a serious medical illness that might have prevented them from participating in a longitudinal research study. Participants were not excluded based on a prior history of depression, although they could not be currently depressed to be included

in the final study sample. Furthermore, if participants met criteria for a diagnosis of depression in the two months preceding study entry, they were disqualified. The final sample of the CVD Project consisted of 347 participants (172 HR and 175 LR; see Alloy et al., 2000 for a complete discussion of sample demographics, between-groups and between-site differences, and sample representativeness). In the present study, data from 345 of the CVD participants were used for analyses; two participants were excluded due to missing data concerning the course of their depressive symptomatology. These 345 participants were representative of the larger CVD sample. Refer to Table 1 for information pertaining to participants included in the present study.

Table 1. Demographic and cognitive style characteristics of the study sample across sites

	Temple University	University of Wisconsin
Sample size	168	177
Age (years)	19.40 (2.39)	18.46 (.92)
Gender (% female)	64.8%	67.6%
Risk Status (% HR)	47.8%	48.2%
CSQ Negative Score	3.92 (1.20)	3.93 (1.15)
DAS Total Score	199.70 (70.44)	200.21 (67.54)
Initial BDI score	12.78 (9.96)	10.94 (8.50)

Unless otherwise noted, statistics are presented as Mean (Standard Deviation). DAS = Dysfunctional Attitudes Scale. CSQ = Cognitive Style Questionnaire. Groups not significantly different on any of the variables included in the table (all ps > .05).

Participants included in the final sample of the CVD Project were administered a Time 1 assessment (including a variety of questionnaires, interviews, and tasks), and were assessed every 6 weeks thereafter for symptomatology since the last study assessment for 2.5 years. Specifically, they were given a Beck Depression Inventory (BDI; Beck & Steer, 1993; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) at each

assessment point. In addition, interviewers blind to risk status administered an expanded Schedule for Affective Disorders and Schizophrenia – Change Version (SADS-C; Spitzer & Endicott, 1978) at each assessment, allowing for an extremely careful and precise tracking of participants' mood and other symptomatology.

Measures

Schedule for Affective Disorders and Schizophrenia – Lifetime Version (SADS-L; Endicott & Spitzer, 1978). The SADS-L is a well-established and widely used semi-structured diagnostic interview. In administering the SADS-L, interviewers can assess for current and past psychological disorders according to RDC criteria. As stated above, the SADS-L was used in Phase II screening of participants. In the CVD Project, the SADS-L was expanded to also allow for DSM-III-R diagnoses to be made. In addition, the original interview structure was rearranged slightly to allow for symptoms related to a given disorder (both past and current) to be presented together, additional questions were included in the depression and anxiety sections, and a greater number of more precise probes were included to allow the interviewers the ability to better assess the persistence of depressed mood. Both the original and expanded versions of the SADS-L have been shown to possess strong psychometric properties and high inter-rater reliability (Alloy et al., 2000; Endicott & Spitzer, 1978). In the CVD Project, the SADS-L was administered by highly-trained interviewers, and inter-rater reliability analyses demonstrated strong interviewer agreement (kappa $\geq$ .90 for all diagnoses).

Schedule for Affective Disorders and Schizophrenia – Change Version (SADS-C; Spitzer & Endicott, 1978). An expanded version of the SADS-C was given to participants at each 6-week prospective assessment in order to carefully track the

symptomatology and course of depression and other Axis I disorders. As with the SADS-L, the SADS-C was expanded diagnostically – in this case to include DSM-IV diagnoses – and was administered by trained interviewers blind to cognitive risk status. Furthermore, to enable extremely precise and careful tracking of the course of depression, the expanded version of the SADS-C also included elements from the Longitudinal Interval Follow-Up Evaluation (LIFE; Shapiro & Keller, 1992). In the CVD Project, diagnostic inter-rater reliability using the SADS-C was high (kappa $\geq$ .90 for all project diagnoses; Alloy et al., 2006). The reliability of symptom dating using the SADS-C was also excellent (mean between-rater correlation = .97; Alloy et al., 2006).

Cognitive Style Questionnaire (CSQ; Alloy et al., 2000). The CSQ is designed to assess participants' inferential styles for 24 hypothetical events (12 positive, 12 negative). The CSQ is a modified version of the Attributional Style Questionnaire (ASQ; Peterson et al., 1982; Seligman, Abramson, Semmel, & von Baeyer, 1979). Domains of internality, stability, and globality of causal attributions are tapped by the CSQ; inferred consequences and self-worth implications are also assessed. In the CVD Project, a composite score for negative events was used in conjunction with the DAS to assign participants to one of the two cognitive risk groups. Previous research has demonstrated that the CSQ possesses excellent internal consistency (.88 and .86 for negative and positive event composites, respectively; Alloy et al., 2000) and one-year test-retest reliability (.80 for both negative and positive event composites; Alloy et al., 2000).

Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978). The DAS is a 40-item self-report measure designed to assess perfectionistic expectations of performance, pessimism, concerns about disapproval, and causal attributions. In the

CVD Project, an additional 24 items were added to the DAS in order to assess dysfunctional beliefs in achievement and interpersonal domains that are relevant to the study sample (i.e., college students). The original version of the DAS is a widely-used instrument, and has well-established psychometric properties (Hammen & Krantz, 1985; Weissman & Beck, 1978), and the expanded version is also psychometrically sound (e.g., internal consistency in the Phase I sample was .90; one year test-retest reliability in the final sample was .78; Alloy et al., 2000). Moreover, cognitive risk status based on both the CSQ and DAS predicted lifetime history of depression (Alloy et al., 2000) and prospective first onset and recurrence of depression (Alloy et al., 2006), speaking to the validity of these indices of cognitive risk.

Beck Depression Inventory (BDI; Beck & Steer, 1993; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI is a widely-used 21-item self-report measure that taps participants' subjective severity of depressive symptoms. The BDI was given at Time 1 and at every 6-week assessment during the prospective follow-up phase of the study. Apart from the degree and severity of baseline depressive symptomatology, the BDI allowed tracking of participants' subjective mood state and symptoms over time. The BDI has adequate and well-established psychometric properties in terms of both reliability and validity (e.g., internal consistency = .81 with non-psychiatric samples; Beck, Steer, & Garbin, 1988).

