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## An Evaluation of the Depressive Attributional Style in a Clinically Depressed and Nondepressed Sample

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AN EVALUATION OF THE DEPRESSIVE ATTRIBUTIONAL STYLE IN A  
CLINICALLY DEPRESSED AND NONDEPRESSED SAMPLE

by

Donna Munic

A Dissertation Submitted to the Faculty of the Graduate School  
of Loyola University of Chicago in Partial Fulfillment  
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## VITA

The author, Donna Munic, is the only child of Morris Munic and Doris (Bernick) Munic. She was born November 22, 1954, in St. Paul, Minnesota. Her elementary education was obtained at Homecroft Grade School, St. Paul, Minnesota. She graduated with Highest Distinction from Highland Park Senior High School in 1972.

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## INTRODUCTION

The prevalence of depression is staggering. Although the true percentage of affective disorders is not actually known, it has been estimated that twenty per cent of Americans will have an affective disorder in their lifetime (Task Panel Reports submitted to the President's Commission on Mental Health, Vol. IV, Appendix, 1978).

Depression cuts across all socio-economic classes and results in both financial burdens and emotional suffering in the depressed individual, in his/her immediate family, and in society as a whole. What is more, depression can be lethal as in suicide. Despite its frequency and its far reaching ramifications, there is still much confusion as to what depression really is, what causes it, and what is the most effective treatment.

Various systematic formulations of depression have been proposed of which one of these has been the learned helplessness model of depression by Martin Seligman and his colleagues. Central to the learned helplessness theory is that it is not trauma per se that produces interference with later adaptive responding, but not having control over the trauma. The learning that trauma is uncontrollable has three main effects, i.e., motivational, cognitive, and affective or emotional. First, it is motivational in the sense that if a person has previously learned that his responses have no effect, future expectations will be lowered. This is believed to underlie the



passivity, intellectual slowness, and social impairment in learned helplessness and depression. Second, it is cognitive in the sense that by learning that responding does not produce relief it is more difficult to learn that responding at another time and at another place does produce relief. This is thought of as being responsible for the negative cognitive set of depressed people. Third, it is affective or emotional in the sense that if a person learns he cannot control an event, initial anxiety produced by this traumatic event is displaced by affective components of depression. This is thought to elicit the feelings of uselessness and sadness (Seligman, 1975).

Historically the learned helplessness model was formulated on the basis of laboratory experiments with animals whereby exposure to inescapable shock resulted in interference in subsequent escape-avoidance learning (Seligman, 1975). Investigators then began extending this paradigm to research with human subjects (See Miller & Norman, 1979 for a review). A number of inadequacies in the original learned helplessness model became evident in these human helplessness studies. To address these inadequacies Abramson, Seligman, and Teasdale (1978) proposed an attributional reformulation model of learned helplessness. According to their reformulation, a person first learns that certain outcomes and responses are independent and then he/she makes an attribution about the cause. This attribution effects subsequent expectations for future noncontingency. These expectations, in turn, determine the generality, chronicity, and type of helplessness. These researchers suggest that there is a depressive attributional

style, whereby individuals who typically tend to attribute failure to global, stable, and internal factors are most prone to general and chronic helplessness depressions with low self-esteem.

To examine predictions made by the reformulated model, Seligman, Abramson, Semmel, and von Baeyer (1979) employed an Attributional Style Questionnaire (ASQ). These researchers found that depressed students differed from nondepressed students in the predicted directions on attributions for bad and good outcomes.

The present study further examined predictions of the reformulated attributional theory using the ASQ, but on a clinical population. Moreover, it compared the attributions of mildly to moderately depressed patients and severely depressed patients to determine possible attributional differences between varying degrees of depression. Males and females served as subjects in order to detect for possible sex differences in depressive attributional style. Finally, this study assessed whether the attributional style predicted for clinically depressed patients is uniquely related to depression or whether it is a feature of psychopathology per se.

## REVIEW OF RELATED LITERATURE

### Original Learned Helplessness Model

Seligman and Maier (1967) and Overmier and Seligman (1967) found that dogs who were exposed to unavoidable, inescapable shock, later failed to avoid or escape traumatic shock in another situation where shock was avoidable by performing a simple response. In describing this phenomenon, the term learned helplessness was used and refers to the perception or learning of independence between an organism's response and the outcome which, in turn, leads to an expectation of uncontrollability. This expectation of uncontrollability in learned helplessness results in three interrelated deficits: motivational; cognitive, and emotional. Specifically as hypothesized by Seligman, learned helplessness

(1) reduces the motivation to control the outcome; (2) interferes with learning that responding controls the outcome; and if the outcome is traumatic, (3) produces fear for as long as the subject is uncertain of the uncontrollability of the outcome and then produces depression. (Seligman, 1975, p. 56)

With respect to depression, Seligman (1975) cites six parallels between the laboratory-induced phenomena of learned helplessness and naturally occurring depression in man. These parallels are: (1) decreased initiation of voluntary responses; (2) negative cognitive set; (3) dissipation in time; (4) decreased aggression; (5) loss of libido and loss of appetite; and (6) physiological changes of nor-epinephrine depletion and cholinergic activity. To Seligman, these

parallels suggest that "depression, as well as learned helplessness, has its roots in the belief that valued outcomes are uncontrollable" (Seligman, 1975, p. 105).

Although the occurrence of learned helplessness was originally found in dogs, it was later demonstrated in cats (e.g., Thomas, 1975); fish (e.g., Padilla, Padilla, Ketterer, & Giacolone, 1970); and rats (e.g., Seligman, Rosselle, & Kozak, 1975). Maier and Seligman (1975) provide a review of the animal research on learned helplessness.

Human helplessness studies were carried out to replicate the animal findings in humans (e.g., Hiroto & Seligman, 1975) and to test the claim that learned helplessness is a laboratory model for depression in humans (e.g., Miller & Seligman, 1975). Miller and Norman (1979) provide a review of the learned helplessness research using human subjects. Suffice it to say that as investigators began extending the paradigm to research with human subjects and began applying the theoretical constructs from animal helplessness to human helplessness, a number of problems surfaced. Abramson, Seligman, and Teasdale (1978) have identified four major inadequacies of the original model of learned helplessness: (1) The expectation of uncontrollability is not sufficient for depressed affect in that there are many uncontrollable events in people's lives that do not sadden them. Indeed, only uncontrollable events where highly aversive outcomes are perceived as probable or where highly desired outcomes are believed as improbable, bring on depression. (2) Lowered self-esteem in depression is not explained by the original model. (3) The tendency of

depressed individuals to make internal attributions for failure is not explained. (4) Variations in generality, chronicity, and intensity of depression are not explained.

