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Toward an understanding of the cognitive etiology of depressive reactions to life stressors: An evaluation of the hopelessness theory of depression

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TOWARD AN UNDERSTANDING OF THE COGNITIVE ETIOLOGY OF DEPRESSIVE REACTIONS TO LIFE STRESSORS: AN EVALUATION OF THE HOPELESSNESS THEORY OF DEPRESSION

BY

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To my parents,
Joan B. Gibbons and Michael Asdigian

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ABSTRACT

TOWARD AN UNDERSTANDING OF THE COGNITIVE ETIOLOGY OF DEPRESSIVE REACTIONS TO LIFE STRESSORS: AN EVALUATION OF THE HOPELESSNESS THEORY OF DEPRESSION

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Abramson, Metalsky, and Alloy's (1989) theory of hopelessness depression is the most recent model of depression to emerge from the learned helplessness tradition (Seligman, 1975). Hopelessness theory describes an etiological pathway by which a unique subtype of depression-- hopelessness depression-- is believed to emerge.

The most proximal cause of hopelessness depression is the formation of a hopelessness expectancy. Experiencing a significant life stressor and either making stable and global causal attributions, anticipating adverse consequences, or inferring derogratory self-attributes is thought to contribute to hopelessness. In turn, generalized tendencies to make stable and global causal attributions, expect negative consequences, or perceive personal deficiencies in response to life stress purportedly increase the likelihood of making hopelessness-inducing inferences, and thus increase the risk of hopelessness and depressive symptoms.

The present research sought to a.) provide a comprehensive assessment of the proposed etiological pathway and b.) evaluate the competing predictions made by the hopelessness model and its most immediate theoretical precursor, the reformulated theory of learned

helplessness depression (Abramson, Seligman, & Teasdale, 1978). In a two-wave panel design, college undergraduates (N = 247) completed measures assessing the constructs of both models. Higher levels of dysfunctional cognitive styles and event-based inferences predicted increases in depressive symptoms only among subjects who experienced an upsetting interpersonal stressor that was also perceived as uncontrollable. Little support was obtained for the hypothesis that hopelessness mediates associations between depressive symptoms and either maladaptive cognitive styles or event-inferences.

Consistent with the postulates of hopelessness theory, dysfunctional cognition appeared to render individuals vulnerable to depressive symptomatology in the face of life stress. However, the present findings call into question hopelessness theory's elimination of control perceptions in its etiological pathway. Continued study of the control construct is encouraged as are future tests of hopelessness theory that use more sophisticated assessments of life stress and fine-grained measures of hopelessness expectancies.

INTRODUCTION

The cognitive approach to depression has emerged over the past several decades as one of the leading paradigms guiding the study as well as the treatment of depressive disorders (Gilbert, 1984). Although numerous theories of depression align themselves with such a perspective, (e.g, Beck, 1967; Beck, Rush, Shaw, & Emery, 1979; Ellis, 1985; Seligman, 1975), the assumption that cognitive structures and processes are intimately involved in the etiology of depressive illnesses lies at the heart of each theory.

A substantial amount of theoretical and empirical work on the cognitive underpinnings of depression has been inspired by Seligman's (1975) influential treatise on learned helplessness. The most recent model of depression to emerge from the learned helplessness tradition is Abramson, Metalsky, and Alloy's (1989, 1988; Abramson, Alloy, & Metalsky, 1988) hopelessness theory of depression. Abramson et al.'s (1989) model posits the existence of a unique subtype of unipolar depression--hopelessness depression--and outlines an etiological pathway by which the hypothesized depressive subtype is believed to emerge. A comprehensive assessment of the causal pathway and etiological processes specified by Abramson et al. has not yet been undertaken, although more restricted tests of specific components of the hopelessness model have been generally supportive (e.g., Alloy & Clements, 1992; Metalsky, Abramson, Seligman, Semmel, & Peterson, 1982; Metalsky, Halberstadt, & Abramson, 1987; Metalsky & Joiner, 1992; Metalsky,

Joiner, Hardin, & Abramson, 1993). The present research was designed to test the full causal model offered by Abramson et al. (1989) and to evaluate the competing predictions made by hopelessness theory and its theoretical forerunner, the reformulated theory of learned helplessness depression (Abramson, Seligman, & Teasdale, 1978).

The Nature and Prevalence of Depressive Disorders

Mood disorders represent a pervasive form of psychopathology, afflicting up to 100 million people worldwide (Charney & Weismann, 1988). Of the different types of mood disturbances, major depression is one of the most prevalent. More people suffer from a major depressive disorder than all other psychiatric disturbances combined. Using DSM-III-R criteria (American Psychiatric Association; APA, 1987), major depressive episodes are diagnosed in individuals who experience either depressed mood or anhedonia for a period of 2 weeks or more. The presence of additional features such as unintentional weight changes (i.e., weight loss or gain), sleep disturbances (i.e., insomnia or hyposomnia), psychomotor retardation, feelings of guilt or worthlessness, is also required for a diagnosis of major depression.

In their review of current epidemiological data, Charney and Weissman (1988) reported that as many as 3.5% of U.S. citizens suffer a major depressive episode during any six-month period. Substantially higher prevalence rates (i.e., 13-20%) have been observed for less severe but clinically significant depressive symptomatology as well as for major depression in some high risk groups (e.g., women). Moreover, up to 6.7% of the U.S. population has experienced an episode of major depression sometime during their life and incidence rates of depressive

disorders have steadily increased during the last decade (cf. Seligman, 1990).

Depression also tends to be either chronic or recurrent (APA, 1987; Charney & Weismann, 1988). With respect to the former, evidence indicates that as many as one-quarter of major depressive episodes are superimposed on dysthymic disturbances. Dysthymia is a moderate but chronic form of depression that persists for a large majority of sufferers (roughly 60%) after more severe depressive episodes remit. Additionally, 20% of major depressive episodes among individuals without a history of dysthymia persist for intervals longer than two years. With respect to recurrence, between 50% and 85% of individuals who experience an episode of major depression will have at least one subsequent episode in their lifetime.

In addition to the distress and functional impairment experienced by those who suffer from it, depression takes its toll in numerous other ways. For example, of all individuals diagnosed with DSM-III-R disorders, those with mood disorders represent the greatest portion of mental health service users (Charney & Weissman, 1988). Furthermore, the children of depressed parents are two to three times more likely than children of nondepressives to experience a range of pathological disorders, especially depression. The former are also at increased risk for a variety of behavioral problems including school difficulties, social skill deficits, and substance abuse (Charney & Wiessman, 1988; see also Hammen, 1991)).

Finally, depression is responsible for a large majority of attempted and completed suicides. Weissman (1974) found that 80% of

patients hospitialized for a suicide attempt received a diagnosis of major depression. Depressives are also more likely than other suicide-prone individuals (e.g., those with borderline personality disorder) to complete their suicide attempts (Boyer & Guthrie, 1986).

The Classification of Depressive Disorders

Psychopathologists have long regarded unipolar depression as a heterogenous disorder with respect to both underlying etiology and clinical manifestation (Depue & Monroe, 1978). Researchers have made significant advances in uncovering both biological and psychological factors that contribute to the onset of depression (Shelton, Hollon, Purdon, & Loosen, 1991). In addition, numerous subtypes have been proposed for both unipolar major depression (e.g., primary, secondary, psychotic, neurotic, endogenous, reactive, familial pure depressive disease, depression spectrum disease, sporadic depressive disease) and dysthymic disorders (e.g., subaffective dysthymia; character spectrum disorder) (Leber, Beckham, & Danker-Brown, 1985; Rush, 1986).

Unfortunately, the correspondence between etiology and depressive subtypes has not been unequivocally established (Abramson et al., 1988a; Leber et al., 1985; Rush, 1986). For example, stressful life events are believed to contribute to the onset of reactive depression whereas endogenous depression is thought to arise independently of environmental events. Depressives falling in both diagnostic categories, however, report stressful life events as precipitants of their symptoms (Rush, 1986). Conversely, biological correlates believed to distinguish endogenous depression have been observed in varying degrees in both endogenous and reactive depressives (Leber et al., 1985). Furthermore,

psychopharmacological treatments for depression are effective in individuals diagnosed with a variety of depressive disorders (Shelton et al., 1991).

Largely because of the lack of conclusive etiological evidence, DSM-III-R criteria for the differential diagnosis of depressive disorders reflect only clinical data (i.e., variability in presenting signs and symptoms and/or treatment response). The etiology of those disorders is merely inferred (Leber et al., 1985; Millon, 1991; Rush, 1986). For example, biological dysfunction is believed to underlie DSM-III-R's category of melancholic depression. The diagnosis of melancholia, however, is based only on the presence of a variety of vegetative symptoms (e.g., weight loss, insomnia) (APA, 1987). As Rush (1986, p. 9) aptly stated:

> In our current state of knowledge, psychiatric syndromes are nonspecific etiologically... In affective disorder the field is replete with ideas, but no current model is specific enough for us to understand fully the etiology or pathogenetic mechanisms in any given case...In light of these problems, DSM-III diagnoses are simply based on the apparent phenomenology (the particular signs and symptoms) and do not imply a specific etiology.

An etiologically-based classification system for depressive disorders would represent a significant advance over our current understanding of the illness of depression. An etiological (rather than descriptive) approach to the identification of depression subtypes would also be of great value to clinicians in choosing appropriate treatment regimens. A model of depression, based on such an approach, has recently been offered by Abramson et al. (1989). Abramson and colleagues proposed that hopelessness depression is an etiologically distinct subtype of depression. According to the model, a series of

events, occurring in a causal chain, contributes to the development of a hopelessness expectancy. Hopelessness is characterized by the expectation that undesirable events are likely to occur in the future and that there is nothing one can do to prevent the occurrence of those events. This hopelessness expectancy is regarded by Abramson et al. as a necessary and sufficient cause of a unique constellation of symptoms with a distinct clinical course and specific treatment implications (i.e., hopelessness depression).

Evolution of the Hopelessness Theory of Depression: Learned Helplessness Theory

Abramson et al.'s (1989) hopelessness model of depression traces its roots to observations made by Seligman and colleagues (e.g., Overmier, 1968, Overmier & Seligman, 1967, Seligman & Maier, 1967) over two decades ago. Seligman and others demonstrated in numerous experimental investigations that organisms exhibit striking behavioral deficits after exposure to uncontrollable events. In a prototypical experiment, Seligman and Maier (1967) initially exposed mongrel dogs to a series of either escapable or inescapable electric shocks. In the former condition, each shock presentation could be terminated if the dogs made a prespecified panel-press response. In the latter condition, however, no response was effective in terminating the shock. Thus, for dogs in this inescapable-shock condition, the shock presentations were uncontrollable (i.e., the probability of shock offset given a response was equal to the probability of shock offset given no response). A no-shock control group was also used in this initial, training phase of the experiment.

In a subsequent testing phase, the experimental dogs were placed in a shuttle-box and again exposed to a series of electric shocks. This time, however, all of the dogs could terminate the shocks by hurdling a barrier that separated the two compartments of the shuttle-box. Dogs in the no-shock control and escapable-shock training conditions quickly learned to hurdle the barrier and escape the shock. In marked contrast, the dogs previously exposed to inescapable shocks exhibited extreme deficits in escape responding. For example, Seligman and Maier (1967) reported that 75% of the dogs in the latter group failed to hurdle the barrier on nine or more of the 10 test trials and took almost twice as long as the other dogs to hurdle the barrier when escape was attempted. Moreover, the dogs initially exposed to inescapable shock responded maladaptively in the testing phase. They became passive and immobile during shock presentations, lying in the corner of the shuttle box while the shocks were being delivered. They even failed to escape the shock after one or two trials in which hurdling the barrier successfully terminated the shock.

Observations such as those described above led to the development of learned helplessness theory (Seligman, 1975; Maier & Seligman, 1976). According to the theory, organisms are capable of integrating contingencies for responding and contingencies for not responding. That is, organisms can simultaneously represent the probability of outcome occurrence given a response and the probability of outcome occurrence given no response. As such, they are able to detect instances in which outcomes are as likely to occur when a response is emitted as when a response is not emitted (i.e., response noncontingency). Seligman

(1975) believed that when organisms perceive outcomes as response-noncontingent they develop an expectation that future outcomes will also occur independently of their responses. He further proposed that this expectation of future response-outcome independence is the "causal condition" (p. 48) for a state of learned helplessness. The latter is characterized by a failure to initiate voluntary responses in an attempt to control future outcomes that truly are controllable (motivational deficit), difficulty perceiving response-outcome contingencies that do exist, (cognitive deficit), and depressed mood (affective deficit)

In explaining the emergence of the motivational deficit among organisms exposed to uncontrollability, Seligman (1975) argued that organisms remain passive in the absence of an incentive to do otherwise. The incentive that underlies voluntary behavior, according to Seligman, is the expectation that such behavior will increase the likelihood of desired outcomes (e.g., obtaining food, terminating shock). Thus, by eliminating the expectation that outcomes are contingent upon responses, experience with uncontrollability eliminates the incentive to behave and produces response deficits. Seligman (1975, p. 50) summarized his reasoning by stating that: "For voluntary responding to occur, an incentive must be present in the form of an expectation that responding may succeed. In the absence of such an expectation, that is, when an organism believes responding is futile, voluntary responding will not occur."

Viewed through learned helplessness theory, the extreme passivity of the dogs in Seligman and Maier's (1967) inescapable-shock condition becomes more understandable. According to the theory, these dogs

learned during the training phase of the experiment that shock offset was response-noncontingent; it occurred independently of their behavior. When presented in the shuttle-box, the dogs expected that shock would again be uncontrollable and therefore made no attempt to either avoid or terminate it. Now devoid of any incentive to attempt escape, the dogs sat passively in the shuttle-box and accepted the shock.

In addition to motivational impairments, Seligman (1975) suggested that helpless organisms suffer cognitive deficits. The cognitive component of helplessness manifests itself as difficulty detecting contingencies that do exist between responses and outcomes. That is, organisms who expect outcomes to be response-noncontingent have difficulty learning that some outcomes are indeed controllable. Seligman accounted for the emergence of this cognitive deficit using the notion of proactive interference. Proactive interference refers to instances in which information learned at one point in time inhibits the acquisition of contradictory information at a later point in time. In the context of helplessness, the representation of response-outcome independence simply interferes with the representation of response-outcome dependence. That is, once organisms learn that outcomes are uncontrollable, their ability to learn the converse--that outcomes can be controlled--is impaired.

Seligman's (1975) insight into the cognitive component of helplessness sheds additional light on the seemingly inexplicable behavior exhibited by the dogs who received inescapable shock in the training phase of Seligman and Maier's (1967) investigation. Recall that in the testing phase of that experiment, some of the dogs previously

exposed to inescapable shock hurdled the shuttle-box barrier and successfully escaped the shock on several initial trials. Unlike the dogs in the escapable- and no-shock training conditions, however, the former did not continue to hurdle the barrier to escape the shock even though that response was effective in terminating the shock. For those dogs, the expectation of response-outcome noncontingency superseded the experience of shock offset upon hurdling the barrier and the contingency went unlearned. In reference to this observation, Seligman (1975, p. 51) noted that:

> When [a dog who initially receives inescapable shock] goes to the shuttle box and jumps the barrier, in reality causing shock termination, the dog has trouble learning this. This is because . . . he still expects that shock will be just as likely to go off if he fails to jump the barrier. Such a dog will revert to taking shock passively even after he makes one or two successful jumps. In contrast, a naive dog has no interfering expectation that shock termination is independent of responding, so one jump over the barrier resulting in shock termination is sufficient for him to catch on.

Finally, Seligman (1975) conceived of learned helplessness as having an affective component. More specifically, he believed organisms experience a state of depression upon learning that important outcomes are uncontrollable. According to Seligman, the initial emotional response to an aversive event is fear. When confronted with such an event, organisms will attempt to exert control over the event in order to reduce the ensuing trauma. If the event can be brought under control, the fear response dissipates and is replaced by effective responding. If, on the other hand, the aversive event can not be brought under the organism's control, the initial fear reaction, because it is effortful and no longer useful, gives way to depression.

Although Seligman (1975) believed that uncontrollable trauma

produced depression, he suggested that depressive reactions could also arise from uncontrollable events that were positive in valence. Seligman based this speculation on his belief that positive affect does not emerge from reinforcement per se, but from effective responding or mastery over the environment. Thus, receiving "free" reinforcement was, according to Seligman, sufficient to engender depression. He states, for example:

> I suggest that what produces self-esteem and a sense of competence, and protects against depression, is not only the absolute quality of experience, but the perception that one's own actions controlled the experience. To the degree that uncontrollable events occur, either traumatic or positive, depression will be predisposed and ego strength undermined. To the degree that controllable events occur, a sense of mastery and resistance to depression will result (p. 99).

This, then, is the model that Seligman (1975) developed to account for the behavior exhibited by organisms exposed to uncontrollable outcomes. Experience with response-noncontingent outcomes fosters an expectation of uncontrollability and the latter brings about deficits in motivation, contingency learning, and affect.

Although Seligman's (1975) formulation of learned helplessness was derived from experimental observations of nonhuman animals, researchers were quick to evaluate the theory's ability to predict human responses to uncontrollability. For example, Hiroto and Seligman (1975) exposed college students to aversive tones that were either controllable or uncontrollable, or to discrimination tasks that were either soluble or insoluble. Half of the subjects in each of these four training conditions were subsequently exposed to aversive tones that could be terminated (or avoided) by moving a manipulandum to the opposite side of a hand shuttle-box. The remaining subjects in each training condition

were asked to solve (soluble) anagrams.

As would be predicted from learned helplessness theory, Hiroto and Seligman (1975) found that subjects who were initially exposed to uncontrollable outcomes performed worse on the test-phase tasks relative to subjects who initially received controllable outcomes. The former solved fewer anagrams and successfully escaped aversive tones on fewer trials than did their counterparts in the controllable training conditions. When they were successful in either solving test-phase anagrams or terminating the aversive tone, subjects in the two uncontrollable training conditions took longer to do so than did subjects in either controllable training condition. Furthermore, performance deficits were not affected by the degree of correspondence between training- and test-phase tasks. For example, subjects who were initially exposed to an insoluble discrimination task performed just as poorly on the noise-escape test task as they did on the anagram test task. This finding, as well as similar results observed among nonhuman animals (e.g., Altenor, Kay, & Richter, 1977; Braud, Wepman, & Russo, 1969; Rosellini & Seligman, 1975), lends support to the suggestion that helplessness expectations generalize to, and produce deficits when dealing with a range of outcomes.

In an analogous investigation, Thornton and Jacobs (1971) first exposed college students to either escapable or inescapable electric shocks and then assessed shock-avoidance/escape responding. The students who first received inescapable shocks failed to either avoid or escape shock on more test trials than did their counterparts who initially experienced escapable shock. Indeed, a full 65% of the

subjects in the former condition failed to make even one escape response across 10 test trials. Moreover, 60% of the subjects in the inescapable-shock condition spontaneously stated in post-experimental interviews that they did not attempt to avoid or escape shock on the test trials because they felt as if they had no control over the shocks and therefore expected responding to be futile.

Noting similarities in symptomatology, etiology, cure, and prevention, Seligman (1975) applied his learned helplessness model to human depression, suggesting that experimentally-induced helplessness is analogous to naturally occurring human depressions. With respect to symptoms, Seligman pointed out that both human depressives and organisms exposed to uncontrollability suffer motivational/motoric impairments. Like helpless organisms who remain passive and immobile instead of initiating responses that could effectively control important outcomes, depressives tend not to engage in even the most effortless activities and are much slower in the behaviors that they do perform.

Seligman additionally argued that depressives exhibit the same types of cognitive deficits that characterize helpless organisms. That is, depressives expect their behavior to be ineffective in altering important outcomes. Moreover, the results of an experiment conducted by Miller and Seligman (1973, cited in Seligman, 1975) suggest that these expectancies are resistant to change in the face of contrary evidence (i.e., experience with response-outcome contingency). Miller and Seligman examined changes in success expectancies among depressed and nondepressed subjects as they worked on tasks involving either skill-determined or chance-determined outcomes. When asked to estimate the

likelihood of success on successive trials, expectancy changes as a function of previous task performance were larger among nondepressed subjects who worked on skill tasks than among nondepressed subjects who worked on tasks of chance. Specifically, nondepressives lowered their success expectancies more after experiencing failure on a skill task than after experiencing failure on a chance task. They likewise increased their success expectancies more after success on the skill task than after success on the chance task.

Among depressed subjects, however, previous performance had as little impact on success expectancies for the skill task as it did on success expectancies for the chance task. That is, depressed subjects in the skill-task condition were no more likely to alter their success expectancies in response to past successes and failures than were depressed subjects in the chance-task condition. Whereas nondepressives appropriately perceived the skill and chance components of the respective tasks, depressed subjects apparently perceived their performance on both types of tasks as chance-determined. In other words, depressives failed to see the relation between their behavior and their task performance when working on the skill task.

Interestingly, Miller and Seligman (1974, cited in Seligman, 1975) found that the pattern of expectancy changes exhibited by nondepressed subjects exposed to inescapable noise paralleled that exhibited by the untreated depressives described above. In contrast, nondepressives exposed to either escapable or no noise behaved like the untreated nondepressives described above. In discussing the implications of the latter findings, Seligman (1975, p. 87) suggested that "These results

show experimentally that both depression as found in the real world and helplessness induced by uncontrollable events result in a negative cognitive set, the belief that success and failure are independent of one's efforts."

A subsequent investigation by Miller and Seligman (1975) demonstrated that laboratory-induced helplessness and naturally occurring depression converge not only with respect to cognitive deficits but also in regard to affective symptoms. In this study, depressed and nondepressed subjects were assigned to either an inescapable-noise, escapable-noise, or no-noise training condition. Subjects were then presented with a series of anagrams, all of which were scrambled according to the same pattern, and asked to solve each anagram within 100 seconds. Post-training performance on the anagrams task was indexed by average latency to solution, number of anagrams left unsolved after 100 seconds, number of trials required for learning the anagram pattern (with the latter defined as the point at which solutions are reached within 15 seconds), and number of anagrams successfully solved before learning the anagram pattern. Finally, Miller and Seligman assessed levels of pre- to post-training change in depressed mood among subjects in each training condition.

Consistent with previous demonstrations of the helplessness phenomena, Miller and Seligman (1975) found that nondepressed subjects who received inescapable-noise performed worse than nondepressed subjects in the escapable- and no-noise conditions on all performance measures. More importantly, however, Miller and Seligman demonstrated that untreated (i.e., no-noise control group) depressives also exhibited

a greater degree of impairment on all performance measures than did nondepressed subjects in the escapable- and no-noise training conditions. Untreated depressives performed just as poorly on the anagrams task as did nondepressives pretreated with inescapable noise.

The effects of inescapable noise were not, however, limited to performance deficits on the anagrams task. Among initially nondepressed subjects, exposure to inescapable noise resulted in larger increases in post-training levels of depressed mood than did exposure to escapable or no noise. Although post-training anxiety and hostility levels also increased among nondepressives exposed to inescapable-noise, they did so to a lesser extent than depressed mood. Among initially depressed subjects, however, exposure to inescapable noise had no such effect on post-treatment changes in levels of depressed mood. Finally, subjects' self-reported depression levels were highly positively correlated with the degree of task impairment on the four performance measures (rs ranged from .69 to .86). Taken together, the results of Miller and Seligman's research program provide evidence of substantial overlap between naturally occurring depression and laboratory-based helplessness.

Guided by his laboratory observations, Seligman (1975) advanced a theory of human reactive depression in which experience with uncontrollability served a central etiological role. According to Seligman's learned helplessness model of depression, individuals become depressed when they learn that important outcomes, either positive or negative in valence, are noncontingently related to their actions. Seligman believed that such experience with response-outcome

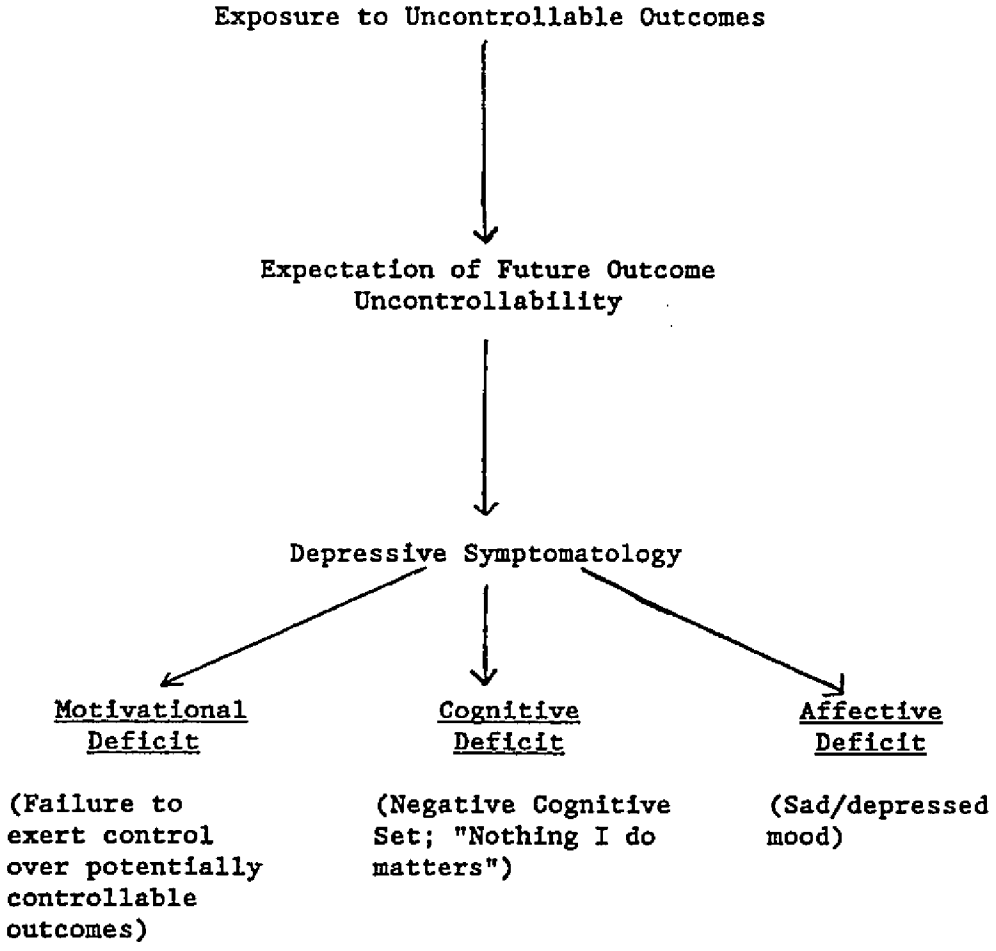
noncontingency gives rise to an expectation that future outcomes will also be uncontrollable. The syndrome of depression, he argued, is the result of this future uncontrollability expectation, and consists of the motivational, cognitive, and affective deficits characterizing the state of learned helplessness (see Figure 1). The motivational deficit manifests itself in human depression as psychomotor retardation as well as cognitive and social dullness; the cognitive deficit manifests itself as a "negative cognitive set," the depressive's belief that all actions are futile (e.g., "nothing I do matters"); and the affective deficit is expressed as sad or depressed mood.

Attributional Reformulation of Learned Helplessness Theory

Shortly after its development, researchers began to recognize that Seligman's (1975) theory of learned helplessness was unable to adequately account for the course and characteristics of helplessness deficits. Instances in which organisms failed to become helpless after exposure to uncontrollability were attributed post hoc to previous (but unknown) experiences with control that served to immunize those organisms from helplessness (Abramson et al., 1978; Seligman, 1975, 1991). Furthermore, the original theory had difficulty explaining individual differences in the nature of helplessness deficits that did occur following experience with response-noncontingent outcomes.

To remedy these explanatory problems, an attributional reformulation of the learned helplessness model was proposed by Abramson et al. (1978). The reformulation allowed researchers to predict, a priori, 1.) under what conditions exposure to uncontrollable outcomes would give rise to an expectation of future outcome uncontrollability

Figure 1. Learned Helplessness Model of Depression
(Adapted from Seligman, 1975)



(and thus to learned helplessness deficits), 2.) whether the future uncontrollability expectancy would be relatively short-lived or relatively long-lasting, 3.) whether the future uncontrollability expectancy would remain specific to the outcome experienced as uncontrollable or generalize to a variety of other outcomes, and 4.) whether lowered self-esteem would accompany the other helplessness deficits. In addition, a revised theory of human depression, based on the attributional reformulation of learned helplessness theory, was advanced.

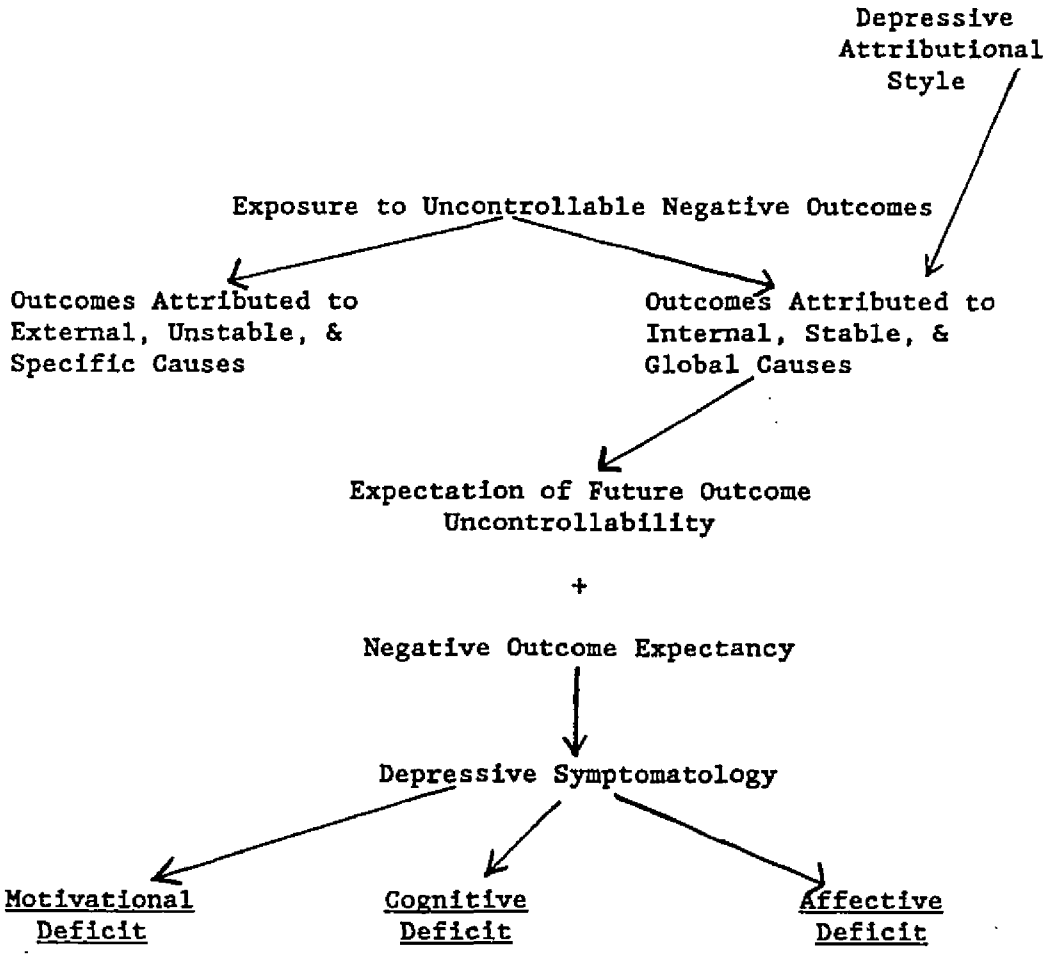
As in the original learned helplessness theory, Abramson et al. (1978) maintained that an expectation of future uncontrollability directly precipitates the motivational, cognitive, and affective helplessness deficits. In contrast to Seligman's (1975) suggestion, however, perceived response-outcome noncontingency was no longer regarded as a sufficient condition for the development of this future uncontrollability expectation. According to the reformulation, the perception of response-outcome noncontingency motivates an attributional search whereby individuals attempt to determine the cause of the uncontrollability. It is the causal attributions that individuals make for uncontrollable outcomes, not the outcomes themselves, that lead to the future uncontrollability expectation. Specifically, the reformulation suggested that individuals develop future uncontrollability expectations only when they attribute response-noncontingent outcomes to causes that are internal (something about themselves), stable (long-lasting), and global (affecting many other outcomes). When response-noncontingent outcomes are attributed to

external (something about others or the environment), unstable (short-lived), and specific (affecting only a narrow range of outcomes) causes, expectations of future uncontrollability, and consequent learned helplessness deficits are not predicted to arise (see Figure 2).

The reformulated theory also suggested that the temporal and situational parameters of the future uncontrollability expectation, as well as the occurrence of self-esteem deficits, could be accounted for by the nature of the causal attributions made for uncontrollable outcomes.[1] Abramson et al. (1978) hypothesized that three orthogonal dimensions of causal attributions--internal/ external, stable/unstable, and global/specific--influence the future uncontrollability expectation and thus, the nature of helplessness deficits.

The internal-external dimension reflects the extent to which the causes of response-noncontingent outcomes are believed to reflect something about the self or something about the environment, respectively. Internal causes for failing an academic task despite studying might include low academic ability or poor study habits. External causes for the same outcome might include the difficulty of the task and poor academic instruction. According to Abramson et al. (1978), attributing uncontrollable negative outcomes to internal causes results in a state of personal helplessness. Personally helpless individuals expect that they will be unable to control the future occurrence of negative events but believe that others possess the responses that can control those events. For example, a student who believes that her academic failure was caused by low ability might expect that she will be unable to attain future academic success but

Figure 2. Reformulated Learned Helplessness Model of Depression
Abramson, Seligman, & Teasdale (1978)



Unique Roles Assigned To Attributional Dimensions

1. Internality ---> Self esteem loss will accompany motivational, cognitive, and affective deficits

2. Stability ---> Chronicity of future uncontrollability expectation and thus duration of depressive symptoms

3. Globality ---> Generality of future uncontrollability expectation (i.e., range of outcomes over which a lack of control is expected) and thus pervasiveness of depressive symptoms

that her fellow classmates will be able to do so.

Individuals who attribute noncontingency to external causes, on the other hand, are believed to experience a state of universal helplessness characterized by the expectation that neither they nor similar others are able to control the occurrence of future negative outcomes. A student who believes that her academic failure was caused by poor instruction might expect that both she and her fellow classmates will be unable to succeed on future academic tasks in the same class.

Given that the expectation of future uncontrollability is present among personally and universally helpless individuals, both are predicted to experience the motivational and cognitive deficits of learned helplessness. Because they believe that the causes of aversive uncontrollability reflect something about themselves, however, only personally helpless individuals should experience self-esteem loss in addition to the other deficits. According to the reformulation, then, both the personally helpless and universally helpless students mentioned above might stop trying to control future academic outcomes (motivational deficit) and erroneously perceive a lack of control over other academic outcomes (cognitive deficit). Only the personally helpless student, however, should experience lowered self-esteem in response to the noncontingent academic outcome.

The stable-unstable attributional dimension represents the extent to which causes are perceived, respectively, as enduring or transitory. Stable attributions for the uncontrollable academic outcome mentioned above might include low aptitude for that academic subject or chronic unfairness on the part of the instructor. Unstable attributions might

include a temporary illness or an unusually heavy work load. According to the reformulation, the temporal persistence or chronicity of future uncontrollability expectations is dependent on the perceived stability of the causes attributed to uncontrollable negative outcomes.

Expectations of future noncontingency (and the resulting helplessness deficits) are believed to persist for an extended period of time when uncontrollable outcomes are attributed to stable causes, but to be short-lived when outcomes are attributed to unstable causes. When a student attributes an uncontrollable academic outcome to a stable cause such as low aptitude, it suggests that she will also be unable to control similar academic outcomes that occur well into the future. That student should thus remain helpless as long as she expects those outcomes to be uncontrollable. On the other hand, a student who attributes an uncontrollable academic outcome to an unstable cause should not expect that cause to render similar outcomes uncontrollable long into the future. The future uncontrollability expectation, and resulting helplessness deficits should thus dissipate quickly.

The third dimension of causal attributions discussed by Abramson et al. (1978) is the global-specific dimension. Global causes are those that affect a wide variety of outcomes whereas specific causes affect only a limited array of outcomes. Continuing with the example of academic failure, global causes might include low general intelligence or incompetent faculty at a particular institution. An inability to understand the particular academic task or the instructor's inability to explain the concepts relevant to that particular task reflect specific causes.