Self-Referent Information-Processing Task Battery (SRIP Task Battery; Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997). The SRIP Task Battery consisted of four tasks administered at Time 1 that made use of 40 adjectives that varied on two dimensions: Valence (positive or negative), and Content (depression-relevant or

depression irrelevant). Thus, adjectives each belong to one of four domains; namely positive depression-relevant (PDR; e.g., "competent"), negative depression-relevant (NDR; e.g., "failure"), positive depression-irrelevant (PDI; e.g., "polite"), and negative depression-irrelevant (NDI; e.g., "offensive"). The tasks included in the battery are designed to assess the degree to which participants demonstrate information-processing biases. These tasks include a computerized presentation of the adjectives in which participants classified each as descriptive or not descriptive of themselves; response times (RTs) for making these judgments were assessed ("Self-descriptiveness Judgments and Latency"), a written exercise in which participants provided specific evidence for words they felt are self-descriptive ("Behavioral Descriptions"), a ratings task in which participants gauged the probability that they would behave in a way related to several of the Valence x Content type adjectives in a hypothetical situation ("Behavioral Predictions"), and an incidental recall task based on the words presented in the computerized judgment task ("Free Recall"). In all, the four tasks yield five dependent measures (see Alloy et al., 1997 for additional details concerning development and validation of the SRIP Task Battery). In the present study, a negative composite score related to all five measures was used in all analyses. With respect to this score, negative values indicate relatively positive SRIP, whereas positive values indicate relatively negative SRIP.

Sociotropy-Autonomy Scales (SAS; Beck, Epstein, Harrison, & Emery, 1983). The SAS contains two 30-item self-report measures designed to assess the constructs of sociotropy (the tendency to value investment in positive interchange with others) and autonomy (the tendency to value investment in preserving and increasing mobility,

independence, and personal rights). Participants are presented with a series of hypothetical statements, and asked to rate how much each applies to them on a 5-point scale. The SAS has adequate psychometric properties (e.g., alpha coefficients of .90 and .80 and 4-6 week test-retest reliability of .75 and .69 for sociotropy and autonomy, respectively; Robins, 1985), and was administered at the Time 1 assessment. In the present sample, alpha coefficients were .85 and .86 for sociotropy and autonomy, respectively. The SAS includes three subscales each within the sociotropy and autonomy scales; in the present study, these subscale scores were used because previous research suggested different associations between various SAS subscales and indicators of depressive course (Iacoviello, Grant, Alloy, & Abramson, 2009).

Self-Consciousness Scale (SCS; Fenigstein, Scheier, & Buss, 1975). The SCS is a 23-item self-report measure designed to assess an individual's tendency to engage in self-focused attention in the private, public, and social domains. Previous research has demonstrated that the SCS has adequate psychometric properties (Carver & Glass, 1976; Fenigstein et al., 1975), and in the present sample, $\alpha = .79$. It was given to participants at the Time 1 assessment. In the present study, SCS subscale scores (i.e., Private, Public, and Social Anxiety subscales) were used.

Conceptualization of Depressive Course Variables

In the present study, several course variables were of interest, and each had to be operationalized. In defining depressive course with respect to the pattern of symptoms over time, the extant literature provides general, empirically-supported guidelines (e.g., Frank et al., 1991; Rush et al., 2006), and these were used wherever possible. With respect to participants in the midst of a clinically significant depressive episode, partial

remission was ascribed after at least three weeks of a return of their symptoms to levels of non-clinical significance (i.e., such that the symptoms no longer met diagnostic criteria), and full remission was ascribed following at least three weeks in the absence of significant depressive symptoms. Note that full remission in many cases may have followed a period of partial remission, but also may have occurred without preceding partial remission. If a participant's full remission lasted for at least four months, the participant was considered to have recovered from the depressive episode. If the individual's symptoms returned to diagnostic threshold level after remission was achieved, but before the individual reached the recovery stage, this was considered a continuation of the depressive episode, and was termed a relapse. If the individual, once recovered, experienced a return of clinically significant depressive symptoms for a sufficient period of time to warrant a diagnosis, this was conceptualized as an entirely new episode, or recurrence. In addition to the recurrent/episodic nature of depression, the present study also examined factors that are predictive of long-term unremitting episodes and of long-term presence of subthreshold depressive symptoms (i.e., long-term partial remission without recovery).

Participant Grouping

In the present study, several participant groups were created based on relevant depressive course variables, for the purposes of statistical analyses. Participants' mood state (i.e., never-depressed, non-depressed, depressed, in partial remission, in full remission, in recovery) was tracked on a day-by-day basis for the duration of their time in the prospective follow-up phase of the study in order to have an extremely precise index of level of symptomatology over time. Mood state was tracked using diagnostic and

symptom information from both the SADS-C and BDI, which were administered frequently throughout the prospective phase. Participants were first grouped based on occurrence (n = 177) versus non-occurrence (n = 168) of depression over the prospective phase of the study. Within the group experiencing depression over the course of the study, subgroups were created based on the occurrence of remission, recovery, relapse, and recurrence. The groups created for the purposes of the present study were as follows: No Prospective Episode (NE; n = 168), Single Episode With Recovery (SR; n = 54), Single Episode, Non-Recovery (SN; n = 18), Recurrent With Recovery (RR; n = 39), and Recurrent, non-recovery (RN; n = 66). Refer to Table 2 for demographics of these participant groups. More details concerning specific between-groups comparisons are presented below.

Data Analysis

Using the aforementioned participant groups, stepwise discriminant function analysis was employed to determine which variables maximally distinguished between the various groups of participants. Discriminant function analysis is a procedure by which group or category membership is predicted using one or more independent variables. The procedure is relatively theory-free and aims to elucidate the independent variable (or combination of independent variables) that best serve to differentiate between previously defined groups of participants. The procedure first produces a series of discriminant functions that serves to optimize discrimination between groups, based on statistically-guided combinations of predictor variables. By definition, any such functions are identified in descending order based on the proportion of unique between-groups variability they account for; that is, the first function will account for the greatest

Table 2. Characteristics of the study sample across participant groups defined by course

	NE	SR	SN	RR	RN
Sample size	168	54	18	39	66
Age (years)	19.14 (2.15)	18.94 (1.55)	19.18 (2.65)	18.59 (1.18)	18.39 (.88)
Gender (% female)	60.7%	64.7%	77.8%	64.1%	69.7%
Risk Status (%HR)[a]	35.7%	47.1%	50.0%	53.8%	68.2%
Days followed	772.02 (337.29)	822.81 (86.94)	782.22 (153.80)	826.62 (167.59)	792.05 (110.81)

NE = No Episode Group; SR = Single Episode Recovery Group; SN = Single Episode Non-Recovery Group; RR = Recurrent Recovery Group; RN = Recurrent Non-Recovery Group. Unless otherwise noted, statistics are presented as Mean (Standard Deviation).
[a]Groups different at $p < .001$.

proportion of variability, the second function will account for the second most, and so on. Additionally, each of these discriminant functions is independent (orthogonal) relative to the others; that is, they account for unique between-groups variability.