#### Reformulated Learned Helplessness Model of Depression

To address the majority of the inadequacies cited above, Abramson et al. (1978) have proposed an attributional reformulation of learned helplessness. According to their reformulation, once a person perceives that certain outcomes and responses are independent, he then makes an attribution about the cause of his helplessness. The cause can be internal or external, stable or unstable, and global or specific. Internal factors stem from within the person, i.e., personal responses and individual characteristics, whereas external factors stem from outside the person, i.e., the situation and the environment. Stable factors are long-lived and recurrent as compared to unstable factors which are short-lived and intermittent. Global factors affect a wide variety of outcomes while specific factors are more unique to the original situation of helplessness. The attribution chosen affects subsequent expectations for future independency or noncontingency. These expectations, in turn, determine the generality, the chronicity, and the type of helplessness. Abramson et al. (1978) predict that internal attributions are more likely to be characterized by loss of self-esteem than external attributions. They further contend that attributions to stable factors produce deficits with greater chronicity than attributions to unstable factors. Moreover, deficits attributed to global factors are expected to generalize further than deficits attributed

to specific factors. Also, the strength or certainty of the expectation of uncontrollability is considered as determining the intensity of the deficits. Abramson et al. speculate that there is a depressive attributional style, whereby those people who tend to make internal, stable, and global attributions for failure are at high risk for depression.

Several studies have looked at attributional predictions in learned helplessness research. Klein, Fencil-Morse, and Seligman (1976) directly manipulated the attributions of depressed and nondepressed college students on an unsolvable task by assigning students to one of three conditions. In the internal attribution condition subjects were informed that 55% of previous students succeeded on all four discrimination problems and only 1% failed all problems. In the external attribution condition subjects were told that no one solved all the problems and 90% failed all the problems. A third group of subjects received no attributional instructions. Following these instructions, subjects were exposed to random reinforcement of the discrimination problems and then tested on an anagram task. Results revealed that the type of attributional instructions did not significantly effect the performance of nondepressed subjects on the subsequent anagrams. However, attributional instructions did have a major impact on depressed subjects' performances. When depressed subjects attributed failure to task difficulty, i.e., external attribution condition, rather than to personal incompetency, i.e., internal attribution condition, their performance on the anagram task improved.

Kuiper (1978) investigated the effects of depression not only on the causal attributions for failure, but also on the causal attributions for success. Following a word association task, depressed and nondepressed female students made attributions for their success or failure by choosing from four designated factors of effort, ability, luck, and task difficulty. As expected, depressed females selected internal attributions (i.e., ability and effort) whereas nondepressed females selected external attributions (i.e., luck and task difficulty) for failure. In successful outcomes, no differences were found between depressed and nondepressed students as both groups made internal attributions for success. An analysis of the stability dimension failed to reveal any significant differences between depressed and nondepressed groups.

Rizley (1978) also studied the causal attributions of depressed and nondepressed subjects in failure and success situations. Like Kuiper (1978), Rizley (1978) found that depressed subjects significantly rated an internal factor, i.e., effort, as a more important cause of failure than nondepressed subjects. However, unlike Kuiper (1978), Rizley (1978) noted that depressed subjects also rated an internal factor, i.e., ability, as a less important cause of success than nondepressed subjects.

In each of the above studies subjects were manipulated into success and failure situations with the attributional dimensions (i.e., internal versus external; stable versus unstable) based on preconceived notions of ability, effort, luck, or task difficulty. This procedure

can be problematic in two ways. First, subjects' responses are limited to only the causes anticipated by the researcher in his assessment questionnaire. Other causes not assessed may be just as, or even more, important to the subjects. Second, causes can fall at varying intervals along the dimensional continua depending upon an individual's perspective. For example, although most people would consider luck an external variable, someone may personalize luck and thus perceive luck more as an internal variable, i.e., "I'm an unlucky person." (See Abramson et al., 1978, p. 58 for a more detailed explanation of the unclear link between a specific cause and a conceptual attributional dimension.)

In order to try and to rectify the problem cited above, Seligman, Abramson, Semmel, and von Baeyer (1979) employed an Attributional Style Questionnaire (ASQ) on a sample of depressed and nondepressed college students. Basically, the ASQ assesses the attributional dimensions separately and exhaustively by asking subjects to provide a free response to various positive and negative outcomes, indicating the one major cause of each outcome. Subjects then rate this causal explanation on four dimensions: internality, stability, globality, and importance. In addition to filling out the ASQ, subjects were asked to complete two depression self-report inventories, i.e., the Beck Depression Inventory (BDI) and the Multiple Affect Adjective Checklist (MAACL). As expected, these researchers found that depressed students as compared to nondepressed students had greater ratings of internality, stability, and globality for bad outcomes. Moreover, depressed



subjects were more external and unstable in their attributions to good outcomes than nondepressed subjects. It should be noted that the relationship between ASQ indices for good outcomes and depression was not as strong as the relationship between ASQ indices for bad outcomes and depression.

Blaney, Behar, and Head (1980) employed Seligman et al.'s (1979) ASQ on two college samples using BDI scores as the measure of depressive affect. Although these researchers found some significant correlations between ASQ indices and depression, their correlations were generally much smaller than those reported by Seligman et al. (1979) and considered by them as mostly being "unimpressive in absolute terms." Specifically, Blaney et al.'s (1980) correlations between ASQ indices of internality and stability for bad outcomes and depression ranged from .07 to .15, whereas correlations between internality, stability, and globality for good outcomes and depression ranged from .02 to -.19. The only exception in their findings was for globality for bad outcomes in which case the correlations between globality for bad outcomes and depression were generally high and at a level consistent with Seligman et al.'s (1979) findings.

Golin, Sweeney, and Shaeffer (1981) studied the causal role of attributions in depression by administering the ASQ and the BDI to 180 undergraduates on two different occasions. Results showed that internality, stability, globality, and composite scores for bad outcomes were significantly correlated with depression. In addition, internality, stability, globality, and composite scores for good

outcomes were negatively correlated with depression, but only in the second testing session. However, all the correlations were generally small and only accounted for a small percentage of the variance. A cross-lagged panel correlational analysis on the data provided evidence that stable and global attributions for bad outcomes and unstable attributions for good outcomes may cause depression. There was no support that internal attributions for bad outcomes and external or specific attributions for good outcomes may cause depression nor was there support that depression may cause attributional style.

Several studies have looked at individuals' causal attributions in naturally occurring, personally significant, situations in contrast to the hypothetical situations on the ASQ. Forsyth and McMillan (1981) asked 233 college students various questions concerning their performances on a recent introductory psychology examination. In line with the reformulated model of learned helplessness, there was a strong positive correlation between affective response and controllability. Students who felt that their performance was caused by controllable factors were more satisfied and happy than students who thought that their performance was caused by factors beyond their control. This consistent relationship between positive affect and controllability was noted in cases both when the students did well or did poorly on the test. In addition, more positive affective responses were reported by students who attributed success to internal factors or who attributed failure to external factors.