According to the reformulated theory, the perceived globality of causes influences the generality of helplessness deficits or the range of outcomes over which an individual expects to lack future control. A student who attributes an uncontrollable negative outcome to a global cause might come to expect that the many outcomes affected by that cause will also be uncontrollable in the future. The helplessness deficits exhibited by that student should thus be highly generalized and manifest themselves in a wide variety of situations. For example, a global cause such as low general intelligence might be expected to adversely affect a variety of outcomes including one's performance in many different academic subjects or one's occupational success. A student who makes such an attribution after experiencing an uncontrollable academic failure might then become helpless in her other classes and/or give up the lofty career goals to which she once aspired.

Unlike global attributions, specific causes render only a limited array of outcomes uncontrollable. Being unable to understand a particular task or concept, for example, has little relevance to tasks requiring other skills. An individual who makes such an attribution might expect to lack control over only future tasks requiring the unattainable skill. That individual should not, however, expect other unrelated outcomes to also be uncontrollable.

The reformulation also made predictions about the severity of helplessness deficits and the extent of self-esteem loss among personally helpless individuals. Abramson et al. (1978) suggested that the motivational and cognitive deficits of helplessness increase in severity as the expectation of future uncontrollability increases in

certainty. The severity of affective deficits and the intensity of self-esteem loss was hypothesized to be a function of both the certainty of the future uncontrollability expectation and the importance of the outcome over which a lack of control is expected. Individuals who are highly certain that an outcome will be uncontrollable should thus exhibit more extreme passivity and impairment in learning when outcomes truly are controllable. When that certainty concerns a highly important outcome (e.g., the ability to obtain a desired job), the intensity of depressive affect and magnitude of self-esteem loss (if an internal causal attribution is made) should also increase.

To reiterate, the reformulated theory is an attributional model of learned helplessness that makes specific predictions about the occurrence and nature of helplessness deficits arising from perceived noncontingency. According to the reformulation, self-esteem loss accompanies helplessness deficits only among personally helpless individuals who attribute noncontingency to internal causes. The chronicity and generality of helplessness deficits are influenced, respectively, by the perceived stability and globality of the causes of noncontingent outcomes. Finally, the strength of the future uncontrollability expectation and the importance of the outcome(s) expected to be uncontrollable influence the intensity of helplessness deficits.

The developers of the reformulated model also addressed the important question of when noncontingent outcomes are likely to be attributed to internal, stable, and/or global causes. They suggested that both "bottom-up" and "top-down" processes influence beliefs about

the causes of uncontrollable outcomes. With respect to the former, Abramson et al. (1978) followed the lead of other attribution theorists (e.g., Kelley, 1967) in noting that causal attributions for specific outcomes are often derived from situational information (e.g., the nature of the outcome, the contexts in which it occurs, whether others also experience it). For example, a student who continually fails exams that the majority of her classmates pass is likely to make an internal attribution for those failures. But Abramson et al. also speculated that causes might be attributed to outcomes in a "top-down" fashion. That is, people's generalized styles of attributing causality to outcomes might shape their perceptions of the causes of specific outcomes. Abramson et al. further proposed that some people possess a "depressogenic attributional style," or a general tendency to attribute a wide variety of negative outcomes to internal, stable, and global causes. Individuals who possess this depressogenic attributional style are thought to be prone to helplessness deficits and depressive reactions when noncontingent negative outcomes occur.

Abramson et al. (1978) also revised Seligman's (1975) original helplessness model of human depression. According to the reformulated theory, "helplessness depression" is a subtype of depression comprised of motivational, cognitive, affective, and self-esteem deficits. In contrast to Seligman's (1975) proposal that each of these deficits follow directly from the expectation of future uncontrollability, Abramson et al. hypothesized that the affective deficits of depression arise only when the expectancy of future uncontrollability co-occurs with a negative outcome expectancy. That is, people experience sadness

or depressed mood only when they anticipate either being unable to bring about highly desired outcomes or being unable to prevent the occurrence of highly undesired outcomes. Passivity and a negative cognitive set, however, arise when future outcomes of any valence are perceived as uncontrollable.

Abramson et al. (1978) also noted that the original helplessness model of depression could not explain the low self-esteem frequently observed among depressives, nor could it account for variability in the time course or generality of depressive symptoms. In line with their attributional reformulation of learned helplessness, Abramson et al. suggested self-derogation and low self-worth should be exhibited by depressives who attribute their helplessness to internal causes. Likewise, variability in the duration and generality of depressive symptoms was accounted for by individual differences in the perceived stability and globality of the causes of one's helplessness, respectively.

Abramson et al.'s (1978) reformulated model of helplessness depression thus restricted the affective component of human depression to expectations regarding the uncontrollable occurrence of negative outcomes (or nonoccurrence of positive outcomes), provided an account of the depressive's low self-esteem, and explained individual differences in the chronicity and generality of depressive symptomatology. Finally, it is important also to note that the reformulation regarded a negativistic attributional style as a vulnerability factor for depression. Abramson et al. suggested that people who generally attribute negative outcomes to internal, stable, and global causes

possess a "depressive personality," and are thus vulnerable to depressive reactions in response to uncontrollable stressors (see also Abramson et al., 1989; Metalsky et al., 1982).

Hopelessness Theory: A Revision and Extension of the Attributional Reformulation of Helplessness Depression

Since its appearance in the literature, the reformulated model of helplessness depression has been the subject of an extraordinary amount of research, not all of which has been supportive (for reviews see Brewin, 1985; Coyne & Gotlib, 1983; Peterson, Villanova, & Raps, 1985; Robins, 1988; Sweeney, Anderson, & Bailey, 1986). In light of the accumulating evidence, and in response to various critiques of the reformulated model, Abramson et al. (1989) recently revised and extended their 1978 statement of the model. Abramson et al. refer to this revision as the hopelessness theory of depression. The fundamental postulate of hopelessness theory is that "hopelessness depression" represents a subtype of depression that is distinguished primarily by its etiology, but which is also unique in symptomatology, clinical course, and treatment/ prevention implications.

With respect to its etiology, Abramson et al. (1989) proposed that the formation of a hopelessness expectancy is the most proximal cause of the symptoms of hopelessness depression. The hopelessness expectancy is comprised of two necessary components: a negative outcome expectancy and a helplessness expectancy. The former refers to the belief that either highly undesired outcomes are likely to occur in the future or that highly desired outcomes are unlikely to occur. The latter reflects the belief that the occurrence of those outcomes can not be controlled. In

essence, people feel hopeless when they expect their futures to be bleak and believe that there is nothing they can do to change that pessimistic forecast.

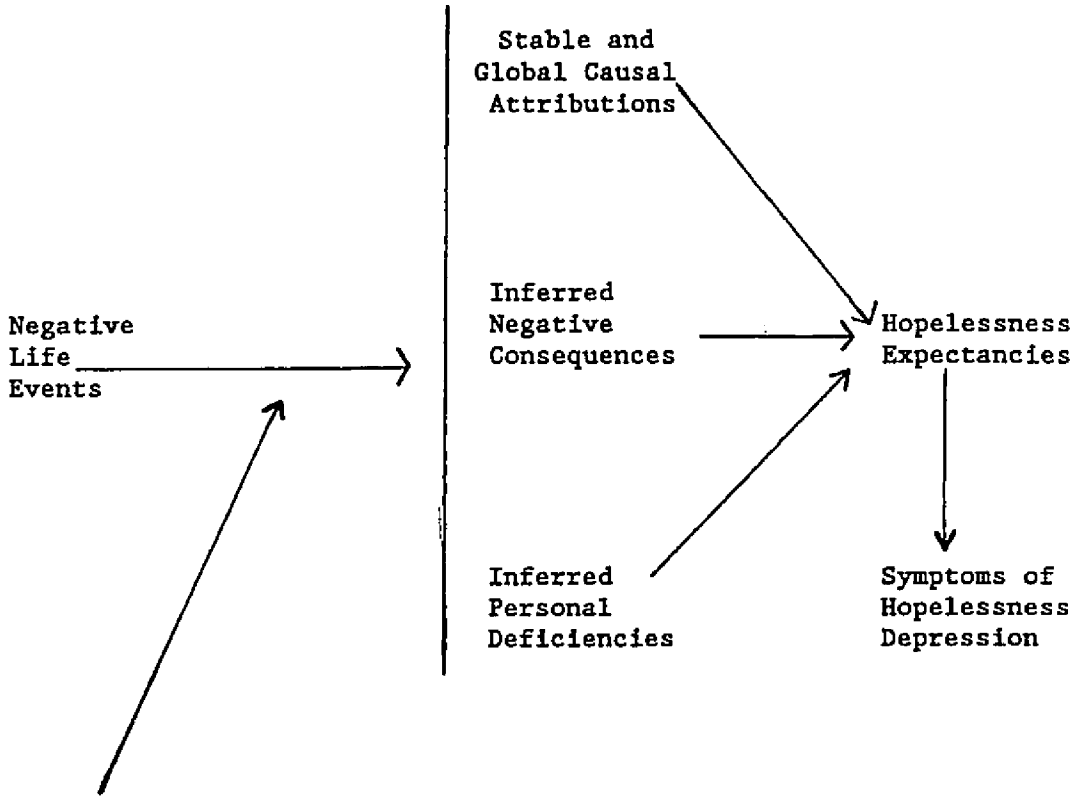
The formal theoretical status of the hopelessness expectancy is that of a necessary and sufficient proximal cause of hopelessness depression. It is a sufficient cause because, according to the model, the presence of a hopelessness expectancy guarantees the onset of depressive symptoms. A hopelessness expectancy is a necessary cause of hopelessness depression because it defines that subtype of depression. Of course, Abramson et al. do not suggest that hopelessness is a necessary cause of all depressive disorders or symptoms. Finally, hopelessness is conceived as a proximal cause because its occurrence directly precedes the onset of depressive symptoms.

In addition to specifying hopelessness as the proximal cause of depressive symptoms, Abramson et al. (1989) outlined an etiological pathway by which hopelessness expectancies are believed to develop (see Figure 3). That causal pathway describes a series of sequentially occurring events, each of which contributes to, but is neither necessary nor sufficient for the formation of helplessness expectancies. The events along the proposed causal chain are therefore formally regarded as contributory causes of hopelessness depression.

The causal sequence leading up to the hopelessness expectancy is initiated by the perceived occurrence of one or more negative or stressful life events. As mentioned above, negative life events are contributory causes and are not by themselves sufficient for a hopelessness expectancy to occur. According to the hopelessness model,

Figure 3. Causal chain Specified in the Hopelessness Model of Depression

(Adapted from Abramson, Metalsky, and Alloy (1989))



depressogenic inferences drawn from negative life events moderate the relation between those events and hopelessness. Whether the occurrence of a stressful event results in a hopelessness expectancy depends on the inferences an individual draws about: 1.) the cause of the event, 2.) the consequences resulting from the event, or 3.) the characteristics of the self given the event's occurrence. Note that in contrast to the reformulated model, the depressogenic inferences specified in hopelessness theory include, but are no longer limited to, causal attributions.

Although causal attributions are not accorded exclusive status, Abramson et al. (1989) do suggest that attributing negative events to stable and global causes can give rise to hopelessness expectancies. Hopelessness theory further speculates that stable and global attributions for negative events contribute to hopelessness expectancies only when those events are regarded as important. Finally, internal attributions for negative events play no role in the development of hopelessness expectancies according to the current model.

Abramson et al. (1989) suggest that regardless of how causality is attributed to negative events, people might also become hopeless if they expect those events to bring about a variety of undesired consequences. Thus an individual might believe that she performed poorly on an important job assignment because of inadequate preparation (an unstable and specific causal attribution), but still become hopeless if the poor performance is expected to undermine her ability to secure a desired promotion. In addition, hopelessness is thought to be more likely to occur when the expected negative consequences are regarded as highly

important, unchangeable, and as affecting many aspects of one's life.

The final moderating variable included in the hopelessness model concerns the inferences an individual draws about herself upon experiencing a negative life event. Hopelessness is predicted to result when negative events adversely impact an individual's self-conception. In other words, hopelessness arises when negative events are perceived as diagnostic of personal deficiencies such as being unworthy, unlovable, unintelligent, or incapable. As with inferred negative consequences, the development of hopelessness expectancies is believed to be more probable when the negative characteristics ascribed to the self are perceived as highly incapacitating (i.e., preventing one from attaining valued goals).

Although they are conceived as orthogonal causes, Abramson et al. (1989) acknowledge that it may be difficult to distinguish inferred negative self-characteristics from causal attributions. For example, the inference, "I failed the exam, therefore I must be unintelligent," includes the ascription of a negative trait to the self as well as an implicit internal attribution for the cause of the failure. Despite their cautionary comments, however, the difference between internal attributions (which currently have no causal role in the development of hopelessness) and inferred negative traits is unclear, and hopelessness theory currently offers no adequate resolution to this discrepancy.

The three classes of inferences discussed above are regarded as proximal contributory causes of hopelessness depression because their occurrence in the causal chain closely precedes the development of hopelessness expectancies (which then produce the symptoms of

hopelessness depression). However, the causal pathway proposed by Abramson et al. (1989) also incorporates more remote (i.e., distal) causal factors that contribute indirectly to the formation of hopelessness expectancies. These distal contributory causes, which are referred to as "depressogenic cognitive styles," represent generalized thought patterns or styles of perceiving the environment that are believed to render individuals vulnerable to depressive symptomatology.

Abramson et al. (1989) suggested that individuals who chronically attribute negative events to stable and global causes, habitually anticipate negative consequences, or infer personal deficiencies when a variety of negative events occur, are at risk of becoming hopeless in the face of life stressors. Depressogenic cognitive styles are considered risk factors for hopelessness because the individuals who exhibit them are likely to make corresponding inferences when specific negative life events occur (i.e., attribute specific stressors to stable and global causes, expect negative consequences to result from those stressors, or infer derogatory characteristics about the self when those stressors occur).

Hypotheses regarding the relation between attributional style and depressive symptomatology are, of course, not new to hopelessness theory. Such a relation was initially suggested by the reformulators of helplessness theory (Abramson et al., 1978), elaborated in subsequent theoretical statements (e.g., Peterson & Seligman, 1984; Seligman, Abramson, Semmel, & von Baeyer, 1979), and empirically validated by helplessness researchers (for a review, see Sweeney et al., 1986). Like causal attributions for specific events, however, the role of

attributional style in putting individuals at risk for depression is de-emphasized as other inferential styles are incorporated into the current model. In addition, hopelessness theory considers only the tendency to make stable and global attributions for negative events to be a risk factor for depressive symptoms. In contrast to previous statements, the tendency to attribute negative events to internal causes is no longer regarded as a risk factor.

The hopelessness model also differs from its theoretical predecessor in postulating a "specific vulnerability hypothesis" with regard to attributional style. The specific vulnerability hypothesis holds that depressogenic attributional styles are domain-specific and therefore increase the likelihood of depressive symptomatology only when vulnerable individuals encounter stressors in a corresponding domain. Specifically, Abramson et al. (1989) suggest that individuals habitually make stable and global attributions for either negative achievement outcomes or negative interpersonal outcomes. The former group should then be prone to depression when they encounter stressors in the achievement domain, but not when they experience stressful outcomes of an interpersonal or social nature. The converse is true of individuals whose depressogenic attributional style is limited to interpersonal outcomes.

Abramson et al.'s (1989) notion of domain specific attributional styles is similar to Beck's (e.g., Beck, 1983) suggestions regarding sociotropic and autonomous personality styles. The former refers to tendencies toward socially dependency and is believed to render individuals vulnerable to depression when loss or disruption of social

relationships is experienced. The latter is characterized by excessive achievement striving and independence needs. An autonomous personality style is believed to increase the likelihood of depressive reactions in the face of events that threaten one's goal attainment.

Relations between the aforementioned depressogenic cognitive styles and hopelessness expectancies are thought to be moderated by the occurrence of negative life events and the inferences drawn from those events. As such, hopelessness theory is regarded as a diathesis-stress model of depression. Depressogenic cognitive styles serve as "cognitive diatheses" that increase the likelihood of depression only in the presence of life stress. In the absence of negative life events (or in the presence of positive life events), individuals who exhibit the hypothesized inferential styles should be no more hopeless or depressed than individuals who do not exhibit those cognitive styles.

A note about the proposed symptoms and clinical course of hopelessness depression is also in order. The constellation of symptoms believed to characterize hopelessness depression includes the motivational and affective deficits originally discussed by Seligman (1975) and subsequently retained in the (1978) reformulation. According to the current statement, psychomotor decrements stem from the helplessness component of the hopelessness expectancy. Depressed or sad mood, on the other hand, stems from the negative outcome expectancies accompanying hopelessness. The cognitive deficit (i.e., negative cognitive set) discussed in the original and reformulated models is no longer included in the symptom constellation comprising hopelessness depression. According to Abramson et al. (1978), its exclusion is based

on evidence suggesting that depressives do not erroneously underestimate the degree of control they have over outcomes. Rather, nondepressives appear to distort optimistically their degree of control, perceiving personal control over outcomes when none exists in reality (cf. Alloy & Abramson, 1979, 1988; Taylor & Brown, 1988).

Abramson et al. (1989) suggest that hopelessness depression might also be characterized by rumination and consequent disturbances in concentration and sleep. Furthermore, research by Beck (e.g., Beck, Kovacs, & Weismann, 1975) has shown that hopelessness expectancies are strong predictors of suicidal ideation and suicide attempts. As such, the latter are also considered likely concomitants of hopelessness depression.

Finally, Abramson et al. (1988) make predictions as to when self-esteem loss will be present amid the other symptoms of hopelessness depression. In contrast to the reformulation which held that self-esteem deficits stem exclusively from internal attributions, hopelessness theory predicts that internal attributions lead to self-esteem loss only when they are combined with stable and global attributions. Self-esteem deficits are not expected to occur when negative outcomes (e.g., academic failure) are attributed to causes perceived as internal, unstable, and specific (e.g., lack of adequate preparation). An alternative route to self-esteem reduction suggested by hopelessness theory is the ascription of negative characteristics to the self in response to the occurrence of a negative life event (see above discussion of contributory causes of hopelessness).

Hopelessness depression is believed also to have a distinct

clinical course. Briefly, Abramson et al. (1989) speculate that the continued presence of hopelessness expectancies predicts the duration (i.e., chronicity) of a given depressive episode. In turn, an individual's perceptions regarding the stability of event causes, inferred consequences, or inferred self-deficiencies might contribute to the maintenance of hopelessness expectancies.

Differences Between Hopelessness Theory and the Reformulated Model of Helplessness Depression

Several differences between the hopelessness model and the reformulated model of helplessness depression were mentioned in the preceding discussion. Because of their importance to the present research, the differences between the two models are elaborated and more completely discussed below.

First, and perhaps most striking, Abramson et al. (1989) eliminated perceived uncontrollability from the etiological sequence leading to hopelessness depression. Hopelessness theory does not require negative events to be perceived as uncontrollable in order for hopelessness expectancies and depressive symptoms to emerge. As discussed above, a perceived lack of control over negative outcomes was central to both Seligman's (1975) original theory of learned helplessness and to the 1978 reformulated theory of helplessness. According to Seligman, exposure to uncontrollable outcomes directly precipitates helplessness expectancies. In the reformulation, exposure to uncontrollable negative outcomes brings about helplessness expectancies only when those outcomes are attributed to internal, stable, and global causes.

According to hopelessness theory, perceiving negative events as uncontrollable is no longer a necessary prerequisite for the formation of hopelessness expectancies. Abramson et al. (1989) specifically assert that,

> In contrast to the 1978 reformulation (but consistent with later statements such as those of Peterson & Seligman, 1984), we begin the etiological chain with the perceived occurrence of a negative life event, rather than an uncontrollable event, because the logic of the hopelessness theory requires only the occurrence of the former, rather than the latter, to initiate the series of causes hypothesized to culminate in hopelessness and, in turn, the symptoms of hopelessness depression ... (p. 360).

Other than the above statement, Abramson et al. (1989) offer no empirical or theoretical rationale for hopelessness theory's elimination of perceived uncontrollability (nor did Peterson and Seligman, 1984). It appears to be based only on the assumption that stressful life events, combined with the hypothesized depressogenic inferences, are sufficient conditions for the development of hopelessness expectancies, regardless of the perceived controllability of those events.

Contrary to Abramson et al.'s (1989) recent assumptions, however, the results of recent investigations demonstrate the importance of perceived uncontrollability to the onset of depressive symptomatology. Several researchers (Benassi, Sweeney, & Asdigian, 1990; Brown & Siegel, 1988; Pagel, Becker, & Coppel, 1985) have shown that individuals who attribute negative events to internal, stable and/or global causes exhibit elevated levels of depressive symptoms only when those events are perceived as relatively uncontrollable. Attributions for negative events perceived as controllable were not associated with elevated levels of depressive symptomatology. These results suggest that Abramson et al.'s removal of perceived uncontrollability from the

hopelessness model is unwarranted. Instead, perceiving a lack of control over negative events might indeed be a necessary precursor of hopelessness expectancies, and thus to the symptoms associated with hopelessness depression.

Whereas perceived uncontrollability has been removed, other variables not previously included in the reformulation have been incorporated into the hopelessness model. Unlike the reformulation, the current model no longer regards causal attributions as the sole predictors of hopelessness expectancies, nor does it propose that attributional style is exclusive in rendering individuals vulnerable to depressive symptoms.

With respect to the former, the current statement suggests that expectations of negative consequences and inferred personal deficiencies, in addition to stable and global causal attributions for negative life events, might also lead to hopelessness expectancies. Numerous investigations have demonstrated a relation between depressive symptoms and stable and global causal attributions for negative events (e.g., Benassi et al., 1990; Brown & Siegel, 1988; Follete & Jacobson, 1987; Metalsky et al., 1987, but see above for conditions under which this relation is not obtained). There is little evidence available, however, with which to evaluate the latter two predictions.

In support of the moderating effects of inferred negative consequences, Abramson et al. (1989) cite several investigations conducted by Hammen and associates (i.e., Gong-Guy & Hammen, 1980, Hammen & Cochran, 1981) which purport to demonstrate a relation between expectations of negative consequences and depressive symptomatology. A

close examination of the measures used in those studies, however, suggests that inferred negative consequences might not have been adequately assessed. For example, Gong-Guy and Hammen (1980) and Hammen and Cochran (1981) found that, relative to nondepressives, depressed subjects perceived recent life stressors as causing a greater degree of uncertainty in their lives. Based only on subjects' responses to this measure, the authors suggested that cognitions about the consequences of stressful life events play a role in the onset of depressive symptoms.

This conclusion appears premature given that the single-item measure of life uncertainty used in each study might not adequately reflect expectations concerning the negative consequences of stressful life experiences. Moreover, the cross-sectional nature of these studies leaves open the possibility that perceived life uncertainty is a consequence rather than an antecedent of depressive symptomatology. Although suggestive, the findings obtained by Hammen and colleagues do not provide unequivocal support for the hypothesized effects of inferred negative consequences. A more definitive test of this component of hopelessness theory is thus required.

The model's predictions regarding the etiological role of inferred negative self-characteristics are likewise in need of evaluation. As the basis for these predictions, Abramson et al. (1989) cite clinical evidence that depressed patients engage in self-derogation and infer personal deficiencies when they experience negative events (e.g., inferring that one is worthless upon the termination of an important relationship, p. 361). From this evidence, of course, it is impossible to determine whether such inferences serve as causes or consequences of

depressed affect. Abramson et al.'s notion of inferred negative self-characteristics appears conceptually similar to the notion of self-blame. Although self-blame is believed by some to be a symptom of depression (Beck, 1967), the precise role of self-blame in depression remains equivocal (cf. Shaver & Drown, 1986). The ability of inferred personal deficiencies to predict future depressive affect (or more specifically, the development of hopelessness expectancies) needs to be established in order for the model's predictions to be supported.

The diathesis-stress component of the hopelessness model has also been expanded to include cognitive styles other than the depressogenic attributional style as vulnerability factors for depression. The predictions that Abramson et al. (1989) do make about attributional style, however, are similar to those advanced by the reformulation (except for hopelessness theory's exclusion of the internality dimension as a risk factor). As discussed above, hopelessness theory predicts that stable and global attributions for specific negative events are more probable among individuals who are predisposed to attribute negative events to stable and global causes. As such, this subset of individuals is at greater risk of becoming hopeless and therefore depressed when specific negative events are encountered.

Abramson et al. (1989) additionally suggest that some individuals might habitually infer either negative consequences or personal deficiencies in response to a variety of negative events. According to hopelessness theory, these individuals are likely to make similar inferences in response to specific negative events and are therefore at risk of becoming hopeless. Abramson et al. liken these inferential

styles to Beck's (Beck, Rush, Shaw, & Emery, 1979) notion of depressogenic assumptions and to Ellis' (1977) notion of irrational beliefs, both of which are described below.

According to Beck et al. (1979), depressogenic assumptions reflect deeply ingrained, maladaptive beliefs that predispose individuals to depression (p. 244). These beliefs primarily take the form of "personal contracts" or contingencies specifying the conditions required for happiness and perceived self-worth. Beck et al. (1979, p. 246) offered examples of the types of dysfunctional beliefs that foster depressive reactions, including the following: "In order to be happy, I have to be successful in whatever I do"; "To be happy, I must be accepted by all people at all times"; and "If I make a mistake, it means that I am inept." Note that each of the above statements implies that one's value depends on prespecified external factors (e.g., being successful in one's endeavors, being regarded highly by others) (cf. Olinger, Kuiper, & Shaw, 1987).

Beck argued that the above beliefs, and others like them, lay the groundwork for depressive affect because they require excessively demanding conditions that frequently can not be met. Furthermore, they are vaguely defined (e.g., "success") and therefore might never be fully satisfied. When those conditions are not met (e.g., when failure or rejection is encountered), the individual's sense of self-worth or personal security is severely threatened and extreme reactions of depressed affect are likely to result.

Although not explicitly framed in terms of a diathesis-stress model, Beck's cognitive theory of depression clearly conforms to such a

model (cf. Abramson et al., 1990). For example, Beck et al. (1979, p. 270) suggest that dysfunctional beliefs by themselves do not lead to negative affect. Individuals who adopt such beliefs function quite well when they encounter acceptance, love, success, etc.; they are at risk for depression only when those factors are absent or are removed. Moreover, Beck et al. (1979, p. 16) do not regard life stressors by themselves to be sufficient for extreme depressive reactions. Such reactions are expected only among individuals who interpret life stressors through negativistic or depressogenic schemas. Thus, Beck's conception of depressogenic assumptions as vulnerability factors for depressive symptomatology provides a theoretical basis for including such beliefs among the other cognitive diatheses specified by hopelessness theory.

Beck et al. (1979) additionally suggested that depressogenic assumptions underlie various information processing errors that serve to distort the depressive's interpretations of her experiences. The errors outlined by Beck include such things as: 1.) drawing extreme conclusions based on limited information (i.e., overgeneralization), 2.) magnifying the significance of negative events while minimizing that of positive events, 3.) thinking in absolutist and dichotomous terms (e.g., people are either perfect or a failure), 4.) expecting severe consequences when bad things happen (i.e., catastrophizing), 5.) accepting excessive personal responsibility for negative events (i.e., personal causality), 6.) inappropriately expecting the persistence of negative events (i.e., temporal causality), and 7.) making excessively punitive and moralistic judgments about the self. These illogical thought patterns are believed

to follow from the inappropriate contingencies dictated by the depressive's maladaptive beliefs. For example, a person who believes that making mistakes renders one inept is likely to infer, upon making a mistake, that she is indeed inept or completely incapable, and therefore will never be able function competently at important tasks or achieve desired goals. These interpretive distortions contribute to the negative cognitive triad, which according to Beck, directly precipitates depressive symptomatology.

The cognitive errors that Beck ascribes to depressives appear similar to the negativistic inferences that contribute to hopelessness expectancies in Abramson et al.'s (1989) model. For example, "catastrophizing" is analogous to expecting negative consequences to result from specific negative events. Likewise, harsh moralistic and punitive self-judgments are analogous to inferring personal deficiencies from negative life events. Furthermore, Beck's conception of the relationship between depressogenic assumptions and cognitive errors is similar to (and provides theoretical support for) Abramson et al.'s suggestion that individuals who hold dysfunctional assumptions are likely to make the depressogenic inferences that contribute to hopelessness.

As with depressogenic assumptions, Abramson et al. (1989) suggest that irrational beliefs (Ellis, 1977) are similar to the dysfunctional inferential styles that serve as vulnerability factors in the hopelessness model. The notion of irrational beliefs is at the heart of Ellis' Rational-Emotive Therapy (R-ET) as well as the theory of psychopathology upon which R-ET is based. Ellis (1977) contends that

illogical beliefs or thoughts about negative environmental events, and not the events themselves, are the primary causes of emotional disturbance (including, but not limited to, severe depressive affect).

Ellis (1977, p. 10) suggested that most irrational beliefs can be subsumed under the following categories 1.) unrealistic and absolutist demands regarding what the self, others, and world "should" and/or "must" be like; 2.) "awfulizing" beliefs regarding how terrible it is when people (including the self) or conditions deviate from what they should or must be like; 3.) beliefs regarding one's inability to tolerate conditions that deviate from what they should or must be like (i.e., "i-can't-stand-its"); and 4.) beliefs regarding how terrible people (including oneself) are when they fail to behave as they should or must behave. Ellis' (1977) conception of the nature and emotional consequences of irrational beliefs lends support to Abramson et al.'s (1989) contention that such beliefs predispose individuals to depressive affect. To a large extent, Ellis believes that irrational thinking reflects tendencies to overgeneralize from the data at hand and negatively distort objective realities. Accordingly, he suggests that many irrational thoughts involve, "unempirical or unrealistic statements," and "irrational or illogical conclusions from limited data" (p. 8). Irrational thinking might lead a person to conclude, for example, that because certain environmental conditions are currently undesirable, they will <u>always be</u> undesirable, or that because another person has behaved badly, s/he <u>is</u> a bad person. Irrational thinkers place overly stringent demands on themselves, others, and their environments. They overestimate or overgeneralize the "awfulness" of

negative events as well as the "awfulness" of people (including themselves) who do not live up to their lofty standards. Individuals who advocate irrational beliefs should therefore be highly likely to expect negative consequences and/or infer personal deficiencies when they encounter life stressors, as is suggested by hopelessness theory.

An additional difference between the reformulation and hopelessness theory concerns the precursors of self-esteem deficits. The reformulation suggested that the internal-external attributional dimension was uniquely and independently predictive of self-esteem deficits. Individuals who made internal attributions for negative outcomes were predicted to experience self-esteem loss, regardless of the stability or globality of those attributions. The hopelessness model, however, no longer regards internal attributions as sufficient for the appearance of self-esteem loss among depressives. Self-esteem deficits are now believed to result only when negative events are attributed to internal causes that are also perceived as stable and global. Hopelessness theory makes the following predictions regarding symptomatology associated with causal attributions: 1.) attributing negative life events to stable and global causes leads to the formation of a hopelessness expectancy, which in turn, leads to depressed affect, motoric lethargy, concentration difficulties, insomnia, etc, and 2.) attributing negative life events to internal, stable, and global causes predicts the occurrence of hopelessness expectancies, the symptom constellation described above, and self-esteem loss.

Abramson et al. (1989) instituted this revision in response to evidence indicating that internal attributions are sometimes associated

with positive emotional and behavioral outcomes. Dweck (1975), for example, encouraged helplessness-prone children to attribute academic failures to low effort (an internal but unstable cause). Children who received this attributional retraining after initial failure experiences perservered in the face of subsequent academic failure to a greater extent than did children who experienced initial task success. These results suggest that internal attributions might sometimes be associated with hopefulness rather than hopelessness, and thus lend support to the revised predictions presented by hopelessness theory.

The results of the few studies that have actually examined attributional precursors to self-esteem loss favor Abramson et al.'s (1989) current predictions over those made by the reformulated model. For example, both Zautra, Guenther, and Chartier (1985) and Brewin and Furnham (1986) failed to observe a unique relation between internal attributions and self-esteem loss. Zautra et al. found that internal, stable, and global attributions for both hypothetical (i.e., ASQ) and real negative events were each significantly associated with self-esteem deficits. Subsequent analyses showed that internal attributions correlated significantly with self-esteem when those attributions were relatively stable but not when they were relatively unstable. Using a regression approach, Brewin and Furnham (1986) found that both internal and global attributions were uniquely related to low self-esteem.

Although the evidence presented above is consistent with Abramson et al.'s (1989) most recent predictions regarding self-esteem loss, a more direct evaluation of the competing predictions is necessary before the revision is ultimately verified. Abramson et al.'s (1989)

additional speculation that self-esteem deficits stem from inferred personal deficiencies is also in need of assessment.

I. METHODOLOGY

The present investigation was designed to test the etiological postulates of the hopelessness theory of depression (Abramson et al., 1989) and to evaluate the divergent predictions made by the hopelessness and reformulated learned helplessness models (Abramson et al., 1978) of depression. To achieve these research objectives, a two-wave longitudinal methodology was used in which subjects responded to measures of each construct in the two models over a three week period. During the first assessment session, subjects responded to measures of dysfunctional cognitive styles, self-esteem, hopelessness expectancies, and depressive symptomatology. Measures of life stress, event-based inferences and cognitions, self-esteem, hopelessness, and depressive symptoms were administered three weeks later when subjects returned for the follow-up assessment session.

Regression analyses were conducted to examine the degree to which higher levels of maladaptive cognitive styles and event-based cognitions were associated with T1 to T2 increases in depressive symptomatology among subjects who experienced highly stressful life events. Additional tests evaluated hypotheses concerning the mediational roles of event-cognitions and hopelessness expectancies as well as those regarding the cognitive precursors of self-esteem deficits. The role of perceived control in moderating the effects of life stress was assessed by performing the primary analyses separately for subjects who perceived

their most upsetting stressors as relatively controllable and as relatively uncontrollable.

Research Participants

Two hundred forty seven (94 male and 153 female) undergraduates enrolled in Introductory Psychology classes at the University of New Hampshire participated in exchange for course credit. The hopelessness model is appropriately tested using a sample of nondepressed college-student subjects (rather than clinically depressed patients) for several reasons. First, hopelessness theory addresses itself to the etiology of depressive symptoms and can therefore be adequately assessed only by using a sample of initially nondepressed subjects. Second, as mentioned above, the hopelessness model is an etiological theory of moderate depressive reactions as well as depressive episodes of a more severe nature (cf. Metalsky et al., 1982). The depressive reactions that nonclinical student populations exhibit in response to life stressors are thus appropriately used in an evaluation of hopelessness theory (cf. Vredenburg, Flett, & Krames, 1993).

Materials

Dysfunctional Cognitive Styles

Attributional Style. Generalized tendencies to attribute negative achievement and interpersonal events to stable and global causes were assessed using Metalksy et al.'s (1987) Expanded Attributional Style Questionnaire (EASQ). In contrast to the original ASQ (Peterson et al, 1982; Seligman et al., 1979) which includes six positive and six negative hypothetical events, the EASQ is comprised of 12 hypothetical negative events. Six of the EASQ events fall in the achievement domain

(e.g., "You take an exam and receive a low grade on it") and the remaining six fall in the interpersonal domain (e.g., "Your relationship with your boyfriend/girlfriend ends even though you would like it to continue).

For each event on the EASQ, subjects wrote down the one major factor that would have caused it. They then rated each causal factor on seven-point bipolar externality-internality, instability-stability, and specificity-globality subscales. Responses to each subscale were summed across the six achievement events and across the six interpersonal events to yield total achievement-domain and interpersonal-domain EASQ scores on each attributional dimension. Total subscale scores can therefore range from six to 42, with higher scores reflecting stronger tendencies to attribute negative events to internal, stable, and global causes.

The EASQ was developed, in part, to bolster the low subscale reliabilities typically obtained using the original ASQ (cf., Peterson & Seligman, 1984). Metalsky et al. (1987) noted high internal consistency coefficients for composite stability and globality indexes in both the achievement (alpha = .77) and interpersonal (alpha = .79) domains. Metalsky et al. also demonstrated that scores on the EASQ are highly correlated with the attributions that college students make for specific negative events. They obtained a correlation of $r = .60$ between composite stability and globality scores in the achievement domain and scores on an analogous composite assessing causal attributions for failure on an important course exam. Consistent with the hopelessness model's specific vulnerability hypothesis (see above), attributions for

exam failure were less strongly correlated (r = .30, p > .10) with scores on the interpersonal-domain stability/ globality composite. These data suggest that EASQ is both a reliable and valid measure of attributional styles in the achievement and interpersonal domains.