Assuming that at least one significant discriminant function is identified that separates the groups, the procedure then assigns cases (participants) to one of the predefined groups solely according to these functions, to determine the degree to which the function(s) correctly classify cases. Importantly, this classification can be completed using several different sets of specifications. Probability of assigning a case to a given group can be computed using the assumption that all group sizes are equal (a more conservative classification scheme), or utilizing the sizes of the predetermined groups (in which a group with a greater number of participants would have a higher probability of having a case assigned to it, and a group with fewer participants would have a lower probability). In the present analyses, classification was undertaken using the assumption of equal group sizes even though the groups were decidedly unequal in size, because this is the more conservative method of classification. Further, one may specify that

classification be conducted using discriminant functions computed using all participant data, or else by means of a "leave-one-out" classification method. In the latter method, when a given participant is being classified, the discriminant function(s) used for classification are re-computed using data from all other participants, but disregarding the data from the participant in question. Thus, group classification accomplished using this framework (as was done in the present study) has a cross-validation procedure included, minimizing the likelihood that participants are assigned to a given group because their own data influenced the discriminant functions.

Discriminant function analysis relies on several statistical assumptions (Tabachnick & Fidell, 2007), including underlying normality of the data, homoscedasticity, the absence of outliers in the data set, and non-multicollinearity. Unequal group sizes are acceptable. However, it should be noted that discriminant function analysis is a relatively robust test, even if one or more of these assumptions is violated (e.g., deviations from the normal distribution are acceptable if they are due to skewness and/or kurtosis rather than to outliers; independent variables may be significantly correlated if they are not functions of each other and tolerance values do not approach zero in the analyses).

In the present study, variables entered initially were participant scores on the CSQ, DAS, initial BDI, SAS subscales, SCS subscales, and SRIP, as well as days followed in the study. Analyses then determined which of these variables, and in what combinations, best discriminated between never-depressed participants and those who experienced a prospective episode of depression. Furthermore, similar analyses employing the groups who experienced depressive episodes yielded variables that best

distinguished participants who remitted and/or recovered from those who did not, those who experienced relapse and/or recurrence from those who did not, and those who experienced chronic, unremitting symptomatology from those who did not.

Power Analyses

The characteristics of the present study afforded adequate power to detect small-to-medium effects of $f^2 = .10$. The sample size needed to detect a small-to-medium effect of $f^2 = .10$ with power of .80 using the specified model (alpha = .05; conservatively taking into account the potential need to compare up to 6 groups using 20 variables) is 96. In the present analyses, based on the most conservative comparisons (i.e., those between four participant groups using 15 variables and a sample size of 177), the observed power to detect small-to-medium effects was .99.

CHAPTER 3
RESULTS

Preliminary Analyses

Potential predictor variables included in the analyses were CSQ negative composite score, DAS total score, initial BDI score, SRIP negative composite score, SAS subscales (three each for sociotropy and autonomy), SCS subscales (Public, Private, and Social Anxiety), and number of days followed in the study (although this variable was not significantly different between groups, it was included in the analyses to control for any between-groups variability accounted for by this variable). Table 3 provides the means and standard deviations of these variables for the five groups; Table 4 displays the between-groups correlation matrix for these variables.

Assumptions for the use of discriminant function analysis were met. Deviations from normal distributions were observed for several variables (i.e., Initial BDI score, DAS total score, and SCS Public subscale); however, although multiple data points occurred toward the tails of the distributions for many of the variables, no significant outliers were detected. Thus, according to preliminary analyses, these deviations from normality were attributable to skewness and/or kurtosis of the group distributions rather than to outliers; as such, this is acceptable for use in discriminant function analysis (Tabachnick & Fidell, 2007). Furthermore, Initial BDI score was the only variable for which distributions differed significantly from normality for all participant groups due to its positive skew. Heteroscedasticity was generally not an issue, as Levene's statistic was nonsignificant (all ps > .05) for all variables, although the value of Levene's statistic for Initial BDI score approached significance ($p = .056$). Finally, although sizeable and

Table 3. Means and standard deviations of predictor variables included in the analyses

	NE	SR	SN	RR	RN
Sample size	168	54	18	39	66
CSQ Neg. Score[a]	3.59 (1.03)	3.83 (1.14)	4.09 (1.32)	4.19 (1.16)	4.66 (.99)
DAS Total Score[a]	181.36 (61.07)	190.34 (67.00)	206.22 (79.52)	217.68 (68.82)	242.82 (66.61)
Initial BDI Score[a]	9.58 (7.98)	11.22 (8.17)	12.94 (9.18)	16.67 (12.33)	15.08 (9.51)
SRIP Neg. Score[b]	-.11 (.57)	-.15 (.48)	.16 (.67)	-.08 (.43)	.19 (.66)
SAS-S 1[b]	18.72 (8.41)	18.44 (8.96)	18.39 (8.20)	20.53 (8.74)	23.95 (8.65)
SAS-S 2[c]	35.20 (10.56)	35.92 (12.05)	34.30 (7.21)	36.49 (9.04)	39.99 (9.76)
SAS-S 3	16.89 (5.29)	17.04 (6.00)	17.03 (4.70)	18.00 (6.10)	19.18 (4.62)
SAS-A 1[b]	40.67 (9.01)	40.02 (8.60)	35.66 (11.86)	37.62 (8.07)	35.55 (11.07)
SAS-A 2	32.27 (9.13)	31.67 (9.45)	29.19 (8.18)	31.92 (8.63)	29.91 (8.11)
SAS-A 3	13.17 (4.52)	13.19 (4.58)	12.44 (3.82)	12.96 (4.93)	12.05 (4.85)
SCS Private[b]	23.45 (5.29)	24.58 (5.79)	22.24 (4.89)	25.65 (4.89)	25.85 (4.87)
SCS Public[a]	20.21 (5.45)	21.22 (6.10)	20.15 (5.81)	22.82 (6.69)	24.84 (5.19)
SCS Social Anx.[a]	16.94 (7.76)	18.43 (8.75)	19.12 (8.03)	22.22 (8.11)	22.30 (8.71)

NE = No Episode; SR = Single Episode Recovery; SN = Single Episode Non-Recovery; RR = Recurrent Recovery; RN = Recurrent Non-Recovery. Unless otherwise noted, statistics presented as Mean (Standard Deviation). CSQ = Cognitive Style Questionnaire; DAS = Dysfunctional Attitudes Scale; BDI = Beck Depression Inventory; SRIP = Self-Referent Information Processing Task Battery; SAS = Sociotropy-Autonomy Scale; SAS-S = SAS-Sociotropy; SAS-A = SAS-Autonomy; SCS = Self-Consciousness Scale.
[a]Groups different at $p < .001$; [b]Groups different at $p < .01$; [c]Groups different at $p < .05$

significant correlations were found between several of the predictor variables, tolerance statistics provided by the discriminant function analyses indicated that multicollinearity was not an issue in the present data set, as none of the specified tolerance values approached zero for any of the predictor variables. Conventional transformations (e.g., natural log, square, square root, etc.) were attempted to allow Initial BDI score to conform more closely to the statistical assumptions of discriminant function analysis; however, these did little to correct the non-normality of the distributions. Accordingly, and because discriminant function analysis is robust in the face of minor violations of normality, no data transformations were performed. However, results including initial BDI score should be interpreted with some degree of caution.