Harvey (1981) had 45 depressed and 46 nondepressed female college

students provide their own untutored explanations of the causes of their recent important personal events. Findings revealed that depressed females viewed negative events as being more internally caused and less controllable. In addition, depressed females perceived significantly fewer internal causes for their positive events than nondepressed females. No differences between depressed and nondepressed groups on the stability dimension could be detected. Moreover, the globality dimension could not be reliably inferred from the data at hand. Thus, there was only partial support for the reformulated model of learned helplessness.

In another study, moderately depressed, nondepressed but highly stressed, and nondepressed undergraduates were asked to identify the causal explanations concerning the five most upsetting events in their lives. Contrary to the reformulated model of learned helplessness, the three groups did not differ in overall attributional ratings, i.e., composite scores of control, locus, intentionality, stability, and globality. However, major differences between groups were found in their nonattributional cognitions whereby students in the depressed group reported greater upset and more uncertainty than students in the other two groups (Hammen & Cochran, 1981).

Hammen and DeMayo (1982) examined the relationship between causal attributions associated with teacher stress and depression in 75 urban high school teachers. Depression was measured by the Center for Epidemiological Studies-Depression Scale (CES-D). Results were not supportive of the reformulated model of learned helplessness in that

depression was neither related to locus of causality nor perceived stability of causes. However, consistent with the reformulated model, depression was associated with a perceived lack of control over stress factors in teaching.

Feather and Davenport (1981) investigated depression-attribution linkages among unemployed people. Contrary to the learned helplessness model of depression, subjects were not passive and less motivated to find work nor did they blame themselves for their lack of work. Specifically, findings showed that more depressed unemployed people, as compared to less depressed unemployed people, reported higher levels of present need and effort to find a job and they blamed their unemployment status on external difficulties (e.g., the economy, government inactivity, policies of private industry, etc.). Nevertheless, as these researchers point out, their depression measure was situation-specific, tapping only how subjects felt about being unemployed, and not a generalized chronic depression measure. Second, the sample was limited to a group of unemployed youth who were in contact with employment helping agencies and who were still presumably actively searching for job opportunities.

All of the reported studies on the reformulated model of learned helplessness up to this point have used college students as subjects, except for the two studies where teachers and unemployed youth served as subjects. In general, the findings have only been partially supportive of the model. Adding to the inconclusiveness of these results is the fact that none of the subjects in the above studies were

diagnosed as clinically depressed, nor had they sought psychiatric treatment for depression. Indeed, depression was based solely on various types of self-rating scales.

There are potential problems associated with identifying depression only by using self-report measures. Specifically, DePue and Monroe (1978) point out that elevated scores on depression scales could result from a number of independent factors including: an individual who is relatively normal, but who is momentarily unhappy, sad, or lonely; an individual who is suffering from an object loss; an individual who is suffering from a loss of self-esteem; an individual who is suffering from a medical or psychiatric disorder and who has secondary depression; as well as an individual who has a major primary depressive disorder. In addition, the meaning of items on rating scales may be viewed from different perspectives by patients and by mildly depressed normals, but be rated the same. Amenson and Lewinsohn (1981) have shown that high scores on a depression self-report inventory (i.e., CES-D) were correlated with youth, divorce/separation, low education, and unemployment as well as a diagnosis of depression, but only divorce/separation was significantly related to a diagnosis of depression.

#### Attributions of Psychiatric Patients

Taking into account the possible problems in depression self-report measures cited above, it seems important to test the reformulated model of learned helplessness on a clinical population. In this way, it can be shown whether or not mild depression in a student

population, or situational depression due to unemployment or teacher stress, is quantitatively different versus qualitatively different from clinical depression.

Costello (1982) looked at the relationship between depression and locus of control in depressed psychiatric outpatients, nondepressed controls, depressed undergraduates, and nondepressed undergraduates. She found that depression and externality were strongly correlated, with the correlation increasing when age was partialled out. Her findings, thus, suggest that the depression in a student population using the BDI is on a single continuum with the depression in psychiatric depressed outpatients, implying a quantitative difference between college students and the clinically depressed.

Three other studies have specifically looked at attributional style in clinically depressed patients. Gong-Guy and Hammen (1980) asked 72 depressed and nondepressed outpatients the causes and consequences of recent, personally stressful, life events. Using their own questionnaire, no differences were found between depressed and nondepressed groups when all stressful events were taken into account. Yet, there were differences between depressed and nondepressed outpatients when only the most upsetting events (i.e., scores of six or seven out of seven possible) were considered. Relative to nondepressed patients, depressed patients characterized the causes of their most upsetting events as significantly more internal and more intended. In addition, although not statistically significant, depressed patients tended to view the causes as being more global, as being more expected,

and as being more stable than nondepressed patients. Thus, there was only weak support for the reformulated model.

Raps, Peterson, Reinhard, Abramson, and Seligman (1982) measured depressive attributional style in clinically diagnosed unipolar depressed males, male schizophrenics, and medical-surgical male patients using the ASQ. Results were generally supportive of a depressive attributional style. Specifically, depressed inpatients, as compared to schizophrenics, were more likely to attribute bad events to internal and stable causes and tended to attribute bad events to global causes. Relative to medical-surgical inpatients, depressed inpatients made more internal, global and stable attributions for bad events and made more external and unstable attributions for good events. Composite evenhandedness scores were also assessed, and as predicted, depressed inpatients judged the causes of bad and good events to be more similar than either schizophrenic patients or medical-surgical patients.

Miller, Klee, and Norman (1982) assessed the generality of depressive attributional style by asking depressed and nondepressed inpatients for their causal explanations and other cognitions regarding three types of situations: hypothetical events (i.e., three negative and three positive outcomes); experimental tasks; and their most stressful life event. These researchers found that depressed patients exhibited a significantly greater depressive attributional style, based on composite scores of internality-externality plus stability-variability plus generality-specificity, but only for their most stressful life events. Depressed and nondepressed patients did not differ in attributional style for hypothetical events nor for

experimental tasks. Also, correlations between these three types of situations were mostly nonsignificant, thus suggesting little cross-situational consistency between the measures.

In brief, the few studies on depressive attributional style using clinically depressed samples, as in the studies using nonpsychiatric samples, show mixed results depending on various factors, including the instruments employed and the methods of analysis.