Dysfunctional Attitudes. Subjects also responded to Wiessman's (1979) Dysfunctional Attitudes Scale (DAS-Form A). The items on the DAS were derived from clinical reports of the thought content of depressed patients and include 40 statements such as: "I must be a useful, productive, creative person or life has no purpose," "A person should think less of himself if other people do not accept him," and "If I fail at my work, then I am a failure as a person." Subjects used seven-point (strongly disagree to strongly agree) Likert scales to indicate their level of agreement with each statement. Total scores can range from 40-280, with higher scores reflecting more dysfunctional attitudes.

Although Weissman (1979) demonstrated that the original 100-item DAS was multi-factorial, she reported alpha coefficients of .86 and .87 for both 40-item versions of this measure. Dobson and Breiter (1983) obtained comparable reliability coefficients for males (alpha = .90) and for females (alpha = .88). In addition to being a reliable measure of depressogenic assumptions, the DAS has been shown to correlate in theoretically predicted ways with measures of depression-related cognitions and depressive symptomatology. Using a college-student sample, Weissman (1979) obtained a correlation of r = .52 between DAS scores and scores on Krantz and Hammen's (1976) Story Completion Test of cognitive distortion. Dobson and Breiter (1983) observed significant correlations (r = .43 for males; r = .36 for females) between DAS scores

and scores on Hollon and Kendall's (1980) Automatic Thoughts Questionnaire. In addition, Weissman (1979) and Dobson and Breiter (1983) found DAS scores to correlate between .30 and .36 with scores on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

Irrational Beliefs. Kassinove, Crisci, and Tiegerman's (1977) 33-item Idea Inventory (IdI) was used to measure irrational beliefs falling in three of the four categories described by Ellis (1977): 1.) unrealistic demands (i.e., "shoulds" and "musts"); 2.) awfulizing statements, and 3.) blame and self-derogation. The IdI consists of Ellis' 11 original irrational beliefs and two additional statements corresponding to each of the original beliefs. Example statements on the IdI include: "People need the love or approval of almost everyone one they consider important" (one of Ellis' original irrational beliefs), "I feel inadequate and worthless when I fail at school or work," and "I cant help but feel depressed and rejected when others let me down." Responses to each item were recorded on Likert scales ranging from 1 (disagree) to 3 (agree). Total IdI scores can range from 33 to 99, with higher scores reflecting stronger irrational ideation.

To assess the internal consistency of the IdI, Kassinove et al. (1977) correlated each third of the total inventory with each of the other two-thirds and obtained reliability coefficients ranging from .84 to .91. Vestre (1983) administered the IdI to a sample of college students on two occasions separated by a 4-week interval and obtained a test-retest reliability coefficient of .81. Also using a college-student sample, Kassinove et al. found that IdI scores (reverse coded in

their study) correlated negatively with neuroticism (r = - .57) and adjustment (rs ranging from -.35 to -.58) scores. With respect to depressive symptomatology, Vestre and Budd (1980; cited in Vestre, 1983) obtained a significant correlation of -.40 between IdI and BDI scores. Finally, Vestre (1983) found that relative to low and moderate scorers, high IdI scorers exhibited the most intense levels of negative affect across a 2-4 week time period.

Overgeneralization. The seven-item Overgeneralization subscale of Carver and Ganellen's (1983) Attitudes Toward Self Scale (ATS) was included as a measure of tendencies to generalize the implications of specific negative events to one's self worth. The Overgeneralization index was developed to assess one aspect of self-punitiveness that appears to overlap with the tendency, described to Abramson et al. (1988), to perceive personal deficiencies in response to life stressors. Subscale items include statements such as "How I feel about myself overall is easily influenced by a single mistake," and "When even one thing goes wrong I begin to feel bad and wonder if I can do well at anything." Subjects used a five-point Likert scale to rate the extent of their agreement with each statement. Scores on this measure can range from 7-35 with higher scores reflect stronger overgeneralizing tendencies.

In Carver and Ganellen's (1983) factor analysis of the ATS (which also includes subscales assessing self-imposed high standards and self-criticism), all Overgeneralization items loaded onto a single factor with an alpha coefficient of .82. In addition, of the three ATS subscales, only Overgeneralization was significantly associated with

concurrently measured depressive symptomatology levels, accounting for 23% of the variance in BDI scores among college undergraduates. Similar results using the ATS were obtained in a subsequent investigation conducted by Carver, La Voie, Kuhl, and Ganellen (1988). In addition, Ganellen (1988) reported that Overgeneralization was uniquely associated with depressive symptomatology--subscale scores continued to predict clinical ratings of depressive symptoms after controlling for levels of anxiety.

Locus of Control. Generalized control expectancies were assessed using the Powerful Others and Chance subscales of Levenson's (1981) multidimensional Locus of Control Scale. The eight-item Powerful Others scale measures the degree to which outcomes are believed to be controlled by people in positions of power (e.g., "Although I may have good ability, I will not be given leadership responsibility without appealing to those in positions of power"). The eight-item Chance scale measures the degree to which events are perceived to be random (e.g., Whether or not I get to be a leader depends on whether I'm lucky enough to be in the right place at the right time"). Subjects rated the extent of their agreement with each statement on six-point Likert scales such that higher scores reflected a more external control orientation. A composite locus of control measure, with a possible score range of 16 to 96, was created by summing responses across all 16 items.[2]

Stressful Achievement and Interpersonal Life Events

An Achievement Events Questionnaire (AEQ) and an Interpersonal Events Questionnaire (IEQ) were developed to assess the recent occurrence and perceived stressfulness of a variety of negative

achievement and interpersonal events, respectively. Both questionnaires were adapted from several published life-stress measures, including Cochrane and Robertson's (1973) Life Events Inventory (LEI), Sarason, Johnson, and Siegel's (1978) Life Experiences Survey (LES), and Andersen's (1990) Life Events Questionnaire (LEQ). Numerous researchers have demonstrated that life-stress scores derived from the above inventories (by themselves and/or in combination with various measures of cognitive styles) successfully predict depressive symptomatology (e.g., Andersen, 1990; Hammen, Marks, Mayol, and de Mayo, 1985; Olinger et al., 1987; Robins & Block, 1988; Wise & Barnes, 1986). In developing the AEQ and the IEQ, only negative events relevant to the experiences of college students were selected from existing inventories. In addition, events were chosen such that they clearly fell into either achievement or interpersonal domains and did not overlap in content. Several events, not listed on any of the existing life stress measures, were also added. Finally, a scale on which subjects could rate the degree of stress associated with life events was adapted from Sarason et al.'s (1978) LES.

The AEQ consists of 16 negative achievement events typically encountered by students in their first semester or year of college. Example events include: "I received a lower grade on an exam or paper than anticipated," "I am having trouble adjusting to the academic life at college (e.g., workload, time-management, effective study habits), "I chose a college major but now I realize that I do not like it," and "I am having difficulty balancing school responsibilities with my other commitments (e.g., job, sports, clubs, fraternity or sorority)." The

IEQ lists 20 negative interpersonal events typical of the same population. Example events from the IEQ include: "I am having difficulty adjusting to the social life at college (e.g., difficulty making friends or "fitting in")," "I was rejected by someone I am attracted to," and "I had an argument/disagreement with a friend."

On each inventory, subjects placed a check next to each event they experienced in the three weeks since the first assessment session. Additional space was provided for subjects to write in up to five negative events that they experienced during the same time period but which were not listed on the inventory. Subjects were also asked to rate the degree of stress associated with each event that was either checked or written in. Stress ratings were made on seven-point Likert scales ranging from 1 (not at all stressful) to 7 (extremely stressful) (cf. Sarason et al., 1978). On both the AEQ and the IEQ, subjects were asked to select the one event that they experienced as most stressful and indicate (on two seven-point Likert scales) how important the event was to them.

Depressogenic Inferences: Perceived Control, Expected Consequences, Personal Deficiencies, Self-Blame, and Causal Attributions

For their most upsetting achievement and interpersonal stressor, subjects completed four-item measures of perceived control, expected negative consequences, perceived personal deficiencies, behavioral blame, and characterological blame. Subjects were also asked to identify the one major cause of their most stressful achievement and interpersonal event and to rate each cause on four-item scales assessing the attributional dimensions of internality-externality, stability-

instability, and globality-specificity. Ratings on each of these measures were made on seven-point Likert scales, anchored such that high scores reflect more depressogenic responses.

Hopelessness Expectancies

Generalized hopelessness expectancies were assessed with the Beck Hopelessness Scale (BHS; Beck et al., 1974, Beck & Steer, 1988). The BHS consists of 20 clinically-derived statements reflecting both pessimistic and optimistic attitudes toward the future (e.g., "I might as well give up because there is nothing I can do about making things better for myself," "When I look ahead to the future, I expect that I will be happier than I am now"). Subjects provided true (coded as "1") or false (coded as "0") responses depending on whether each statement described their feelings or attitudes throughout the past week. Responses to the positively keyed (optimistic) items were reverse scored so that higher BHS scores (which can range from 0-20) reflected higher levels of hopelessness.

Beck and Steer (1988) reviewed the results of numerous studies that examined the psychometric properties of the BHS and reported internal consistency coefficients ranging from .82 to .93 as well as test-retest correlations of .69 (one week) and .66 (six weeks). They also found that BHS scores correlate significantly with clinical ratings of hopelessness (*rs* ranging from .62 to .74), severity of depressive symptomatology (*rs* ranging from .46 to .76), and scores on a BDI-item assessing pessimism toward the future (*rs* ranging from .42 to .74).

Beck et al. (1974) factor analyzed BHS data obtained in a sample of suicide attempters and observed three dimensions respectively

reflecting feelings about the future, loss of motivation, and future expectations. These factors overlap sufficiently with the helplessness and negative outcome expectancy components of the proposed proximal cause of hopelessness depression.

Affect and Self-Esteem Measures

Depressive Symptomatology. The Beck Depression Inventory (BDI; Beck et al., 1979) was used to measure the symptomatology associated with hopelessness depression. The BDI is a widely used instrument that assesses the severity with which individuals experience a variety of somatic, motivational, emotional, and cognitive symptoms of depression. It consists of 21 sets of four statements that reflect increasingly severe levels of depressive symptoms (e.g., (a) "I do not feel sad," (b) "I feel sad," (c) "I am sad all the time and I can't snap out of it," (d) "I am so sad or unhappy that I can't stand it"). Subjects were asked to choose the one statement in each set that best represented how they felt during the previous week. Each response was coded on a 4-point scale ranging from 0 (least severe statement) to 3 (most severe statement). Total scores on the 21-item inventory can range from 0 to 63.

Using meta-analytic techniques, Beck, Steer, and Garbin (1988) reviewed studies conducted over the past 25 years that evaluated the psychometric properties of the BDI. The results of their review revealed that the BDI provides a reliable and valid indicator of depressive symptoms in both psychiatric and nonpsychiatric populations. Among the latter, the mean alpha coefficient associated with the BDI was .81. Beck et al. (1988) reported stability coefficients for the BDI

ranging from .60 to .90 in nonpsychiatric samples. In addition, Oliver and Burkham (1979) reported a test-retest reliability coefficient of .78 for BDI scores assessed across a three-week interval in a college-student sample.

Beck et al.'s (1988) analysis also showed that BDI scores obtained by nonpsychiatric respondents are highly correlated with clinical ratings of depression (rs range from .60 to .80) and scores on other self-report depression inventories (rs range from .60 to .86). Looking specifically at college students, Bumberry, Oliver, and McClure (1978) reported that BDI scores correlated .77 with symptom ratings obtained concurrently from psychiatric interviews. Using a similar procedure, Hammen (1980) obtained a correlation of .80. These data indicate that the BDI provides a valid assessment of the severity of depressive symptoms experienced by subjects sampled from college-student populations.

Self-Esteem Deficits. Rosenberg's (1965) Self-Esteem Scale (RSES) was used to measure levels of self-esteem. The RSES is a brief (10 items) but widely used inventory that primarily assesses a self-acceptance component of self-esteem. Using a five-point Likert scale, subjects indicated the extent of their agreement with statements such as, "I feel that I'm a person of worth, at least on an equal basis with others," and "All in all, I am inclined to feel that I am failure." Scale values ranged from 1 (extremely true) to 5 (extremely untrue), yielding a possible score distribution of 10-50. Higher scores reflect higher self-esteem.

Robinson and Shaver (1973) reported that the RSES is both reliable

over time (.85) and internally consistent (.92). Zautra et al. (1985) administered the RSES to college students on two occasions separated by a two-week interval and reported a test-retest reliability coefficient of $\underline{r}$ = .81. With respect to concurrent validity, RSES scores have been shown to correlate between .27 and .83 with other self-report measures of self-esteem and with clinical ratings of self-esteem (Robinson & Shaver, 1973). Zautra et al. (1985) obtained correlations of .45 and .58 between the RSES and two depression inventories (one of which was the BDI). Zautra et al. also demonstrated that the two depression measures used in their research correlated significantly more highly with one another than they did with the RSES. The latter suggests that scores on the RSES reflect more than depressed or negative affect.

Procedure

Subjects participated in two assessment sessions in mixed sex groups of approximately 15. The initial two-hour sessions were conducted during October and November of 1991. Follow-up sessions, requiring only one-hour of time, were held three weeks later. The nature and participation requirements of the study were described to subjects at the outset of the initial session. Subjects were asked to sign informed consent forms as an indication of their willingness to participate in this research. Subjects then received folders containing all of the measures to be completed during the session as well as answer sheets for their responses.

During the first assessment session, subjects completed measures in the following order: EASQ, BDI, DAS, IdI, BHS, RSES, ATS-OVERGEN, and LOC. At the follow-up session, the measures were ordered in the

following way: BHS, RSES, BDI, AEQ, and IEQ. Written instructions were available for each measure and additional oral instructions were provided for several of the measures (EASQ, AEQ, and IEQ). Subjects responded to each measure at their own pace and were allowed to take rest breaks as needed.

After completing their participation in both assessment sessions, subjects received a written debriefing statement describing the purpose of this research as well as the nature of the materials they completed. The debriefing statement included my name and phone number and subjects were encouraged to contact me if they wanted to discuss the nature or results of this research in the future.

II. RESULTS AND DISCUSSION

Descriptive and Reliability Data

Means, medians, standard deviations, and coefficient alpha reliabilities for the Time 1 (T1) measures of dysfunctional cognitive style, the Time 2 (T2) event stress and event cognition measures, and the T1 and T2 hopelessness, mood, and self-esteem scales for subjects who completed both sessions are presented in Tables 1 through 4, respectively. Scores on the stability and globality subscales of the Extended Attributional Style Questionnaire (EASQ; Metalsky et al., 1987) were summed to form "attributional generality" scales in the achievement and interpersonal domains (see Table 1). Similar composites were created from the scales assessing stability and globality attributions for subjects' most stressful achievement and interpersonal events (see Table 3). The use of these composite attributional scales is consistent with hopelessness model's contention that only the stability and globality dimensions of causal attributions play a role in the development of hopelessness depression (Abramson et al., 1989; Metalsky et al., 1982, 1987).

An achievement stress variable was created by multiplying subjects' ratings of the stressfulness of their most upsetting achievement event by their ratings of the importance of that event. The interpersonal stress variable likewise represents the multiplicative product of subjects' stressfulness and importance ratings for their most upsetting interpersonal event. Methodological as well as theoretical

Table 1

Descriptive Statistics for Cognitive Style Measures

Measure	M	Mdn	SD	Alpha	N
EASQ-ACGEN	42.40	43.0	10.80	.79	247
EASQ-ACINT	30.20	30.0	4.84	.48	247
EASQ-IPGEN	45.22	45.0	10.03	.72	247
EASQ-IPINT	26.54	26.0	5.08	.41	247
DYSATT	130.39	129.0	20.61	.79	246
IRRIDEAS	65.14	65.0	10.14	.82	243
OVERGEN	19.09	19.0	6.36	.87	244
LOCUS	45.43	46.0	10.91	.82	228

Note. EASQ-ACGEN=Extended Attributional Style Questionnaire: Achievement Events Generality Composite; EASQ-ACINT=Extended Attributional Style Questionnaire: Achievement Events Internality Subscale; EASQ-IPGEN=Extended Attributional Style Questionnaire: Interpersonal Events Generality Composite; EASQ-IPINT=Extended Attributional Style Questionnaire: Interpersonal Events Internality Subscale; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

Table 2

Descriptive Statistics for Event Stress Measures

Measure	M	Mdn	SD	Alpha	N
ACSTRESS	31.09	33.0	12.43	NA	230
IPSTRESS	28.16	27.5	13.35	NA	225

Note. ACSTRESS=Achievement Event Stress; IPSTRESS=Interpersonal Event Stress.

Table 3

Descriptive Statistics for Achievement- and Interpersonal-Event Cognition Measures

Measure	M	Mdn	SD	Alpha	N
AEQ-GEN	6.81	7.0	2.36	.76	231
AEQ-INT	4.23	4.5	1.91	.93	231
AEQ-CNS	3.45	3.3	1.58	.87	230
AEQ-DEF	2.64	2.3	1.47	.88	230
AEQ-CNT	3.67	3.8	1.53	.79	230
AEQ-BBL	3.98	4.0	1.88	.86	230
AEQ-CBL	3.17	3.0	1.80	.84	230
IEQ-GEN	7.45	7.3	2.33	.80	221
IEQ-INT	3.39	3.5	1.93	.92	223
IEQ-CNS	3.34	3.5	1.67	.93	225
IEQ-DEF	2.73	2.3	1.68	.91	225
IEQ-CNT	4.78	4.8	1.42	.75	225
IEQ-BBL	3.28	3.0	1.84	.88	225
IEQ-CBL	2.96	2.5	1.82	.88	225

Note. AEQ=Achievement Events Questionnaire; IEQ=Interpersonal Events Questionnaire; GEN=Attributional Generality Subscale; INT= Internality Subscale; CNS=Expected Consequences Subscale; DEF= Personal Deficiencies Subscale; CNT=Perceived Control Subscale; BBL=Behavioral Blame Subscale; CBL=Characterological Blame Subscale.

Table 4

Descriptive Statistics for Time 1 (T1) and Time 2 (T2) Hopelessness, Depressive Symptom, and Self-Esteem Measures

Measure	M	Mdn	SD	Alpha	N
T1 BHS	4.24	3.0	3.74	.84	247
T2 BHS	3.88	3.0	3.56	.83	234
T1 BDI	9.57	8.0	6.36	.82	247
T2 BDI	7.88	6.0	6.87	.88	226
T1 RSES	38.65	39.0	7.31	.88	244
T2 RSES	39.97	42.0	7.32	.90	234

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; RSES=Rosenberg Self-Esteem Scale.

considerations guided the construction of the event stress variables in this way. With respect to the former, recall that subjects provided stress ratings for the one achievement and interpersonal event that they experienced as most stressful. Given these instructions, it was not surprising to find that the distributions of stress ratings for both achievement and interpersonal events were highly negatively skewed (skewness = -.824 and -1.04, respectively). Combining stress and importance ratings produced event upsettingness variables that were more normally distributed. On a theoretical level, the stress measure used here is consistent with the claim that the etiological pathway leading to hopelessness depression is triggered by the occurrence of a negative life event and the perception of that event as important (Abramson et al., 1988). Moreover, hopelessness theorists themselves have used a similarly constructed stress variable in previous tests of the model (Metalsky et al., 1987).[3] Inspection of the alpha coefficients in Tables 1-4 reveals that all but several of the measures possessed adequate levels of internal consistency. The low reliability obtained for the internality subscale of the EASQ (see Table 1) proved to be the exception to this pattern (see Cutrona, Russell, & Jones, 1984; Peterson & Seligman, 1984; Peterson, Semmel, von Baeyer, Abramson, Metalsky, & Seligman, 1982; Peterson et al., 1985 for similar findings regarding the internality dimension of attributional style).

Subject-Attrition

Differences between the 12 subjects who failed to return to the follow-up session (noncompleters) and the 235 subjects who participated in both sessions (completers) were assessed through a series of

multivariate analyses of variance (MANOVAs) and correlational analyses on the T1 cognitive style, hopelessness, depressive symptom, and self esteem measures (cf. Metalsky & Joiner, 1992).[4] Noncompleters did not differ from completers on any of the T1 measures (all *p*s > .33),[5] nor was completion status significantly correlated with scores on any of the T1 measures (*r*s range from -.05 to .07, ns). These data provide no evidence that subject attrition biased the results of this investigation in any way.

Test-Retest Reliabilities and Mean Differences Between Scores on the T1 and T2 Measures

Scores on the hopelessness, depressive symptom, and self esteem measures were highly stable across assessment sessions (test-retest reliability coefficients = .64, .63, and .76 respectively). A series of repeated measures analyses of variance (ANOVAs), with time of administration serving as the within-subjects factor, was conducted to examine mean differences in scores on the T1 and T2 measures. Based on a Bonferroni correction for the number of tests performed (Kirk, 1984), interpretation was restricted to differences that were significant at or beyond the $p = .017$ level.

Using this criterion, T2 BDI scores were significantly lower than T1 BDI scores (F [1, 225] = 21.76, $p < .001$) and T2 RSES scores were reliably higher than T1 RSES scores (F [1, 230] = 18.76, $p < .001$). The reduction in BHS scores from T1 to T2 did not meet the adjusted alpha criterion, F (1, 233) = 4.00, $p < .05$. The T1 to T2 decreases in distress levels that were observed in this study are similar to those found by other researchers using student samples (e.g., Barnett &

Gotlib, 1988) and may reflect, in part, measurement reactivity (cf. Zimmerman, 1986).

Gender Differences

Gender differences on the T1 and T2 measures were evaluated in several steps. A one-way between-subjects MANOVA was first performed on T1 RSES, BDI, and BHS scores. A significant multivariate effect of subject gender emerged in that analysis, F (3, 240) = 2.68, $p < .05$). Due to the magnitude of the intercorrelations among these measures (rs range from .54 to .60, all $ps < .01$), stepdown analysis was used to assess the significance of the individual measures.[6] Higher priorities were given to measures assessing more stable aspects of functioning, resulting in the following ordering of measures: RSES, BHS, and BDI. A five percent familywise error rate was maintained by setting the alpha level for each test to $p = .017$.

In accord with the stepdown procedure, gender differences on the T1 RSES measure were assessed in a univariate ANOVA. Scores on all other measures were examined in separate univariate analyses of covariance (ANCOVAs) in which subject gender served as the between-groups factor and higher-priority measures were used as covariates. As shown in Table 5, the only effect to emerge was a marginally significant gender difference in T1 RSES scores. Female subjects reported lower levels of self-esteem (M = 37.86) than male subjects (M = 39.94) did. Note that after controlling for this difference in self-esteem, males and females did not significantly differ in levels of depressive symptomatology (adjusted BDI Ms = 9.05 and 9.94, respectively).

Gender differences on the remaining T1 measures were evaluated

Table 5

Results of Univariate and Stepdown Analyses Assessing Gender Differences on Time 1 Self-Esteem, Hopelessness, and Depressive Symptom Measures

Measure	Univariate F	df	p[a]	Stepdown F	df	p	Eta^2
RSES	4.70	1,242	.03	4.70	1,242	.03	.02
BHS	0.50	1,242	.48	0.46	1,241	.50	.002
BDI	4.92	1,242	.03	2.84	1,240	.09	.02

Note. RSES=Rosenberg Self-Esteem Scale; BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory.

[a] Significance levels can not be properly evaluated because tests are not independent. The significance levels presented here are those that would have been obtained in a univariate context.

controlling for pre-existing differences in self-esteem. In the first analysis, scores on the cognitive style measures were subjected to a one-way multivariate analysis of covariance (MANCOVA). Subject gender was the between-groups factor and T1 RSES served as a covariate.[7] This analysis yielded a highly significant gender effect, F (8, 211) = 3.42, p = .001. Due to the moderate to large correlations among most scales within this set (see Table 7 below), stepdown analysis was used to evaluate the significance of the individual cognitive style measures. T1 RSES was used as a covariate in all stepdown tests and the cognitive style scales were prioritized in the order in which they are listed in Table 6. A five percent family-wise error rate was maintained by restricting interpretation to differences that were significant at or beyond the p = .006 level.

None of the tests met or surpassed this criterion, although a number of marginal effects were observed (see Table 6). Relative to males, females exhibited a stronger tendency to attribute negative achievement outcomes to internal causes (adjusted Ms = 30.64 and 29.29, respectively). Conversely, males were more likely than females to make stable and global causal attributions for negative achievement outcomes (adjusted Ms = 47.56 and 43.75) and for negative interpersonal outcomes (adjusted Ms = 47.56 and 43.75). Consistent with observations made in other investigations (e.g., Barnett & Gotlib, 1988), females (adjusted M = 127.75) scored somewhat lower than males (adjusted M = 133.60) on the Dysfunctional Attitudes Scale. Finally, Overgeneralization scores were slightly higher among females (adjusted M = 19.06) than among males (adjusted M = 18.76).

Table 6

Results of Univariate and Stepdown Analyses Assessing Gender Differences on Cognitive Style Measures

Measure	Univariate F	df	p[a]	Stepdown F	df	p	Eta^2
EASQ-ACINT	4.19	1,218	.04	4.19	1,218	.04	.02
EASQ-ACGEN	5.00	1,218	.03	4.67	1,217	.03	.02
EASQ-IPINT	3.55	1,218	.06	2.71	1,216	.10	.02
EASQ-IPGEN	8.07	1,218	.01	4.36	1,215	.04	.04
DYSATT	5.39	1,218	.02	3.43	1,214	.07	.02
IRRIDEAS	0.004	1,218	.95	1.83	1,213	.18	.000
OVERGEN	0.25	1,218	.62	3.38	1,212	.07	.001
LOCUS	3.38	1,218	.07	1.88	1,211	.17	.02

Note. EASQ-ACINT=Extended Attributional Style Questionnaire: Achievement Events Internality Subscale; EASQ-ACGEN=Extended Attributional Style Questionnaire: Achievement Events Generality Composite; EASQ-IPINT=Extended Attributional Style Questionnaire: Interpersonal Events Internality Subscale; EASQ-IPGEN=Extended Attributional Style Questionnaire: Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] Significance levels can not be properly evaluated because tests are not independent. The significance levels presented here are those that would have been obtained in a univariate context.

Gender differences on the T2 measures of self-esteem, hopelessness, and depressive symptomatology were examined in a one-way MANCOVA with T1 self-esteem scores used as covariates.[8] Stepdown analysis (measures ordered: 1.) RSES, 2.) BHS, 3.) BDI) revealed that the multivariate gender effect found across this set of measures (F [3, 218] = 3.12, p = .03) was specific to T2 BHS scores. After controlling for T1 and T2 levels of self-esteem, males (adjusted BHS M = 4.45) exhibited higher levels of hopelessness at T2 than females did (adjusted BHS M = 3.45), F (1, 219) = 8.31, p < .01. Gender differences were not obtained for either T2 RSES scores (F [1, 220] = 0.47, p = .49) or T2 BDI scores (F [1, 218] = 0.59, p = .44).

Multivariate analysis of the T2 achievement and interpersonal event stress measures (controlling for T1 RSES scores) also yielded a significant gender difference (F [2, 214] = 3.29, p < .05). Given the modest correlation (r = .29) between achievement and interpersonal stress, as well as the difficulty of appropriately prioritizing these measures, the results of the univariate rather than stepdown tests were interpreted. The negative achievement events reported by females were rated as more stressful (adjusted M = 32.78) than those reported by males (adjusted M = 28.99), F (1, 215) = 4.92, p < .03. A similar, but nonsignificant effect emerged in the analysis of interpersonal- event stress (adjusted Ms = 29.39 and 26.12 for females and males, respectively), F (1, 215) = 3.24, p = .07. Despite these differences in the perceived stressfulness of life events, scores on the event cognition measures in the achievement (F [7, 218] = 1.70, p = .11) and interpersonal (F [7, 211] = 1.81, p = .09] domains did not reliably

differ as a function of gender.

Considering the number of measures used in this investigation, relatively few gender differences were observed. The differences that did emerge generally reflected higher levels of emotional distress among women and higher levels of dysfunctional cognition among men. For example, females reported lower levels of self-esteem at T1 and higher levels of stress associated with the recent occurrence of negative achievement and interpersonal events. Males did, however, report higher levels of hopelessness at T2 relative to females.

With respect to the cognition measures, males endorsed more dysfunctional attitudes than females did and were more likely to attribute negative achievement and interpersonal outcomes to stable and global causes. Overgeneralization and internalization tendencies, on the other hand, were slightly stronger among females. Notwithstanding these differences, the results of the gender analyses provided little indication that it was necessary to perform the primary analyses separately for male and female subjects.

Dysfunctional Cognitive Styles

Diathesis-Stress Predictions of Hopelessness Theory

A primary tenet of the hopelessness model is that individuals who possess certain dysfunctional cognitive styles are at increased risk of becoming hopelessness and thus developing the symptoms of hopelessness depression when they experience stressful events. These cognitive diatheses, which are formally referred to in the hopelessness model as "distal contributory causes" of hopelessness depression, include tendencies to attribute the causes of negative events to stable and

global factors, expect negative events to result in an array of adverse consequences, and view the self as personally flawed or deficient when negative events occur. The hopelessness model hypothesizes that individuals who possess these dysfunctional cognitive styles will be more likely than their counterparts to exhibit elevated levels of depressive symptomatology under conditions of high stress. In the absence of stress (or in the presence of positive life events), little or no difference is expected in the degree of depressive symptomatology manifested by individuals who do and do not possess the hypothesized cognitive diatheses.

The diathesis-stress predictions advanced by hopelessness theory were evaluated in the present investigation by examining the degree to which levels of dysfunctional cognitive style interact with the severity of a recent life stressor to predict T1 to T2 increases in depressive symptomatology.[9] Specifically, Analysis of Partial Variance (APV; Cohen & Cohen, 1983) was used to examine whether residual changes in BDI scores from T1 to T2 (i.e., change in BDI scores from T1 to T2, adjusted for T1 BDI score) could be predicted from interactions of subjects' ratings of the stressfulness of a recent negative life event and their scores on several different measures of dysfunctional cognitive style (cf. Metalsky et al., 1987). An extension of Analysis of Covariance (ANCOVA), APV assesses relations between a covariate-adjusted change score and one or more quantitative (rather than group membership) research factors, which are also adjusted for the covariate, and is more appropriate than the simple pre-test/post-test change score method for predicting change over time (Cohen & Cohen, 1983).

In each analysis conducted here, residualized change scores were created by partialling out the variance in T2 BDI scores that was predictable from T1 BDI scores. Residualized BDI change scores were then regressed on the main effects of event stressfulness and dysfunctional cognitive style. The interaction of event stressfulness and dysfunctional cognitive style was always evaluated on the third and final step, after the variance due to T1 BDI scores and the two main effects had been removed from both T2 BDI scores and the interaction term. The squared partial correlation (pr^2) represents the portion of variance in residualized BDI change scores uniquely accounted for by each predictor variable, whereas the R^2 increment on each step represents the total variance in T2 BDI scores explained by the set of variables entered on that step (Cohen & Cohen, 1983). Support for the diathesis-stress predictions of the hopelessness model would be obtained if the partial correlations corresponding to the cognitive style X stress interaction terms are significantly greater than zero.

Each cognitive style variable and its interaction with achievement and interpersonal stress was tested in a separate analysis. Following the recommendations of Jaccard, Turrisi, and Wan (1990), as well as those of Cohen and Cohen (1983), all stress and cognitive style variables were "centered" prior to their entry in the regression models. Centering is accomplished by subtracting the mean from scores on relevant variables. Its use serves to reduce multicollinearity problems (i.e., unstable regression coefficients) that result when interaction terms are created from scores on main effect variables. Multiplicative composites representing interactions of dysfunctional cognitive styles

and either achievement or interpersonal stress were formed from centered scores.

Zero-order correlations among the variables used in the diathesis-stress regression models are presented in Table 7. In general, associations among the cognitive style, event-stress, and depressive symptomatology measures were moderate to strong in magnitude. The one exception to this pattern occurred for the achievement stress variable, which was relatively weakly correlated with measures of dysfunctional cognitive style and depressive symptomatology. The analyses appearing in Table 8 test hypotheses regarding the interactive effects of cognitive style and achievement stress on depressive symptomatology. As can be seen, a large portion of the variance in T2 BDI scores (40%) was predictable from T1 BDI scores. Such a finding is understandable in light of the fact that the first and second assessment sessions were separated by only three weeks. A similar result was obtained in Metalsky and Joiner's (1992) investigation which used a five week interval between assessment sessions.

The main effect of achievement-event stress was a significant predictor of residualized BDI change scores, uniquely accounting for an additional two to three percent of the residualized variance across analyses. Significant although weak positive relations were also obtained between residualized BDI change scores and scores on the Overgeneralization and Irrational Ideas main effect variables. Except for a marginally significant Locus of Control X Achievement Stress effect, none of the cognitive style X achievement stress interactions contributed to the prediction of change in BDI scores from T1 to T2.

Table 7

Intercorrelations Among Cognitive Style, Stress, and Depressive Symptomatology Measures

Measure	1	2	3	4	5	6	7	8	9	10
1. T1 BDI	-									
2. T2 BDI	.63	-								
3. ACSTRESS	.29	.31	-							
4. IPSTRESS	.35	.40	.29	-						
5. EASQ-ACGEN	.31	.23	.16[a]	.12[b]	-					
6. EASQ-IPGEN	.39	.39	.23	.27	.69	-				
7. DYSATT	.45	.34	.10[a]	.25	.23	.40	-			
8. IRRIDEAS	.45	.43	.26	.23	.22	.32	.53	-		
9. OVERGEN	.60	.47	.26	.32	.32	.45	.48	.46	-	
10. LOCUS	.43	.35	.14[a]	.24	.19	.31	.50	.48	.49	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; IPSTRESS=Interpersonal Event Stress; EASQ-ACGEN=Extended Attributional Style Questionnaire: Achievement Events Generality Composite; EASQ-IPGEN=Extended Attributional Style Questionnaire: Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .05$. [b] ns.

Table 8

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Achievement Stress, Dysfunctional Cognitive Style, and Achievement Stress X Dysfunctional Cognitive Style Interactions (N = 226)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	EASQ-ACGEN				.03	0.50
	ACSTRESS	.02	3.47[c]	3,222	.17	2.54[b]
3	EASQ-ACGEN X ACSTRESS	.00	0.03	4,221	.01	0.19
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	DYSATT				.07	1.09
	ACSTRESS	.02	3.95[c]	3,222	.17	2.63[b]
3	DYSATT X ACSTRESS	.002	0.71	4,221	.06	0.84
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	IRRIDEAS				.18	2.77[b]
	ACSTRESS	.04	7.29[b]	3,222	.15	2.19[c]
3	IRRIDEAS X ACSTRESS	.005	1.81	4,221	.09	1.35
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	OVERGEN				.13	1.88[d]
	ACSTRESS	.03	5.16[b]	3,222	.16	2.39[c]
3	OVERGEN X ACSTRESS	.00	0.0003	4,221	.001	0.02
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	LOCUS				.09	1.39
	ACSTRESS	.02	4.34[b]	3,222	.17	2.58[b]
3	LOCUS X ACSTRESS	.01	3.42[e]	4,221	.12	1.85[e]

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; EASQ-ACGEN=Extended Attributional Style Questionnaire-Achievement Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$. [e] $p = .07$.

A similar pattern of results emerged when cognitive style scores, interpersonal stress levels, and their corresponding interaction terms were used to predict residual changes in T2 BDI scores (see Table 9). The significant main effect of interpersonal stress was twice as strong as that of achievement stress, explaining between four and five percent of the variance in residualized BDI scores. Scores on the Attributional Style (Interpersonal Domain) and Irrational Ideas measures were also significant predictors. As in the previous set of analyses, none of the cognitive style X interpersonal stress interaction terms were reliably different from zero.

Competing Predictions Derived From the Reformulated Theory of Learned Helplessness

One of the primary differences between the 1989 hopelessness model and the 1978 reformulation of learned helplessness theory pertains to the role of perceived control in the development of depressive symptomatology. Whereas the reformulation began the causal chain leading to learned helplessness depression with the perception of negative events as uncontrollable, the 1989 statement eliminated perceived uncontrollability as a factor in the development of hopelessness depression. Unlike the hopelessness model, the reformulated theory of learned helplessness would predict that dysfunctional cognitive styles are associated with increases in depressive symptomatology only among individuals who experience highly stressful life events that are also perceived as uncontrollable.