Table 4. Correlation matrix of predictor variables included in analyses

	CSQ	DAS	BDI	SRIP	S1	S2	S3	A1	A2	A3	PRIV	PUB	SOC
CSQ	--	.808**	.481**	.456**	.613**	.421**	.417**	-.368**	.088	-.090*	.238**	.378**	.302**
DAS		--	.536**	.444**	.703**	.506**	.493**	-.430**	.106	-.137*	.275**	.419**	.316**
BDI			--	.311**	.446**	.314**	.314**	-.181**	.171**	.064	.194**	.204**	.146**
SRIP				--	.324**	.120*	.168**	-.282**	-.007	-.024	.061	.195**	.236**
S1					--	.785**	.747**	-.059	.487**	.221**	.311**	.201**	-.048
S2						--	.710**	.112*	.480**	.101	.180**	.040	-.236**
S3							--	.064	.386**	.198**	.206**	.067	-.180**
A1								--	.580**	.585**	.011	-.516**	-.657**
A2									--	.607**	.194**	-.230**	-.452**
A3										--	.171**	-.252**	-.397**
PRIV											--	.562**	.297**
PUB												--	.764**
SOC													--

Pearson correlations reported. *significant at the .05 level (2-tailed); **significant at .01 level (2-tailed). CSQ = CSQ negative composite score; DAS = DAS total score; BDI = BDI initial score; SRIP = SRIP Task Battery negative composite score; S1 = SAS Sociotropy subscale 1; S2 = SAS, Sociotropy subscale 2; S3 = SAS, Sociotropy subscale 3; A1 = SAS, Autonomy subscale 1; A2 = SAS, Autonomy subscale 2; A3 = SAS, Autonomy subscale 3; PRIV = SCS, Private subscale; PUB = SCS; Public subscale; SOC = SCS, Social Anxiety subscale.

Analysis I: Differentiating the Episode and No Episode Groups

In an analysis aiming to differentiate between participants who experienced at least one prospective depressive episode (i.e., SR, SN, RR, and RN groups; n = 177) and those who did not (i.e., the NE group; n = 168), the stepwise procedure retained three predictors. In step 1, CSQ negative composite score was included in the model ($F(1, 343) = 30.910$, $p < .001$); in the second step, the SCS social anxiety subscale score was included ($F(2, 342) = 20.216$, $p < .001$); in the third, initial BDI score was included ($F(3, 341) = 15.396$, $p < .001$). One discriminant function was calculated, with a combined $\chi^2 (3) = 43.381$, $p < .001$. By default, because there was only one function generated, it accounted for 100% of between-groups variability. This model correctly classified 64.9% of cross-validated grouped cases using prior probabilities derived from the assumption of equal group sizes (classification statistics in all analyses made use of prior probabilities derived from the assumption of equal group sizes, which is a more conservative classification method than using prior probabilities derived from pre-existing group sizes), and suggested that CSQ negative composite score best differentiated between the defined participant groups. DAS total score also loaded highly on the discriminant function, but was not retained as a significant predictor. See Table 5 for a summary of the results from this analysis.

Analysis II: Differentiating All Five Groups

In the analysis between all five participant groups (i.e., NE, SR, SN, RR, and RN), the stepwise procedure retained three predictors. In step 1, CSQ negative composite score was included in the model ($F(4, 340) = 12.617$, $p < .001$); in the second step, SCS

Table 5. Summary of Information from Analysis I

	Function 1
Structure Matrix[a]	
CSQ Negative Score	.816
DAS Total Score	.731
Initial BDI Score	.670
SCS Social Anxiety Subscale	.636
SCS Public Subscale	.580
SAS Autonomy Subscale 1	-.526
SAS Sociotropy Subscale 1	.426
SRIP Negative Composite	.420
SCS Private Subscale	.295
SAS Sociotropy Subscale 3	.215
SAS Autonomy Subscale 2	-.194
SAS Sociotropy Subscale 2	.191
SAS Autonomy Subscale 2	-.077
Days Followed	.030
Functions at Group Centroids[b]	
Episode	.358
No Episode	-.377

[a]Pooled within-groups correlations between discriminating variables and standardized canonical discriminant functions. Variables ordered by absolute size of correlation within function. [b]Unstandardized canonical discriminant functions evaluated at group means.

public subscale score was included ($F(8, 678) = 8.158$, $p < .001$); in the third step, initial BDI score was also included ($F(12, 894) = 6.368$, $p < .001$). Three discriminant functions were calculated, with a combined $\chi^2 (12) = 73.734$, $p < .001$. After removal of the first function, which accounted for 88.3% of between-groups variance, there was no significant association between groups and predictors, $\chi^2 (6) = 9.332$, $p = .156$. The first discriminant function maximally separated the Recurrent (i.e., RR, RN) groups from the Single-Episode (i.e., SR, SN) groups from the No-Episode (NE) group. The second function maximally separated the RR group from the SN group from the SR and NE groups from the RN group. The third function maximally separated the RR group from

the SR, RN, and NE groups from the SN group. As in Analysis I, DAS total score also loaded highly on the discriminant function, but was not retained as a significant predictor. The results suggest that of all variables examined, CSQ negative composite score best differentiated between participant groups. This model correctly classified 41.7% of cross-validated grouped cases. See Table 6 for a summary of the results from this analysis.

Table 6. Summary of Information from Analysis II

	Function 1	Function 2	Function 3
Structure Matrix[a]			
CSQ Negative Score	.837*	-.200	-.510
DAS Total Score	.747*	.005	-.257
SCS Public Subscale	.686*	-.255	.682
SCS Social Anxiety Subscale	.508*	-.228	.502
SAS Sociotropy Subscale 1	.505*	.068	-.275
SAS Autonomy Subscale 1	-.447*	.156	-.215
SCS Private Subscale	.412*	-.080	.348
SRIP Negative Composite	.371*	.025	-.151
Days Followed	.323*	.053	-.058
SAS Sociotropy Subscale 3	.303*	.069	-.252
SAS Sociotropy Subscale 2	.291*	.078	-.284
Initial BDI Score	.614	.778*	-.134
SAS Autonomy Subscale 3	-.123	.178*	-.161
SAS Autonomy Subscale 2	.027	.178	-.280*
Functions at Group Centroids[b]			
SR	-.099	-.012	.012
SN	.006	.093	-.331
RR	.414	.371	.050
RN	.782	-.155	.007
NE	-.372	-.031	.017

[a]Pooled within-groups correlations between discriminating variables and standardized canonical discriminant functions. Variables ordered by absolute size of correlation within function. [b]Unstandardized canonical discriminant functions evaluated at group means. *Largest absolute correlation between each variable and any discriminant function.