The present study attempts to clarify these inconclusive findings in the reformulated learned helplessness model of depression. First, an attempt is made to replicate the Seligman et al.'s (1979) study as close as possible in a clinically depressed population. Specifically, like Seligman et al. (1979), this study employs the full ASQ and uses the BDI and the MAACL-D. It also uses the MMPI-D scale (unlike Seligman et al., 1979) as a further measure of depressive affect. Second, unlike the Raps et al.'s (1982) study which used only unipolar males, depressed males and females in the present investigation vary in the diagnoses of depression. As Seligman (1978) suggests, "learned helplessness is a subclass of depression caused by the expectation that important events are uncontrollable and that this subtype might cut across preexisting descriptive subtypes of depression" (Seligman, 1978, p. 166). Moreover, there appears to be a major theoretical problem with using unipolar depressives. DePue and Monroe (1978) in reviewing the parallels between learned helplessness and depression as set down by Seligman (1975), concluded that some of the symptom parallels, e.g.,



passivity and lack of aggression, more adequately apply to bipolar depression or some form of endogenous depression rather than unipolar depression or some form of reactive/neurotic depression. Indeed, DePue and Monroe (1978) point out that unipolar depression is characterized by active pacing, agitation, hostility, and irritability. Third, unlike the other learned helplessness studies reviewed, depressed clinical patients in this study are divided into three groups of depression, namely, no depression, mild to moderate depression, and severe depression. Thus, it can be determined whether or not mild depression is quantitatively different versus qualitatively different from severe depression, without having a possible confound of subject population, i.e., college students versus patients. Fourth, up to this point there has been little research that has attempted to sort out the effects of depression versus global psychopathology. Consequently, in the current study, subjects are classified into high psychopathology or low psychopathology groups as well as classified into one of the three depressed groups.

### Sex Differences

Differences in behavior between males and females have been documented as early as the first year of life (Goldberg & Lewis, 1969). Sex differences would thus seem to be an important variable in research, and especially important in depression research as women are more likely to experience depression than men. In a comprehensive review of epidemiological studies from 1936 through 1973 on sex differences in depression, Weissman and Klerman (1977) conclude that, in general,

women in the United States are twice as likely to be depressed as males. Not only is a sex difference observed in diagnosed cases of depression, it is also found in nondiagnosed cases. Weissman and Klerman (1977) report on United States community surveys, based upon a random sample of treated and untreated cases of depression, and again more women are depressed than men. These researchers note that the preponderance of female depressives is not confined to the United States, but is observed in other Western industrialized societies as well. Moreover, at any given age, rates of depression are higher for females than for males. In an even more recent community survey, Amenson and Lewinsohn (1981) also found a significantly higher percentage of women than men meeting the criteria for unipolar depression. Thus, it seems sex differences in depression continue to be a consistent and general finding in the literature.

General explanations for sex differences in depression have been summarized by Weissman and Klerman (1977), Amenson and Lewinsohn (1981), and King and Buchwald (1982) and other researchers. These explanations include the artifact hypothesis, biological hypotheses, and psychosocial hypotheses. The artifact hypothesis contends that the actual prevalence of depression among men and women is equal but women are simply more likely to admit and to seek help for depressive symptoms. The biological hypotheses include theories concerning genetic transmission (i.e., X-linked dominant trait) and female endocrinological causes (i.e., premenstrual tension, use of oral contraceptives, and postpartum). The psychosocial explanations take many forms including

social status differences (i.e., women have less education, lower occupational levels and less power than men); legal and economic discriminations (i.e., women make less money than men); and women's internalization of role expectations (i.e., stereotypic views of women characterized as dependent, passive, and emotional and men characterized as independent, competent, and active), all of which may result in "relative helplessness" and depression. As noted by Amenson and Lewinsohn (1981) other sources of potential psychosocial explanations can be derived from existing cognitive theories (e.g., Abramson et al., 1978; Beck, 1967; Ellis, 1962); reinforcement theories (e.g., Lewinsohn, 1974); and stress theories (Paykel, 1969). What is intriguing for the purposes of this study is the possibility that attributional style as outlined by Abramson et al. (1978) may be a factor in why more females than males are depressed.

Indirect support that attributional style may be an important determinant of the sex difference in depression comes from work done by Dweck and others in learned helplessness studies done with children. Dweck and Reppucci (1973) looked at 20 male and 20 female fifth graders. Findings revealed that boys, relative to girls, were more likely to attribute failure to lack of effort (i.e., an internal, unstable, specific attribution), and lack of effort was more associated with persistence than helplessness.

Nichols (1975) observed that boys blame their failure on bad luck (i.e., an external and unstable attribution) whereas girls blame their failure on lack of ability (i.e., an internal, stable, global

attribution). In addition, boys had higher expectations for success than girls. Moreover, boys, but not girls, spent more time on an angle matching task when failing than succeeding, thus they were more persistent during failure.

Dweck and Bush (1976) found that male and female children reacted differently to failure feedback from adults. Specifically, boys attributed failure to lack of effort, which resulted in improved performance on a subsequent task. On the other hand, girls attributed failure to lack of ability and their performance on the task was impaired.

Dweck, Goetz, and Strauss (1981) further examined sex role differences in learned helplessness in children in two separate studies. In the first study, fifth grade children received failure feedback after each of four trials and then they were assigned to one of the following conditions: new task, new evaluator, new task and new evaluator, or no change. Results showed that expectancies of all children dropped by the fourth trial with girls tending to have even lower expectancies than boys. In addition, boys revised their expectations upward when the evaluator changed, but girls did not raise their expectations. In the second study, fourth, fifth, and sixth graders were asked for their expectations concerning school grades. As expected, boys had higher expectations than girls at the beginning of the school term, despite the girls' previous school records.

In summary, these studies all point to a greater incidence of learned helplessness (i.e., the perception of uncontrollability in the

face of failure) in girls accompanied by a specific attribution. Indeed, girls blame their failure on lack of ability with ability being viewed as an internal, stable, and global attribution. Boys, on the other hand, blame failure on external (e.g., bad luck, evaluator) or motivational (e.g., lack of effort) factors. Girls' expectations for success are lower than boys, and girls' expectations after failure are less resilient than boys' expectations after failure. Finally, girls relative to boys are less persistent and show performance deficits in response to failure. Although the dimension of depression was not assessed in these children, it should be remembered that the reformulated learned helplessness model of depression sees both depression and learned helplessness as sharing common parallels, including attributional style.

Turn now to the studies on the reformulated model using adults as subjects. Here the evidence of sex difference is inconclusive or lacking. Amenson and Lewinsohn (1981) in their community sample of 998 males and females did not find a consistent relationship between attributing failure to internal causes and unipolar depression. However, contrary to their predictions, they found that men relative to women, were less likely to attribute success to internal causes and less likely to attribute failure to external causes. Note, these findings are inconsistent with the results of Dweck and her associates with male and female children.