This prediction was evaluated in a series of regression analyses similar to those described above. This time, however, the analyses were

Table 9

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Interpersonal Stress, Dysfunctional Cognitive Style, and Interpersonal Stress X Dysfunctional Cognitive Style Interactions (N = 226)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	EASQ-IPGEN				.15	2.20[c]
	IPSTRESS	.04	8.69[b]	3,222	.21	3.17[b]
3	EASQ-IPGEN X IPSTRESS	.002	0.80	4,221	.06	0.90
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	DYSATT				.04	0.62
	IPSTRESS	.03	6.34[b]	3,222	.22	3.42[b]
3	DYSATT X IPSTRESS	.001	0.52	4,221	.05	0.72
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	IRRIDEAS				.19	2.89[b]
	IPSTRESS	.05	10.55[a]	3,222	.22	3.32[b]
3	IRRIDEAS X IPSTRESS	.01	2.38	4,221	.10	1.54
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	OVERGEN				.11	1.72[d]
	IPSTRESS	.04	7.69[b]	3,222	.21	3.27[b]
3	OVERGEN X IPSTRESS	.0002	0.07	4,221	.02	0.26
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	LOCUS				.08	1.12
	IPSTRESS	.03	6.80[b]	3,222	.22	3.39[b]
3	LOCUS X IPSTRESS	.0003	0.13	4,221	-.02	-0.35

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .09$.

performed separately for subjects who perceived their most stressful achievement event as either relatively controllable (N = 126) or relatively uncontrollable (N = 100) and for subjects who perceived their most stressful interpersonal event as either relatively controllable (N = 111) or relatively uncontrollable (N = 107). Low and high perceived control groups were formed through median splits on the measures assessing perceived control over negative achievement (Mdn = 3.8) and interpersonal (Mdn = 4.8) events. As before, the cognitive style X stress interaction terms were formed from centered scores on the cognitive style and either achievement or interpersonal stress variables. Diathesis-stress predictions derived from the reformulated theory of learned helplessness would receive support if cognitive style scores interacted with event stressfulness to predict T1 to T2 increases in BDI scores among subjects who perceived negative achievement or interpersonal events as relatively uncontrollable but not among subjects who perceived those events as relatively controllable.

Achievement Stress. Tables 10 and 11 present zero-order correlations among the cognitive style, achievement stress, and depressive symptom measures separately for subjects who perceived their most stressful achievement event as relatively controllable and uncontrollable, respectively. Regression results are presented first for subjects who perceived negative achievement events as controllable (see Table 12). Higher levels of achievement stress as well as higher scores on measures of Irrational Ideas, Overgeneralization, and Locus of Control Orientation (indicating greater externality), were significantly associated with residual increases in BDI scores from T1 to T2. None of

Table 10

Intercorrelations Among Cognitive Style, Achievement Stress, and Depressive Symptomatology Among Subjects Who Perceived Events as Controllable (N = 126)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.65	-						
3. ACSTRESS	.33	.35	-					
4. EASQ-ACGEN	.29	.18[a]	.24	-				
5. DYSATT	.45	.37	.09[b]	.22[a]	-			
6. IRRIDEAS	.45	.44	.26	.31	.51	-		
7. OVERGEN	.51	.48	.30	.43	.44	.46	-	
8. LOCUS	.37	.38	.09[b]	.09[b]	.50	.50	.47	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; EASQ-ACGEN=Extended Attributional Style Questionnaire: Achievement Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS= Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .05$. [b] ns.

Table 11

Intercorrelations Among Cognitive Style, Achievement Stress, and Depressive Symptomatology Among Subjects Who Perceived Events as Uncontrollable (N = 100)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.62	-						
3. ACSTRESS	.23[a]	.25[a]	-					
4. EASQ-ACGEN	.33	.29	.06[b]	-				
5. DYSATT	.46	.32	.10[b]	.23[a]	-			
6. IRRIDEAS	.45	.43	.24[a]	.10[b]	.55	-		
7. OVERGEN	.70	.48	.21[a]	.21[a]	.53	.46	-	
8. LOCUS	.54	.36	.22[a]	.31	.51	.47	.51-	

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; EASQ-ACGEN=Extended Attributional Style Questionnaire: Achievement Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS= Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .05$. [b] ns.

Table 12

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Achievement Stress, Dysfunctional Cognitive Style, and Achievement Stress X Dysfunctional Cognitive Style Interactions Among Subjects Who Perceived Events as Controllable (N = 126)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	EASQ-ACGEN				-.05	-0.58
	ACSTRESS	.02	2.45[c]	3,122	.20	2.20[c]
3	EASQ-ACGEN X ACSTRESS	.005	1.14	4,122	-.10	-1.07
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	DYSATT				.13	1.44
	ACSTRESS	.03	3.35[c]	3,122	.20	2.24[c]
3	DYSATT X ACSTRESS	.00	0.00	4,121	-.001	-0.01
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	IRRIDEAS				.20	2.22[c]
	ACSTRESS	.04	4.83[b]	3,122	.17	1.85[d]
3	IRRIDEAS X ACSTRESS	.0004	0.09	4,121	.03	0.30
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	OVERGEN				.19	2.16[c]
	ACSTRESS	.04	4.68[b]	3,122	.16	1.81[d]
3	OVERGEN X ACSTRESS	.0002	0.05	4,121	.02	0.23
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	LOCUS				.20	2.31[c]
	ACSTRESS	.04	5.05[b]	3,122	.20	2.28[c]
3	LOCUS X ACSTRESS	.01	1.65	4,121	.12	1.29

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; EASQ-ACGEN=Extended Attributional Style Questionnaire-Achievement Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .07$.

the cognitive style X achievement stress interaction terms approached significance, however. The results of analogous tests for subjects who perceived a lack of control over negative achievement events appear in Table 13. No significant main effects or interactions involving the achievement stress and cognitive style variables were obtained. Thus, for negative achievement events, no evidence was obtained to support the etiological status accorded to perceptions of uncontrollability in the reformulated learned helplessness theory.

Interpersonal Stress. Tables 14 and 15 present zero-order correlations among the cognitive style, interpersonal stress, and depressive symptom measures separately for subjects who perceived their most stressful interpersonal event as relatively controllable and uncontrollable, respectively. A very different pattern of findings emerged when the regression analyses were repeated for interpersonal stress. Among subjects who perceived negative interpersonal events as relatively controllable, only the stress, Irrational Ideas, and Locus of Control main effect variables predicted temporal increases in BDI scores (see Table 16). Among subjects who perceived a lack of control over negative interpersonal events, event stress as well as Attributional Style (Interpersonal Domain) were significant main effect predictors of T1 to T2 increases in BDI scores (see Table 17). More important, however, all but one of the cognitive style X interpersonal stress interaction terms accounted for a significant portion of the variance (ranging from three to eight percent) in residualized BDI scores. Although small in magnitude, the size of these interaction effects is comparable to that reported in similar investigations (e.g., Alloy &

Table 13

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Achievement Stress, Dysfunctional Cognitive Style, and Achievement Stress X Dysfunctional Cognitive Style Interactions Among Subjects Who Perceived Events as Uncontrollable (N = 100)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	EASQ-ACGEN				.12	1.16
	ACSTRESS	.02	1.56	3,96	.14	1.35
3	EASQ-ACGEN X ACSTRESS	.01	1.59	4,95	.13	1.26
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	DYSATT				.03	0.33
	ACSTRESS	.01	0.92	3,96	.13	1.32
3	DYSATT X ACSTRESS	.01	1.53	4,95	.13	1.24
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	IRRIDEAS				.18	1.81[d]
	ACSTRESS	.03	2.54[e]	3,96	.11	1.06
3	IRRIDEAS X ACSTRESS	.02	3.04[e]	4,95	.18	1.74[e]
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	OVERGEN				.08	0.81
	ACSTRESS	.02	1.20	3,96	.13	1.26
3	OVERGEN X ACSTRESS	.00001	0.001	4,95	-.003	-0.03
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	LOCUS				.02	0.21
	ACSTRESS	.01	0.89	3,96	.13	1.28
3	LOCUS X ACSTRESS	.01	1.49	4,95	.12	1.22

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; EASQ-ACGEN=Extended Attributional Style Questionnaire-Achievement Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .0001$. [d] $p = .07$. [e] $p = .08$.

Table 14

Intercorrelations Among Cognitive Style, Interpersonal Stress, and Depressive Symptomatology Among Subjects Who Perceived Events as Controllable (N = 111)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.61	-						
3. IPSTRESS	.35	.34	-					
4. EASQ-IPGEN	.44	.36	.27	-				
5. DYSATT	.50	.37	.24[a]	.49	-			
6. IRRIDEAS	.44	.44	.25	.38	.55	-		
7. OVERGEN	.66	.47	.36	.38	.51	.36	-	
8. LOCUS	.53	.46	.28	.44	.54	.42	.62	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; EASQ-IPGEN= Extended Attributional Style Questionnaire: Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS= Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .05$. [b] ns.

Table 15

Intercorrelations Among Cognitive Style, Interpersonal Stress, and Depressive Symptomatology Among Subjects Who Perceived Events as Uncontrollable (N = 100)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.68	-						
3. IPSTRESS	.35	.49	-					
4. EASQ-IPGEN	.36	.41	.28	-				
5. DYSATT	.38	.32	.26	.34	-			
6. IRRIDEAS	.45	.42	.21[a]	.27	.54	-		
7. OVERGEN	.52	.49	.28	.54	.48	.55	-	
8. LOCUS	.32	.23[a]	.20[a]	.17[b]	.46	.57	.33	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. BDI=Beck Depression Inventory; IPSTRESS= Interpersonal Event Stress; EASQ-IPGEN= Extended Attributional Style Questionnaire: Interpersonal Events Generality Composite; DYSATT= Dysfunctional Attitudes Scale; IRRIDEAS= Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .05$. [b] ns.

Table 16

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Interpersonal Stress, Dysfunctional Cognitive Style, and Interpersonal Stress X Dysfunctional Cognitive Style Interactions Among Subjects Who Perceived Events as Controllable (N = 111)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	EASQ-IPGEN				.09	0.93
	IPSTRESS	.02	1.77	3,107	.14	1.50
3	EASQ-IPGEN X IPSTRESS	.002	0.36	4,106	-.06	-0.60
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	DYSATT				.07	0.68
	IPSTRESS	.02	1.57	3,107	.15	1.58
3	DYSATT X IPSTRESS	.005	0.08	4.106	-.03	-0.29
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	IRRIDEAS				.22	2.37[c]
	IPSTRESS	.05	4.20[c]	3,107	.13	1.40
3	IRRIDEAS X IPSTRESS	.001	0.16	4,106	.04	0.41
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	OVERGEN				.08	0.86
	IPSTRESS	.02	1.71	3,107	.14	1.47
3	OVERGEN X IPSTRESS	.02	2.69	4,106	-.16	-1.64
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	LOCUS				.18	1.92[d]
	IPSTRESS	.04	3.22[c]	3,107	.14	1.45
3	LOCUS X IPSTRESS	.001	0.19	4,106	-.04	-0.43

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$.

Table 17

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Interpersonal Stress, Dysfunctional Cognitive Style, and Interpersonal Stress X Dysfunctional Cognitive Style Interactions Among Subjects Who Perceived Events as Uncontrollable (N = 107)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	EASQ-IPGEN				.19	1.98[c]
	IPSTRESS	.09	9.32[b]	3,103	.32	3.44[b]
3	EASQ-IPGEN X IPSTRESS	.04	8.46[b]	4,102	.28	2.91[b]
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	DYSATT				.02	0.25
	IPSTRESS	.07	7.13[b]	3,103	.34	3.69[b]
3	DYSATT X IPSTRESS	.02	3.60[d]	4,102	.18	1.90[d]
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	IRRIDEAS				.16	1.61
	IPSTRESS	.08	8.57[b]	3,103	.34	3.72[b]
3	IRRIDEAS X IPSTRESS	.02	4.13[c]	4,102	.20	2.03[c]
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	OVERGEN				.17	1.76[e]
	IPSTRESS	.08	8.84[b]	3,103	.33	3.60[b]
3	OVERGEN X IPSTRESS	.03	7.56[b]	4,102	.26	2.75[b]
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	LOCUS				-.02	-0.23
	IPSTRESS	.07	7.12[b]	3,103	.35	3.77[b]
3	LOCUS X IPSTRESS	.0002	0.04	4,102	.02	0.21

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$. [e] $p = .08$.

Clements, 1992; Metalsky et al., 1987; Metalsky & Joiner, 1992).

The unstandardized regression coefficients obtained for the four significant interaction terms in Table 17 were compared in series of *t*-tests with the unstandardized regression coefficients obtained for the corresponding interaction terms in Table 16. The purpose of these comparisons was to determine whether the cognitive style X stress interactions were reliably larger among subjects who perceived negative interpersonal events as uncontrollable than among subjects who perceived negative interpersonal events as controllable (cf. Williams, 1985). The results are presented in Table 18. Significant differences were obtained for the interactions involving Attributional Style (Interpersonal Domain) and Overgeneralization. Although both were in the predicted direction, the Interpersonal Stress X Dysfunctional Attitudes interaction was only marginally higher in the low control group and the Interpersonal Stress X Irrational Ideas interaction did not reliably differ in low and high in perceived control groups.

Despite the somewhat inconsistent results of these comparisons, an effort was made to clarify the nature of the four cognitive style X interpersonal stress interactions. Toward this end, the relationship between each of the cognitive style variables and residual T1 to T2 changes in BDI scores was evaluated at low, average, and high values of uncontrollable interpersonal stress. Using the procedures outlined by Cohen and Cohen (1983) and Jaccard et al. (1990), the slope of residualized BDI change scores on each cognitive style measure was calculated at one standard deviation below the mean, at the mean, and one standard deviation above the mean on the interpersonal stress

Table 18

Comparisons of Cognitive Style X Interpersonal Stress Regression Coefficients Obtained Among Subjects Who Perceived Events as Uncontrollable and Subjects Who Perceived Events as Controllable

Interaction Term	b_1	$S.E._1$	b_2	$S.E._2$	t	p[a]
EASQ-IPGEN X IPSTRESS	.013	.005	-.002	.004	2.34	<.025
DYSATT X IPSTRESS	.003	.001	-.0006	.002	1.61	<.06
IRRIDEAS X IPSTRESS	.006	.003	.002	.004	0.92	<.25
OVERGEN X IPSTRESS	.01	.005	-.01	.007	2.33	<.025

Note. b_1=Unstandardized regression coefficient obtained among subjects who perceived interpersonal events as uncontrollable (N = 107). $S.E._1$=Standard error of regression coefficient obtained among subjects who perceived interpersonal events as uncontrollable. b_2=Unstandardized regression coefficient obtained among subjects who perceived interpersonal events as controllable (N = 111). $S.E._2$=Standard error of regression coefficient obtained among subjects who perceived interpersonal events as controllable. $t = [(b_1 - b_2)/((S.E._1)^2 + (S.E._2)^2)]^{1/2}$. $df = N_1 + N_2 - 4$. IPSTRESS= Interpersonal Event Stress; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS= Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale.

[a] One-tailed.

measure among subjects low in perceived control. The results of these calculations appear in Table 19.

The form of the interaction was the same in each case. At low values of interpersonal stress, there was no relation between levels of dysfunctional cognition and residual changes in BDI scores (i.e., the regression coefficient did not differ from zero). At medium values of stress, significant positive relations emerged between residualized BDI change scores and scores on the Overgeneralization and Attributional Style (Interpersonal Domain) measures. At high values of stress, significant positive relations were obtained for all four measures of dysfunctional cognitive style. Among subjects who felt unable to control the occurrence of highly stressful interpersonal events, higher levels of dysfunctional cognition were associated with larger increases in levels of depressive symptomatology.

The present results provide evidence that dysfunctional cognitive styles render individuals vulnerable to depressive symptomatology, and support the general diathesis-stress framework that was made explicit in hopelessness theory. They do not, however, support the removal of perceived uncontrollability from the etiological model specified by hopelessness theorists. Increases in depressive symptomatology among individuals who possessed dysfunctional cognitive styles were observed only when highly stressful uncontrollable interpersonal events occurred. Analogous relations were not observed among subjects who experienced highly stressful but controllable interpersonal events.[10] This pattern of results is exactly what would be predicted by Abramson et al.'s (1978) reformulation of learned helplessness theory.

Table 19

Slopes of Residual BDI Scores on Cognitive Style Variables at Low, Medium, and High Levels of Uncontrollable Interpersonal Stress

Measure	b_{low}	t_{low}	b_{med}	t_{med}	b_{high}	t_{high}
EASQ-IPGEN	-.01	-0.13	.10	2.32[c]	.21	3.50[a]
DYSATT	-.04	-0.001	.001	0.96	.05	1.61[d]
IRRIDEAS	.00	0.0003	.08	1.68	.16	2.42[b]
OVERGEN	.06	0.66	.19	2.38[c]	.33	2.87[b]

Note. EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale.

[a] $p < .001$. [b] $p < .01$. [c] $p = .02$. [d] $p < .05$.

Hopelessness Theory's Specific Vulnerability Hypothesis

The specific vulnerability hypothesis put forth by hopelessness theory predicts that individuals who possess a depressogenic attributional style in a particular domain (e.g., the interpersonal domain) are at risk for developing depressive symptomatology only when they encounter stressors in the same domain (e.g., social stressors). The fact that, in the present investigation, subjects who tended to make stable and global attributions for negative interpersonal outcomes increased in levels of depressive symptomatology when they also experienced high levels of interpersonal stress is consistent with this hypothesis, but does not by itself provide unequivocal support for it.

In order to demonstrate the specificity of the interactive relation between attributional style and stress, it must be shown that 1.) individuals who possess a depressogenic attributional style for negative interpersonal outcomes do not exhibit increases in depressive symptomatology under conditions of elevated achievement stress, and 2.) individuals who possess a depressogenic attributional style for negative achievement outcomes do not exhibit increases in depressive symptomatology under conditions of elevated interpersonal stress.

The specific-vulnerability hypothesis was tested in two additional regression analyses that evaluated the predictive utility of "incongruent" attributional style X stress interactions among subjects who perceived negative life events as uncontrollable. As shown in the upper panel of Table 20, the interaction of Achievement Stress and Attributional Style (Interpersonal Domain) was not significant. Consistent with the specific vulnerability hypothesis, the tendency to

Table 20

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Domain-Incongruent Attributional Style X Stress Interactions Among Subjects Who Perceived Events as Uncontrollable (N = 107)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	EASQ-IPGEN				.17	1.72
	ACSTRESS	.03	2.37	3,96	.12	1.21
3	EASQ-IPGEN X ACSTRESS	.02	2.94	4,95	.17	1.72
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	EASQ-ACGEN				.10	0.98
	IPSTRESS	.07	7.64[b]	3,103	.35	3.80[b]
3	EASQ-ACGEN X IPSTRESS	.03	5.91[c]	4,102	.23	2.43[c]

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; IPSTRESS=Interpersonal Event Stress; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; EASQ-ACGEN=Extended Attributional Style Questionnaire- Achievement Events Generality Composite.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

make stable and global attributions for negative interpersonal outcomes did not confer a risk for depressive symptomatology at high levels of achievement stress.

The results presented in the lower panel of Table 20, however, suggest a very different conclusion about the validity of the specific vulnerability hypothesis. Significant effects were found for both Interpersonal Stress and the interaction of Interpersonal Stress and Attributional style (Achievement Domain). The results of a slope analysis performed on that interaction revealed that, as the stressfulness of negative interpersonal events increased, the tendency to make stable and global attributions for negative achievement events became more strongly associated with increases in depressive symptoms.

This finding is of special interest in light of the fact that scores on all other cognitive style measures used in this research, with the exception of Locus of Control, also predicted increases in depressive symptoms at high levels of (uncontrollable) interpersonal stress. Stress stemming from the occurrence of negative achievement events did not moderate either the nature or the strength of any cognitive style-depression relation. Although each cognitive style measure was developed to assess a unique type of dysfunctional cognition, the diathesis-stress results reported thus far suggest some commonality that underlies these constructs and leaves individuals particularly susceptible to depressive symptoms in the face of interpersonal difficulties. The analyses described in the following section were conducted to more specifically address this suggestion.

Assessing the Unique Effects of the Diathesis-Stress Interactions

Although hopelessness theorists described various dysfunctional cognitive styles that might serve as vulnerability factors for depressive symptomatology, they left open the question of whether these cognitive diatheses were orthogonal. For example, when discussing tendencies to anticipate negative consequences and attribute personal deficiencies to the self when stressors occur, Abramson et al. (1989, p. 362) remarked that "We do not know whether such cognitive styles are independent of the hypothesized depressogenic attributional style."

In addition to the results reported above, the recent findings of Metalsky and Joiner (1992) suggest that the cognitive diatheses specified in the hopelessness model are not independent but reflect a higher-order vulnerability factor. When assessed in separate analyses, Metalsky and Joiner found that tendencies to make stable and global attributions for negative outcomes, expect adverse consequences to result from negative events, and attribute negative characteristics to the self when negative events occur each interacted with scores on a measure of life stress (the number of stressors recently experienced) to predict temporal increases in depressive symptomatology. When tested in the same analysis, none of the interaction terms attained significance, leading Metalsky and Joiner (p. 673) to speculate that "it was the variance shared by the three vulnerability factors that conferred a risk for depressive reactions. Thus, the variance shared by the three vulnerability factors may constitute a common 'core' liability."

In the same vein, the diathesis-stress interaction terms that reliably predicted changes in depressive symptoms in the present

research were assessed simultaneously to determine if they exert unique effects. The T1 BDI covariate was entered on the first step of this regression analysis, followed by the entry of five main effect terms on the second step: ratings of interpersonal stress and scores on the Overgeneralization, Attributional Style (Interpersonal Domain), Irrational Ideas, and Dysfunctional Attitude measures. All four cognitive style X interpersonal stress interaction terms entered the equation on the third step. Unique predictive utility would be attributed to each interaction term that continued to be significantly related to residualized BDI change scores after all other interaction terms were statistically controlled.

Table 21 shows a pattern of results similar to that reported by Metalsky and Joiner (1992). Of the main effects evaluated on the second step of the analysis, only interpersonal stress accounted for a significant portion (11%) of the variance in BDI change scores. None of the cognitive style main effect or interaction variables contributed unique variance to the prediction of BDI change scores. These results strengthen speculations that a higher-order cognitive vulnerability factor, which is tapped by a number of existing measures, contributes to the onset of depressive symptomatology under conditions of high (uncontrollable) interpersonal stress.

Hopelessness as a Mediator of the Relation Between Dysfunctional Cognitive Style and Depressive Symptomatology

As the proximal sufficient cause of depression in Abramson et al.'s (1989) revised model, hopelessness is believed to mediate the effects of all cognitive style and stress variables on depressive

Table 21

Regression Models Assessing the Unique Contribution of Each Significant Cognitive Style X Interpersonal Stress Interaction Among Subjects Who Perceived Events as Uncontrollable (N = 107)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	EASQ-IPGEN				.15	1.52
	DYSATT				-.09	-0.91
	IRRIDEAS				.13	1.33
	OVERGEN				.06	0.60
	IPSTRESS	.10	4.33[b]	6,100	.33	3.45[b]
3	EASQ-IPGEN X IPSTRESS				.16	1.59
	DYSATT X IPSTRESS				-.07	0.71
	IRRIDEAS X IPSTRESS				.06	0.60
	OVERGEN X IPSTRESS	.05	2.94[c]	10,96	.10	1.02

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

symptomatology. According to the model, individuals who possess dysfunctional cognitive styles become hopeless (i.e., come to believe that negative outcomes are likely to occur in the future and that nothing can be done to prevent their occurrence) under conditions of high stress and therefore exhibit elevated levels of depressive symptomatology. Thus the relation between dysfunctional cognitive style and depression hypothesized by Abramson et al. (1989), and demonstrated in the present investigation among subjects who perceived interpersonal stressors as uncontrollable, is thought to be an indirect one, mediated by the formation of hopelessness expectancies.

Support for this mediational hypothesis is very limited. Ironically, although future outcome and control expectancies have always played a key role in learned helplessness theorizing, empirical tests of these models (e.g., Cutrona, 1983; Follette & Jacobson, 1987; Metalsky et al., 1982; Metalsky et al., 1987; O'Hara, Rehm, & Campbell, 1982; Rothwell & Williams, 1983) frequently fail to include measures of hopelessness (but see Alloy & Clements, 1992; Andersen, 1990; Metalsky et al., 1993; Metalsky & Joiner, 1992; Riskind, Rholes, Brannon, Burdick, 1987 for exceptions). When future expectancies have been measured so that the mediational hypotheses of the hopelessness model could be evaluated, the results have not always been in line with predictions. Metalsky and Joiner (1992), for example, found that interactions of dysfunctional cognitive style and life stress continued to predict increases in depressive symptomatology after controlling for concurrent increases in hopelessness expectancies (see Alloy & Clements, 1992 for similar results). Metalsky et al. (1993), however, did find

that hopelessness accounted for the relation between negativistic attributional styles and depressive reactions among students who received failing grades on a midterm exam.

The regression analyses described below were conducted in an effort to determine whether hopelessness expectancies (as assessed by the BHS) mediate the interactive effects of dysfunctional cognitive style and stress that were observed in the present research. For each significant cognitive style X stress interaction effect, two regression equations were estimated to determine whether the conditions required for hopelessness mediation were satisfied (cf. Baron & Kenny, 1986; Metalsky & Joiner, 1992). The first analysis in each set tested whether the interaction of cognitive style and stress predicts T2 BHS scores after controlling for T1 BHS scores and the cognitive style and stress main effects (Condition 1). The second analysis in each set tested whether T2 BHS scores predict residualized increases in T2 BDI scores (Condition 2), and whether the cognitive style X stress interaction continues to predict BDI change scores after T2 BHS scores are controlled (Condition 3).

The mediating role of hopelessness expectancies predicted by Abramson et al. (1989) would be demonstrated if high levels of dysfunctional cognitive style in combination with high levels of uncontrollable interpersonal stress predicted increases in hopelessness, if increases in hopelessness were significantly associated with increases in depressive symptomatology, and if the cognitive style X stress interactions that previously predicted changes in depressive symptoms were no longer significant when hopelessness was included in

the model. Partial mediation would be demonstrated if Conditions 1 and 2 were satisfied and the interactive effects of cognitive style and stress on depressive symptoms were significantly reduced in strength (as opposed to being eliminated in the case of complete mediation) after controlling for hopelessness.

Tables 22 through 25 present the results of the mediational analyses. None of the cognitive style X stress interaction effects were mediated by hopelessness. Interactions involving Interpersonal Stress and Dysfunctional Attitudes, Irrational Ideas, and Overgeneralization failed to predict changes in BHS scores (Condition 1) and, although increases in hopelessness were reliably associated with increases in depressive symptomatology (Condition 2), those interaction terms continued to be significant predictors of BDI change scores after controlling for hopelessness (Condition 3). This pattern of findings suggests a direct relation between dysfunctional cognitive styles and depressive symptomatology under conditions of elevated interpersonal stress.

BHS change scores were predicted by the interaction of Interpersonal Stress and Attributional Style (Interpersonal Domain) (see Table 25). Removing the variance associated with hopelessness, however, had only a trivial effect on the strength of the relation between BDI change scores and the Interpersonal Stress X Attributional Style interaction. Table 26 shows that the regression coefficients corresponding to each interaction term did not significantly decrease in magnitude after T2 BHS scores were controlled. The development of hopelessness expectancies was not, therefore, responsible for the

Table 22

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Dysfunctional Attitudes and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		DYSATT				-.05	-0.55
		IPSTRESS	.04	3.35[c]	3,103	.25	2.59[b]
3		DYSATT X IPSTRESS	.001	0.13	4,102	.04	0.36
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		DYSATT				.02	0.25
		IPSTRESS	.07	7.02[b]	4,102	.34	3.66[b]
3		T2BHS	.12	32.36[a]	5,101	.49	5.69[a]
4		DYSATT X IPSTRESS	.01	3.71[d]	6,100	.19	1.93[d]

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; DYSATT=Dysfunctional Attitudes Scale; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$.

Table 23

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Irrational Ideas and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		IRRIDEAS				-.05	-0.56
		IPSTRESS	.04	3.36[c]	3,103	.25	2.59[b]
3		DYSATT X IPSTRESS	.01	0.73	4,102	.08	0.86
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		IRRIDEAS				.16	1.60
		IPSTRESS	.08	8.44[b]	4,102	.34	3.69[b]
3		T2BHS	.12	34.07[a]	5,101	.50	5.84[a]
4		IRRIDEAS X IPSTRESS	.01	3.41[e]	6,100	.18	1.85[e]

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; IRRIDEAS=Irrational Ideas Inventory; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$. [e] $p = .07$.

Table 24

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Overgeneralization and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		OVERGEN				.02	0.25
		IPSTRESS	.04	3.23[c]	3,103	.23	2.42[c]
3		OVERGEN X IPSTRESS	.01	2.31	4,102	.15	1.52
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		OVERGEN				.17	1.75[c]
		IPSTRESS	.08	8.72[b]	4,102	.33	3.58[b]
3		T2BHS	.11	31.90[a]	5,101	.49	5.65[a]
4		OVERGEN X IPSTRESS	.02	4.71[c]	6,100	.21	2.17[c]

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scales; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$. [e] $p = .07$.

Table 25

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Dysfunctional Attributional Style (Interpersonal Domain) and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		EASQ-IPGEN				.18	1.89[d]
		IPSTRESS	.06	5.08[b]	3,103	.20	2.08[c]
3		EASQ-IPGEN X IPSTRESS	.02	3.85[c]	4,102	.19	1.96[c]
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		EASQ-IPGEN				.19	1.96[c]
		IPSTRESS	.08	9.16[b]	4,102	.32	3.43[b]
3		T2BHS	.10	28.82[a]	5,101	.47	5.37[a]
4		EASQ-IPGEN X IPSTRESS	.02	5.38[d]	6,100	.23	2.32[c]

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$. [e] $p = .08$.

Table 26

Comparisons of Cognitive Style X Interpersonal Stress Regression Coefficients With those Obtained in Hopelessness Mediational Analysis (N = 107)

Interaction Term	b_1	$S.E._1$	b_2	$S.E._2$	t	p[a]
DYSATT X IPSTRESS	.003	.001	.002	.001	0.71	>.05
IRRIDEAS X IPSTRESS	.006	.003	.005	.003	0.24	>.05
OVERGEN X IPSTRESS	.01	.005	.009	.004	0.15	>.05
EASQ-IPGEN X IPSTRESS	.013	.005	.006	.003	1.21	>.05

Note. b_2=Unstandardized regression coefficient obtained in hopelessness mediational analysis. $S.E._2$=Standard error of regression coefficient obtained in hopelessness mediational analysis. $t = [(b_1 - b_2)/((S.E._1)^2 + (S.E._2)^2)]^{1/2}$. $df = N_1 + N_2 - 4$. IPSTRESS=Interpersonal Event Stress; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite.

[a] One-tailed.

increases in depressive symptomatology observed among subjects who possessed higher levels of dysfunctional cognitive style and experienced high levels of uncontrollable interpersonal stress.

Summary of Dysfunctional Cognitive Style Findings

Taken together, the results reported in this section provide little support for Abramson et al.'s (1989) predictions regarding the etiological role of dysfunctional cognitive styles in hopelessness depression. Scores on measures of Locus of Control, Overgeneralization, Attributional Style, Irrational Ideas, and Dysfunctional Attitudes failed to predict T1 to T2 increases in depressive symptomatology at high levels of either achievement or interpersonal stress. The reformulation's (Abramson et al., 1978) competing prediction that dysfunctional cognitive styles would be positively associated with depressive symptoms when negative outcomes were perceived as uncontrollable fared much better.

Among subjects who perceived a lack of control over negative interpersonal events, those with higher scores on measures of Overgeneralization, Attributional Style (Interpersonal Domain), Irrational Ideas, and Dysfunctional Attitudes exhibited larger increases in depressive symptomatology as the stressfulness of those events increased. Similar relations among dysfunctional cognitive styles, interpersonal stress, and depressive symptomatology were not observed for subjects who perceived negative events as controllable. These findings suggest that highly stressful experiences were not sufficient by themselves to trigger increases in depressive symptomatology among cognitively vulnerable individuals. Rather, dysfunctional cognition was

associated with temporal increases in depressive symptoms only when (interpersonal) stressors were perceived as uncontrollable.

Tests of the hopelessness model's supplemental diathesis-stress predictions likewise yielded little support. Consistent with the specific vulnerability hypothesis, the tendency to make stable and global attributions for negative interpersonal outcomes predicted increases in depression at high levels of (uncontrollable) interpersonal stress but not at high levels of (uncontrollable) achievement stress. The converse was not true, however, of attributional style in the achievement domain. Tendencies to make stable and global attributions for negative achievement events predicted increases in depression at high levels of (uncontrollable) interpersonal stress but not at high levels of (uncontrollable) achievement stress.

In addition, no justification was found for conceptualizing the three dysfunctional cognitive styles discussed by hopelessness theorists as independent vulnerability factors. When evaluated simultaneously, none of the previously significant cognitive style X interpersonal stress interaction terms contributed unique variance to the prediction of T1 to T2 change in depressive symptomatology levels. Assuming the adequate measurement of these constructs, tendencies to attribute negative outcomes to stable and global causes, expect adverse consequences to result from life stressors, and draw derogatory inferences about the self when negative events occur appear to be unique manifestations of a more global vulnerability factor rather than independent risk factors.

Finally, hopelessness expectancies did not mediate any of the

interactive effects of dysfunctional cognitive style and stress on depressive symptoms. All but one of the cognitive style X interpersonal stress interaction terms failed to predict changes in hopelessness expectancies. Although the Attributional Style (Interpersonal Domain) X Interpersonal Stress interaction was significantly related to hopelessness, it continued to be reliably associated with changes in levels of depressive symptomatology after controlling for hopelessness. In contrast to the expectations of hopelessness theorists, relations between dysfunctional cognitive styles and depression were direct rather than mediated.

Relations Between Dysfunctional Cognition and Event Inferences: Mediational Predictions of Hopelessness Theory

Hopelessness theory addresses itself to the question of why certain dysfunctional cognitive styles put individuals at risk for depression when negative life events occur. According to the theory, relations between maladaptive cognitive styles and depressive symptomatology are mediated by the inferences that cognitively vulnerable individuals make about the stressors they experience. Abramson et al. (1989) hypothesized that attributing specific negative events to stable and global causes, expecting those events to bring about undesired consequences, and inferring personal deficiencies when those events occur, each increase the likelihood of hopelessness and, in turn, hopelessness depression. Although they acknowledge that events themselves provide the "raw data" for the types of inferences that are made, Abramson et al. made the reasonable prediction that individuals who habitually make stable and global attributions, anticipate adverse

consequences, or view themselves as personally flawed when negative events occur, will be more likely than their counterparts to make corresponding inferences when particular stressful events occur. Thus, it is the propensity of cognitively vulnerable individuals to make hopelessness-inducing inferences about stressful life events that is believed to increase their risk of depressive symptomatology when such events occur.

The analyses described below evaluate mediational predictions concerning associations between dysfunctional cognitive styles and inferences made in response to the occurrence of uncontrollable interpersonal stressors. A series of hierarchical regression analyses were performed to assess the extent to which interactions of dysfunctional cognitive style and interpersonal stress predicted scores on event-based measures of attributional generality (i.e., the stable and global attributional composite), expected negative consequences, and inferred personal deficiencies among subjects low in perceived control. Because they have been implicated as etiological factors for depression (e.g., Janoff-Bulman, 1979), scores on measures of behavioral and characterological blame for uncontrollable interpersonal stressors also served as criterion variables in these analyses.

Separate regression equations were estimated to assess the predictive utility of interactions involving Interpersonal Stress and Attributional Style (Interpersonal Domain), Dysfunctional Attitudes, Irrational Ideas, and Overgeneralization. Event inference scores were regressed on the (centered) cognitive style and interpersonal stress main effect variables on the first step of each analysis. Cognitive

style X interpersonal stress interaction terms, formed from centered scores on their respective main effect variables, were always assessed on the second step of the analysis. Zero-order correlations among the cognitive style, interpersonal stress, and inference measures are presented in Table 27. In addition to being moderately correlated among themselves, scores on most event-inference measures were significantly positively associated with scores on each cognitive style measure expect the Dysfunctional Attitudes Scale and Irrational Ideas Inventory.