Analysis III: Differentiating Recovered, Non-Recovered, and No Episode Groups

In an analysis differentiating between participants recovering from a given episode of depression (SR and RR groups; n = 93) from those who did not recover (SN and RN groups; n = 84) relative to participants who did not experience an episode of depression (NE; n = 168), the stepwise procedure retained two predictors. In step 1, CSQ negative composite score was included in the model ($F(2, 342) = 21.726$, $p < .001$); in the second step, SCS social anxiety subscale score was also included ($F(4, 682) = 12.898$, $p < .001$). Two discriminant functions were calculated, with a combined $\chi^2 (4) = 49.808$, $p < .001$. After removal of the first function, which accounted for 97.9% of between-groups variance, there was no significant association between groups and predictors, $\chi^2 (1) = 1.119$, $p = .290$. The first discriminant function maximally separated all three participant groups from each other (i.e., the function differentiated between the Recovery, Non-Recovery, and No Episode groups). The second function maximally separated the Recovery group from the Non-Recovery and No Episode groups. The results suggest that of all variables examined, CSQ negative composite score best differentiated between participant groups. DAS total score also loaded highly on the discriminant function, but was not retained as a significant predictor. This model correctly classified 50.4% of cross-validated grouped cases. See Table 7 for a summary of the results from this analysis.

Analysis IIIa: Differentiating Recovered and Non-Recovered Groups

In a further analysis including only participants who experienced at least one episode of depression, differentiating between participants recovering from a given

Table 7. Summary of Information from Analysis III

	Function 1	Function 2
Structure Matrix[a]		
CSQ Negative Score	.909*	-.418
DAS Total Score	.724*	-.244
SCS Public Subscale	.560*	.515
SAS Autonomy Subscale 1	-.516*	-.409
SRIP Negative Composite	.394*	-.058
Initial BDI Score	.390*	-.193
SCS Private Subscale	.278*	.129
SCS Social Anxiety Subscale	.614	.789*
SAS Sociotropy Subscale 2	.190	-.497*
SAS Autonomy Subscale 2	-.084	-.496*
SAS Sociotropy Subscale 3	.211	-.441*
SAS Sociotropy Subscale 1	.413	-.437*
SAS Autonomy Subscale 3	-.215	-.328*
Days Followed	.107	-.235*
Functions at Group Centroids[b]		
Recovery	.101	.093
Non-Recovery	.600	-.049
No Episode	-.356	-.027

[a]Pooled within-groups correlations between discriminating variables and standardized canonical discriminant functions. Variables ordered by absolute size of correlation within function. [b]Unstandardized canonical discriminant functions evaluated at group means. *Largest absolute correlation between each variable and any discriminant function.

episode of depression (SR and RR groups; n = 93) from those who did not recover (SN and RN groups; n = 84), the stepwise procedure retained one variable. SRIP composite score was included in the model ($F(1, 175) = 13.478$, $p < .001$). One discriminant function was calculated, $\chi^2 (1) = 12.947$, $p < .001$. By default, this discriminant function accounted for 100% of between-group variability. In examining the structure matrix from this analysis, no other predictors loaded highly on the function; although CSQ negative composite score and DAS total score had the second- and third-highest loadings, respectively, neither was retained as a significant predictor. This model correctly

classified 61.0% of cross-validated grouped cases. See Table 8 for a summary of the results from this analysis.

Table 8. Summary of Information from Analysis IIIa

	Function 1
Structure Matrix[a]	
SRIP Negative Composite	1.000
CSQ Negative Score	.481
DAS Total Score	.476
SAS Sociotropy Subscale1	.369
Initial BDI Score	.358
SAS Autonomy Subscale 1	-.269
SAS Sociotropy Subscale 3	.247
Days Followed	.237
SCS Social Anxiety Subscale	.230
SAS Sociotropy Subscale 2	.158
SCS Public Subscale	.106
SAS Autonomy Subscale 2	.075
SAS Autonomy Subscale 3	.035
SCS Private Subscale	.004
Functions at Group Centroids[b]	
Recovery	-.262
Non-Recovery	.290

[a]Pooled within-groups correlations between discriminating variables and standardized canonical discriminant functions. Variables ordered by absolute size of correlation within function. [b]Unstandardized canonical discriminant functions evaluated at group means.

Analysis IV: Differentiating Single Episode, Recurrent Episode, and No Episode Groups

In the analysis differentiating between participants with a single prospective episode of depression (SR and SN groups; n = 72) from those with recurrent episodes (RR and RN groups; n = 105) relative to participants who did not experience an episode of depression (NE; n = 168), the stepwise procedure retained three predictors. In step 1, CSQ negative composite score was included in the model ($F(2, 342) = 22.298$, $p < .001$);

in the second step, SCS social anxiety subscale score was included ($F(4, 682) = 14.388$, $p < .001$); in the third step, initial BDI score was also included ($F(6, 680) = 10.973$, $p < .001$). Two discriminant functions were calculated, with a combined $\chi^2 (6) = 60.026$, $p < .001$. After removal of the first function, which accounted for nearly 100% of between-groups variance, there was no significant association between groups and predictors, $\chi^2 (2) = .020$, $p = .990$. The first discriminant function maximally separated all three participant groups from each other (i.e., the function differentiated between the Single Episode, Recurrent, and No Episode groups). The second function maximally separated the Recurrent and No Episode groups from the Single Episode group. Again, DAS total score also loaded highly on the first discriminant function, but was not retained as a significant predictor. This model correctly classified 50.1% of cross-validated grouped cases. See Table 9 for a summary of the results from this analysis.