Of the studies reviewed on depressive attributional style in the last section, four studies used only one sex in their sample groups

(Costello, 1982 in her outpatient group; Harvey, 1981; Kuiper, 1978; Raps et al., 1982). Eight studies did not directly focus on the variable of sex either because there were no significant differences in sex distribution between depressed and nondepressed groups or presumably because sex difference was not viewed as a potentially significant confounding variable (Forsyth & McMillan, 1981; Golin et al., 1981; Gong-Guy & Hammen, 1980; Hammen & Cochran, 1981; Klein et al., 1976; Miller et al., 1982; Rizley, 1978; Seligman et al., 1979). The remaining studies (Blaney et al., 1980; Costello, 1982 in her undergraduate groups; Feather & Davenport, 1981; Hammen & DeMayo, 1982) reported no evidence of sex differences in attributional style. However, subjects in these studies were not drawn from a clinical population and only subjects in Blaney et al.'s (1980) study filled out the complete ASQ.

In light of the lack of conclusive evidence on sex differences, it seems worthwhile to focus on sex as a variable in depressive attributional style. In the present study, it is expected that females more than males should attribute bad outcomes to internal, stable, and global causes and attribute good outcomes to external, unstable, and specific causes.

### Hypotheses

Basically this study examines the reformulated model of learned helplessness in a clinical population. The specific hypotheses being tested include: (1a) Clinically depressed subjects have higher ratings of internality, stability, globality, and composite scores for bad outcomes than nondepressed subjects; (1b) Clinically depressed

subjects have lower ratings of internality, stability, globality, and composite scores for good outcomes than nondepressed subjects; (1c) Clinically depressed subjects' bad and good outcome composite scores are more equal than nondepressed subjects' bad and good outcome composite scores; (2a) Severely depressed patients have higher ratings of internality, stability, globality, and composite scores for bad outcomes than mildly to moderately depressed patients; (2b) Severely depressed patients have lower ratings of internality, stability, globality, and composite scores for good outcomes than mildly to moderately depressed patients; (2c) Severely depressed patients' bad and good outcome composite scores are more equal than mildly to moderately depressed patients' bad and good outcome composite scores; (3a) Females have higher ratings of internality, stability, globality, and composite scores on bad outcomes than males; (3b) Females have lower ratings of internality, stability, globality, and composite scores on good outcomes than males; (3c) Females' bad and good outcome composite scores are more equal than males' bad and good outcome composite scores; and (4) Psychopathology, per se, makes no difference in subjects' attributions.

## METHOD

### Subjects

A total of 123 individuals voluntarily participated as subjects in this study. All subjects had a minimum of an eighth grade education (range was eighth grade to post-graduate work) and sufficient reading and comprehension ability to complete the self-report measures. One hundred and one subjects were psychiatric inpatients and outpatients at the following Indianapolis-based medical facilities: Larue D. Carter Hospital (52 patients); Indiana University Hospital (11 patients); Veterans Administration Hospital (33 patients); and Long Outpatient Clinic (5 patients). All of these psychiatric patients were in treatment for less than three months at the time they completed the various questionnaires. Of these 101 patients, 54 were males and 47 were females. Ages ranged from 17.5 years old to 67 years old ( $M = 35.09$ ,  $SD = 12.33$ ). Marital status was as follows: 37 single, 18 divorced, 32 married, 13 separated, and 1 widowed.

A remaining group of 22 subjects were drawn from an adult non-psychiatric population and were all voluntary participants of a weight loss group in the Indianapolis area. They had been attending group sessions for less than two months at the time of testing. Nine were males and 13 were females. Ages ranged from 18 years old to 58 years old ( $M = 41.09$ ,  $SD = 11.46$ ). Marital status was as follows: 4 single, 16 married, 1 widowed, and 1 marital status unknown.



## Measures

Four questionnaires are employed in this study. These questionnaires are the Attributional Style Questionnaire (ASQ), the Beck Depression Inventory (BDI), the Multiple Affect Adjective Check List (MAACL), and the Minnesota Multiphasic Personality Inventory (MMPI). Each questionnaire is described below.

The ASQ (Seligman, Abramson, Semmel, & von Baeyer, 1979) is a self-administered, relatively new test which assesses attributional style. It consists of 12 hypothetical situations with six of the situations describing bad outcomes and six of the situations describing good outcomes. Six of the situations have an affiliation orientation while the other six situations have an achievement orientation. Testees are asked to first vividly imagine each situation and decide what they feel would be the major cause of the situation if it happened to them and record this cause on the test booklet. They then are required to rate each generated cause on a seven-point scale for degree of internality (i.e., from "totally due to the other person or circumstances" to "totally due to me"), for degree of stability (i.e., from "will never again influence what happens" to "will always influence what happens"), and for degree of globality (i.e., from "influences just this particular situation" to "influences all situations in my life"). Also, testees rate on a seven-point scale how important each situation would be if it happened to them. Internal reliabilities as reported by Seligman et al. (1979) for the individual subscales using alpha coefficients are: internality for bad outcomes = .44;

internality for good outcomes = .39; stability for bad outcomes = .63; stability for good outcomes = .54; globality for bad outcomes = .64; globality for good outcomes = .58. Peterson et al. (cited in Raps et al., 1982) report higher reliabilities on composite scores based on the sum of internality, stability, and globality scores. Specifically, reliability for composite scores on bad outcomes is .72 and reliability for composite scores on good outcomes is .75. Test-retest correlations over five weeks approach the internal reliabilities for individual subscales and for composite scores. In terms of validity of the ASQ, Peterson et al. (cited in Raps et al., 1982) report that the ASQ predicts attributions made by people about actual life events; predicts the generality of the helplessness deficits produced in experiments; and predicts the reports of depressive symptoms following failure on a test. See Appendix A for the specific instructions and content of the ASQ.

The BDI (Beck, 1967) is a self-report inventory which measures depth of depression by taking into account both the total number of depressive symptoms and the severity of the symptoms. Testees are asked to read 21 multiple choice statements and within each item choose the one best statement that describes the way they feel.<sup>1</sup>

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<sup>1</sup>Originally the BDI was administered by a trained interviewer who would read aloud the statements to the patients and mark down their answers (Beck, 1967). Learned helplessness studies have not used an interviewer, but have had respondents answer the BDI by themselves. DePue and Monroe (1978) perceive this difference in test administration as a potentially inherent problem in learned helplessness research. However, in one of the original cross-validation studies, Metcalfe and

If desired, however, they are permitted to choose more than one statement in each item. These statements correspond to previously identified affective (e.g., dejected mood, crying), cognitive (e.g., low self-evaluations, negative expectations), motivational (e.g., loss of motivation), and physical (e.g., loss of appetite, sleep disturbance) factors of clinical depression. Reliability measures using protocols of 200 cases reveal a split-half reliability of .86 (Beck, 1967). The BDI significantly correlates with other depressive inventories, including the MAACL-D (Nussbaum, Witting, & Hanlon, 1963) and the MMPI-D (Burkhart, Gynther, & Fromuth, 1980; Nussbaum et al., 1963). Strong correlations between BDI scores and psychiatric ratings range from .61 to .67 (Beck, 1967; Metcalfe & Goldman, 1965; Nussbaum et al., 1963). In a recent study, Bumberry, Oliver, and McClure (1978) have shown that BDI scores can distinguish between nondepressed (BDI values 0-9); mildly depressed (BDI values 10-15); moderately depressed (BDI value 16-23); and severely depressed subjects (BDI values 24-63). These values are used in this study to distinguish between varying degrees of depression.