Hierarchical regression results for the Attributional Style (Interpersonal Domain) measure appear in Table 28. The main effects of both attributional style and interpersonal stress significantly predicted scores on each event-inference measure. Tendencies to make stable and global attributions for negative interpersonal outcomes were associated with higher scores on scales assessing stable and global event attributions, negative consequence expectancies, perceived deficiencies, and behavioral as well as characterological blame for uncontrollable interpersonal stressors. Between four and 10% percent of the variance in scores on these measures was predictable from the Attributional Style main effect variable. In addition, the more stressful subjects' uncontrollable interpersonal events were, the higher their scores were on each inference measure. Stress ratings were most strongly related to expectations about the negative consequences of uncontrollable interpersonal experiences, accounting for 22% of the variance in scores on that measure. Stress was only weakly related to behavioral blame, accounting for a marginally significant three percent of the variance in scores on that measure. The Attributional Style

Table 27

Intercorrelations Among Cognitive Style, Interpersonal Stress and Event-Cognition Measures Among Subjects Low In Perceived Control (N = 107)

Measure	1	2	3	4	5	6	7	8	9	10
1. EASQ-IPGEN-										
2. DYSATT	.34	-								
3. IRRIDEAS	.27	.54	-							
4. OVERGEN	.54	.48	.55	-						
5. IPSTRESS	.28	.26	.21[a]	.28	-					
6. IEQ-GEN	.37	.16[b]	.15[b]	.29	.38	-				
7. IEQ-CNS	.35	.10[b]	.21[a]	.31	.52	.48	-			
8. IEQ-DEF	.37	.15[b]	.14[b]	.32	.34	.26	.42	-		
9. IEQ-BBL	.35	.10[b]	.05[b]	.32	.26	.23[a]	.28	.80	-	
10. IEQ-CBL	.26	.08[b]	.08[b]	.27	.26	.18[b]	.18[b]	.78	.89	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; IPSTRESS= Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS= Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 28

Regression Models Predicting Inferences About Uncontrollable Negative Interpersonal Events from Interpersonal Stress, Attributional Style (Interpersonal Domain), and Interpersonal Stress X Attributional Style Interactions

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	IEQ-GEN	EASQ-IPGEN				.30	3.17[b]
		IPSTRESS	.22	14.53[a]	2,104	.31	3.32[b]
2		EASQ-IPGEN X IPSTRESS	.002	0.20	3,103	.04	0.45
1	IEQ-CNS	EASQ-IPGEN				.26	2.72[b]
		IPSTRESS	.32	24.29[a]	2,104	.47	5.42[a]
2		EASQ-IPGEN X IPSTRESS	.001	0.17	3,103	.04	0.41
1	IEQ-DEF	EASQ-IPGEN				.31	3.31[b]
		IPSTRESS	.20	12.95[a]	2,104	.27	2.80[b]
2		EASQ-IPGEN X IPSTRESS	.0003	0.03	3,103	-.02	-0.18
1	IEQ-BBL	EASQ-IPGEN				.30	3.19[b]
		IPSTRESS	.15	9.17[a]	2,104	.18	1.87[d]
2		EASQ-IPGEN X IPSTRESS	.002	0.22	3,103	-.05	-0.47
1	IEQ-CBL	EASQ-IPGEN				.21	2.15[c]
		IPSTRESS	.11	6.13[a]	2,104	.20	2.07[c]
2		EASQ-IPGEN X IPSTRESS	.00	0.00	3,103	.001	0.01

Note. EASQ-IPGEN=Extended Attributional Style Questionnaire-Interpersonal Events Generality Composite; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$.

(Interpersonal Domain) X Interpersonal Stress interaction was unrelated to scores on all of the inference measures examined. The hopelessness model's mediational hypothesis regarding the attributional diathesis was not supported. Tendencies to make stable and global attributions for negative interpersonal outcomes were associated with negativistic inferences for uncontrollable interpersonal events at all levels of stress.

Table 29 presents regression results for the Dysfunctional Attitudes Scale. Moderate to strong positive associations were once again observed between stress ratings and scores on all inference measures. However, neither the main effect of Dysfunctional Attitudes nor the Dysfunctional Attitudes X Interpersonal Stress interaction effect accounted for a significant portion of the variance in scores on any event inference measure. An identical pattern of results was obtained in the analyses involving scores on the Irrational Ideas Inventory (see Table 30).

As shown in Table 31, scores on the Overgeneralization main effect variable were, along with stress ratings, significant predictors of the inferences subjects made about uncontrollable negative interpersonal events. Stronger tendencies to magnify the implications of negative events were associated with higher scores on all measures of depressogenic inferences across all levels of stress. Like the attributional diathesis, the positive relation between Overgeneralization and inferences did not vary as a function of the stressfulness of negative uncontrollable interpersonal events.

Hypothesized relations between dysfunctional cognitive styles and

Table 29

Regression Models Predicting Inferences About Uncontrollable Negative Interpersonal Events from Interpersonal Stress, Dysfunctional Attitudes, and Interpersonal Stress X Dysfunctional Attitudes Interactions

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	IEQ-GEN	DYSATT				.07	0.68
		IPSTRESS	.15	8.92[b]	2,104	.35	3.85[b]
2		DYSATT X IPSTRESS	.01	0.91	3,103	.09	0.95
1	IEQ-CNS	DYSATT				-.05	-0.49
		IPSTRESS	.27	19.40[a]	2,104	.51	6.12[a]
2		DYSATT X IPSTRESS	.01	0.97	3,103	.10	0.98
1	IEQ-DEF	DYSATT				.07	0.72
		IPSTRESS	.12	7.04[b]	2,104	.31	3.37[b]
2		DYSATT X IPSTRESS	.0001	0.01	3,103	.01	0.10
1	IEQ-BBL	DYSATT				.03	0.31
		IPSTRESS	.07	3.77[c]	2,104	.24	2.55[b]
2		DYSATT X IPSTRESS	.001	0.08	3,103	.03	0.28
1	IEQ-CBL	DYSATT				.02	0.17
		IPSTRESS	.07	3.68[c]	2,104	.24	2.57[b]
2		DYSATT X IPSTRESS	.0002	0.02	3,103	.01	0.14

Note. DYSATT=Dysfunctional Attitudes Scale; IPSTRESS= Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

Table 30

Regression Models Predicting Inferences About Uncontrollable Negative Interpersonal Events from Interpersonal Stress, Irrational Ideas, and Interpersonal Stress X Irrational Ideas Interactions

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	IEQ-GEN	IRRIDEAS				.08	0.83
		IPSTRESS	.15	9.05[b]	2,104	.36	3.92[b]
2		IRRIDEAS X IPSTRESS	.00	0.002	3,103	.004	0.04
1	IEQ-CNS	IRRIDEAS				.12	1.26
		IPSTRESS	.28	20.32[a]	2,104	.50	5.87[a]
2		IRRIDEAS X IPSTRESS	.01	1.59	3,103	.12	1.26
1	IEQ-DEF	IRRIDEAS				.08	0.78
		IPSTRESS	.12	7.09[b]	2,104	.32	3.45[b]
2		IRRIDEAS X IPSTRESS	.002	0.21	3,103	.05	0.46
1	IEQ-BBL	IRRIDEAS				-.002	-0.02
		IPSTRESS	.07	3.72[c]	2,104	.25	2.68[b]
2		IRRIDEAS X IPSTRESS	.00	0.001	3,103	.004	0.04
1	IEQ-CBL	IRRIDEAS				.03	0.26
		IPSTRESS	.07	3.70[c]	2,104	.25	2.60[b]
2		IRRIDEAS X IPSTRESS	.0005	0.05	3,103	.02	0.23

Note. IRRIDEAS=Irrational Ideas Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

Table 31

Regression Models Predicting Inferences About Uncontrollable Negative Interpersonal Events from Interpersonal Stress, Overgeneralization, and Interpersonal Stress X Overgeneralization Interactions

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	IEQ-GEN	OVERGEN				.21	2.17[c]
		IPSTRESS	.18	11.39[a]	2,104	.32	3.49[b]
2		OVERGEN X IPSTRESS	.001	0.02	3,103	-.01	-0.13
1	IEQ-CNS	OVERGEN				.20	2.05[c]
		IPSTRESS	.30	22.12[a]	2,104	.48	5.52[a]
2		OVERGEN X IPSTRESS	.004	0.54	3,103	.07	0.73
1	IEQ-DEF	OVERGEN				.25	2.60[b]
		IPSTRESS	.17	10.58[a]	2,104	.28	2.93[b]
2		OVERGEN X IPSTRESS	.00	0.0002	3,103	.001	0.01
1	IEQ-BBL	OVERGEN				.26	2.76[b]
		IPSTRESS	.13	7.80[b]	2,104	.19	1.96[c]
2		OVERGEN X IPSTRESS	.001	0.12	3,103	-.03	-0.34
1	IEQ-CBL	OVERGEN				.21	2.23[c]
		IPSTRESS	.11	6.33[b]	2,104	.20	2.05[c]
2		OVERGEN X IPSTRESS	.0000	0.002	3,103	.004	0.04

Note. OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; IPSTRESS=Interpersonal Event Stress; IEQ-GEN= Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

inferences about uncontrollable interpersonal stressors failed to receive support in all tests of the hopelessness model's mediational component. Although higher attributional style and overgeneralization scores were associated with increases in depressive symptomatology among subjects who experienced highly upsetting uncontrollable interpersonal stressors, the inferences made about those events were not predictable by any of the cognitive style X stress interactions. Dysfunctional cognitive style was either unrelated to interpersonal event inferences or positively related to inferences across all levels of interpersonal stress. In no case were maladaptive cognitions associated with negative inferences for only the most stressful of subjects' uncontrollable interpersonal experiences.

Relations Between Event Inferences and Depressive Symptomatology

Proximal Contributory Cause Predictions of Hopelessness Theory

The results described above suggest that maladaptive inferences did not mediate the dysfunctional cognition-depressive symptom relations observed among subjects who experienced high levels of uncontrollable interpersonal stress. The hopelessness model's proposed causal chain linking dysfunctional cognition to maladaptive inferences about negative life events, and maladaptive inferences to depressive symptoms (via hopelessness expectancies) was not, therefore, empirically confirmed. The failure to provide support for this etiological process does not, of course, nullify or in any way diminish the importance of the direct effects that dysfunctional cognitive styles had on increases in depressive symptomatology. Likewise, a lack of support for the mediational component does not preclude the possibility that maladaptive

inferences about highly upsetting events will affect levels of depressive symptoms independently of dysfunctional cognitive styles.

An additional set of analyses was therefore performed to evaluate the proximal contributory cause component of the hopelessness model. The APV procedure described previously was used to test the hypothesis that attributing highly upsetting life events to stable and global causes, expecting an array of negative consequences to result from those events, and/or making derogatory character inferences would be associated with increases in depressive symptomatology. Relations between depressive symptom levels and behavioral as well as characterological blame for life stressors were also explored. The etiological role of maladaptive inferences postulated by hopelessness theorists would be demonstrated if event-inference X stress interaction terms accounted for a significant portion of the variance in residualized BDI change scores. Such findings would indicate that higher scores on the inference measures were associated with increases in residual BDI change scores among subjects who experienced highly stressful negative life events.

Tables 32 and 33 present zero-order correlations among the depressive symptom, event-inference, and stress measures used in the analyses involving negative achievement and negative interpersonal events, respectively. Results of the achievement- and interpersonal-event regressions appear in Tables 34 and 35. Both achievement stress and interpersonal stress were positively related to residualized BDI change scores. In addition, significant main effects were obtained for achievement and interpersonal event attributional generality, negative

Table 32

Intercorrelations Among Achievement Stress, Achievement Event Inferences, and Depressive Symptomatology (N = 226)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.63	-						
3. ACSTRESS	.29	.31	-					
4. AEQ-GEN	.22	.34	.35	-				
5. AEQ-CNS	.28	.44	.49	.58	-			
6. AEQ-DEF	.35	.45	.30	.42	.51	-		
7. AEQ-BBL	.08[b]	.14[a]	.13[b]	.12[b]	.24	.50	-	
8. AEQ-CBL	.17[a]	.21	.08[b]	.20	.23	.64	.76	-

Note. Unless otherwise indicated, correlations are significant at the $p < .01$ level. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; AEQ-GEN=Achievement Events Questionnaire-Attributional Generality Subscale; AEQ-CNS= Achievement Events Questionnaire-Expected Consequences Subscale; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 33

Intercorrelations Among Interpersonal Event Inferences, Interpersonal Stress, and Depressive Symptomatology (N = 226)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.63	-						
3. IPSTRESS	.35	.40	-					
4. IEQ-GEN	.24	.38	.40	-				
5. IEQ-CNS	.33	.50	.50	.48	-			
6. IEQ-DEF	.25	.45	.36	.30	.45	-		
7. IEQ-BBL	.07[a]	.22	.28	.19	.23	.72	-	
8. IEQ-CBL	.12[a]	.28	.26	.20	.22	.77	.85	-

Note. Unless otherwise indicated, correlations are significant at the $p < .01$ level. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS= Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] ns.

Table 34

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Achievement Stress, Achievement Event Inferences, and Achievement Stress X Event Inference Interactions (N = 226)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	AEQ-GEN				.22	3.30[b]
	ACSTRESS	.05	8.96[b]	3,222	.10	1.50
3	AEQ-GEN X ACSTRESS	.003	1.14	4,221	.07	1.07
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	AEQ-CNS				.30	4.76[a]
	ACSTRESS	.07	15.01[a]	3,222	.02	0.34
3	AEQ-CNS X ACSTRESS	.001	0.37	4,221	-.04	-0.61
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	AEQ-DEF				.28	4.36[a]
	ACSTRESS	.06	13.13[a]	3,222	.11	1.65
3	AEQ-DEF X ACSTRESS	.002	0.80	4,221	-.06	-0.89
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	AEQ-BBL				.09	1.33
	ACSTRESS	.02	4.25[c]	3,222	.16	2.43[c]
3	AEQ-BBL X ACSTRESS	.01	2.14	4,221	.10	1.46
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	AEQ-CBL				.12	1.85[d]
	ACSTRESS	.03	5.10[b]	3,222	.17	2.55[b]
3	AEQ-CBL X ACSTRESS	.01	3.22[d]	4,221	.12	1.80[d]

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; AEQ-GEN=Achievement Events Questionnaire-Attributional Generality Subscale; AEQ-CNS=Achievement Events Questionnaire-Expected Consequences Subscale; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .07$.

Table 35

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Interpersonal Stress, Interpersonal Event Inferences, and Interpersonal Stress X Event Inference Interactions (N = 226)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	IEQ-GEN				.23	3.57[b]
	IPSTRESS	.06	12.84[a]	3,222	.14	2.11[c]
3	IEQ-GEN X IPSTRESS	.002	0.75	4,221	-.06	-0.87
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	IEQ-CNS				.32	5.03[a]
	IPSTRESS	.09	19.50[a]	3,222	.08	1.14
3	IEQ-CNS X IPSTRESS	.02	9.76[b]	4,221	.21	3.12[b]
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	IEQ-DEF				.33	5.21[a]
	IPSTRESS	.09	20.45[a]	3,222	.13	1.97[c]
3	IEQ-DEF X IPSTRESS	.00	0.00	4,221	-.001	-0.02
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	IEQ-BBL				.17	2.50[b]
	IPSTRESS	.05	9.43[b]	3,222	.18	2.74[b]
3	IEQ-BBL X IPSTRESS	.00	0.00	4,221	-.002	-0.04
1	T1 BDI	.40	146.39[a]	1,224	.63	12.10[a]
2	IEQ-CBL				.21	3.22[b]
	IPSTRESS	.06	11.59[a]	3,222	.18	2.74[b]
3	IEQ-CBL X IPSTRESS	.001	0.20	4,221	.03	0.45

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL= Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

consequences, and personal deficiencies, as well as behavioral and characterological blame for negative interpersonal events. However, with only one exception, neither set of analyses yielded any significant inference X stress interaction effects. Given the paucity of support for the hopelessness model's predictions, the significant stress and inference main effects that emerged from these analyses will not be further detailed nor will the single significant interaction be described.

Competing Predictions Derived From the Reformulated Theory of Learned Helplessness

The regression analyses described in the preceding paragraphs were repeated to evaluate predictions derived from the 1978 reformulation of learned helplessness theory about the etiological role of maladaptive inferences in the onset of depressive symptomatology. The reformulation postulated that expectations of future uncontrollability arise when individuals make internal, stable, and global attributions about the causes of uncontrollable negative outcomes. Although the reformulation limited itself to a discussion of the attributional precursors of helplessness depression, it would not be inconsistent with the logic of the 1978 model to predict that negative consequence expectancies, inferred personal deficiencies, and/or self-blame associated with uncontrollable negative events would also be predictive of increases in depressive symptomatology. After all, it is reasonable to expect that inferences of these nature, like internal, stable, and global causal attributions, would be associated with expectations that one will be unable to prevent the occurrence of highly undesired outcomes in the

future or unable to bring about the occurrence of highly desired outcomes.

Tests of these hypotheses were performed by examining the predictive utility of inference X stress interactions separately for subjects who perceived their most negative achievement event as relatively controllable (N = 126) or relatively uncontrollable (N = 100) and for subjects who perceived their most negative interpersonal event as either relatively controllable (N = 111) or relatively uncontrollable (N = 107). Median splits on the achievement and interpersonal control measures were again used to classify subjects into low and high perceived control groups. All other aspects of the analyses were the same as those previously described.

Achievement Stress. Zero-order correlations among the depressive symptom, inference, and stress variables appear in Table 36 for subjects who perceived achievement stressors as controllable and in Table 37 for subjects who perceived a lack of control over achievement stressors. An examination of the correlations in Tables 36 and 37 shows that relations among depressive symptoms, achievement stress, and event inferences were comparable in magnitude among subjects who perceived high and low levels of control over negative achievement events. Behavioral and characterological blame for uncontrollable achievement stressors were, however, more highly positively correlated with depressive symptoms and scores on most inference measures than were behavioral and characterological blame for controllable achievement stressors.

As shown in Tables 38 and 39, respectively, scores on the event inference measures did not interact with levels of achievement stress to

Table 36

Intercorrelations Among Achievement Event Inferences, Achievement Stress, and Depressive Symptomatology Among Subjects High in Perceived Control (N = 126)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.65	-						
3. ACSTRESS	.33	.35	-					
4. AEQ-GEN	.19[a]	.25	.30	-				
5. AEQ-CNS	.24	.25	.40	.47	-			
6. AEQ-DEF	.39	.44	.38	.44	.54	-		
7. AEQ-BBL	-.01[b]	.10[b]	.24	.09[b]	.31	.40	-	
8. AEQ-CBL	.09[b]	.15[b]	.12[b]	.19[a]	.24	.56	.67	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; AEQ-GEN=Achievement Events Questionnaire-Attributional Generality Subscale; AEQ-CNS= Achievement Events Questionnaire-Expected Consequences Subscale; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 37

Intercorrelations Among Achievement Event Inferences, Achievement Stress, and Depressive Symptomatology Among Subjects Low in Perceived Control (N = 100)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.62	-						
3. ACSTRESS	.23[a]	.25[a]	-					
4. AEQ-GEN	.23[a]	.38	.39	-				
5. AEQ-CNS	.31	.57	.56	.66	-			
6. AEQ-DEF	.33	.55	.23	.45	.53	-		
7. AEQ-BBL	.29	.43	.17[b]	.37	.46	.70	-	
8. AEQ-CBL	.37	.49	.15[b]	.40	.45	.81	.80	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; AEQ-GEN=Achievement Events Questionnaire-Attributional Generality Subscale; AEQ-CNS=Achievement Events Questionnaire-Expected Consequences Subscale; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 38

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Achievement Stress, Achievement Event Inferences, and Achievement Stress X Event Inference Interactions Among Subjects High in Perceived Control (N = 126)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	AEQ-GEN				.12	1.36
	ACSTRESS	.03	3.23[c]	3,122	.16	1.74[d]
3	AEQ-GEN X ACSTRESS	.005	1.12	4,121	.10	1.06
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	AEQ-CNS				.07	0.82
	ACSTRESS	.02	2.63[d]	3,122	.15	1.71[d]
3	AEQ-CNS X ACSTRESS	.0002	0.05	4,121	.02	0.21
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	AEQ-DEF				.21	2.41[c]
	ACSTRESS	.05	5.29[b]	3,122	.13	1.39
3	AEQ-DEF X ACSTRESS	.0002	0.04	4,121	-.02	-0.19
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	AEQ-BBL				.08	0.93
	ACSTRESS	.03	2.73[d]	3,122	.16	1.83[d]
3	AEQ-BBL X ACSTRESS	.005	1.01	4,121	.09	1.01
1	T1 BDI	.41	87.61[a]	1,124	.64	9.36[a]
2	AEQ-CBL				.09	1.05
	ACSTRESS	.03	2.85[d]	3,122	.18	2.04[c]
3	AEQ-CBL X ACSTRESS	.01	2.45	4,121	.14	1.57

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; AEQ-GEN=Achievement Events Questionnaire-Attributional Generality Subscale; AEQ-CNS=Achievement Events Questionnaire-Expected Consequences Subscale; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p < .09$.

Table 39

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Achievement Stress, Achievement Event Inferences, and Achievement Stress X Event Inference Interactions Among Subjects Low in Perceived Control (N = 100)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	AEQ-GEN				.28	2.89[b]
	ACSTRESS	.06	5.12[b]	3,96	.03	0.26
3	AEQ-GEN X ACSTRESS	.0001	0.01	4,95	.01	0.12
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	AEQ-CNS				.50	5.63[a]
	ACSTRESS	.16	17.02[a]	3,96	-.17	-1.69
3	AEQ-CNS X ACSTRESS	.004	0.87	4,95	-.10	-0.93
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	AEQ-DEF				.43	4.64[a]
	ACSTRESS	.12	11.80[a]	3,96	.07	0.67
3	AEQ-DEF X ACSTRESS	.004	0.80	4,95	-.09	-0.89
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	AEQ-BBL				.30	3.12[b]
	ACSTRESS	.07	5.81[b]	3,96	.10	1.01
3	AEQ-BBL X ACSTRESS	.007	1.22	4,95	.11	1.10
1	T1 BDI	.38	60.89[a]	1,98	.62	7.80[a]
2	AEQ-CBL				.33	3.47[b]
	ACSTRESS	.08	7.00[b]	3,96	.12	1.15
3	AEQ-CBL X ACSTRESS	.006	1.13	4,95	.11	1.06

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; AEQ-GEN=Achievement Events Questionnaire-Attributional Generality Subscale; AEQ-CNS=Achievement Events Questionnaire-Expected Consequences Subscale; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$.

predict increases in depressive symptomatology among subjects who perceived negative achievement events as relatively controllable or as relatively uncontrollable. It is interesting to note, however, that a different pattern of results obtained in the two sets of analyses. Higher scores on measures of attributional generality, expected negative consequences, behavioral blame, and characterological blame for uncontrollable achievement stressors, but not for controllable achievement stressors, were associated with increases in residualized BDI scores.

Interpersonal Stress. Tables 40 and 41 present zero-order correlations among the depressive symptom, inference, and stress variables for subjects who perceived negative interpersonal stressors as controllable and uncontrollable, respectively. The strength of interrelations among these measures was similar in both groups although depressive symptoms at T1 tended to be more highly correlated with the inferences subjects made about controllable rather than uncontrollable interpersonal stressors.

As can be seen in Table 42, scores on the attributional generality, negative consequence, personal deficiency, and characterological blame main effect variables were positively associated with residual T1 to T2 increases in depressive symptoms among subjects who experienced controllable interpersonal stressors. The main effect of interpersonal stress was nonsignificant, however, as were four of the five inference X interpersonal stress interaction effects tested. The only interaction effect to reliably predict residualized changes in BDI scores (accounting for 6% of the variance) was that between

Table 40

Intercorrelations Among Interpersonal Event Inferences, Interpersonal Stress, and Depressive Symptomatology Among Subjects High in Perceived Control (N = 111)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.61	-						
3. IPSTRESS	.35	.34	-					
4. IEQ-GEN	.30	.39	.43	-				
5. IEQ-CNS	.43	.53	.48	.49	-			
6. IEQ-DEF	.34	.49	.41	.36	.49	-		
7. IEQ-BBL	.09[b]	.18[b]	.37	.18[b]	.19[a]	.60	-	
8. IEQ-CBL	.16[b]	.29	.30	.24[a]	.25	.71	.78	-

Note. Unless otherwise indicated, all correlations are significant at the $p < .01$ level. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS= Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL= Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 41

Intercorrelations Among Interpersonal Event Inferences, Interpersonal Stress, and Depressive Symptomatology Among Subjects Low in Perceived Control (N = 107)

Measure	1	2	3	4	5	6	7	8
1. T1 BDI	-							
2. T2 BDI	.68	-						
3. IPSTRESS	.35	.49	-					
4. IEQ-GEN	.17[b]	.38	.38	-				
5. IEQ-CNS	.21[a]	.47	.52	.48	-			
6. IEQ-DEF	.14[b]	.38	.34	.26	.42	-		
7. IEQ-BBL	.07[b]	.22[a]	.26	.23[a]	.28	.80	-	
8. IEQ-CBL	.08[b]	.23[a]	.26	.18[b]	.18[b]	.78	.89	-

Note. Unless otherwise indicated, all correlations are significant at the $p < .01$ level. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS= Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL= Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 42

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Interpersonal Stress, Interpersonal Event Inferences, and Interpersonal Stress X Event Inference Interactions Among Subjects High in Perceived Control (N = 111)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	IEQ-GEN				.22	2.36[c]
	IPSTRESS	.05	4.19[c]	3,107	.07	0.70
3	IEQ-GEN X IPSTRESS	.01	1.51	4,106	-.12	-1.23
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	IEQ-CNS				.33	3.68[b]
	IPSTRESS	.08	8.25[b]	3,107	.02	0.17
3	IEQ-CNS X IPSTRESS	.03	6.38[b]	4,106	.24	2.53[b]
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	IEQ-DEF				.34	3.70[b]
	IPSTRESS	.08	8.36[b]	3,107	.04	0.41
3	IEQ-DEF X IPSTRESS	.004	0.82	4,106	-.09	-0.91
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	IEQ-BBL				.10	1.08
	IPSTRESS	.02	1.93	3,107	.11	1.13
3	IEQ-BBL X IPSTRESS	.001	0.12	4,106	-.03	-0.35
1	T1 BDI	.37	64.65[a]	1,109	.61	8.04[a]
2	IEQ-CBL				.20	2.09[c]
	IPSTRESS	.04	3.56[c]	3,107	.10	1.06
3	IEQ-CBL X IPSTRESS	.0003	0.06	4,106	.02	0.24

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL= Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

interpersonal stress and expected negative consequences.

A markedly different pattern of results emerged for subjects who perceived a lack of control over negative interpersonal events (see Table 43). Significant main effects were obtained for interpersonal stress as well as for attributional generality, negative consequences, and inferred personal deficiencies. Weaker and only marginally significant effects were obtained for scores on the behavioral and characterological blame main effect variables. In addition, scores on measures of expected negative consequences, inferred personal deficiencies, behavioral blame, and characterological blame interacted with levels of interpersonal stress to predict residual T1 to T2 increases in BDI scores (although interactions involving the latter two measures were only marginally significant).

Table 44 shows the results of comparisons between the interpersonal stress X event inference interactions obtained among subjects low and high in perceived control over negative interpersonal events. Only the interaction of interpersonal stress and perceived personal deficiencies was reliably larger among low control subjects. Results pertaining to the interactions of stress with behavioral and characterological blame were in the predicted direction but nonsignificant. Contrary to expectations, the unstandardized regression coefficient for the interpersonal stress X negative consequences interaction was nonsignificantly larger in the high control group than in the low control group.

Slope analyses (Cohen & Cohen, 1983; Jaccard et al., 1990) were performed to illustrate the relation between event inferences and

Table 43

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Interpersonal Stress, Interpersonal Event Inferences, and Interpersonal Stress X Event Inference Interactions Among Subjects Low in Perceived Control (N = 107)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	IEQ-GEN				.28	2.92[b]
	IPSTRESS	.10	11.93[a]	3,103	.25	2.67[b]
3	IEQ-GEN X IPSTRESS	.0001	0.03	4,102	.02	0.16
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	IEQ-CNS				.33	3.54[b]
	IPSTRESS	.12	14.23[a]	3,103	.17	1.76[d]
3	IEQ-CNS X IPSTRESS	.02	5.28[c]	4,102	.22	2.30[c]
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	IEQ-DEF				.31	3.27[b]
	IPSTRESS	.11	13.17[a]	3,103	.26	2.74[b]
3	IEQ-DEF X IPSTRESS	.03	6.68[b]	4,102	.25	2.59[b]
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	IEQ-BBL				.16	1.63
	IPSTRESS	.08	8.60[b]	3,103	.31	3.29[b]
3	IEQ-BBL X IPSTRESS	.01	3.16[e]	4,102	.17	1.78[d]
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	IEQ-CBL				.17	1.70
	IPSTRESS	.08	8.74[b]	3,103	.31	3.28[b]
3	IEQ-CBL X IPSTRESS	.01	3.05[d]	4,102	.17	1.75[d]

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .08$.

Table 44

Comparisons of Inference X Interpersonal Stress Regression Coefficients Obtained Among Subjects Who Perceived Events as Uncontrollable and Subjects Who Perceived Events as Controllable

Interaction Term	b_1	$S.E._1$	b_2	$S.E._2$	t	p[a]
IEQ-CNS X IPSTRESS	.039	.017	.063	.025	-0.80	<.25
IEQ-DEF X IPSTRESS	.050	.019	.022	.024	2.40	<.01
IEQ-BBL X IPSTRESS	.036	.020	.008	.023	1.47	<.08
IEQ-CBL X IPSTRESS	.035	.020	.006	.024	0.97	<.25

Note. b_1=Unstandardized regression coefficient obtained among subjects who perceived interpersonal stressors as uncontrollable (N = 107). $S.E._1$=Standard error of regression coefficient obtained among subjects who perceived interpersonal stressors as uncontrollable. b_2=Unstandardized regression coefficient obtained among subjects who perceived interpersonal stressors as controllable (N = 111). $S.E._2$=Standard error of regression coefficient obtained among subjects who perceived interpersonal stressors as controllable. $t = [(b_1 - b_2)/((S.E._2)^2 + (S.E._2)^2)]^{1/2}$. $df = N_1 + N_2 - 4$. BDI=Beck Depression Inventory; IPSTRESS= Interpersonal Event Stress; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF= Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] One-tailed.

residual T1 to T2 changes in BDI scores at low, average, and high values of uncontrollable interpersonal stress. The results of these analyses for the negative consequence, personal deficiency, behavioral blame, and characterological blame measures appear in Table 45. The form of the interaction was consistent across the four event inference measures. The inferences that subjects made about relatively unstressful interpersonal events (i.e., stress rating of one standard deviation below the mean) were unrelated to residualized BDI change scores (i.e., the slope of T2 BDI scores on each inference variable was not significantly different from zero, all ps > .10). Only the negative consequence and personal deficiency inferences that subjects made about interpersonal events that were average in stressfulness were positively related to residual T1 to T2 change scores (ps = .002 and .03, respectively). Among subjects who experienced highly upsetting uncontrollable interpersonal stressors (stress rating of one standard deviation above the mean), however, higher scores on measures of expected negative consequences, personal deficiencies, behavioral blame, and characterological blame were each reliably associated with increases in residualized BDI scores. Thus, as the stressfulness of uncontrollable negative interpersonal events increased, higher scores on measures of maladaptive event inferences became more strongly associated with residual T1 to T2 increases in depressive symptomatology.

Assessing the Unique Effects of the Inference X Stress Interactions

Given the moderate to strong intercorrelations that were observed among negative consequence expectancies, inferred personal deficiencies, behavioral blame, and characterological blame for uncontrollable

Table 45

Slopes of Residual BDI Scores on Event Inference Variables at Low, Medium, and High Levels of Uncontrollable Interpersonal Stress

Measure	b_{low}	t_{low}	b_{med}	t_{med}	b_{high}	t_{high}
IEQ-CNS	.29	0.91	.82	3.12[a]	1.38	3.66[a]
IEQ-DEF	-.08	-0.26	.59	2.15[b]	1.29	2.95[a]
IEQ-BBL	-.18	-0.51	.35	1.30	.91	2.22[b]
IEQ-CBL	-.19	-0.60	.28	1.07	.77	1.79[c]

Note. IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire -Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .01$. [b] $p < .05$. [c] $p > .05 < .10$.

interpersonal stressors (see Table 41), it is important to determine whether the stress X event inference interactions described above remain significant when assessed simultaneously. The independent effects of the interactions were therefore assessed by entering stress ratings along with scores on each inference measure on the second step of a hierarchical regression analysis, after the variance explainable by T1 BDI scores was removed on the first step. The four stress X event inference interactions were entered as a set on the third step of the analysis. Results are presented in Table 46.

As a set, the main effect variables accounted for 15% of the variance in residualized BDI change scores. Only negative consequence expectancies and inferred personal deficiencies were individually predictive, however, respectively explaining six and four percent of residual BDI score variance. The set of stress X event inference interaction terms did not contribute to the prediction of T2 BDI scores after T1 BDI scores, stress ratings, and the four event inference main effects were statistically controlled. These findings provide no evidence upon which to posit distinct roles of negative consequence expectancies, personal deficiency inferences, and self-blame in the etiology of depressive symptomatology.

Hopelessness as a Mediator of the Relation Between Event Inferences and Depressive Symptomatology

The final series of regression analyses to be reported in this section evaluated predictions about the role of hopelessness as a mediator of relations between depressive symptomatology and inferences about highly upsetting uncontrollable interpersonal stressors. Only

Table 46

Regression Model Assessing the Unique Contribution of Each Significant Event Inference X Interpersonal Stress Interaction Among Subjects Who Perceived Events as Uncontrollable (N = 107)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.44	83.63[a]	1,105	.67	9.15[a]
2	IEQ-CNS				.25	2.62[b]
	IEQ-DEF				.21	2.10[c]
	IEQ-BBL				-.12	-1.25
	IEQ-CBL				.06	0.57
	IPSTRESS	.15	7.40[b]	6,100	.14	1.41
3	IEQ-CNS X IPSTRESS				.13	1.33
	IEQ-DEF X IPSTRESS				.06	0.56
	IEQ-BBL X IPSTRESS				.02	0.23
	IEQ-CBL X IPSTRESS	.03	1.91	10,96	.03	0.25

Note. BDI=Beck Depression Inventory; IPSTRESS=Interpersonal Event Stress; IEQ-GEN=Interpersonal Events Questionnaire-Attributional Generality Subscale; IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

indirect associations between negativistic event inferences and depressive symptoms are predicted by hopelessness theorists. Such inferences are believed to be related to depression only insofar as they contribute to the development of hopelessness expectancies. In the context of the present research, support for this mediational hypothesis would be obtained by demonstrating that 1.) the interpersonal stress X event inference interactions that predicted T1 to T2 increases in depressive symptoms also predict T1 to T2 increases in hopelessness expectancies, 2.) T2 hopelessness expectancies are positively related to the severity of depressive symptoms exhibited at T2, and 3.) the interactive effects of interpersonal stress and event inferences on T2 depressive symptoms are eliminated (or diminished, in the case of partial mediation) when levels of T2 hopelessness are statistically controlled.

The results of regression analyses designed to evaluate these predictions appear in Tables 47 through 50. The analyses involving negative consequence expectancies and inferred personal deficiencies revealed no evidence of hopelessness mediation. As can be seen in Table 47, the residualized BHS change scores obtained by subjects who perceived a lack of control over negative interpersonal events were not predictable from the interaction of stress ratings and scores on the measure of negative consequence expectancies. Moreover, the Interpersonal Stress X Negative Consequence interaction continued to predict residualized BDI change scores with no significant reduction in magnitude (see Table 51) after hopelessness expectancies at T2 were controlled. Table 48 shows a similar pattern of results for the

Table 47

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Negative Consequence Expectancies and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		IEQ-CNS				.15	1.52
		IPSTRESS	.06	4.43[b]	3,103	.14	1.42
3		IEQ-CNS X IPSTRESS	.00	0.00	4,102	.00	0.01
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		IEQ-CNS				.33	3.53[b]
		IPSTRESS	.12	14.07[a]	4,102	.17	1.74
3		T2BHS	.10	29.09[a]	5,101	.47	5.39[a]
4		IEQ-CNS X IPSTRESS	.02	6.57[b]	6,100	.25	2.56[b]

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; IEQ-CNS=Interpersonal Events Questionnaire: Expected Consequences Subscale; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$.