Analysis IVa: Differentiating Single Episode and Recurrent Episode Groups

In a further analysis including only participants who experienced at least one episode of depression, differentiating between participants who experienced a single episode of depression (SR and SN groups; n = 72) from those who experienced recurrent episodes (RR and RN groups; n = 105), the stepwise procedure retained two variables. In the first step, DAS total score was included in the model ($F(1, 175) = 13.776$, $p < .001$); in the second step, SCS public subscale score was also included ($F(2, 174) = 9.956$, $p < .001$). One discriminant function was calculated, $\chi^2 (2) = 18.852$, $p < .001$. By default, this discriminant function accounted for 100% of between-group variability. The discriminant function maximally separated the two groups from one another. In examining the structure matrix from this analysis, CSQ negative composite score also

Table 9. Summary of Information from Analysis IV

	Function 1	Function 2
Structure Matrix[a]		
CSQ Negative Score	.802*	-.503
DAS Total Score	.713*	-.319
Initial BDI Score	.657*	-.244
SCS Public Subscale	.567*	.462
SAS Autonomy Subscale 1	-.525*	-.355
SRIP Negative Composite	.409*	-.103
SCS Private Subscale	.278*	.105
Days Followed	.215*	.095
SCS Social Anxiety Subscale	.633	.737*
SAS Sociotropy Subscale 2	.165	-.519*
SAS Sociotropy Subscale 1	.396	-.483*
SAS Autonomy Subscale 2	-.081	-.482*
SAS Sociotropy Subscale 3	.190	-.465*
SAS Autonomy Subscale 3	-.192	-.305*
Functions at Group Centroids[b]		
Single Episode	-.042	-.015
Recurrent	.648	.003
No Episode	-.387	.004

[a]Pooled within-groups correlations between discriminating variables and standardized canonical discriminant functions. Variables ordered by absolute size of correlation within function. [b]Unstandardized canonical discriminant functions evaluated at group means. *Largest absolute correlation between each variable and any discriminant function.

loaded highly on the function but was not retained as a significant predictor. This model correctly classified 66.7% of cross-validated grouped cases. See Table 10 for a summary of the results from this analysis.

Table 10. Summary of Information from Analysis IVa

	Function 1
Structure Matrix[a]	
DAS Total Score	.829
SCS Public Subscale	.774
CSQ Negative Score	.664
SCS Social Anxiety Subscale	.595
SAS Autonomy Subscale 1	-.557
SAS Sociotropy Subscale 1	.483
SCS Private Subscale	.426
SRIP Negative Composite	.393
Initial BDI Score	.376
SAS Sociotropy Subscale 3	.301
Days Followed	.297
SAS Sociotropy Subscale 2	.293
SAS Autonomy Subscale 3	-.239
SAS Autonomy Subscale 2	-.049
Functions at Group Centroids[b]	
Single Episode	-.406
Recurrent	.279

[a]Pooled within-groups correlations between discriminating variables and standardized canonical discriminant functions. Variables ordered by absolute size of correlation within function. [b]Unstandardized canonical discriminant functions evaluated at group means.

CHAPTER 4

DISCUSSION

The main hypothesis of the present study was supported, in that statistically-guided analytical techniques were able to distinguish between predefined participant groups at rates significantly better than chance. That is, measures of cognitive vulnerability to depression given at the outset of the study were capable of prospectively predicting the course of participants' depressive symptoms over a significant follow-up period; furthermore, the combination of these measures that maximally differentiated between participant groups were elucidated through actuarial data analytic strategies. Variables emerged from the analyses that differentiated participants who experienced an episode of depression during the prospective phase of the study from those who did not, as well as those that differentiated between participants based on several important course variables (i.e., recovery versus non-recovery from a given episode of depression; a single prospective episode versus recurrent episodes).

That scores on measures of cognitive vulnerability to depression were related to the course of depression is not especially surprising in general terms; as noted previously, a wealth of previous research has demonstrated associations between cognitive variables and the course of depression. However, the suggestion that such variables can predict the course of depression prospectively is a promising finding, especially when the present results suggest that statistically-derived combinations of these variables consistently predict depressive course at a rate higher than chance.

In particular, the degree to which the CSQ emerged as a significant predictor of depressive course is certainly noteworthy. In all of the main analyses, CSQ negative

composite score emerged as the most robust predictive variable; this was true whether differentiating groups based on presence versus absence of a prospective depressive episode (as in Analysis I), recovery versus non-recovery from a given episode of depression (as in Analysis III), or the episodic nature of symptoms over time (i.e., single versus recurrent episodes; as in Analysis IV). CSQ negative composite score also emerged as the variable that best distinguished between participants in a more fine-grained analysis, in which they were grouped based on both recovery status and episodic nature of symptoms (in Analysis II). It should be noted that DAS total score also loaded highly on functions in each of these analyses; however, in each case, CSQ simply accounted for more between-groups variability, and was thus consistently retained over the DAS score. This might suggest that, although DAS score is an important indicator of cognitive vulnerability to depression (i.e., in Analysis IVa, it was the variable that best predicted whether a participant would experience a single episode of depression or multiple episodes over the course of the study), the CSQ may represent a more sensitive tool in its predictive accuracy vis-à-vis the course of depression as defined in the present study. Another interesting finding related to this was the pattern of group means on the CSQ; that is, these showed a trend such that higher mean negative composite scores were associated with groups displaying more "complex" depressive course (i.e., No Episode < Single Episode < Recurrent). Furthermore, within the groups that experienced at least one depressive episode, those who recovered had a lower mean CSQ negative composite score than those who did not.

Although the foregoing analyses suggest that the CSQ may represent a valuable predictive tool, it is of note that several other variables emerged as significant prospective

predictors of depressive course. In Analyses I, II, III, IV, and IVa, subscales from the SCS emerged as predictors retained in addition to CSQ negative composite score. Although the functions associated with the SCS were consistently non-significant after removal of the first discriminant functions (i.e., after removing the between-groups variance first incremented by the CSQ), this is an interesting finding nonetheless. Specifically, this suggests that the SCS may also serve as a useful predictive tool when used in conjunction with a more sensitive measure such as the CSQ. Interestingly, only the Public and Social Anxiety Self-Consciousness subscales emerged as significant predictors; the Private Self-Consciousness subscale was not retained in any of the analyses. It may be that, for individuals at relatively high risk for depression (as assessed with the CSQ, for example), items focusing on awareness of, and excessive concerns with, issues such as one's appearance and how one presents oneself to others (as assessed by the Public subscale), or items assessing shyness and discomfort with strangers (as with the Social Anxiety subscale) are simply more pertinent in predicting future depressive symptoms than are items tapping such constructs as self-awareness and awareness of one's emotional state (as assessed with the Private subscale). Perhaps this discrepancy is due to some degree of conceptual overlap between an individual's risk status and private self-consciousness; that is, perhaps individuals at high cognitive risk are generally more (or less) prone to reflect on their internal states and to try to "figure themselves out" than are individuals at low cognitive risk. If this were the case, the other two subscales of the SCS would simply account for more unique variability between groups (i.e., over and above the variability already accounted for by the variable(s) assessing cognitive risk),

and would thus be more likely to be retained as predictors in analyses seeking to maximize differences between groups.

Initial BDI score also emerged as a significant predictor in Analysis I, II , and IV, suggesting that one's relative level of depressive symptomatology at any given point in time is one potentially useful predictor of future symptomatology. Importantly, however, it should be noted that across analyses, discriminant functions associated with BDI score, like those associated with SCS subscale scores, were not significant in the absence of those primarily associated with the CSQ; furthermore, as noted above, the importance of the BDI as a predictor must be viewed with some degree of caution due to the fact that this was the most problematic variable with respect to conformity with the assumptions of the present analyses.