The MAACL (Zuckerman & Lubin, 1965) is a brief, self-administered check list which provides a measure of three clinically relevant

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Goldman (1965) had those clinical patients who were sufficiently alert read the statements to themselves while a psychologist or nurse was in the room. Moreover, King and Buchwald (1982) found that the type of administration had no effect on BDI scores in college students. Thus, in the present study, for consistency purposes, subjects are asked to complete the BDI by themselves.

negative affects: anxiety, depression, and hostility. Twenty-one of the items are scorable on the anxiety key, 40 items are scorable on the depression key, 28 items are scorable on the hostility key, and the remaining 43 items are buffer items. The MAACL can be used as either a state measure or a trait measure. For the purposes of the present study, the focus is on the MAACL-Depression Scale (MAACL-D) as a state measure. Testees are simply asked to check all the words that describe their feelings at the time of testing. Internal reliabilities for the MAACL-D range from .65 to .92 (Zuckerman & Lubin, 1967) and test-retest reliabilities range from .21 in college students, to .79 in psychiatric patients (Zuckerman & Lubin, 1967). Since its inception, the MAACL, as a research tool, has been used by several investigators concerned with evaluating a wide variety of effects including sensory deprivation, examinations, frustration, failure, pain, stress, drug treatment and therapy (Kelly, 1972).

The MMPI (Hathaway & McKinley, 1943) is a self-administered, true-false item questionnaire which is considered among the most useful psychometric instruments in many clinical environments. It is composed of nine clinical scales, i.e., Hypochondriasis, Depression, Hysteria, Psychopathic Deviate, Masculinity-Femininity, Paranoia, Psychoasthenia, Schizophrenia, and Hypomania. In addition, there is a Social Introversion scale. The MMPI has four validity scales, which are unanswered questions (?), Lie (L), Frequency (F), and Correction (K). In the current investigation the MMPI provides a third measure of depressive affect and assesses severity of psychopathology. The

depression scale on the MMPI (MMPI-D) has sixty items which tap apathy, dissatisfaction, lack of optimism, physical symptoms, etc. Test-retest reliability coefficients based on up to two week intervals range from .72 to .89 for psychiatric patients and range from .69 to .96 for nonpsychiatric patients (Dahlstrom, Welsh, & Dahlstrom, 1975, pp. 253-258). Although it is generally standard practice in clinical interpretations to look at an individual's configuration of scores, Endicott and Jortner (1966) provide some evidence for absolute scaling of depression on the MMPI. Specifically, the MMPI-D correlated .51 with clinically rated depression for psychiatric inpatients and outpatients. Moreover, Zuckerman, Persky, Eckman, and Hopkin (1967) found that in their sample of clinical patients the MMPI-D scale correlated .59 with clinical ratings of depression. Thus, in the present study absolute depression scores, rather than profile configurations of depression are used. As noted, the MMPI is also employed in this study to assess global psychopathology. A variety of MMPI indices are used to measure the severity of psychiatric illness. Of these measures, Shaffer, Ota, and Harion (1964) found that the best single MMPI index was Peterson signs (Peterson, 1954), the Paranoia scale, and the F scale. For all practical purposes the differences between these measures and their ability to predict the Total Morbidity Scale derived from the Multidimensional Scale for Rating Psychiatric Patients were slight. McAdoo and Connolly (1975) found that parents who were seeking help for themselves in an adult outpatient clinic had a significantly higher number of Peterson six signs than child guidance parents for whom the child was the identified patient. This

finding was consistent with their results that adult outpatient parents, relative to child guidance parents, scored significantly higher on several other indices of psychopathology, including mean profile deviations, inverted V on the vector of validity scales, F, Tamkin's pathology scale (Tamkin, 1959), Pa, and Ip (Sines & Silver, 1963). In the current study, Peterson six signs are used to measure severity of psychiatric illness, per se, and they serve as a global measure of psychopathology. The MMPI-Peterson six signs are: (1) T scores on 4 or more clinical scales over 70; (2) F greater than 65; (3) Sc greater than Pt; (4) Pa or Ma greater than 70; (5) Pa or Sc or Ma greater than Hs and D and Hy; (6) D greater than Hs and Hy.

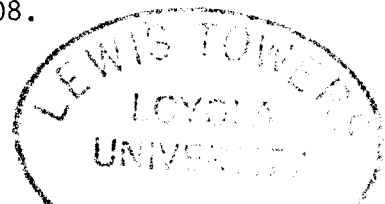
#### Procedure

All subjects were administered the questionnaires either individually or in small groups. When patients were approached about the study, they initially were asked to fill out a consent form. See Appendix B. They were then given a packet containing the ASQ, the BDI, and the MAACL. All instructions for responding to the scales were included with each scale and subjects were allowed to proceed with the questionnaires at their own pace. The majority of the subjects took between 20 minutes and 75 minutes, averaging approximately 40 minutes, to complete the three questionnaires in the packet. The primary investigator was available for all but 19 subjects to answer any questions about the material. The 19 subjects who had no contact with the primary investigator, nevertheless, were able to get any necessary assistance from a qualified clinician who had become familiar with the above tests.

Since the MMPI was frequently used in routine diagnostic evaluations for psychiatric patients at the various institutions, it was usually given by trained hospital or clinic personnel before these subjects were asked to participate in this study and before they had filled out the three other questionnaires. Thus, after subjects had completed the packet containing the ASQ, BDI, and MAACL, they were asked for their permission to use their MMPI profile scores, if they, in fact, had already completed the MMPI. No subject refused to give the necessary permission. If, however, subjects did not have an MMPI within the last three months, they were asked to fill one out at the time of the first testing or at a later prearranged session. The time lag for all patients between the MMPI testing and the administration of the ASQ, BDI, and MAACL was  $\underline{M} = 7.37$  days,  $\underline{SD} = 10.55$  days. The control group of Nondepressed-Low Psychopathology subjects had already taken the MMPI one week prior to when they were administered the ASQ, BDI, and MAACL. Finally, all subjects were thanked for their cooperation.