Table 48

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Personal Deficiency Inferences and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		IEQ-DEF				.16	1.60
		IPSTRESS	.06	4.55[b]	3,103	.18	1.88[d]
3		IEQ-DEF X IPSTRESS	.01	1.38	4,102	.12	1.77
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		IEQ-DEF				.31	3.26[b]
		IPSTRESS	.11	13.01[a]	4,102	.26	2.71[b]
3		T2BHS	.10	28.83[a]	5,101	.47	5.37[a]
4		IEQ-DEF X IPSTRESS	.02	5.32[c]	6,100	.22	2.31[c]

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; IEQ-DEF=Interpersonal Events Questionnaire: Personal Deficiencies Subscale; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

Table 49

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Behavioral Blame and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		IEQ-BBL				.21	2.19[c]
		IPSTRESS	.07	5.73[b]	3,103	.19	1.94[d]
3		IEQ-BBL X IPSTRESS	.03	5.44[c]	4,102	.22	2.33[c]
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		IEQ-BBL				.16	1.62
		IPSTRESS	.08	8.48[b]	4,102	.31	3.26[b]
3		T2BHS	.11	29.36[a]	5,101	.47	5.42[a]
4		IEQ-BBL X IPSTRESS	.002	0.65	6,100	.08	0.81

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; IEQ-BBL=Interpersonal Events Questionnaire: Behavioral Blame Subscale; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .06$.

Table 50

Regression Model Testing the Role of Hopelessness as a Mediator of the Relation Between Characterological Blame and Depressive Symptomatology Among Subjects Who Perceived Negative Interpersonal Events as Uncontrollable (N = 107)

Step	Criterion	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T2BHS	T1BHS	.29	43.44[a]	1,105	.54	6.59[a]
2		IEQ-CBL				.14	1.45
		IPSTRESS	.05	4.30[c]	3,103	.20	2.10[c]
3		IEQ-CBL X IPSTRESS	.03	5.45[c]	4,102	.23	2.34[c]
1	T2BDI	T1BDI				.61	7.81[a]
		T1BHS	.44	41.54[a]	2,104	.04	0.37
2		IEQ-CBL				.17	1.69
		IPSTRESS	.08	8.61[b]	4,102	.31	3.26[b]
3		T2BHS	.11	30.15[a]	5,101	.48	5.49[a]
4		IEQ-CBL X IPSTRESS	.002	0.56	6,100	.07	0.75

Note. BHS=Beck Hopelessness Scale; BDI=Beck Depression Inventory; IEQ-CBL=Interpersonal Events Questionnaire: Characterological Blame Subscale; IPSTRESS=Interpersonal Event Stress.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

Table 51

Comparisons of Regression Coefficients for Event-Inference X Interpersonal Stress With Those Obtained In Hopelessness Mediational Analyses

Interaction Term	b_1	$S.E._1$	b_2	$S.E._2$	t	p
IEQ-CNS X IPSTRESS	.039	.017	.038	.015	0.43	>.25
IEQ-DEF X IPSTRESS	.050	.019	.040	.017	0.40	>.25
IEQ-BBL X IPSTRESS	.036	.020	.015	.019	0.71	<.25
IEQ-CBL X IPSTRESS	.035	.020	.014	.019	0.75	<.25

Note. b_2=Unstandardized regression coefficient obtained in hopelessness mediational analysis. $S.E._2$=Standard error of regression coefficient obtained in hopelessness mediational analysis. $t = [(b_1 - b_2)/((S.E._1)^2 + (S.E._2)^2)]^{1/2}$. $df = N_1 + N_2 - 4$. IPSTRESS=Interpersonal Event Stress. IEQ-CNS=Interpersonal Events Questionnaire-Expected Consequences Subscale; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

personal deficiency inference measure (see also Table 51).

Somewhat stronger support for hopelessness theory's mediational predictions was obtained when behavioral and characterological blame were assessed. With respect to the former, the Interpersonal Stress X Behavioral Blame interaction term accounted for a significant 5% of the variance in residualized BHS change scores (see Table 49). Higher levels of behavioral blame were associated with T1 to T2 increases in hopelessness among subjects who experienced very stressful uncontrollable negative interpersonal events. Increases in hopelessness were significantly associated with corresponding increases in depressive symptom severity-- T1 to T2 changes in BHS scores predicted a full 22% of the variance in BDI change scores. Finally, the Interpersonal Stress X Behavioral Blame interaction term that previously predicted BDI scores (albeit marginally, see Table 43) was no longer significant after T2 BHS scores were controlled. As shown in Table 50, an identical pattern of results emerged for characterological blame.

The three conditions required to establish hopelessness mediation (cf. Baron & Kenny, 1986) were therefore satisfied in the analyses involving behavioral and characterological blame. Subjects who perceived a lack of control over highly stressful interpersonal events but blamed either their actions or their characters for the occurrence of those events exhibited increases in hopelessness expectancies from T1 to T2. Those expectations about the future occurrence of uncontrollable negative outcomes served to increase the severity of depressive symptomatology experienced by those subjects. It is important to note, though, that the regression coefficients obtained for the behavioral and

characterological blame interaction terms in the mediational analyses on T2 BDI scores were not significantly different than those obtained in the analyses that did not control for T2 BHS scores (see Table 51).

Summary of Event Inference Findings

Once again, little support for predictions derived from hopelessness theory was found in the present data. Hypothesized relations between dysfunctional cognitive styles and inferences about negative life events, as well as those between event inferences and depressive symptomatology, were not empirically demonstrated. Although higher scores on measures of Attributional Style (Interpersonal Domain) and Overgeneralization were reliably associated with more depressogenic inferences for uncontrollable interpersonal stressors, none of the cognitive style variables specifically predicted inferences about the most upsetting uncontrollable interpersonal stressors.

Hopelessness theory's predictions regarding relations between event inferences and depressive symptomatology fared no better. Despite several significant stress and event-inference main effects, changes in depressive symptomatology were not reliably predicted by any event-inference X achievement or interpersonal stress interaction. When data from subjects high and low in perceived control were analyzed separately, somewhat stronger support was obtained for an alternative set of predictions derived from the learned helplessness reformulation. Levels of stress did not interact with inferences made about controllable or uncontrollable negative achievement events to predict depressive symptoms. In addition, all but one of the stress X interpersonal event inference interactions were unrelated to BDI scores

among subjects who perceived interpersonal stressors as controllable. The exception to this pattern occurred for negative consequence expectancies, which predicted increases in depressive symptoms among subjects who experienced highly stressful but controllable interpersonal events.

Among subjects low in control, however, interpersonal stress interacted with negative consequence expectancies, perceived personal deficiencies, behavioral blame, and characterological blame to predict depressive symptomatology. Inspection of the interactions revealed that the magnitude of positive associations between negativistic inferences and T1 to T2 changes in BDI scores increased as the stressfulness of subjects' uncontrollable interpersonal events also increased. However, the strength of the interactions involving behavioral and characterological blame was not significantly different in the high and low control groups and the interpersonal stress X negative consequences interaction was nonsignificantly larger in the high control group. It therefore seems appropriate to conclude that the interpersonal stress X negative consequences interaction predicted depressive symptoms among all subjects and only the interaction of interpersonal stress X personal deficiencies predicted depressive symptoms exclusively among subjects low in perceived control. The former favors the hopelessness model, and the latter, the reformulated model of learned helplessness.

Finally, analyses assessing the degree to which hopelessness expectancies mediated the observed relations between event inferences and depressive symptoms yielded few conclusive findings. Hopelessness expectancies were not predicted by interactions of uncontrollable

interpersonal stress and either negative consequence expectancies or perceived personal deficiencies. Moreover, when the analyses on residualized BDI change scores were repeated using T2 BHS scores as controls, the predictive utility of these two interaction effects did not diminish in magnitude.

Results of the mediational analyses using behavioral and characterological blame as predictors were more ambiguous. Interpersonal stress interacted with scores on both measures to predict residual increases in hopelessness among subjects low in control and the two marginally significant stress X blame interaction effects on BDI change scores dropped out when hopelessness expectancies were statistically controlled. However, additional analyses revealed no difference in the strength of the behavioral and characterological blame interaction effects with and without the hopelessness covariate. Rather than applying a mediational interpretation to these findings, it may be more appropriate to conclude that behavioral and characterological blame are more highly related to hopelessness than to depressive symptoms among subjects who experienced highly upsetting uncontrollable interpersonal stressors.

Self-Esteem Deficits

A final set of analyses was conducted to evaluate hopelessness theory's revision of the predictions that the 1978 reformulation made about the cognitive precursors of self-esteem loss. The reformulated model postulated an exclusive association between self-esteem deficits and the internality-externality dimension of causal attributions. Attributing uncontrollable negative outcomes to internal causes

(regardless of the stability or globality of those causes) was linked to a state of personal helplessness characterized by the belief that others, but not the self, could control the occurrence of important future outcomes. Only individuals who felt personally helpless were believed to suffer self-esteem loss in addition to the other symptoms of helplessness depression.

A growing body of evidence indicating that internal attributions are sometimes associated with positive behavioral outcomes led to a restatement of these predictions in the hopelessness model. Self-esteem loss is now believed to occur among individuals who either attribute negative outcomes to internal, stable and global causes or ascribe derogatory characteristics to themselves when stressful life events occur. Abramson et al. (1988, p. 363) explicitly state that

> If people make internal, stable, global attributions, then they expect that others could attain the outcomes about which they feel hopeless and therefore would feel inadequate compared with others. In addition, lowered self-esteem should occur in cases of hopelessness depression when people have inferred negative characteristics about themselves that they view as important to their general self-concept and not remediable or likely to change.

The APV procedure described throughout this paper was employed again to test the alternative predictions that the hopelessness and reformulated learned helplessness models make about the cognitive precursors of self-esteem deficits. In each regression model estimated, event-inference and stress main effect variables, as well as inference X stress interaction terms, served as predictors of residualized changes in RSES scores from T1 to T2. The first set of regression equations tested interactions between achievement stress and composite internal, stable, and global attributions for an achievement stressor, internal

attributions alone, perceived personal deficiencies, behavioral blame, and characterological blame. These analyses were then performed separately for subjects who perceived high and low levels of control over their most upsetting achievement stressor. Parallel tests were conducted using the attributional and cognition measures corresponding to stressful interpersonal events.

The purpose of the analyses reported in this section was not to predict changes in self-esteem that occur independently of changes in depressive symptoms. The predictions made in both the reformulated theory of learned helplessness and the hopelessness model pertain to the conditions under which self-esteem deficits will accompany depressive symptoms rather than the conditions that give rise exclusively to self-esteem loss. The above quote from Abramson et al. (1988) illustrates this point from the perspective of hopelessness theory. Thus, although the overlap between depressive symptoms and self-esteem at T2 was substantial ($r = -.67$, $p < .01$), T2 BDI scores were not controlled in any of the self-esteem analyses.

Achievement Stress

Zero-order correlations among the self-esteem, achievement stress, and event-cognition measures used in the first set of analyses appear in Table 52. Note that although internal attributions were not related to self-esteem levels, higher levels of achievement stress, as well as higher scores on the internal, stable, and global attributional composite and on the personal deficiencies, behavioral blame, and characterological blame measures were reliably associated with RSES scores.

Table 52

Intercorrelations Among Achievement Event Inferences, Achievement Stress, and Self-Esteem (N = 226)

Measure	1	2	3	4	5	6	7	8
1. T1 RSES	-							
2. T2 RSES	.76	-						
3. ACSTRESS	-.17[a]	-.23	-					
4. AEQ-INT	-.13[b]	-.11[b]	-.06[b]	-				
5. AEQ-ATTR	-.19	-.26	.25	.59	-			
6. AEQ-DEF	-.33	-.43	.30	.24	.42	-		
7. AEQ-BBL	-.16[a]	-.20[a]	.13[b]	.53	.12[b]	.50	-	
8. AEQ-CBL	-.21[a]	-.25[a]	.08[b]	.49	.20	.64	.76	-

Note. Unless otherwise indicated, all correlations are significant at the $p < .01$ level. RSES=Rosenberg Self-Esteem Scale; ACSTRESS=Achievement Event Stress; AEQ-INT=Achievement Events Questionnaire-Internality Subscale; AEQ-ATTR=Achievement Events Questionnaire-Internal, Stable, and Global Composite; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

RSES scores were more stable across assessment sessions than were BDI scores (see Table 53). Nearly 60% of the variance in T2 RSES scores was predictable from T1 RSES scores, compared to the 40% of T2 BDI variance that was predictable from T1 BDI scores. This should not be surprising though, given that core beliefs about the self are more stable than depressed mood. Achievement stress, as well as the internal, stable, and global attributional composite, personal deficiency, and characterological blame main effect variables significantly predicted portions (ranging from 1-7%) of the remaining variance in T2 RSES scores. Higher scores on each of these measures were associated with T1 to T2 decreases in self-esteem. The internal attribution main effect was not significantly related to residualized RSES scores.

Only the characterological blame X achievement stress interaction was reliably associated with changes in self-esteem, accounting for two percent of the variance in residualized RSES scores. A slope analysis of this interaction revealed a significant negative relationship between characterological blame and self-esteem only among subjects who experienced the most stressful negative achievement events. Finally, it should be noted that weak and marginally significant effects were also observed for the interactions of achievement stress with composite internal, stable, and global attributions ($p = .10$) and with behavioral blame ($p = .09$).

The results of analyses performed separately for subjects who perceived high and low levels of control over their most stressful achievement events revealed few differences between groups (see Tables

Table 53

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized RSES Scores from Achievement Stress, Achievement Event Inferences, and Achievement Stress X Event Inference Interactions (N = 226)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	AEQ-ATTR				-.16	-2.37[c]
	ACSTRESS	.02	5.52[b]	3,222	-.12	-1.76
3	AEQ-ATTR X ACSTRESS	.01	2.78	4,221	-.11	-1.67
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	AEQ-INT				-.03	-0.41
	ACSTRESS	.01	2.74	3,222	-.15	-2.33[c]
3	AEQ-INT X ACSTRESS	.0001	0.07	4,221	-.02	-0.26
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	AEQ-DEF				-.27	-4.20[a]
	ACSTRESS	.04	11.67[a]	3,122	-.08	-1.19
3	AEQ-DEF X ACSTRESS	.001	0.45	4,221	-.05	-0.67
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	AEQ-BBL				-.11	-1.61
	ACSTRESS	.01	3.98	3,222	-.14	-2.14
3	AEQ-BBL X ACSTRESS	.01	2.97	4,221	-.12	-1.72
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	AEQ-CBL				-.14	-2.09[c]
	ACSTRESS	.02	4.90[b]	3,222	-.15	-2.23[c]
3	AEQ-CBL X ACSTRESS	.01	4.32[c]	4,221	-.14	-2.08[c]

Note. RSES=Rosenberg Self-Esteem Scale; ACSTRESS=Achievement Event Stress; AEQ-INT=Achievement Events Questionnaire-Internality Subscale; AEQ-ATTR=Achievement Events Questionnaire-Internal, Stable, and Global Composite; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p \leq .01$. [c] $p < .05$.

54 and 55 for zero-order correlations among the measures used in these analyses and Tables 56 and 57 for regression results). Although the attribution and event-cognition main effects were stronger predictors of residualized RSES change scores among subjects low in control, a significant achievement stress X composite attribution interaction emerged among subjects high in control and a marginally significant achievement stress X characterological blame interaction emerged among subjects low in control. Inspection of both interactions showed that event-cognition scores were negatively associated with residual RSES change scores at higher levels of stress.

Interpersonal Stress

A very different pattern of results occurred when residual changes in RSES scores were regressed on attributions and cognitions about negative interpersonal events (see Table 58 for intercorrelations and Table 59 for regression results). Collapsed across levels of perceived control, higher scores on measures of interpersonal stress, perceived personal deficiencies, behavioral blame, and characterological blame predicted residual decreases in self-esteem from T1 to T2. In addition, a highly significant interaction, accounting for four percent of the variance in RSES change scores, emerged between interpersonal stress and internal causal attributions.

Unexpectedly, examination of this interaction revealed a pattern opposite to that predicted by the reformulated model. Specifically, more internal attributions were associated with increases in self-esteem at higher levels of interpersonal stress. At first glance, the association between internal attributions for interpersonal stressors and increased

Table 54

Intercorrelations Among Achievement Event Inferences, Achievement Stress, and Self-Esteem Among Subjects High in Perceived Control (N = 126)

Measure	1	2	3	4	5	6	7	8
1. T1 RSES	-							
2. T2 RSES	.77	-						
3. ACSTRESS	-.25	-.30	-					
4. AEQ-INT	-.13[b]	-.07[b]	.04[b]	-				
5. AEQ-ATTR	-.17[b]	-.23[a]	.28	.52	-			
6. AEQ-DEF	-.45	-.50	.38	.12[b]	.44	-		
7. AEQ-BBL	-.21[a]	-.19[a]	.24	.44	.34	.40	-	
8. AEQ-CBL	-.19[a]	-.19[a]	.12[b]	.41	.40	.56	.67	-

Note. Unless otherwise indicated, all correlations are significant at the $p < .01$ level. RSES=Rosenberg Self-Esteem Scale; ACSTRESS=Achievement Event Stress; AEQ-INT=Achievement Events Questionnaire-Internality Subscale; AEQ-ATTR=Achievement Events Questionnaire-Internal, Stable, and Global Composite; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 55

Intercorrelations Among Achievement Event Inferences, Achievement Stress, and Self-Esteem Among Subjects Low in Perceived Control (N = 100)

Measure	1	2	3	4	5	6	7	8
1. T1 RSES	-							
2. T2 RSES	.76	-						
3. ACSTRESS	-.07[b]	-.13[b]	-					
4. AEQ-INT	-.17[b]	-.21[a]	-.05[b]	-				
5. AEQ-ATTR	-.22[a]	-.32	.28	.52	-			
6. AEQ-DEF	-.18[b]	-.35	.23[a]	.12[b]	.55	-		
7. AEQ-BBL	-.15[b]	-.30	.18[b]	.44	.48	.70	-	
8. AEQ-CBL	-.26[a]	-.40[a]	.15[b]	.41	.51	.81	.80	-

Note. Unless otherwise indicated, all correlations are significant at the $p < .01$ level. RSES=Rosenberg Self-Esteem Scale; ACSTRESS=Achievement Event Stress; AEQ-INT=Achievement Events Questionnaire-Internality Subscale; AEQ-ATTR=Achievement Events Questionnaire-Internal, Stable, and Global Composite; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 56

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized RSES Scores from Achievement Stress, Achievement Event Inferences, and Achievement Stress X Event Inference Interactions Among Subjects High in Perceived Control (N = 126)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 RSES	.57	162.62[a]	1,124	.75	12.75[a]
2	AEQ-ATTR				-.11	-1.25
	ACSTRESS	.02	2.80	3,122	-.15	-1.63
3	AEQ-ATTR X ACSTRESS	.02	6.53[b]	4,121	-.23	-2.56[b]
1	T1 RSES	.57	162.62[a]	1,124	.75	12.75[a]
2	AEQ-INT				.05	0.58
	ACSTRESS	.01	2.15	3,122	-.18	-2.00
3	AEQ-INT X ACSTRESS	.001	0.22	4,121	-.04	-0.47
1	T1 RSES	.57	162.62[a]	1,124	.75	12.75[a]
2	AEQ-DEF				-.25	-2.81[b]
	ACSTRESS	.04	6.05[b]	3,122	-.10	-1.08
3	AEQ-DEF X ACSTRESS	.0002	0.05	4,121	-.02	-0.23
1	T1 RSES	.57	162.62[a]	1,124	.75	12.75[a]
2	AEQ-BBL				-.02	-0.17
	ACSTRESS	.01	2.00	3,122	-.17	-1.92
3	AEQ-BBL X ACSTRESS	.001	0.17	4,121	-.04	-0.41
1	T1 RSES	.57	162.62[a]	1,124	.75	12.75[a]
2	AEQ-CBL				-.06	-0.71
	ACSTRESS	.02	2.24	3,122	-.17	-1.94
3	AEQ-CBL X ACSTRESS	.003	0.85	4,121	-.08	-0.92

Note. RSES=Rosenberg Self-Esteem Scale; ACSTRESS=Achievement Event Stress; AEQ-INT=Achievement Events Questionnaire-Internality Subscale; AEQ-ATTR=Achievement Events Questionnaire-Internal, Stable, and Global Composite; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [d] $p = .07$.

Table 57

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized RSES Scores from Achievement Stress, Achievement Event Inferences, and Achievement Stress X Event Inference Interactions Among Subjects Low in Perceived Control (N = 100)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 RSES	.57	132.57[a]	1,98	.76	11.51[a]
2	AEQ-ATTR				-.23	-2.28[c]
	ACSTRESS	.03	3.27[c]	3,96	-.05	-0.50
3	AEQ-ATTR X ACSTRESS	.0004	0.09	4,95	.03	0.30
1	T1 RSES	.57	132.57[a]	1,98	.76	11.51[a]
2	AEQ-INT				-.14	-1.34
	ACSTRESS	.01	1.54	3,96	-.12	-1.22
3	AEQ-INT X ACSTRESS	.001	0.14	4,95	.04	0.37
1	T1 RSES	.57	132.57[a]	1,98	.76	11.51[a]
2	AEQ-DEF				-.31	-3.25[b]
	ACSTRESS	.05	5.98[b]	3,96	-.05	-0.44
3	AEQ-DEF X ACSTRESS	.003	0.64	4,95	-.08	-0.80
1	T1 RSES	.57	132.57[a]	1,98	.76	11.51[a]
2	AEQ-BBL				-.28	-2.87[b]
	ACSTRESS	.04	4.80[b]	3,96	-.07	-0.67
3	AEQ-BBL X ACSTRESS	.01	2.59	4,95	-.16	-1.61
1	T1 RSES	.57	132.57[a]	1,98	.76	11.51[a]
2	AEQ-CBL				-.30	-3.12[b]
	ACSTRESS	.04	5.56[b]	3,96	-.08	-0.75
3	AEQ-CBL X ACSTRESS	.01	3.37[d]	4,95	-.19	-1.84[d]

Note. RSES=Rosenberg Self-Esteem Scale; ACSTRESS=Achievement Event Stress; AEQ-INT=Achievement Events Questionnaire-Internality Subscale; AEQ-ATTR=Achievement Events Questionnaire-Internal, Stable, and Global Composite; AEQ-DEF=Achievement Events Questionnaire-Personal Deficiencies Subscale; AEQ-BBL=Achievement Events Questionnaire-Behavioral Blame Subscale; AEQ-CBL=Achievement Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .07$.

Table 58

Intercorrelations Among Interpersonal Event Inferences, Interpersonal Stress, and Self-Esteem (N = 226)

Measure	1	2	3	4	5	6	7	8
1. T1 RSES	-							
2. T2 RSES	.76	-						
3. IPSTRESS	-.26	-.31	-					
4. IEQ-INT	-.08[b]	-.08[b]	.07[b]	-				
5. IQ-ATTR	-.23	-.25	.35	.66	-			
6. IEQ-DEF	-.37	-.44	.36	.44	.48	-		
7. IEQ-BBL	-.19	-.26	.28	.60	.50	.72	-	
8. IEQ-CBL	-.26	-.30	.26	.60	.50	.77	.85	-

Note. Unless otherwise indicated, all correlations are significant at or beyond the $p < .01$ level. RSES=Rosenberg Self-Esteem Scale; IPSTRESS=Interpersonal Event Stress; IEQ-INT=Interpersonal Events Questionnaire-Internality Subscale; IEQ-ATTR=Interpersonal Events Questionnaire-Internal, Stable, and Global Composite; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 59

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized RSES Scores from Interpersonal Stress, Interpersonal Event Inferences, and Interpersonal Stress X Event Inference Interactions (N = 226)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	IEQ-ATTR				-.07	-0.99
	IPSTRESS	.01	3.74[c]	3,222	-.14	-2.12[c]
3	IEQ-ATTR X IPSTRESS	.01	3.06[d]	4,221	.12	1.75[d]
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	IEQ-INT				-.02	-0.30
	IPSTRESS	.01	3.26[c]	3,222	-.17	-2.53[b]
3	IEQ-INT X IPSTRESS	.01	6.93[b]	4,221	.17	2.63[b]
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	IEQ-DEF				-.22	-3.35[b]
	IPSTRESS	.03	9.00[b]	3,2222	-.10	1.50
3	IEQ-DEF X IPSTRESS	.00	0.001	4,221	.002	0.03
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	IEQ-BBL				-.13	-2.01[c]
	IPSTRESS	.02	5.31[b]	3,222	-.13	-2.00[c]
3	IEQ-BBL X IPSTRESS	.0002	0.13	4,221	.02	0.35
1	T1 RSES	.57	297.34[a]	1,224	.76	17.24[a]
2	IEQ-CBL				-.14	-2.04[c]
	IPSTRESS	.02	5.38[b]	3,222	-.14	-2.09[c]
3	IEQ-CBL X IPSTRESS	.0001	0.04	4,221	.01	0.21

Note. RSES=Rosenberg Self-Esteem Scale; IPSTRESS=Interpersonal Event Stress; IEQ-INT=Interpersonal Events Questionnaire-Internality Subscale; IEQ-ATTR=Interpersonal Events Questionnaire-Internal, Stable, and Global Composite; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$. [d] $p = .08$.

self-esteem appears to support the justification that hopelessness theorists provided for their revised self-esteem predictions. When considered together with a marginally significant ($p = .08$) interpersonal stress X attributional composite interaction that conformed to a similar pattern, however, it becomes clear that neither the hopelessness model nor the reformulation were supported.[11]

The results of parallel analyses performed separately for subjects who perceived high and low levels of control over interpersonal stressors showed that attribution-self-esteem relations similar to those found from the full sample emerged only among subjects who perceived interpersonal stressors as controllable (see Tables 60 and 61 for zero-order correlations among the measures used in these analyses and Tables 62 and 63 for regression results). Specifically, higher scores on the measure of internal attributions and on the internal, stable, and global composite were both associated with <u>increases</u> in self-esteem at higher levels of controllable interpersonal stress. Among subjects who perceived a lack of control over negative interpersonal events, only interpersonal stress levels predicted residualized changes in RSES scores. Subjects who experienced higher levels of uncontrollable interpersonal stress exhibited larger decreases in self-esteem from T1 to T2.

Tests of the self-esteem predictions made by the hopelessness and reformulated learned helplessness models yielded results that were counter to patterns previously observed in the present research. For example, tests of other hypotheses highlighted the role of uncontrollable interpersonal stressors and cognitions associated with

Table 60

Intercorrelations Among Interpersonal Event Inferences, Interpersonal Stress, and Self-Esteem Among Subjects High in Perceived Control (N = 111)

Measure	1	2	3	4	5	6	7	8
1. T1 RSES	-							
2. T2 RSES	.76	-						
3. IPSTRESS	-.34	-.28	-					
4. IEQ-INT	-.10[a]	-.12[a]	.08[a]	-				
5. IQ-ATTR	-.34	-.27	.39	.61	-			
6. IEQ-DEF	-.51	-.53	.41	.27	.44	-		
7. IEQ-BBL	-.28	-.31	.37	.48	.41	.60	-	
8. IEQ-CBL	-.33	-.38	.30	.48	.46	.71	.78	-

Note. Unless otherwise indicated, all correlations ares significant at the $p < .01$ level. RSES=Rosenberg Self-Esteem Scale; IPSTRESS=Interpersonal Event Stress; IEQ-INT=Interpersonal Events Questionnaire-Internality Subscale; IEQ-ATTR=Interpersonal Events Questionnaire-Internal, Stable, and Global Composite; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] ns.

Table 61

Intercorrelations Among Interpersonal Event Inferences, Interpersonal Stress, and Self-Esteem Among Subjects Low in Perceived Control (N = 112)

Measure	1	2	3	4	5	6	7	8
1. T1 RSES	-							
2. T2 RSES	.78	-						
3. IPSTRESS	-.19[b]	-.33	-					
4. IEQ-INT	-.06[b]	-.04[b]	.08[b]	-				
5. IQ-ATTR	-.15[b]	-.22[a]	.34	.63	-			
6. IEQ-DEF	-.26	-.36	.34	.45	.44	-		
7. IEQ-BBL	-.18[b]	-.23[a]	.26	.54	.47	.81	-	
8. IEQ-CBL	-.23[a]	-.24[a]	.26	.54	.43	.79	.89	-

Note. Unless otherwise indicated, all correlations are significant at the $p < .01$ level. RSES=Rosenberg Self-Esteem Scale; IPSTRESS=Interpersonal Event Stress; IEQ-INT=Interpersonal Events Questionnaire-Internality Subscale; IEQ-ATTR=Interpersonal Events Questionnaire-Internal, Stable, and Global Composite; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .05$. [b] ns.

Table 62

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized RSES Scores from Interpersonal Stress, Interpersonal Event Inferences, and Interpersonal Stress X Event Inference Interactions Among Subjects High in Perceived Control (N = 111)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 RSES	.57	146.10[a]	1,109	.76	12.09[a]
2	IEQ-ATTR				-.01	-0.06
	IPSTRESS	.001	0.14	3,107	-.05	-0.48
3	IEQ-ATTR X IPSTRESS	.02	6.29[b]	4,106	.24	2.51[b]
1	T1 RSES	.57	146.10[a]	1,109	.76	12.09[a]
2	IEQ-INT				-.06	-0.65
	IPSTRESS	.003	0.35	3,107	-.05	-0.49
3	IEQ-INT X IPSTRESS	.02	6.04[c]	4,106	.23	2.46[c]
1	T1 RSES	.57	146.10[a]	1,109	.76	12.09[a]
2	IEQ-DEF				-.24	-2.59[b]
	IPSTRESS	.03	3.50[c]	3,107	.02	0.26
3	IEQ-DEF X IPSTRESS	.0001	0.003	4,106	.006	0.06
1	T1 RSES	.57	146.10[a]	1,109	.76	12.09[a]
2	IEQ-BBL				-.14	-1.51
	IPSTRESS	.01	1.28	3,107	-.003	-0.04
3	IEQ-BBL X IPSTRESS	.00	0.001	4,106	.002	0.03
1	T1 RSES	.57	146.10[a]	1,109	.76	12.09[a]
2	IEQ-CBL				-.20	-2.08
	IPSTRESS	.02	2.31	3,107	-.01	-0.08
3	IEQ-CBL X IPSTRESS	.0002	0.06	4,106	-.02	-0.24

Note. RSES=Rosenberg Self-Esteem Scale; IPSTRESS=Interpersonal Event Stress; IEQ-INT=Interpersonal Events Questionnaire-Internality Subscale; IEQ-ATTR=Interpersonal Events Questionnaire-Internal, Stable, and Global Composite; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [c] $p < .05$.

Table 63

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized RSES Scores from Interpersonal Stress, Interpersonal Event Inferences, and Interpersonal Stress X Event Inference Interactions Among Subjects Low in Perceived Control (N = 112)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 RSES	.59	155.48[a]	1,110	.77	12.47[a]
2	IEQ-ATTR				-.06	-0.60
	IPSTRESS	.04	5.73[b]	3,108	-.27	-2.96[b]
3	IEQ-ATTR X IPSTRESS	.001	0.16	4,107	-.04	-0.40
1	T1 RSES	.59	155.48[a]	1,110	.77	12.47[a]
2	IEQ-INT				.15	1.60
	IPSTRESS	.05	6.95[b]	3,108	-.32	-3.48[b]
3	IEQ-INT X IPSTRESS	.002	0.73	4,107	.08	0.85
1	T1 RSES	.59	155.48[a]	1,110	.77	12.47[a]
2	IEQ-DEF				-.18	-1.86[d]
	IPSTRESS	.05	7.44[b]	3,108	-.25	-2.66[b]
3	IEQ-DEF X IPSTRESS	.004	1.08	4,107	-.10	-1.04
1	T1 RSES	.59	155.48[a]	1,110	.77	12.47[a]
2	IEQ-BBL				-.08	-0.83
	IPSTRESS	.04	5.91[b]	3,108	-.28	-3.05[b]
3	IEQ-BBL X IPSTRESS	.003	0.76	4,107	-.08	-0.87
1	T1 RSES	.59	155.48[a]	1,110	.77	12.47[a]
2	IEQ-CBL				-.04	-0.39
	IPSTRESS	.04	5.62[b]	3,108	-.29	-3.16[b]
3	IEQ-CBL X IPSTRESS	.001	0.32	4,107	-.05	-0.56

Note. RSES=Rosenberg Self-Esteem Scale; IPSTRESS=Interpersonal Event Stress; IEQ-INT=Interpersonal Events Questionnaire-Internality Subscale; IEQ-ATTR=Interpersonal Events Questionnaire-Internal, Stable, and Global Composite; IEQ-DEF=Interpersonal Events Questionnaire-Personal Deficiencies Subscale; IEQ-BBL=Interpersonal Events Questionnaire-Behavioral Blame Subscale; IEQ-CBL=Interpersonal Events Questionnaire-Characterological Blame Subscale.

[a] $p < .0001$. [b] $p < .01$. [d] $p = .07$.

those events in the etiology of dysphoric affect. The results of these tests generally favored predictions derived from the reformulation rather than those stemming from hopelessness theory. Self-esteem deficits, on the other hand, were more predictable from cognitions associated with highly stressful achievement events than from those associated with interpersonal stressors. Moreover, relations among achievement stressors, achievement-event cognitions, and self-esteem deficits were more in line with the hopelessness model's revised predictions than with the original 1978 predictions. Subjects who blamed something about their character (presumably an internal, stable, and global factor) for the occurrence of highly upsetting achievement stressors were the most likely to experience a loss of self-esteem from T1 to T2. In addition, this pattern was relatively stable among subjects who perceived high and low levels of control over their negative achievement events.

Analyses of interpersonal-event cognitions yielded findings that were contrary to the theoretical expectations of both models under consideration. Internal causal attributions (and to a lesser extent, composite internal, stable, and global causal attributions) were associated with T1 to T2 <u>increases</u> in self-esteem among subjects who experienced highly upsetting stressors in the interpersonal domain. Subsequent analyses showed that these relations were specific to the subgroup of subjects who perceived interpersonal stressors as controllable. Although the origin of this pattern is unclear,

cognitions about interpersonal stressors did not relate to changes in self-esteem in the ways suggested by either the reformulation or hopelessness theory.

III. Conclusions and Implications

The purpose of this research was to examine the etiological predictions of Abramson et al.'s (1988) hopelessness model of depression and concurrently, to evaluate the divergent postulates of the hopelessness and reformulated learned helplessness theories. Although the findings did not consistently favor one model over another, Abramson et al.'s (1978) learned helplessness reformulation generally received greater support than did the more recently proposed theory of hopelessness depression.

Perceived Control and Depressive Symptoms

The results of the present research particularly highlighted the importance of perceived uncontrollability (especially for negative interpersonal life events) in moderating relations between maladaptive cognitions and depressive symptoms. Tests of the hopelessness model's diathesis-stress and proximal contributory cause components failed to support Abramson et al.'s (1988) assertion that negative life events need not be perceived as uncontrollable in order to trigger the development of depressive symptoms. Collapsed across levels of perceived control, neither interpersonal stress nor achievement stress interacted with scores on measures of dysfunctional cognitive styles to predict changes in depressive symptomatology over time.

When data from subjects who perceived high and low levels of control over recent negative life events were analyzed separately, however, significant cognitive style X stress interactions were obtained

among subjects who perceived a lack of control over interpersonal stressors. Higher scores on measures of Overgeneralization, Attributional Style (Interpersonal Domain), Irrational Ideas, and Dysfunctional Attitudes were each associated with residual increases in depressive symptomatology among subjects who experienced higher levels of uncontrollable interpersonal stress. Similar patterns were <u>not</u> observed among subjects who perceived interpersonal stressors as controllable or among subjects who perceived achievement stressors as either controllable or uncontrollable.

Scores on most measures of negativistic event inferences likewise failed to predict residual changes in depression scores in interaction with the stressfulness of negative achievement and interpersonal events when the data from subjects who perceived high and low levels of control were combined (the one exception to this pattern occurred for negative consequence ratings, which predicted increases in depressive symptoms among subjects who experienced highly upsetting interpersonal stressors). When low and high control groups were examined separately, significant event inference X stress interactions emerged only among subjects who perceived low levels of control over negative interpersonal events (negative consequence ratings once again proved to be an exception to this pattern in interacting with levels of interpersonal stress to predict depressive symptomatology increases among subjects high in control). As the stressfulness of uncontrollable negative interpersonal events increased, higher levels of perceived personal deficiencies, behavioral blame, and characterological blame became more strongly associated with residual increases in depressive symptoms over

the three week study period.