The only analysis that did not retain variables associated with "standard" measures of cognitive vulnerability, such as the CSQ or DAS, was Analysis IIIa, in which the results pertaining to self-referent information processing were of great interest. This factor did not emerge as a significant predictor when all participant groups were considered in the same analysis, or even when participants experiencing recovery versus non-recovery from a given episode of depression were contrasted with those who did not experience a depressive episode; indeed, SRIP composite score was only retained as a predictor when differentiating between participants with a prospective depressive episode experiencing recovery versus non-recovery from that episode. This analysis suggests that SRIP may serve as a differential predictor of one's propensity to recover from an episode of depression; this is consistent with previous research examining relapse and recurrence in depression (e.g., Grant, Alloy, & Abramson, 2009). Here, the difference was in the

direction of participants who recovered (i.e., those in the SR and RR groups) exhibiting more positive (i.e., less negative) SRIP than those who did not recover from a given episode of depression (i.e., those in the SN and RN groups). Perhaps less negative, or more positive, self-referent information processing may be related to the recovery process itself, inasmuch as a person with a relatively less negative cognitive style may be less prone to engage in depressogenic cognitions, may be less prone to perseverate on such cognitions, or may be more adept at disengaging from negative self-referent information processing. The present findings are certainly consistent with this possibility, as well as with emerging research concerning the nature of information processing in depression that suggests depressed individuals may be less able than nondepressed individuals to disengage from negative self-referent information processing (this has been termed the "impaired disengagement hypothesis;" e.g., Koster, De Lissnyder, Derakshan, & De Raedt, 2011). Perhaps future research can shed more light on this matter, and clarify the interesting relationship between SRIP and depressive course.

In sum, the results of the present study are largely consistent with the extant literature concerning cognitive vulnerability to depression. A wealth of previous research has provided compelling evidence suggesting that cognitive variables are related to the onset and course of depression. This is true with respect to a higher rate of onset of symptoms (e.g., Abramson et al., 1999; Alloy et al., 2006; Beck, 1967; 1987; Clark et al. 1999; Ingram, Miranda, & Segal, 1998; Teasdale, 1988), a higher rate of relapse and/or recurrence of depression (e.g., Alloy et al., 2006; Bockting et al., 2005; Burcusa & Iacono, 2007; Iacoviello et al., 2006; Lewinsohn et al., 1999; Mongrain & Blackburn, 2006; Vittengl et al., 2007), and to persistence of depressive symptoms, non-remission,

and non-recovery from depressive episodes (e.g., Dent & Teasdale, 1988; Grant et al., 2009; Iacoviello et al., 2006; Riso et al., 2003; Scott, Harrington, House, & Ferrier, 1996). The results of the present study are certainly consistent with this previous research, and suggest that variables representing cognitive vulnerability factors are useful in predicting the onset and course of depressive symptoms. However, the present study also serves as an interesting extension of previous research, in that statistically-guided analyses were used to maximize prospective predictive accuracy with respect to a variety of aspects of the onset and course of depression. That such variables can be used by these means to consistently predict clinically-relevant outcomes at a rate higher than chance is one noteworthy aspect of these results.

In addition, however, the suggestion that the CSQ may most accurately differentiate between participant groups relative to other measures may have important conceptual and theoretical implications. These measures were created as extensions of cognitive models of depression, with the CSQ arising from the tenets of the hopelessness theory of depression (e.g., Abramson et al., 1989; Alloy et al., 1999), and the DAS stemming from Beck's (1967) theory. As such, to the extent that one measure emerges as a more reliable predictor of depressive course relative to the other, this may provide support for the theory from which the measure originates. In the present study, the majority of the analyses thus seem to favor the hopelessness theory of depression in prospectively predicting who becomes depressed versus who does not, who will recover from a given episode of depression versus who will not (in comparison to those who do not become depressed), and who will experience a single episode of depression versus those who will experience a recurrent course of the disorder (again, in comparison to

those who do not become depressed). In sum, the present results strongly suggest that the CSQ – and by extension the hopelessness theory of depression – are useful in predicting several aspects of depressive course, including recovery from depression, especially when predicting the course of the disorder relative to individuals who do not experience an episode of depression.

Study Strengths

The present study exhibited several significant strengths. The data were from the Temple-Wisconsin CVD Project, a seminal, large-scale, multi-site prospective longitudinal study, and as such, many of the strengths of the original study (e.g., methodological rigor, large sample size, adequate statistical power) are conferred to the present study as well. Additionally, the nature of the present analyses provided a relatively novel take on traditional research questions concerning cognitive vulnerability to depression, in that the present study was more concerned with describing a method and statistical framework that may ultimately prove useful in informing real-world clinical practice rather than with underlying theory per se. In using an actuarial statistical application to elucidate variables that may prospectively predict the onset and course of depression, as well as variables that may be associated with recovery from depression, the present study represents a small but potentially meaningful step toward bridging the gap between science and practice in depression prevention and intervention.

Study Limitations

Although the present study was relatively novel in its aims, and although the analyses conducted were appropriate and possessed sufficient statistical power, there were several notable limitations. First, although the overall sample size was certainly

adequate, the group sizes were clearly unequal; this is especially true with respect to the subgroups of participants experiencing at least one prospective episode of depression. However, discriminant function analysis is relatively robust in the face of group size differences such as those seen in this study (e.g., Tabachnick & Fidell, 2007).

Another methodological limitation concerns the nature of the data; that is, apart from the SRIP task battery, all of the other measures included in the present analyses were self-report questionnaires. To the extent that behaviorally- or task-based data may serve to improve the accuracy of prediction, it would certainly be desirable to have included a wealth of non-self-report data as well. However, it should be reiterated that one of the ultimate aims of the present study was to provide an efficient means to supplement clinical judgment with respect to prediction of depressive course in real-world contexts. As such, although data apart from that obtained through self-report may be highly useful in a predictive sense, given the time and resource constraints of clinical practice, it ultimately may not prove feasible to collect a large amount of high-quality task-based data for predictive purposes outside the research laboratory.

Furthermore, the procedure by which the participant sample used in the present study was selected may limit the generalizability of the results. That is, participants in the CVD Project did not represent an unselected sample; rather, they were selected for their relative degree of cognitive risk. As noted above, only those participants scoring in the highest and lowest quartiles on measures of cognitive risk were included in the final study sample; as such, participants representing a moderate degree of cognitive risk were not included. Thus, although the present results and classification algorithms certainly

apply to the CVD Project participants, this predictive accuracy may not extend to other samples, clinical or otherwise.