Nine dependent measures, each corresponding to an attributional outcome, were employed in this study: (1) Bad Internality; (2) Bad Stability; (3) Bad Globality; (4) Bad Composite; (5) Good Internality; (6) Good Stability; (7) Good Globality; (8) Good Composite; and (9) Good minus Bad Composite called Evenhandedness. Bad Internality looked at the impact of internal attributions as compared to external attributions in bad outcomes. Scores over the six possible bad situations could total from six (i.e., totally external) to 42 (i.e., totally internal). Bad Stability perceived causes in bad outcomes

as either more likely to happen again and be long lived or intermittent and short lived. Possible scores could range from six (i.e., totally unstable) to 42 (i.e., totally stable). Bad Globality portrayed the degree to which causes in bad outcomes were viewed as occurring and affecting a broad range of situations versus viewed only as affecting one specific situation. Total scores could range from six (i.e., totally specific) to 42 (i.e., totally global). Bad Composite reflected the combined total scores of internality, stability, and globality for bad outcomes. Thus, total scores could be between 18 (i.e., totally external, unstable, and specific) to 126 (i.e., totally internal, stable, and global). Good Internality, as in Bad Internality, portrayed a continuum of scores between six (i.e., totally external) to 42 (i.e., totally internal), but had good outcomes. Good Stability showed the extent to which attributions in good outcomes were stable or unstable, and like Bad Stability scores could range from six (i.e., totally unstable) to 42 (i.e., totally stable). Good Globality revealed whether attributions for good outcomes were more global or more specific. Possible scores, as in Bad Globality, could range from six (i.e., totally specific) to 42 (i.e., totally global). Good Composite reflected the combined total scores of internality, stability, and globality for good outcomes. Like Bad Composite, scores could range from 18 (i.e., totally external, unstable, and specific) to 126 (i.e., totally internal, stable, and global). Evenhandedness reflected the general tendency to similarly explain the causes of good and bad outcomes. Absolute scores could range from zero to 108.





There were three basic analyses performed on the first eight dependent measures. These were Pearson product moment correlation coefficients, partial correlations, and analysis of variance. On the ninth dependent measure, i.e., Evenhandedness, only the analysis of variance was performed.

The Pearson product moment correlations were employed to analyze the relationships between the attribution measures and depression and between the attribution measures and psychopathology. Three different depression measures were used: BDI scores, MAACL-D scores, and MMPI-D scores. The psychopathology measure was the number of Peterson signs out of six possible signs on the MMPI.

In addition to the above correlations, 16 partial correlations were carried out using BDI scores as the depressive measure, and MMPI-Peterson six signs as the psychopathology measure, and the ASQ attributional measures. Eight of these partial correlations sought to control for the effects of psychopathology on subjects' attribution scores, and thus reflected the sole impact of depression on attributional style. The remaining eight partial correlations controlled for the effects of depression, thus identifying the exclusive role of psychopathology on people's attributions.

Finally, separate 3 x 2 analyses of variance for three levels of depression using BDI scores (i.e., Nondepressed, Mildly to Moderately Depressed, and Severely Depressed) and two levels of psychopathology using MMPI-Peterson six signs (i.e., Low Psychopathology and High

Psychopathology) were performed on all of the attribution measures.

### Classification of Subjects

On the basis of scores on the BDI and the MMPI-Peterson six signs, subjects were assigned to one of six groups: (1) Nondepressed-Low Psychopathology; (2) Mildly to Moderately Depressed-Low Psychopathology; (3) Severely Depressed-Low Psychopathology; (4) Nondepressed-High Psychopathology; (5) Mildly to Moderately Depressed-High Psychopathology; (6) Severely Depressed-High Psychopathology.

Subjects in the Nondepressed-Low Psychopathology group were from the weight loss sample and served as a nonpsychiatric control group. They had BDI scores of nine or less and had scores of three or less on MMPI-Peterson six signs. In addition, all these subjects were judged as not being clinically depressed and as being low on psychopathology. There were 13 females and nine males in this group.

The remaining five groups were all comprised of psychiatric patients. Subjects in the Mildly to Moderately Depressed-Low Psychopathology group had BDI scores of between 10 and 23 and had scores of three or less on MMPI-Peterson six signs. As in all of the depressed groups, these psychiatric patients were diagnosed as being depressed in their psychiatric work-up, although the diagnoses did not necessarily conform to DSM-III. Ten females and 16 males were in this group.

Subjects in the Severely Depressed-Low Psychopathology group had BDI scores of greater than 24 and had scores of three or less on MMPI-Peterson six signs. They were diagnosed as being depressed by

the clinical staff. Nine females and eight males served as subjects in this group.

Subjects in the Nondepressed-High Psychopathology group had BDI scores of nine or less and had scores of four or more on MMPI-Peterson six signs. These patients were evaluated as not being clinically depressed by the admitting clinicians. Eight females and nine males met the criteria for this group.

Subjects in the Mildly to Moderately Depressed-High Psychopathology group had BDI scores of between 10 and 23 and had scores of four or more on MMPI-Peterson six signs. They were seen as being depressed by the clinical staff. There were 10 females and nine males in this group.

Subjects in the Severely Depressed-High Psychopathology group had BDI scores of greater than 24 and had scores of four or more on MMPI-Peterson six signs. They were viewed as being clinically depressed in their psychiatric work-up. Ten females and 12 males participated in this group.

The means and standard deviations of BDI scores and MMPI-Peterson six signs for all subjects are listed in Table 1.

In addition to the 101 psychiatric patients described above, 84 psychiatric patients were not included in the final sample group for various reasons. Specifically, 13 subjects did not satisfactorily complete the various questionnaire. One subject was over 70 years old.

Table 1

Means and Standard Deviations of BDI Scores and MMPI-Peterson  
Six Signs for All Subjects

	<u>BDI Scores</u>								
	Nondepressed			Mildly to Moderately Depressed			Severely Depressed		
	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>
Low Psychopathology	22	5.23	2.74	26	16.15	3.39	17	30.00	6.96
High Psychopathology	17	5.82	3.13	19	18.63	3.76	22	33.14	5.56

	<u>MMPI-Peterson Six Signs</u>								
	Nondepressed			Mildly to Moderately Depressed			Severely Depressed		
	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>
Low Psychopathology	22	1.41	1.01	26	2.15	.78	17	2.29	.77
High Psychopathology	17	4.76	.75	19	5.05	.71	22	5.09	.81

Thirty-two subjects had depression scores on the BDI of greater than 10, i.e., signifying depression, yet they were not considered depressed by the clinical staff. Two subjects had a diagnosis of depression, but their BDI scores were below 10. Two subjects experienced major diagnostic changes during testing, i.e., went from a manic to a depressive state. Seven patients had an unspecified diagnosis. Finally, 27 psychiatric patients were discarded because they had scores on both the BDI of below 10 and scores of three or less on MMPI-Peterson six signs. Thus, they could be viewed as neither depressed nor high on psychopathology.