Diathesis-stress findings similar to those obtained here have not been observed in other investigations of the interactive effects of dysfunctional cognition and life stress on depression. Although Abramson et al. (1978) accorded perceived uncontrollability a prominent role in the etiology of helplessness depression, researchers have not assessed subjects' perceptions of control over the stressors hypothesized to trigger the putative cognitive vulnerability factors. The neglect of perceived control in this line of research probably stems from a number of factors. As Abramson et al. (1989) acknowledge, the diathesis-stress portion of the reformulated learned helplessness theory was not made explicit in the 1978 presentation of the model. As a result, most research focused on main effect relations between attributional style and depressive symptoms (cf. Sweeney et al., 1986; see Cutrona, 1983; O'Hara et al., 1982; Manly, McMahon, Bradley, & Davidson, 1982 for exceptions). It was not until the publication of hopelessness theory that this component was clearly articulated. With the introduction of the revised model, however, perceived control was eliminated from the proposed etiological pathway leading to depressive symptoms. Thus, when researchers began more consistently evaluating diathesis-stress formulations, perceived control over negative life events was no longer relevant to the validity of the theoretical propositions under consideration.

In addition, rather than focusing on experiences with individual stressors, many diathesis-stress investigations examined interactions of hypothesized vulnerability factors and scores on cumulative indices of

stress (i.e., the total number of stressors experienced over a certain time period) (e.g., Andersen, 1990; Metalsky & Joiner, 1992). It is more difficult, and perhaps less meaningful, to examine perceptions of control over negative life events when such an approach is used. Finally, the diathesis-stress component implicit in Beck et al.'s (1979) cognitive theory of depression does not attribute etiological significance to perceptions of control over life stressors. As such, perceived control has not been examined in tests of Beck et al.'s (1979) diathesis-stress formulation (e.g., Olinger et al., 1987; Wise & Barnes, 1986).

The failure to assess perceptions of control over negative life events might partially account for the mixed findings obtained for diathesis-stress hypotheses. Supportive evidence has been obtained in some investigations (e.g., Andersen, 1990; Cutrona, 1983; Hamilton Sacks & Blunt Bugental, 1987; Metalsky et al., 1987; Metalsky & Joiner, 1992; Metalksy, Joiner, Hardin, & Abramson, 1993; Nolen-Hoeksema, Seligman, & Girgus, 1986; O'Hara, Rehm, & Campbell, 1982; Olinger et al., 1987; Wise & Barnes, 1986 [college student sample]) but not in others (e.g., Barnett & Gotlib, 1988; 1990; Follete & Jacobson, 1987; Manly et al., 1982; Metalsky et al., 1982; Persons & Roa, 1985; Wise & Barnes, 1986 [clinical sample]). That is, variations in perceptions of control among the subjects in these studies might have played a role in the differences in findings across studies. This possibility receives support from a growing body of evidence suggesting that event cognitions (e.g., causal attributions for specific negative life events) predict the onset of depressive symptoms only among individuals who perceive

negative events as uncontrollable. Noting the gradual deemphasis of perceived control in learned helplessness theory and research (e.g., Peterson & Seligman, 1984), as well as the inconsistent and weak relation between event attributions and depression that had emerged from previous research, Brown and Siegel (1988) assessed the degree to which perceptions of control over recent negative life events moderated the relation between causal attributions and depressive symptoms in a sample of normal adolescents. The results of their prospective investigation showed that, among subjects who perceived their most stressful event as uncontrollable (i.e., attributed it to an uncontrollable cause), higher scores on a composite index of internal, stable, and global causal attributions were associated with increases in depressed mood. In contrast, among subjects who perceived their most stressful event as controllable, those with higher scores on the attributional index exhibited <u>decreases</u> in depressed mood. Moreover, when subjects' stressfulness ratings of their most upsetting events were used in a similar analysis in place of perceived control scores, no interactive relation emerged. The latter suggests that the interactive effect of causal attributions and perceptions of control was not merely a function of the overlap between event upsettingness and perceived controllability.

Whereas Brown and Seigel (1988) measured perceptions of event uncontrollability by asking subjects to rate the controllability of the causes of their most stressful events, Benassi et al. (1991) included direct measures of perceived control over life stressors in two cross-sectional studies and observed an identical pattern of results (but see

Flett, Blankstein, & Kleinfeldt, 1990 for a failure to replicate this pattern). Evidence of a moderating role for perceived uncontrollability was also obtained by Pagel et al. (1985) in their analysis of depressive affect among spouse caregivers of Alzheimer's patients.

In Pagel et al.'s (1985) investigation, internal attributions for a spouse's prediagnosis symptoms interacted with control perceptions associated with a spouse's current problem behavior and recent illness-related life changes to predict both concurrent levels of depressed mood and increases in depressive symptomatology over time. Among subjects low in perceived control, internal attributions for a spouse's erratic behavior were associated with both high levels of current depressed affect and temporal increases in depressed mood. Internal attributions were either unrelated or negatively related to depressive symptoms among subjects who perceived more control over the consequences associated with a spouse's illness.

The results obtained in the Brown and Siegel (1988), Benassi et al. (1991), and Pagel et al. (1985) studies converge in their support of the reformulated theory of learned helplessness and are generally consistent with the present diathesis-stress and event-cognition findings. These findings also lend credibility to the suggestion made here that more consistent support for the interaction of dysfunctional cognitive styles and negative life events might have been obtained if the perceived controllability of life stressors had been assessed in previous diathesis-stress investigations. As Brown and Siegel (1988, p. 316) note, "...the ability of causal judgments to predict depression may be diluted or obscured if the causes of negative events are perceived as

controllable and perceptions of control are not taken into account." By analogy, the failure of researchers to examine perceptions of control over life stressors might account for the equivocal support that has heretofore been obtained for diathesis-stress hypotheses. The results of the present study underscore the need to assess perceptions of control over the stressors that are believed to trigger the development of depressive symptomatology in cognitively vulnerable individuals.

Before leaving the topic of perceived control, it is important to note that, shortly after the publication of Abramson et al's (1989) original model, hopelessness theory was revised and extended by Alloy, Kelly, Mineka, and Clements (1990). As implied by its name, Alloy et al.'s "helplessness-hopelessness model of anxiety and depression" seeks to account for the comorbidity of depressive and anxious symptoms within single illness episodes (intra-episode comorbidity) as well as the comorbidity of depressive and anxious episodes across the life span (lifetime comorbidity). The revision of particular relevance to the present discussion is the inclusion of controllability perceptions in the etiological sequence believed to lead to a hopelessness expectancy (now described as a highly certain negative outcome expectancy combined with a highly certain helplessness expectancy).[12,13] Whereas attributing important negative life events to stable and global causes gives rise to the negative outcome expectancy component of hopelessness (and therefore the symptoms of hopelessness depression), Alloy et al. (1990) suggested that a perceived lack of control over negative events fosters a helplessness expectancy which, in turn, leads to the development of depressive symptoms.

It might appear at first glance that, although troublesome to the original hopelessness model, the pattern of event cognition findings obtained in the present study and reported by Brown and Siegel (1988), Benassi et al. (1991), and Pagel et al. (1985) is consistent with Alloy et al.'s (1990) helplessness-hopelessness model. A close inspection of that model, however, shows that this is not the case. The proximal contributory cause component of Alloy et al.'s model regards control perceptions and causal attributions as independent rather than interactive etiological factors. That is, either attributing a negative life event to a stable and global cause or perceiving a lack of control over that event is believed to increase the likelihood of hopelessness expectancies and the subsequent development of depressive symptoms. Alloy et al. (1990) clearly do not suggest that helplessness expectancies stem from stable and global causal attributions that are made exclusively about uncontrollable stressors. As will be shown below, however, the link between hopelessness expectancies and depressive symptoms proposed by both Alloy et al. (1990) and Abramson et al. (1988) is actually better accounted for by an interaction of maladaptive event cognitions and perceptions of uncontrollability than by independent effects of event cognitions and control perceptions. When describing the proximal sufficient cause of hopelessness depression, Alloy et al. (1990, p. 511) note that

> Although helplessness [the expectation that one can not control the future occurrence of negative outcomes] is a necessary component of hopelessness, it is not sufficient to produce hopelessness. For hopelessness to develop, helplessness must be accompanied by a high degree of certainty about the expected occurrence of negative outcomes (Garber, Miller, & Abramson, 1980) [see also Abramson et al. (1988, p. 359)].

The linkages that Alloy et al. (1990) proposed between causal attributions and negative outcome expectancies, and between perceived uncontrollability and helplessness expectancies, do not logically follow from the above claim. If hopelessness expectancies are defined by the joint presence of a highly certain negative outcome expectancy and a highly certain helplessness expectancy, it is unclear how stable and global attributions can be associated with depressive symptoms via only the negative outcome expectancy component or how control perceptions can be associated with depression exclusively through the helplessness expectancy component.

If the formation of hopelessness expectancies was hypothesized to stem from both a perceived lack of control over negative outcomes and stable and global causal attributions, such a logical inconsistency would not exist. The perception of uncontrollability would account for the development of the helplessness expectancy whereas stable and global attributions would account for the development of the negative outcome expectancy. Thus, only an interactive relation between control perceptions and causal attributions is able to adequately explain the occurrence of both components of the proposed proximal sufficient cause of hopelessness depression.

Although Alloy et al.'s (1990) revision of hopelessness theory reintroduces perceptions of event uncontrollability back into the learned helplessness framework, it does so in a manner that is inconsistent with other central theoretical postulates. Careful consideration of the Abramson et al. (1978), Abramson et al. (1989), and Alloy et al. (1990) models suggests that the predictions made by the

former regarding the interactive effects of perceived control and causal attributions have not only been validated at the empirical level but are more theoretically sound.

One final aspect of Alloy et al.'s (1990) revision of hopelessness theory deserves comment here. The diathesis-stress portion of Alloy et al.'s model hypothesizes that a generalized tendency to perceive a lack of control over negative outcomes may render individuals vulnerable to becoming hopeless just as a negativistic attributional style does. Like similar predictions within the hopelessness framework, a "depressogenic perception of control style" (p. 514) is believed to increase the likelihood that particular negative life events will be perceived as uncontrollable and should, therefore, foster the development of helplessness expectancies only when negative life events are encountered. Alloy and Clements (1992) evaluated predictions derived from Alloy et al.'s (1990) expansion of hopelessness theory's diathesis-stress component and obtained generally supportive evidence. Specifically, they evaluated the hypothesis that individuals who exhibit an illusion of control (i.e., perceive a contingency between a particular response and the occurrence of a particular outcome when no such contingency exists) will be less likely than those with more accurate perceptions of response-outcome noncontingency to become hopeless and experience depressive symptoms in response to laboratory-induced and real-life stressors.

Subjects completed a standard judgment of contingency problem and then worked on an insoluble block-design task during the first phase of Alloy and Clements' (1992) investigation. Analysis of pre- to post-task

changes in scores on the MAACL hostility, anxiety, and depression subscales showed that exposure to the uncontrollable failure had no adverse effect on the affective experiences of subjects who exhibited an illusion of control on the contingency problem. In fact, levels of hostility, anxiety, and depressive affect among these subjects actually declined slightly following exposure to the insoluble problems. In contrast, subjects who more accurately perceived a lack of control on the contingency problem displayed residual increases in hostile, anxious, and depressive affect after working on the insoluble block-design task.

The second phase of Alloy and Clement's (1992) study was conducted when subjects returned one month later to complete measures of recent negative life events, event-related stress and discouragement (i.e., hopelessness), and depressive symptomatology. Phase one contingency judgments significantly predicted residual changes in levels of stress and discouragement stemming from the recent occurrence of negative life events. The more control subjects perceived over the response noncontingent outcome, the larger their phase one to phase two decreases were in event-based stress and discouragement. This effect emerged even after statistically controlling for phase one levels of perceived stress, discouragement, and depressive symptomatology.

In line with Alloy et al.'s (1990) diathesis-stress hypotheses, the interaction of subjects' phase one judgment of control scores and the number of negative life events experienced over the previous month was a significant predictor of residual changes in depressive symptomatology. Among subjects who recently experienced a large number

of life stressors, only those who failed to exhibit an illusion of control experienced an increase in depressive symptoms. Phase one to phase two increases in depressive symptom severity were not observed among the subset of highly stressed subjects who fell prey to the illusion of control.

Similar support for Alloy et al.'s (1990) contention that the tendency to perceive a lack of control over negative events predisposes individuals to depressive symptomatology was not obtained in the present research. Of the five cognitive vulnerability measures administered to subjects, only scores on Levenson's (1981) locus of control scale failed to interact with stress ratings to predict residual changes in depressive symptomatology among subjects who perceived a lack of control over negative interpersonal events (see Table 17). Subjects with an external locus of control orientation were no more likely than their internally-oriented counterparts to exhibit increases in depressive symptoms in the face of highly upsetting uncontrollable interpersonal stressors.

An examination of the alpha coefficient reliabilities in Table 1 shows that the internal consistency of the locus of control composite was equal or superior to that of the other cognitive vulnerability measures used in this research. Psychometric inadequacies do not, therefore, appear to account for the failure of locus of control orientation to predict residual depression scores. Thus, despite repeated demonstrations of a significant association between an external locus of control orientation and elevated levels of depressive symptoms (see Benassi, Sweeney, & Dufuor, 1988 for a comprehensive review of this

research), the current findings did not support an etiological role for locus of control orientation in the onset of depressive symptoms.

Specific Vulnerability and the Depressogenic Consequences of Life Stressors

Hopelessness theory's predictions regarding the domain specificity of attributional style and life stressor effects were also not supported in this study. Stronger tendencies to make stable and global attributions for negative interpersonal outcomes and for negative achievement outcomes were associated with T1 to T2 increases in depressive symptomatology among subjects who experienced highly upsetting uncontrollable interpersonal stressors. Neither attributional style in the achievement domain nor attributional style in the interpersonal domain interacted with the severity of subjects' uncontrollable achievement stressors to predict changes in levels of depressed mood.

These findings are inconsistent with the results obtained by Metalsky et al. (1987) in the only other study that explicitly evaluated hopelessness theory's diathesis-stress congruency hypothesis. Metalsky et al. found that attributional style for achievement outcomes, but not for interpersonal outcomes, predicted the severity of undergraduates' depressive reactions to a disappointing performance on a midterm examination. Tests of analogous specific vulnerability hypotheses put forth by Beck (1983) and by various psychodynamic theorists (e.g., Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982) have, however, yielded stronger congruency effects in the interpersonal domain than in the achievement domain (e.g., Hammen et al., 1985; Hammen, Ellicott, &

Gitlin, 1989; Robins, 1990, Study One; Robins & Block, 1988; Segal, Shaw, & Vella, 1989; Segal, Shaw, Vella, & Katz, 1992).

For example, Hammen et al. (1985) assessed levels of depressive symptomatology as well as experience with interpersonal and achievement stressors among undergraduates categorized as either dependent (i.e., an interpersonal domain vulnerability) or self-critical (i.e., an achievement domain vulnerability). Within-group comparisons revealed that, among dependent subjects, depressive symptomatology was more highly correlated with the severity of interpersonal stress experienced over the four month study period than with the severity of achievement event stress. The reverse pattern was not observed among the self-critical group. That is, levels of depressed mood among self-critical subjects were not differentially associated with the severity of achievement and interpersonal stress. In addition, the results of between-group comparisons showed that the magnitude of the interpersonal stress-depression association was larger among dependent subjects than it was among self-critical subjects. The magnitude of the achievement stress-depression relation did not reliably differ, however, in the dependent and self-critical groups.

In a more recent investigation, Segal et al. (1989) found that levels of interpersonal stress experienced over a six month period were significantly correlated with the severity of depressive symptoms and the likelihood of relapse among remitted depressives characterized as dependent. Moreover, relapse among these subjects was more likely to follow periods of increased interpersonal stress than periods of elevated achievement stress. Segal et al. failed to observe

corresponding patterns among a subset of remitted depressives characterized as self-critical.

The results of these and other studies have led researchers to speculate about possible differences between achievement and interpersonal stressors that might influence the strength and nature of their association with depressive symptoms among vulnerable individuals. Hammen et al. (1985) suggested that, because the base rate of negative achievement events is high in student populations, undergraduates might perceive the occurrence of those events as normative. If students do not regard negative achievement events as particularly diagnostic of their individual self-worth or competency level, experience with such events should have little adverse impact on mood. Taking a somewhat different perspective, Segal et al. (1992) pointed out that interpersonal stressors often take the form of discrete losses (e.g., the breakup of a romantic relationship) and result in highly salient disruptions in one's life whereas achievement stressors frequently reflect more gradual deteriorations in existing conditions (e.g., one poor performance on a course exam). As such, the threshold for depressive reactions might be lower for interpersonal stressors and/or the depressogenic effects of negative interpersonal events might become apparent more quickly after their occurrence. Achievement stress, on the other hand, might contribute to depressive symptomatology in an additive or cumulative fashion.

In the present study, high scores on all vulnerability measures (except locus of control) were associated with T1 to T2 increases in depressive symptomatology among individuals who experienced high levels

of uncontrollable interpersonal stress but not among those who experienced high levels of uncontrollable achievement stress. Like those obtained by Hammen et al. (1985) and Segal et al. (1992), these findings also suggest that important contextual and/or perceptual differences exist between achievement and interpersonal stressors. To better understand the nature of those differences, I compared subjects' perceptions of and inferences about their most upsetting T2 achievement and interpersonal stressors. Overall, there were no differences in the degree to which subjects perceived personal deficiencies, expected adverse consequences, or blamed their characters for the occurrence of negative achievement and interpersonal events (ps ranged from .19 to .43). Subjects perceived their negative achievement experiences as more upsetting ($p = .003$) and more internally caused ($p < .0001$) than their negative interpersonal experiences, and were more likely to blame their behavior for the former than for the latter ($p < .0001$). Interpersonal stressors, on the other hand, were perceived as less controllable ($p < .0001$) and as caused by factors that were more stable and global ($p < .0001$).

The results of these comparisons are inconsistent with the proposals offered by both Hammen et al. (1985) and Segal et al. (1992). With respect to the former, achievement stressors were not less likely than interpersonal stressors to be regarded as relevant to one's self-worth. With respect to the latter, subjects did not expect the consequences of interpersonal stressors to be any more severe than those of achievement stressors and actually regarded negative achievement events as more upsetting than negative interpersonal events.

Alternatively, these findings suggest that variations in control perceptions and causal attributions for interpersonal and achievement stressors were responsible for the differential associations between these events and depressive symptoms among vulnerable subjects. It is possible that highly uncontrollable experiences that are attributed to stable and global factors trigger increases in depressive symptoms among vulnerable individuals, regardless of the domain in which those experiences fall. This is, of course, consistent with the predictions of the reformulated learned helplessness model (expect for the fact the 1978 theory also discussed internal attributions as important to the etiology of depressive symptomatology). The finding that levels of dysfunctional cognitive styles interacted exclusively with stressfulness of negative interpersonal events might therefore have less to do with the interpersonal nature of those events than with the placement of those events on the control and attributional dimensions.

This line of reasoning implies that the stressfulness of negative achievement events would have combined with levels of dysfunctional cognitive style to predict changes in depressive symptoms if those events were perceived as uncontrollable as negative interpersonal events were. In an effort to evaluate this prediction, cognitive style X achievement stress interactions were examined among a subset of subjects (n = 56) whose perceived control ratings for their most upsetting T2 achievement stressors fell above 4.5 (the median perceived control rating for subjects' most upsetting T2 interpersonal stressors). As shown in Table 64, the results of regression analyses provided preliminary support for this prediction. Levels of achievement stress

Table 64

Regression Models Predicting Time 1 (T1) to Time 2 (T2) Change in Residualized BDI Scores from Achievement Stress, Dysfunctional Cognitive Style, and Achievement Stress X Dysfunctional Cognitive Style Interactions Among Subjects Who Provided Event Control Ratings Greater Than 4.5 (N = 56)

Step	Predictors in Set	R^2_{inc}	F_{inc}	df	pr	t
1	T1 BDI	.43	40.29[a]	1,54	.64	6.35[a]
2	EASQ-ACGEN				.08	0.58
	ACSTRESS	.03	1.44	3,52	.22	1.65
3	EASQ-ACGEN X ACSTRESS	.003	0.25	4,51	.07	0.50
1	T1 BDI	.43	40.29[a]	1,54	.64	6.35[a]
2	DYSATT				.13	0.97
	ACSTRESS	.04	1.76	3,52	.21	1.56
3	DYSATT X ACSTRESS	.03	2.79[d]	4,51	.23	1.67[d]
1	T1 BDI	.43	40.29[a]	1,54	.64	6.35[a]
2	IRRIDEAS				.31	2.36[b]
	ACSTRESS	.08	4.19[b]	3,52	.22	1.60
3	IRRIDEAS X ACSTRESS	.02	2.16[e]	4,51	.20	1.47[e]
1	T1 BDI	.43	40.29[a]	1,54	.64	6.35[a]
2	OVERGEN				.17	1.22
	ACSTRESS	.04	2.05	3,52	.21	1.56
3	OVERGEN X ACSTRESS	.004	0.38	4,51	-.09	-0.61
1	T1 BDI	.43	40.29[a]	1,54	.64	6.35[a]
2	LOCUS				-.03	-0.19
	ACSTRESS	.03	1.29	3,52	.22	1.60
3	LOCUS X ACSTRESS	.04	4.19[c]	4,51	.28	2.05[c]

Note. BDI=Beck Depression Inventory; ACSTRESS=Achievement Event Stress; EASQ-ACGEN=Extended Attributional Style Questionnaire-Achievement Events Generality Composite; DYSATT=Dysfunctional Attitudes Scale; IRRIDEAS=Irrational Ideas Inventory; OVERGEN=Overgeneralization Subscale of the Attitudes Toward Self Scale; LOCUS=Powerful Others and Chance Locus of Control Composite

[a] $p < .0001$. [b] $p = .01$. [c] $p < .05$. [d] $p = .10$. [e] $p < .15$.

interacted with Locus of Control scores and, marginally, with scores on the Irrational Ideas ($p = .15$) and Dysfunctional Attitudes ($p = .10$) scales. Further examination of these effects showed that as the stressfulness of highly uncontrollable achievement events increased, higher scores on all three measures were more strongly predictive of residual increases in levels of depressive symptoms. Similar relations were not observed for scores on the Overgeneralization and Attributional Style (Achievement Domain) measures.

These findings help to clarify the inconsistent results obtained for interpersonal and achievement stressors in the diathesis-stress and specific vulnerability analyses. They also reinforce conclusions drawn earlier regarding the importance of perceived uncontrollability to the onset of depressive symptomatology, and in doing so, again point to the theoretical superiority of the reformulated learned helplessness model over the hopelessness model.

The results of these supplemental analyses might also prove useful in efforts to determine why tests of specific vulnerability hypotheses have frequently yielded stronger support in the interpersonal domain. The recent focus on life stressors in achievement and interpersonal domains has advanced our understanding of the ways in which negative life events precipitate depressive symptomatology. However, the confounding of event domain with other characteristics such as controllability and causal attributions might ultimately serve to obscure the role that the latter factors play in the etiology of depression. As in the present study, future research might benefit by looking beyond event domain to the contextual and psychological

dimensions on which negative achievement and interpersonal events vary.

Event Cognitions as Mediators of Diathesis-Stress Effects

In their reformulation of learned helplessness theory, Abramson et al. (1978) regarded casual attributions for specific negative outcomes as the mechanisms through which a negativistic attributional style exerted its effects on depressive symptoms. Although hopelessness theorists (Abramson et al., 1989) expanded the domain of proximal contributory causes to include personal deficiency and negative consequence inferences, they did not modify the reformulation's hypotheses about the mediating role of specific event cognitions.

Tests of these mediational hypotheses failed to support the predictions from either model. Event cognition scores were not predictable from interactions of cognitive styles and levels of uncontrollable interpersonal stress. The failure of event cognitions to mediate the interactive relations observed among dysfunctional cognitive styles, uncontrollable interpersonal stress, and depressive symptoms is surprising in light of the fact that higher scores on most event cognition measures predicted increases in depressive symptomatology among subjects who perceived a lack of control over highly stressful interpersonal life events. Rather than playing a mediating role, maladaptive event cognitions appeared to have affected depressive symptoms independently of dysfunctional cognitive styles.

Most of the published research on hopelessness theory has focused exclusively on the model's diathesis-stress hypotheses (e.g., Alloy & Clements, 1992; Andersen, 1990; Metalsky & Joiner, 1992; Metalsky et al., 1993). Despite their prominent and more proximal association with

hopelessness expectancies and depressive symptoms, measures of event cognitions have only infrequently been included in studies that also assess dysfunctional cognitive styles (cf. Hammen, 1988). Mediational predictions specific to Abramson et al.'s (1978) reformulated learned helplessness theory have been evaluated, but only in several investigations (Cutrona, 1983; Follette & Jacobson, 1987; Metalsky et al., 1987). Although more definitive conclusions admittedly await additional research, the evidence collected to date provides, at best, limited support for these predictions.

For example, Cutrona (1983) examined associations between prepartum responses to the six negative outcomes on the Attributional Style Questionnaire (Peterson et al., 1982) and causal attributions for postpartum "maternal blues," highly upsetting child-care stressors, and daily life stressors. Although the ASQ scores of initially nondepressed women predicted levels of depressive symptomatology at two and eight weeks postpartum, attributional style was not significantly correlated with any of the postpartum measures of stressful event attributions. Moreover, two of Cutrona's three postpartum event attribution measures failed to predict levels of concurrent depressed mood, and prepartum ASQ scores remained significant predictors of postpartum depressive symptomatology after the effects of event attributions were statistically controlled.

Follette and Jacobson (1987) and Metalsky et al. (1987) did obtain significant correlations between scores on the Extended Attributional Style Questionnaire and causal attributions for exam performance among students who were disappointed with their exam grade (failure group).

However, in neither of these studies was the same correlation examined among students who were satisfied with their exam performance (success group). Such an approach does not provide a stringent test of mediational hypotheses that predict exclusive associations between dysfunctional cognitive styles and cognitions about highly stressful outcomes (cf. Alloy et al., 1988). Unequivocal support for these predictions requires that maladaptive attributions are made only by cognitively vulnerable individuals who experience a highly a stressful event (i.e., as indicated by a significant cognitive style X stress interaction). At a minimum, Follete and Jacobson (1987) and Metalsky et al. (1987) should have demonstrated that the magnitude of the attributional style-exam attribution relation was stronger in the high stress (failure) group than in the low stress (success) group.

In addition, the attributional style X exam stress interaction was not a reliable predictor of depressive symptoms in Follette and Jacobson's (1987) study. As such, the significant association that they observed between ASQ scores and exam attributions does not bear on the validity of the mediational predictions proposed by learned helplessness reformulators. Metalsky et al. (1987) did find that the interaction of attributional style X exam stress was a significant predictor of subsequent depressed mood. They also demonstrated that, among failure students, exam attributions accounted for a significant portion of the variance in depressive symptoms and that the ASQ scores of those students were no longer predictive of depressed mood after exam attributions were statistically controlled. Although Metalsky et al.'s (1987) findings are generally consistent with the predictions advanced

by the reformulated learned helplessness and hopelessness models, the small number of subjects used in their mediational analyses (n = 23) and the failure to observe a significant relation between exam attributions and depressive symptoms after controlling for ASQ scores weakens any conclusions that can be drawn from that research.

The results obtained in the present mediational analyses were not supportive of model hypotheses insofar as dysfunctional cognitive styles did not exclusively predict cognitions about subjects' most stressful uncontrollable interpersonal experiences. However, the fact that scores on several dysfunctional cognitive style measures were related to subjects' cognitions about all of the uncontrollable negative interpersonal events reported at T2 (i.e., irrespective of the stressfulness of those events) should not be regarded as theoretically unimportant. Subjects with certain cognitive vulnerabilities <u>were</u> more likely than their counterparts to make particular maladaptive inferences about the highly upsetting interpersonal stressors over which they perceived a lack of control. That cognitively vulnerable individuals imposed similar interpretations on less upsetting uncontrollable interpersonal stressors does not negate this fact. In other words, the failure of dysfunctional cognitive styles to confer <u>a specific risk</u> for depressogenic inferences about the most stressful of subjects' experiences does not diminish the fact that depressogenic inferences were made about these experiences.

Although cognitively vulnerable subjects made maladaptive inferences about less upsetting stressors, those inferences were not associated with increases in depressive symptomatology. Perceived

personal deficiencies, negative consequence expectancies, behavioral blame, and characterological blame predicted changes in levels of depressed mood only when they were associated with the most stressful of subjects' uncontrollable interpersonal outcomes. In this sense, the results of the mediational analyses are not at odds with hopelessness theory even though they did not conform to the model's prediction that

> the interaction of the hypothesized cognitive diatheses and negative life events should increase the likelihood that individuals will make negative interpretations (i.e., internal, stable, and global attributions or biased personal inferences) for the particular negative events they encounter (Alloy et al., 1988, p. 36).

The results of the mediational analyses also bear on a number of subsidiary issues related to cognitive style and event cognition associations. Several researchers have expressed doubt about the cross-situational consistency of causal attributions, suggesting that attributions for hypothetical outcomes bear little or no relation to attributions for real-life stressors (Cutrona et al., 1984; Miller, Klee, & Norman, 1982). Cutrona et al. (1984), for example, reanalyzed data from Cutrona's (1983) postpartum depression investigation and reported relatively modest correlations between ASQ scores and causal attributions for post-delivery stressors. Moreover, the magnitude of those associations did not substantially increase when data were examined separately for women whose responses to the six hypothetical ASQ outcomes were most consistent. Similarly, Miller et al. (1982) failed to observe significant associations between the attributions depressed inpatients made about a real life stressor and those made about either hypothetical or experimentally-created negative outcomes.

In contrast to those findings, the tendency to make stable and

global causal attributions for hypothetical negative interpersonal outcomes was predictive of stability and globality attributions for specific (uncontrollable) interpersonal stressors in the present research. It is likely that tests of the cognitive style-event inference relation have yielded inconsistent findings because cognitions about any one event are influenced by a number of different factors. Hopelessness theorists are quick to point out that the interpretation given to any one stressor will be a function of dispositional factors such as attributional style, the specific nature of the event, and the context within which the event occurs. In light of these considerations, it becomes clear that the validity of hypothesized associations between dysfunctional cognitive styles and maladaptive event inferences should not rise and fall based on correlations (or lack thereof) between cognitive style measures and inferences made about a single real life event. In fact, such an approach represents an extremely stringent test of such hypotheses.

Following the lead of attitude-behavior researchers (cf. Ajzen & Fishbein, 1977; Fishbein & Ajzen, 1974; Weigel, Vernon, & Tognacci, 1974), a more fruitful approach to this question might be to assess relations between dysfunctional cognitive styles and inferences made about a number of different real-life stressors. For example, Cutrona et al. (1984) found that ASQ scores were more highly related to attributions averaged over 14 stressors ($r = .263$, $p < .01$) than to attributions averaged over only three stressors ($r = .136$, ns). Thus, before more definitive conclusions are drawn regarding the cross-situational consistency of causal attributions, researchers should await

the results of tests similar to those suggested here.

Finally, it should be noted that a certain level of specificity between dysfunctional cognitive styles and event inferences is implied in the hopelessness model. Hopelessness theory seems to suggest that tendencies to attribute negative outcomes to stable and global causes will be related to stable and global event attributions but not to negative consequence expectancies or perceived personal deficiencies. No such specificity was observed in the present research. Attributional style was related not only to event attributions but to negative consequence expectancies, perceived personal deficiencies, behavioral blame, and characterological blame. The same was true for scores on the overgeneralization measure. This pattern of findings is inconsistent with the specific cognitive style-event inference relations implied by hopelessness theorists and suggests that at least some dysfunctional cognitive styles leave individuals vulnerable to making a host of negativistic inferences when life stressors occur.

Hopelessness as a Mediator of Dysfunctional Cognitive Style and Maladaptive Event Inference Effects

The subtype of depression described by Abramson et al. (1989) is defined by the causal precedence of hopelessness expectancies. Yet the cognitive style and event inference measures that predicted increases in depressive symptoms among subjects who experienced highly upsetting uncontrollable interpersonal stressors were unrelated to levels of hopelessness among the same subjects. The only exceptions to this pattern occurred for interactions of uncontrollable interpersonal stress and behavioral as well as characterological blame. Careful scrutiny of

the latter, however, suggested a closer association with hopelessness expectancies than with depressive symptoms. The general failure of hopelessness expectancies to mediate relations between depressive symptomatology and either dysfunctional cognitive styles or maladaptive event inferences has potentially crucial implications for the validity of Abramson et al.'s (1989) model (and for the 1978 reformulation of learned helplessness theory which makes similar predictions).

Evidence that hopelessness mediates relations between depressive symptoms and either dysfunctional cognitive styles or maladaptive event inferences has also proven somewhat elusive in other investigations that included measures of future outcome and control expectancies. Complete mediation has rarely been established. Metalsky et al.'s (1993) study of depressive symptoms among undergraduate students who recently received feedback regarding their grades on a midterm exam was the only study I located that demonstrated such an effect. In that study, a three-way interaction of attributional style, self-esteem, and exam performance predicted changes in both depressive symptom and hopelessness levels following the receipt of exam grades. Pre- to post-grade increases in depressive symptoms as well as hopelessness expectancies were observed only among low-scoring subjects who reported relatively low levels of achievement self-esteem and who exhibited a tendency to attribute negative achievement outcomes to stable and global causes. Metalsky et al. (1993) established the mediational role of future outcome expectancies by showing that levels of hopelessness reported at one day post-grade predicted levels of depressed mood reported at two, three, and four days post-grade. Moreover, the

attributional style X self-esteem X exam performance interaction that previously predicted post-grade levels of depressive symptomatology no longer did so after the effects of that interaction on hopelessness expectancies were statistically controlled.

Unlike the research just described, most studies have either demonstrated only partial mediation (Alloy & Clements, 1992; Metalsky & Joiner, 1992) or have failed to demonstrate mediation altogether (Andersen, 1990; McEvoy DeVellis & Blalock, 1992; Riskind et al., 1987). Alloy and Clements (1992), for example, found that perception of control styles (i.e., judgments of control over a noncontingent outcome) assessed at T1 interacted with the number of negative life events reported at T2 to predict both the degree of discouragement (i.e., hopelessness) subjects experienced following the occurrence of those stressors and T1 to T2 changes in levels of depressed mood. When discouragement ratings were entered as a control variable in the regression equation predicting residual changes in depressive symptoms, the effect of the control X stress interaction was reduced in strength (i.e., the Beta for the interaction decreased from -.20 to -.16) but remained a significant predictor of residualized depression scores ($p < .05$). Although Alloy and Clements (1992) interpreted the reduction in the control X stress interaction Beta as support for partial mediation, it is important to note that they did not test the two Betas to determine whether they were significantly different (cf. Williams, 1984; see also present study). The absence of evidence indicating that the interaction Betas reliably differed, combined with the fact that the control X stress interaction remained a reliable predictor of depression

scores after discouragement levels were controlled, weakens any conclusions that can be made about hopelessness mediation. At best, the support that Alloy and Clements obtained for hopelessness theory's mediation component appears preliminary.

Only slightly stronger evidence for the mediational predictions of hopelessness theory was obtained when Metalsky and Joiner (1992) examined the degree to which hopelessness expectancies accounted for the interactive effects of life stress and maladaptive cognitive styles on depressive symptomatology observed in their research. Of the three cognitive style X stress interactions that reliably predicted temporal changes in levels of depressed mood, only the effect of negativistic inferences about the self appeared to be mediated by hopelessness. Subjects who demonstrated a tendency to derogate themselves following the occurrence of negative outcomes and who reported a large number of stressful life events exhibited T1 to T2 increases in levels of both depressed mood and hopelessness. Time 2 hopelessness levels were strongly related to depressive symptoms at T2 and the interaction of life stress and self-derogation tendencies failed to account for a significant portion of the residual variance in depression scores when the variance attributable to hopelessness expectancies was statistically controlled.