At a conceptual level, the foregoing analyses also may seem to oversimplify the nature of the onset, maintenance, and amelioration of depressive symptoms to a great degree. For instance, the present analyses and statistical models fail to take into account many contextual factors related to depression, such as the influence of family, schools, neighborhoods, peers, etc. Such contextual factors also may be especially important in selecting adolescents at high risk for developing depression (e.g., Paunesku et al., 2008; Seeley et al., 2009). The present study also was incapable of addressing the complex roles of such factors as underlying neurobiology, social development, or life events in the course of depression. However, it is important to note that the present study was not necessarily concerned at this juncture with taking the full spectrum of risk factors for depression into account; rather, it was concerned solely with specifying a means by which to ultimately afford practicing clinicians and relevant agencies greater predictive accuracy in terms of who is at risk for depression, and for a particular course of depression, while maintaining a high degree of simplicity of administration and interpretation.

In addition, at times the present discussion admittedly blurs the distinction between purely actuarial measures and theory-driven ones. Whereas the foregoing analyses made use of previously-established measures as potential predictors, as well as cognitive vulnerability theories more broadly, it did so for the sake of using statistical processes to achieve the highest degree of predictive accuracy possible. That is, although perhaps a "soft" actuarial tool by virtue of its reliance on previously articulated clinical

hypotheses and theories, the results of the present study nonetheless suggest a choice of cognitive risk factors for depression weighted solely by statistical procedures, which is in essence an actuarial application.

Another major limitation of the present study speaks directly to the research question itself. That is, classification of participants into groups based on depressive course is tremendously difficult in the absence of firm, consensus definitional criteria. Although the Frank and colleagues' (1991) and Rush and colleagues' (2006) criteria were followed as closely as possible, certain executive decisions were necessary nonetheless. For example, should one be considered recovered from an episode of depression if one has a four-month period of residual symptoms that do not qualify as clinically significant? What about the case of a similar period of symptomatology, but of eight months in duration? The decisions made in conducting the present study (i.e., to ascribe non-recovery to both such situations) were in some cases admittedly arbitrary, but a decision along these lines was nevertheless required. This process of classification in the absence of firm and comprehensive classification guidelines speaks to the importance of such guidelines, and hence only bolsters the argument calling for the establishment of, and adherence to, such consensus definitions.

Clinical Implications

The present results speak to several potentially important clinical implications. Foremost among these is the notion that existing measures of cognitive vulnerability may be useful from a predictive standpoint, in terms of providing valuable information as to who is at relatively high risk for developing a future depressive episode, and who may be at relatively high risk for encountering a relatively severe course of the disorder (e.g.,

recurrent episodes; non-recovery from symptoms). Furthermore, these measures serve to predict such outcomes at rates consistently better than chance. To that end, clinicians may choose to institute relatively simple procedures (e.g., administering the CSQ) to aid in predicting clinical course, or to supplement their clinical judgment with respect to prediction. These findings also have notable implications for prevention and early intervention efforts; for example, if the predictive accuracy of the CSQ is found to generalize beyond the CVD Project sample, it would be useful to administer it to adolescents in order to better identify individuals at relatively high risk for depression, or for a severe course of the disorder. Although such prevention efforts are decidedly not simple or straightforward for reasons explained more thoroughly above, the availability of an efficient and relatively straightforward predictive measure would likely bolster such efforts by making them more cost-efficient and less burdensome.

Additionally, the content of these predictive measures may also inform prevention efforts over and above identification of high-risk individuals. For example, because the CSQ has been shown to predict various aspects of depressive course prospectively at rates higher than chance, and because this measure assesses inferential style, a logical clinical extension would be to attempt prevention by targeting inferential style. Here, such efforts might be targeted or universal, but could focus on factors such as psychoeducation around the nature of causal attributions, and training in making realistic causal attributions and other inferences. Such screening and/or preventative education efforts conceivably could be incorporated into existing school curricula, as have other efforts discussed above (e.g., Horowitz et al., 2007), or could be implemented instead in

healthcare settings if the decision were made to undertake such interventions in a more targeted manner.

Future Directions

Future directions in this line of research are many and varied. For example, there are a host of other variables that may well be related to the prediction of depressive course. Variables such as Axis I and II comorbidity, significant medical conditions, presence and quality of treatment (whether it be psychotherapy or pharmacotherapy), and the complex and multifaceted interaction between mood course and life events would be relevant to examine in the context of their association with depressive course. Another area of promising future research concerns self-referent information processing specifically. In the present study, only the SRIP composite score was included in the analyses. However, it would be interesting and informative to conduct analyses using a variety of other data pertaining to self-referent information processing (e.g., data on biases in autobiographical memory, attentional biases, etc.) in its potential association with the course of depressive symptoms.

The results of the present study also require further empirical validation using independent, unselected samples. Although the cross-validation procedure employed in the present analyses is one means by which to ensure the development of an accurate and relatively unbiased classification scheme, there is still a significant possibility that the actuarial methods employed capitalized on chance relationships within the existing data set to maximize predictive accuracy. As such, prospective studies using independent samples will serve as the true test of the predictive accuracy of the present models. In addition, once validated, it may prove feasible to take additional variables, such as those

noted above, into account to provide even more predictive power without necessarily sacrificing all-important efficiency. In the interim, many of these potential shortcomings might be addressed by the delivery of supplemental measures to those identified as high risk for developing depression, or for developing a relatively chronic or complicated course of the disorder. In that way, the efficiency of the screening process itself would not be compromised.

Conclusions

Results of the present study provide preliminary evidence for the utility of actuarial prediction in the realm of clinical decision-making with respect to risk for the onset of depression, as well as certain aspects of depressive course. Specifically, the present results suggest that the CSQ may serve as an especially useful predictor of a given individual's risk for developing depression in the future, as well as a predictor of several depressive course variables, such as recovery versus non-recovery from a given episode of depression. These results also suggest that administration of the CSQ in such contexts may be supplemented with measures such as the SCS and BDI; indeed, various combinations of these measures served to predict depressive onset and course at rates better than chance. Furthermore, these analyses suggest that measures such as the DAS and SRIP task battery may also serve as useful clinical prediction tools.

Thus, although many of the measures examined in the present study have been used traditionally in research contexts, or (as in the cases of measures like the DAS or the BDI), as indicators of current clinical status, their role as relevant indicators of cognitive vulnerability that may also serve an important, real-world predictive role, is underscored. Further validation of these results will speak more broadly to the applicability of such

measures in real-world clinical contexts; for example, in terms of their utility as predictive screening tools. Importantly, if future research should provide independent validation, this also will speak to the potential of highlighting cognitive vulnerability itself (as measured, for example, with the CSQ) as a relevant target in prevention and intervention efforts.

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