Metalksy and Joiner's (1992) examination of attributional style scores showed that they also interacted with levels of life stress to predict changes in hopelessness and depressed mood. These effects were not interdependent, though, as the attributional style X life stress interaction remained reliably related to depression scores after

accounting for hopelessness expectancies. The variance in residualized depression scores explained by this interaction was reduced only by about 2% (from 5.8% to 3.6%) when hopelessness was used as a control variable in the regression equation. It is not clear whether this reduction was a reliable one because Metalsky and Joiner failed to test the difference between the two interaction Betas. Less equivocal results were obtained for the third cognitive style X stress interaction assessed by Metalsky and Joiner. The interaction of generalized negative consequence expectancies and life stress was exclusively predictive of changes in depressed mood. Furthermore, the magnitude of this effect was unaltered when hopelessness expectancies were statistically controlled.

Andersen (1990) also observed a significant interactive effect of attributional style and life stress scores on a measure of hopelessness as well as depressive symptoms, but found neither complete nor partial hopelessness mediation. In addition, both Riskind et al. (1987) and McEvoy DeVellis and Blalock (1992) failed to demonstrate the predicted mediational role of hopelessness expectancies but found evidence for a moderating role instead.

Riskind et al. (1987) found no association between attributional style scores and scores on a concurrently administered measure of hopelessness nor did they find a significant relation between T1 hopelessness expectancies and changes in levels of depressed mood over the six week study period. They did demonstrate, however, that attributional style, by itself, and in conjunction with hopelessness expectancies, predicted residualized changes in depressive

symptomatology levels. Analysis of the attributional style X hopelessness interaction showed that higher levels of hopelessness were associated with increases in depressive symptoms only among subjects who tended to attribute negative outcomes to internal, stable, and global causes. Among subjects who tended to explain negative outcomes in terms of external, unstable, and specific causes, higher levels of hopelessness predicted <u>decreases</u> in depressive symptomatology.

The pattern of results obtained by Riskind et al. (1987) is inconsistent with hopelessness theory's mediational predictions but supports an alternative, moderating model. In the "confluence model" suggested by Riskind and colleagues, a variety of factors in addition to attributional style contribute to the development of hopelessness expectancies; not all individuals with a negativistic attributional style will become hopeless and not all individuals with negative outcome expectancies possess a negativistic attributional style. The model also states, however, that hopelessness will bring about increases in depressive symptoms only among individuals who possess maladaptive attributional tendencies. In other words, a maladaptive attributional style and negative outcome expectancies are independent (i.e., non causally related) risk factors, which, by themselves, are either unrelated to or weakly related to depressive symptomatology. It is only when these vulnerability factors combine, do they contribute to the onset of depressive symptoms. As Riskind et al. (1987, p. 350) note

> According to the confluence hypothesis, the predictive capacity of attributional style is contingent on the degree of correspondence between attributions and expectations; specifically, it states that the working combination of a highly negative attributional style and negative outcome expectations represents the worst case of risk for future depression. Furthermore, negative

attributional style or expectations alone are not as indicative of higher levels of depression in the future.

Proponents of hopelessness theory might point out that the failure to assess the occurrence of negative life events limits the conclusions that can be drawn from Riskind et al.'s (1987) research regarding the role of hopelessness expectancies. That is, it could be argued that the failure to observe a significant relation between attributional style and future outcome expectancies was due to the fact that the stress levels of subjects were not taken into account. Neither hopelessness theory nor the reformulated learned helplessness model predict a main effect relation between attributional style and hopelessness; both models propose that individuals who possess a negativistic attributional style are at risk of becoming hopeless only when they experience high levels of life stress. Thus, a reliable association between attributional style scores and hopelessness expectancies (consistent with hopelessness theory's mediational component) might have emerged in Riskind et al.'s study if that relation had been assessed among a subset of subjects who had recently experienced important life stressors.

Although the failure to assess life stress might explain why Riskind et al. (1987) failed to establish hopelessness mediation, it can not adequately account for a similar observation made by McEvoy DeVellis and Blalock (1992) in their study of attributions, hopelessness expectancies, and depressive symptomatology among rheumatoid arthritis patients. McEvoy DeVellis and Blalock used the internality, stability, and globality dimensions to code spontaneous attributions that a sample of arthritis sufferers made for various aspects of their illness (e.g., onset, course, symptom flare-ups, treatment difficulties). Patients

also completed measures of future control and outcome expectations regarding their disease and reported on their levels of depressed mood at baseline (T1) and again four months later (T2).

McEvoy DeVellis and Blalock (1992) used these data to evaluate mediating and moderating models of the relations among attributions for illness-related events, hopelessness expectancies, and depressive symptomatology. Tests of the mediating model were unsupportive. Controlling for T1 levels of depressed mood and physical functioning, illness attributions (a stability-globality composite and stability scores alone) predicted T2 levels of depressive symptomatology but were unrelated to T1 hopelessness expectancies. Moreover, the use of hopelessness as a third control variable in the model predicting changes in depression levels had no effect on the strength of the attribution effect.

Assessment of the alternative moderating model yielded significant main effect relations between T1 to T2 changes in depressive symptomatology and both illness attributions and hopelessness expectancies. In addition, the interaction of attributions and hopelessness was a significant predictor of residualized depression scores. In an effort to clarify the nature of that interaction, McEvoy DeVellis and Blalock (1992) examined the relation between attributions and depressive symptoms separately for patients who felt relatively hopeless and relatively hopeful about their disease prognosis. Time 1 to Time 2 increases in depressive symptomatology were reliably associated with more stable and global illness attributions only among patients who felt unable to control the future course of their illnesses

and who were pessimistic about the long term prognosis of their condition. Among the more optimistic patients, illness attributions were unrelated to changes in depressive symptom levels over the four month study period.

Taken together, the results obtained by Riskind et al. (1987) and McEvoy DeVellis and Blalock (1992) provide little support for the mediational links among cognitions, hopelessness expectancies, and depressive symptoms proposed by hopelessness theory. The effects of negativistic attributions on depressive symptoms found in these studies were not explained by attribution-hopelessness and hopelessness-depression relations. Neither attributional style nor event attributions predicted subjects' levels of hopelessness. Although hopelessness theorists in no way claim that attributions and hopelessness expectancies will be perfectly correlated, they do suggest a nonzero relation between the two. Alloy et al. (1988, p. 36) specifically state

> The negative interpretations for particular negative life events (attributions or biased inferences) that a person makes should, in turn, increase the likelihood of forming the expectation of hopelessness... Again, because the particular interpretations an individual makes for negative events are hypothesized to contribute to, but not be sufficient for, the formation of hopelessness..., <u>this probability linkage should be greater than 0 but less than 1.0</u>" (emphasis added).

Rather than operating through a mediating process, attributions worked in conjunction with hopelessness expectancies to predict changes in depressive symptomatology. Only the combination of negativistic attributions and pessimistic future expectancies was related to increases in depression.

In an effort to determine whether similar moderated relations

were present in this research, three-way interactions between hopelessness expectancies (as assessed by the BHS), levels of interpersonal stress, and scores on the cognitive style and event inference measures were used to predict residual changes in depression among subjects who perceived a lack of control over negative interpersonal events. Moderator effects would be demonstrated if increases in depressive symptomatology were exhibited only by highly stressed subjects who reported both maladaptive cognitions and high levels of hopelessness. None of the hopelessness X stress X cognition interactions were significantly related to changes in levels of depressed mood. Thus, although the moderating effects of hopelessness expectancies have now been documented in several recent investigations, these findings were not replicated in the present research.

The failure to observe either moderating or mediating effects of hopelessness might be due to the fact that generalized future control and outcome expectancies were assessed whereas all of the cognition X stress findings were domain specific (i.e., pertained only to interpersonal stressors). In their discussion of hopelessness theory, Abramson et al. (1989) distinguished between circumscribed pessimism and generalized hopelessness. The former is said to occur when individuals anticipate a lack of control over a specific set of negative outcomes (e.g., interpersonal events) whereas the latter describes situations in which negative expectancies cut across multiple domains. It is perhaps more reasonable to expect that the maladaptive cognitions subjects held about uncontrollable interpersonal stressors gave rise to circumscribed pessimism regarding future social interactions than to generalized

feelings of hopelessness about many different areas of their lives.

The BHS, which was used in the present study to assess hopelessness, is a measure of generalized future expectancies. If the cognition X interpersonal stress interactions that predicted increases in depressive symptomatology had specific effects on expectancies regarding future interpersonal outcomes, it is unlikely that those effects would have been detected by the items on the BHS. Considered in this light, the failure of the cognition X interpersonal stress effects to either predict changes in BHS scores (consistent with a mediated relation) or interact with BHS scores to predict changes in depressive symptoms (consistent with a moderating relation), becomes more understandable.

A close examination of the hopelessness measures used in other investigations however, suggests that the lack of sensitivity inherent in the BHS can not completely account for the absence of mediational or moderator effects in this study. Moderator effects have been obtained by researchers who used both generalized and domain-specific measures of hopelessness expectancies. In Riskind et al.'s (1987) study, for example, the interaction of attributional style scores and scores on a measure of global outcome expectancies predicted increases in depressive symptoms among college undergraduates. In contrast, McEvoy DeVellis and Blalock (1992) found that illness attributions interacted with expectancies regarding disease prognosis to predict changes in depressive symptomatology among arthritis patients.

In the Metalsky et al. (1993) investigation, which provides the strongest evidence in support of hopelessness mediation, attributional

style for negative achievement outcomes and self-esteem in the achievement-domain interacted with performance on a midterm exam (achievement stress) to predict scores on a measure that assessed generalized hopelessness expectancies as well as expectancies regarding future achievement and interpersonal outcomes. In discussing their use of a broad-based hopelessness inventory, Metalsky et al. (1993, p. 103) commented that "In contrast to the original Hopelessness Scale..., the EHS [Extended Hopelessness Scale] includes 20 achievement-related items, 20 interpersonal-related items, and the 20 original items. Because we were interested in predicting generalized hopelessness, across content domains, we used all 60 items of the EHS" (emphasis added). These results are at odds with the suggestion advanced earlier that global measures of hopelessness are not sufficiently sensitive to detect the effects of domain-specific cognitions on future outcome and control expectancies. Nevertheless, the level at which hopelessness expectancies are assessed remains an important methodological and theoretical issue which might account for variability in findings across studies.

Self-Blame and Depressive Symptoms

Although not included among the set of proximal contributory causes of hopelessness depression, self-blame has been cited as a prominent feature of depressive thinking (Beck, 1967) that co-occurs with a perceived lack of control over negative outcomes (Abramson & Sackheim, 1979; Peterson, 1979). In discussing the relation between self-blame and depressive affect, Janoff-Bulman (1979) distinguished between behavioral blame (i.e., blaming oneself for past actions or

inactions) and characterological blame (i.e., blaming oneself for being a particular type of person) and regarded only the latter as a maladaptive response to negative outcomes capable of bringing about depressive symptomatology.

According to Janoff-Bulman, blaming negative events on one's actions or inactions implies that similar events can be avoided in the future by simply altering one's behavior. Because behavioral blame serves to enhance expectations of future control, it should be associated with positive coping outcomes. Conversely, blaming a faulty character, which is not as readily amenable to modification, implies a lack of control over future negative outcomes and should therefore be associated with helplessness expectancies and depressive affect. Janoff-Bulman (1979, Study 1) attempted to validate this distinction by examining the manner in which relatively depressed and nondepressed female undergraduates allocated blame for hypothetical negative outcomes between their behavior, their character, other people, and the external environment. As predicted, relatively depressed subjects scored higher than their less symptomatic counterparts on the characterological blame measure. The expectation that behavioral blame would be higher among nondepressed subjects was not, however, supported. No significant differences emerged between the two groups in their behavioral blame scores.

The concurrent association that Janoff-Bulman (1979) observed between characterological blame and depressive symptomatology levels was replicated by Major, Mueller, & Hildebrandt (1985) in their study of abortion patients. Women who engaged in more characterological blame

prior to an abortion procedure exhibited higher levels of depressive symptomatology immediately after the procedure. Levels of behavioral blame were not, however, significantly related to the severity of post-procedure depressive symptoms. As a supplement to their cross-sectional data, Major et al. obtained longitudinal data on depressive symptoms from a subset of the women in their sample who attended a follow-up visit three weeks after their abortion. When pre-procedure levels of depressive symptomatology were controlled, neither behavioral nor characterological blame predicted the severity of depressed mood at the three week follow-up. Taken together, Janoff-Bulman's (1979, Study 1) findings, and those reported by Majors et al. (1985), provide little evidence for an etiological role of characterological blame in depressive disorders. Rather, they suggest that characterological blame is a concomitant of depressive symptomatology.

The results of the present study suggest a different conclusion about the etiological importance of characterological and behavioral self-blame. Higher levels of both behavioral and characterological blame for subjects' most upsetting uncontrollable interpersonal stressors were associated with temporal increases in hopelessness expectancies as well as depressive symptomatology (although the latter effects were only marginally significant). The findings pertaining to characterological blame are consistent with Janoff-Bulman's (1979) expectation that blaming negative events on uncontrollable aspects of the self will be associated with maladaptive affective outcomes. The effects of behavioral blame, however, are at odds with Janoff-Bulman's claim that blaming controllable factors for negative events protects individuals

against the development of future helplessness expectancies and elevations in depressed mood.

Because characterological blame was not statistically controlled in the analyses involving behavioral blame, it is possible that the relations found between behavioral blame and both hopelessness expectancies and depressive symptoms were spurious. That is, the effects of behavioral blame might simply reflect the strong positive association (r = .89) between behavioral and characterological blame for uncontrollable interpersonal stressors. To assess this possibility, supplementary analyses of residual hopelessness and depression scores were undertaken with controls for the main effect of characterological blame and its interaction with levels of uncontrollable interpersonal stress. The interaction of behavioral blame and levels of uncontrollable interpersonal stress was not predictive of residual changes in either BHS ($pr2$ = .07, p = .46) or BDI (pr^2 = .04, p = .67) scores in these analyses.

Several additional points are important to the interpretation of these findings. First, in both the hopelessness and depressive symptom analyses, the Betas associated with the behavioral blame main effect (Bs = .36 and .06 for BHS and BDI scores, respectively) and interaction terms (Bs = .12 and .06 for BHS and BDI scores, respectively) remained positive when characterological blame scores were controlled. Thus, it does not appear that characterological blame served to obscure an otherwise negative association between behavioral blame and either helplessness expectancies or depressive symptomatology (cf. Janoff-Bulman, 1979). Second, when the behavioral blame main effect and

interaction terms were used as a covariates in the analyses involving characterological blame, the characterological blame X interpersonal stress interaction no longer predicted changes in either hopelessness expectancies (pr^2 = .08, p = .40) or levels of depressive symptomatology (pr^2 = .06, p = .52) among subjects low in perceived control.

Thus, it was the variance shared by behavioral and characterological blame that was responsible for the emergence of the interaction effects involving both variables. It may be that a more general self-punitive component, which is tapped by both blame measures, fosters hopelessness expectancies and depressive symptomatology in response to uncontrollable interpersonal stressors. Given the potential etiological significance of self-blame suggested by the results of the present study, it would seem a worthy pursuit to attempt to replicate these findings as well as explicate the operative dimension underlying measures of behavioral and characterological blame.

Study Limitations and Future Research Directions

The results of this research add to a growing body of evidence suggesting that dysfunctional cognitive styles represent a risk factor for the development of depressive symptomatology. They also suggest that the specific inferences individuals make about the uncontrollable experiences they encounter moderate the severity of depressive reactions elicited by those events. The present findings, as well as those obtained in related inquiries (e.g., Alloy & Clements, 1992; Metalsky & Joiner, 1992; Metalsky et al., 1987, 1993), suggest an optimistic outlook for the future verification of learned helplessness models of depression. However, such verification ultimately depends on how well

the predictions advanced in these models fare when subjected to rigorous examination in investigations that do not suffer from the methodological shortcomings of this and similar studies.

In the present research, for example, depressive symptomatology and life stressors were assessed within a three week period. With such a brief assessment period, its unlikely that many individuals will experience the types of severe events that are most strongly associated with the onset of depressive symptoms (Brown, 1981; Monroe & Simons, 1991). Only 107 of the 226 participants in this study reported the occurrence of highly stressful uncontrollable interpersonal events and only 56 subjects reported the occurrence of equally uncontrollable achievement stressors. The loss of this many subjects greatly reduced the power of the statistical analyses and, in turn, the strength of the effects that emerged from those analyses. The brevity of the study period might have also caused the depressogenic effects of some stressors to go undetected. Those subjects who experienced a significant stressor shortly before the final assessment session might not have developed a recognizable cluster of depressive symptoms until after the assessment (cf. Depue & Monroe, 1986). "Classroom methodologies," like those used by Metalsky et al. (1987, 1993), are better able to provide the type of access to subjects that multiple assessments and extended observation periods require, as are "daily report methodologies" which require subjects to keep ongoing daily records of life events and emotional experiences (cf. Vestre, 1984).

Methodologies such as these might also prove useful in reducing the interpretive difficulties that arise when individuals

retrospectively report life events that occurred over a certain time period. The subjects in the present study reported on the stressful events they experienced over the three weeks since their initial participation (see Alloy & Clements, 1992; Barnett & Gotlib, 1990, 1991 for examples of similar methodologies). Such a procedure is vulnerable to a number of reporting biases that can obscure true temporal relations between depressive symptoms and life stress. Higher levels of depressive symptomatology at T2 (i.e., when subjects completed the life event measures), for instance, might have contributed to the selective recall of negative life experiences, inflated perceptions of the stressfulness (or uncontrollability) of those events, and/or more negativistic inferences about those events (Depue & Monroe, 1986; Monroe & Simons, 1991). Moreover, these distortions might have been particularly likely to occur among relatively depressed subjects who also possessed higher levels of dysfunctional cognition (Monroe & Simons, 1990).

It is also possible that higher levels of depressive symptomatology, which arose after the first assessment session from factors other than life stress, contributed to the actual occurrence of highly upsetting experiences during the three week interim period. Hammen (1991, p.555) has recently argued that "at least some subsets of depressed people are exposed to considerable stress by virtue of their condition and their characteristics and behaviors and that to some degree, depressed persons generate the stressors that befall them." She went on to demonstrate that clinically depressed women experienced more "behavior-dependent" stressors and more interpersonal stressors

(especially those involving conflicts with others) over a six month period than did psychiatric, medically ill, and normal female controls. Although the depressed women in Hammen's (1991) research reported higher levels of overall stress, they did not experience more "behavior-independent" events than their nondepressed counterparts.

Monroe and Simons (1991) have likewise suggested that individuals who are vulnerable to depression (e.g., those who possess maladaptive cognitive styles) may create stress as a result of either a dysfunctional interactional style and/or chronic, low-level affective symptomatology. They make the frequently overlooked point that

> ... stress is not a random process, but part of a developmental sequence systematically influenced by the diathesis. Whereas the construct of stress may still play an important role in the evolving scheme, it is generated to a considerable degree by the person's behavior, which in turn is likely to be influenced by the diathesis (p. 411).

Individuals who believe, for example, that they are worthy only to the extent that they are loved or regarded highly by others may behave in excessively needy and dependent ways toward others. Such behavior may, in turn, ultimately lead to the very types of conflicted relationships and rejection experiences that they feared originally. For those whose esteem is mastery-based, self-imposed achievement pressures may debilitate performance to the point of failure, again leading to the very outcomes those individuals sought to avoid.

With respect to the present research, the reporting of self-generated stressors by relatively depressed subjects and/or cognitively vulnerable subjects might have artifactually inflated the strength of stress-depression associations among those scoring the highest on various measures of maladaptive cognitive styles. This set of

circumstances would lead to the false conclusion that stress played an etiological role in the onset of depressive symptoms among those who possess cognitive diatheses. It is difficult to unequivocally establish the cause and effect sequencing of life stress and depressive symptoms in two-wave panel designs that rely on retrospective reports of negative life experiences. The use of T1 depression level as a covariate in the analysis of panel data only removes the effects of initial symptom severity from T2 reports of life stress and depressive symptomatology. It makes no adjustment for the effects of either maladaptive cognitive styles or interim changes in symptom severity on T2 reports of life stress. Panel designs, such as the present one, would be better able to disentangle the complex relations among vulnerability factors, life stress, and depressive symptoms if precise datings of event occurrences and symptom changes were obtained (cf. Brown, 1981; Depue & Monroe, 1986).

The use of a "respondent-based" approach to the measurement of life stress might have also contributed to a number of interpretive problems. In contrast to an "investigator-based" assessment method in which details about the occurrence and context of negative life events are systematically collected by trained interviewers, respondent-based questionnaire methods rely solely on the subject for information about life stress (Brown, 1981). The responsibility for resolving issues such as what constitutes a stressful event, what distinguishes interpersonal stressors from achievement stressors, or whether or not a particular occurrence qualifies for a given event category (e.g., "received a low grade on a course examination") falls primarily on the subject. In

addition, judgments regarding the stressfulness or severity of life events are made exclusively by subjects.

Respondent-driven/questionnaire-based methods of measuring stress have been harshly criticized, and their use discouraged by a number of life stress researchers. Brown (1981), Dohrenwend, Dohrenwend, Dodson, and Shrout (1984), and Monroe and Simons (1991), for example, each describe numerous reporting biases that threaten the reliability and validity of such assessment instruments. Relatively depressed individuals, or those who possess highly dysfunctional attitudes, might have a lower threshold for life stress, and thus perceive relatively minor events as extremely stressful or upsetting. In addition, although efforts were made to ensure that the stressors included on the event checklists did not reflect symptoms of disorder (e.g., change in eating habits), it is possible that this goal was not fully realized. Subjects were free to write in other negative events that they experienced during the relevant time period and then to choose one of those events as their most upsetting stressor. The events that subjects supplied were not evaluated to determine whether they represented aspects of disorder. These potential distortions and inaccuracies increase the difficulty of disentangling stress from diathesis and disorder so that the appropriate causal ordering of these factors can be established.

Furthermore, despite explicit instructions about the reference period from which they were to report life stressors (i.e., the 3 weeks since their initial participation), subjects might have reported on events that occurred before that time period if those events were particularly salient. Alternatively, they might have reported chronic

stressors that began in the more distant past but were still present at the time of assessment (e.g., interpersonal difficulties with roommates). No effort was made to distinguish chronic stressors from more acute or discrete occurrences, despite the fact that these two classes of events are likely to bear different relations to the development and maintenance of depressive symptomatology (Depue & Monroe, 1986; Monroe & Simons, 1991). In addition, the lists of events provided to subjects were primarily comprised of minor stressors. More severe or traumatic events (e.g., severe physical illness) were not included because they have low base rates of occurrence in student populations and would have been unlikely to occur within the time frame of this research. Although acute, major stressors are likely to play a larger role in the etiology of depressive symptomatology than are minor events (Brown, 1981), their assessment in a prospective study would have required following subjects over a much longer time period. The inadequate assessment of major life events probably contributed to the relative weakness of the effects obtained in this research.

For these and other reasons, investigator-based methods of stress assessment, such as the contextual threat interview developed by Brown and Harris (1978), have become more common in investigations of stress-disorder relationships (e.g., Hammen, Mayol, deMayo, & Marks, 1986; Hammen, 1991). Using an event checklist as a guide, contextual threat interviewers query respondents about the occurrence of life stressors over a prespecified time period. For each event reported, respondents are asked to pinpoint as precisely as possible the date on which the event occurred. When needed, probes are used by the trained

interviewers to ensure the accurate dating of events. Detailed information is also collected about such things as the circumstances surrounding the event's occurrence, the respondent's previous experiences with event, and the adequacy of his/her resources (e.g., social support) for dealing with the event and its consequences. Interviewers subsequently prepare narrative reports of the details of each event and the context in which it occurred, omitting all information about the respondent's emotional reactions to the event. Independent judges then use explicit criteria to rate the level of threat associated with each event. This procedure yields data, uninfluenced by reporting inaccuracies and distortions, regarding the degree of stress to which an individual has been exposed. Combined with information about changes in depressive symptom levels and the timing of those changes, the contextual threat method enables investigators to better determine whether elevations in symptomatology antedate or follow life stress. Other approaches, such as the study of individuals presently experiencing high levels of stress (e.g., postpartum women, Cutrona, 1983; abortion patients, Majors et al., 1985; individuals with chronic or recurrent medical illnesses, McEvoy DeVellis & Blalock, 1992), also avoid the problems of respondent-based methods of stress assessment, and are becoming more common in investigations concerned with the psychopathological sequelae of life stress. The fact that theoretical predictions of the learned helplessness models of depression received partial support in this study despite the shortcomings associated with the assessment of life stress, suggests that the expense entailed in these more sophisticated approaches is warranted.

Problems associated with the operationalization and assessment of maladaptive cognitive styles might have also served to weaken the findings of this research. Measures of dysfunctional attitudes, irrational ideas, and overgeneralization were used to assess the cognitive styles included in hopelessness theory's expanded diathesis component. Although Abramson et al. (1989, p. 362) likened those cognitive styles (tendencies to exaggerate the adverse consequences of negative life events and to infer personal deficiencies when negative life events occur) to the notions of dysfunctional attitudes and irrational ideas, the overlap between these concepts is not complete. It is possible that stronger diathesis-stress effects and/or cognitive style-event cognition relations would have emerged if more direct measures of these cognitive styles had been used.

Metalsky and Joiner (1992), for example, used the Cognitive Style Questionnaire (CSQ; Abramson & Metalsky, 1986) to measure generalized tendencies to infer negative consequences and personal deficiencies in response to life stress. The CSQ asks subjects to imagine that they experienced each of 12 hypothetical negative events (the same events used on the EASQ), and then rate a.) the likelihood that the event will result in other bad occurrences, and b.) the degree to which the event implies some personal flaw(s). Metalsky and Joiner (1992) found that scores on both measures interacted with the number of negative life events subjects experienced over a five week period to predict residual increases in depressive symptomatology. The stress X negative consequences interaction accounted for seven percent of the variance in residualized depression scores and the stress X personal deficiency

interaction explained four percent of the variance in symptom changes. Only the latter interaction term predicted changes in hopelessness expectancies, however, and this effect was independent of (i.e., did not mediate) the interactive effects of stress and generalized personal deficiency inferences on changes in depressive symptomatology. Thus, Metalsky and Joiner's (1992) direct assessments of the proposed dysfunctional cognitive styles yielded somewhat stronger, but by no means complete, support for hopelessness theory's diathesis-stress and hopelessness mediation predictions.

With respect to the present study, it is also noteworthy that none of the cognitive style X stress interactions effects were uniquely predictive of increases in depressive symptomatology when assessed simultaneously in the same regression analysis (see Metalsky & Joiner, 1992 for a similar result). This finding might simply reflect "shared method variance," in that each measure was administered in the same format during the same assessment session. Alternatively, it might be that the cognitive style measures used in this research assess different aspects of the same construct rather than independent constructs. The results of a factor analysis of the Attributional Style (Interpersonal Domain), Overgeneralization, Dysfunctional Attitudes, and Irrational Ideas scores obtained by subjects who perceived interpersonal stressors as uncontrollable provided preliminary evidence for the latter speculation. Only one factor, accounting for 59% of the score variance, emerged from that analysis (an identical result was obtained when cognitive style data from the full sample was factor analyzed).

If these results are upheld in subsequent investigations, an

important objective for future research would be to specify more explicitly the nature of this general diathesis. For example, the common vulnerability factor being tapped by the different cognitive style measures used in this study might partially overlap with the constellation of traits believed to characterize the depressive personality. The depressive personality encompasses such traits as quiet, introverted, passive, and nonassertive; gloomy, pessimistic, serious, and incapable of fun; self-critical, self-reproaching, and self-derogatory; skeptical, hypercritical, and hard to please; conscientious, responsible, and self-disciplined; brooding and given to worry; preoccupied with negative events, feelings of inadequacy, and personal shortcomings (Akiskal, 1983).

Research by Klein (1990) has shown that psychiatric outpatients who met the criteria for depressive personality scored higher on measures of attributional style, dysfunctional attitudes, self-criticism, introversion, and self-constraint. Klein (1990) did not report the correlations between scores on these measures and the depressive personality index, nor did he perform a factor analysis to determine whether the set of personality and cognitive style scales converge onto a unitary dimension. The results of his research nevertheless suggest that the depressive personality may serve as a useful framework for integrating research on maladaptive cognitive styles and other personality variables that appear to predispose individuals to the development of depressive symptomatology.

END NOTES

[1] Abramson et al. (1978) were inconsistent in their discussion of the role that causal attributions play in the onset and nature of learned helplessness. They sometimes suggested that internal, stable, and global attributions for uncontrollable negative outcomes were necessary for both the onset and the nature (i.e., chronicity and generality) of future uncontrollability expectations. For example, Abramson et al. (1978) state that,

> The old model [Seligman, 1975], however, was vague in specifying the conditions under which a perception that events are noncontingent (past or present oriented) was transformed into an expectation that events will be noncontingent (future oriented). Our reformulation regards the attribution the individual makes for noncontingency between his acts and outcomes in the here and now as a determinant of his subsequent expectations of future noncontingency. These expectations, in turn, determine the generality, chronicity, and type of his helplessness symptoms (p. 52).

At other times, however, Abramson et al. (1978) implied that the perception of negative events as uncontrollable was sufficient for the onset of future uncontrollability expectations and causal attributions determined only the nature of that expectation and thus the nature of helplessness deficits. These suggestions are illustrated in the following quote, "In general, the properties of the attribution predict in what new situations and across what span of time the expectation of helplessness will be likely to recur" (p. 59).

Abramson et al. (1988b) acknowledged this source of confusion in the reformulated model. In an attempt to provide clarification, they offered the following comments,

> The 1978 statement of the hopelessness theory of depression was unclear about whether or not certain events (i.e., causal attributions) in the hypothesized causal chain contributed to the onset of depressive symptoms as well as to their chronicity and generality or only to their chronicity and generality. We believe that the underlying logic of the 1978 statement suggests that the causal events in question contribute to the onset, chronicity, and generality of depressive symptoms, and present the theory accordingly. Consistent with our interpretation of the underlying logic of the 1978 statement, Seligman, Abramson, Semmel, and von Baeyer (1979) wrote, 'According to the reformulated hypothesis, a certain attributional style, when combined with bad outcomes, causes depression' (p. 247).

Unfortunately, these comments leave a logical problem unresolved. If a particular pattern of causal attributions is necessary for symptom onset, there can be no symptom variability. That is, if future uncontrollability expectations develop only when noncontingent outcomes are attributed to internal, stable, and global causes, it would follow

that all cases of learned helplessness or depression would be chronic, highly generalized, and would involve self-esteem loss. It is thus logically inconsistent to postulate that causal attributions serve the dual roles of bringing about future uncontrollability expectations and shaping the nature of those expectations.

[2] The decision to include Levenson's (1981) Multidimensional Locus of Control Scale in this research was made subsequent to the start of the study. As a result, data on this measure were obtained for only 228 of the 247 Time 1 participants.

[3] Metalsky et al. (1987) created an "exam stress" variable by taking the difference between subjects' midterm grade aspirations and their actual exam grade. They then used the multiplicative product of exam stress and scores on the Importance subscale of the EASQ (Achievement Domain) in conjunction with EASQ-Generality scores (Achievement Domain) to predict change in depressive symptoms among the students in their sample. To the extent that Metalsky et al.'s stress variable includes an element of dysfunctional cognition, the precise meaning of the diathesis-stress interaction term that they created is unclear. Given this problem, combined with the fact that the weighted approach used by Metalsky et al. was abandoned in a subsequent "classroom study" conducted by this group (Metalsky et al., 1993), event stress in the present study was weighted by the perceived importance of those events rather than by EASQ importance.

[4] The assumption of homogeneity of variance-covariance matrices was evaluated prior to performing all multivariate analyses of variance reported in this research. No violations of this assumption were revealed.

[5] All multivariate significance tests were based on Wilks' criterion.

[6] When a MANOVA is performed on a set of correlated measures, it is inappropriate to interpret the results of univariate ANOVAs performed on the individual measures following a significant multivariate effect. A significant univariate effect might emerge for a given measure, not because that measure is necessarily related to the independent variable, but because it shares variance with another measure that is affected by the independent variable. Moreover, the univariate *F*'s associated with correlated measures are not independent and the alpha levels for those tests can not easily be adjusted to control overall error rates (Tabachnick and Fidell, 1989).

Stepdown analysis is the appropriate strategy for evaluating the significance of correlated measures following a significant MANOVA. As described by Tabachnick and Fidell (1989, pp. 400-401), stepdown analysis is similar to hierarchical regression. Each measure is first prioritized in order of its importance to the independent variable. The significance of the highest-priority measure is then assessed in a univariate ANOVA. Each remaining measure is tested in an ANCOVA with higher priority measures serving as covariates. The alpha levels for the individual tests are adjusted using a Bonferonni procedure to

control the overall (familywise or experimentwise) error rate.

Stepdown analysis is a conservative strategy and its use here was somewhat problematical because at times there was no clear ordering of the individual measures (see Tabachnick & Fidell, 1984, p. 402 for a discussion of this problem). An effort was made, however, to impose the most meaningful order on the measures within each set. To the extent that this goal was not realized, caution must be used when interpreting the results of each stepdown analysis.

[7] The homogeneity of regression assumption was evaluated prior to conducting all MANCOVAs. Unless otherwise indicated, the assumption was met in all cases.

[8] A significant subject gender X T1RSES interaction indicated a violation of the homogeneity of regression assumption. The results of this analysis should be interpreted with caution.

[9] Data from only those subjects whose negative achievement or interpersonal events occurred within the three weeks prior to the second assessment session were used in these and all other analyses.

[10] It might be argued that the subset of interpersonal stressors that were perceived as uncontrollable differed from those that were perceived as controllable on some other important dimension besides controllability, and that those other differences contributed to the cognitive style X stress regression results. For example, if uncontrollable stressors were also perceived as more stressful or more likely to result in negative consequences, that might account for the fact that significant diathesis-stress interaction effects were obtained only among subjects who experienced uncontrollable interpersonal stressors. To examine whether this was the case, I compared the stress and cognition ratings provided by the 107 subjects who perceived their most upsetting interpersonal stressor as relatively uncontrollable with those of the 111 subjects who perceived their most upsetting interpersonal stressor as relatively controllable. Controllable and uncontrollable interpersonal stressors did not differ in perceived stressfulness ($p = .81$), attributional generality ($p = .27$) or anticipated negative consequences ($p = .53$). As might be expected, attributional internality, perceived personal deficiencies, behavioral blame, and characterological blame ratings were higher for controllable versus uncontrollable interpersonal stressors (all $ps < .0001$). Additional comparisons revealed that subjects who perceived their most upsetting interpersonal stressor as relatively uncontrollable were no more depressed at T1 than were their counterparts ($p = .99$), nor did the former report the occurrence of more stressful life events in the T1 to T2 interim ($p = .81$). Thus, it does not appear that perceptions of control were confounded with another dimension that could also account for the diathesis-stress results obtained in this research.

[11] It might be argued that this effect occurred simply because the attributional composite was comprised in part of scores on the internality dimension. To test this hypothesis, an additional analysis

was performed on T2 RSES scores after removing the internality dimension from the attributional composite. Although the interaction from this analysis was nonsignificant (p = .69), the corresponding regression coefficient was positive (b = .004), suggesting a pattern of results similar in form to that found for the interaction of interpersonal stress and scores on the internal, stable, and global composite.

A similar finding was obtained when the same analysis was performed separately for subjects who perceived interpersonal stressors as controllable. That is, a positive regression coefficient (b = .02) was obtained for the marginally significant (p = .16) interaction of interpersonal stress and composite stability/globality scores. These findings run counter to the argument that the internality dimension was solely responsible for the form of the interpersonal stress X internal, stable, and global attributional composite interaction.

[12] No mention is made of personal deficiency inferences or negative consequence expectancies in Alloy et al.'s (1990) discussion of the proximal contributory causes of hopelessness expectancies. Although their etiological status in the "helplessness-hopelessness theory of anxiety and depression" is unclear, researchers testing hopelessness theory (Metalsky & Joiner, 1992) have continued to measure both types of event-based cognitions even after the 1990 publication Alloy et al.'s model. Thus, it seems appropriate to infer that the hypotheses regarding personal deficiency inferences and negative consequence expectancies outlined in Abramson et al.'s (1989) original model are still valid.

[13] Alloy et al. (1990) likewise make no mention of the predisposing effects of tendencies to infer personal deficiencies or expect negative consequences when negative life events occur. As hopelessness theorists (Metalsky & Joiner, 1992) have also continued to assess these constructs in empirical investigations, it would appear that the original hypotheses remain valid.

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