## RESULTS

The depressive attributional style outlined by Seligman and his colleagues was generally supported. Before presenting the data, however, three general comments need to be made. First, one of the main concerns in this study was to examine any differences between males and females in terms of a depressive attributional style. Since none of the correlations between sex and the attributional measures even approached significance, scores for males and females were combined in all of the reported findings. Second, there has been no indication that weighted scores as compared to unweighted scores increased the association between attribution measures and degrees of depression (Blaney et al., 1980). Consequently, scores were not weighted for subjects' ratings of importance of outcome in each attribution item. Third, as noted, three different depression scales were used, i.e., BDI, MAACL-D, and MMPI-D, in the original correlation matrix to examine the relationship between attribution and depression. The Pearson product moment correlation coefficients between these scales were highly significant, thus signalling strong concurrent validity for these depression measures. Specifically, the correlation between BDI scores and MAACL-D scores was  $r(121) = .71, p < .001$ ; the correlation between BDI scores and MMPI-D scores was  $r(121) = .65, p < .001$ ; and the correlation between MAACL-D scores and MMPI-D scores was  $r(121) = .59, p < .001$ .

Turning now to the findings, for organizational purposes separate subheadings are used for each one of the attributional measures, that is, Bad Internality, Bad Stability, Bad Globality, Bad Composite, Good Internality, Good Stability, Good Globality, Good Composite, and Evenhandedness. Under each subheading, statistical results pertinent to that attribution are reported, specifically Pearson product moment correlation coefficients, partial correlations, and analysis of variance, with depression and psychopathology as independent variables. Following these nine subheadings, a brief results summary section is included.

#### Bad Internality

It was hypothesized that depressed patients have greater internality scores for bad outcomes than nondepressed individuals. Simple correlations did not support this hypothesis. Specifically, the correlation between BDI scores and Bad Internality was  $r(121) = .17$ , ns; the correlation between MAACL-D scores and Bad Internality was  $r(121) = .13$ , ns; the correlation between MMPI-D scores and Bad Internality was  $r(121) = -.03$ , ns; However, when psychopathology was partialled out the correlation between BDI scores and Bad Internality was significant,  $r(118) = .20$ ,  $p < .05$ . Analysis of variance did not show any main effect for depression,  $F(2,117) = 1.53$ , ns. The means (and standard deviations) of the depressed groups are: Nondepressed = 27.05 (5.21); Mildly to Moderately Depressed = 27.98 (6.25); Severely Depressed = 29.59 (6.40). However, analysis of variance revealed a significant interaction between depression and psychopathology for Bad

Internality,  $F(2,117) = 4.40$ ,  $p < .05$ . The means and standard deviations of each of the groups are listed in Table 2. A subsequent Neuman-Keuls test was performed on these means and results revealed that at a .01 level of significance only the Severely Depressed-High Psychopathology group had a significantly higher Bad Internality score than the Nondepressed-High Psychopathology group. A less robust testing at the .05 level of significance showed that all groups, except for the Mildly to Moderately Depressed-High Psychopathology group, had higher Bad Internality scores than the Nondepressed-High Psychopathology group.

It was also hypothesized that psychopathology per se does not effect Bad Internality scores. There was a nonsignificant correlation between MMPI-Peterson six signs and Bad Internality scores,  $r(121) = .10$ , ns, and the partial correlation between MMPI-Peterson six signs and Bad Internality scores with depression controlled for was also nonsignificant,  $r(118) = -.16$ , ns. Moreover, there was not a significant main effect for psychopathology on the analysis of variance,  $F(1,117) = 1.53$ , ns.

### Bad Stability

It was predicted that depressed people have higher stability scores for bad outcomes than nondepressed people. This prediction was supported in that BDI scores correlated significantly with Bad Stability,  $r(121) = .19$ ,  $p < .05$ ; MAACL-D scores correlated with Bad Stability,  $r(121) = .24$ ,  $p < .02$ ; and MMPI-D scores correlated with Bad Stability,  $r(121) = .22$ ,  $p < .05$ . Moreover, when the effects of



Table 2

Means and Standard Deviations of Internality Scores  
for Bad Outcomes for All Subjects

	Nondepressed			Mildly to Moderately Depressed			Severely Depressed		
	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>
Low Psychopathology	22	29.55	3.66	26	27.85	6.42	17	28.76	5.47
High Psychopathology	17	23.82	5.22	19	28.16	6.18	22	30.23	7.09

psychopathology were controlled for, the correlation between BDI scores and Bad Stability continued to be significant,  $r(118) = .19$ ,  $p < .05$ . Analysis of variance also revealed a significant main effect for depression,  $F(2,117) = 3.25$ ,  $p < .05$ . A subsequent Neuman-Keuls test showed that at a .05 level of significance the Severely Depressed group had greater Bad Stability scores than the Nondepressed group. The means (and standard deviations) of each of the groups are: Nondepressed = 27.74 (6.30); Mildly to Moderately Depressed = 29.13 (6.48); Severely Depressed = 31.00 (5.95).

As predicted, no significant differences were associated with psychopathology on Bad Stability scores. Specifically, the correlation between MMPI-Peterson six signs and Bad Stability was nonsignificant,  $r(121) = .05$ , and the partial correlation between MMPI-Peterson six signs and Bad Stability with depression controlled for was also not significant,  $r(118) = .00$ . In addition, the analysis of variance revealed no main effect for psychopathology,  $F(1,117) = .01$ , ns, nor was there an interaction effect between psychopathology and depression for Bad Stability,  $F(2,117) = 1.42$ , ns.

### Bad Globality

The hypothesis that depressed subjects as compared to nondepressed subjects are more global for bad outcomes was supported. The correlation between BDI and Bad Globality was  $r(121) = .34$ ,  $p < .001$ ; the correlation between MAACL-D and Bad Globality was  $r(121) = .32$ ,  $p < .001$ ; and the correlation between MMPI-D and Bad Globality was  $r(121) = .34$ ,  $p < .001$ . By partialling out psychopathology, the

correlation between BDI scores and Bad Globality was  $r(118) = .31$ ,  $p < .01$ . With respect to the analysis of variance, there was a significant main effect for depression,  $F(2,117) = 7.03$ ,  $p < .005$ . The means (and standard deviations) of each of the depressed groups are: Non-depressed = 24.56 (8.40); Mildly to Moderately Depressed = 27.80 (7.20); Severely Depressed = 30.77 (7.08). A Neuman-Keuls test revealed that the Severely Depressed group had significantly higher Bad Globality scores than the Nondepressed group at a .01 level of significance, whereas at a .05 level of significance both the Mildly to Moderately Depressed group and the Severely Depressed group had higher Bad Globality scores than the Nondepressed group.

It was hypothesized that Low Psychopathology and High Psychopathology groups do not differ in their ratings for globality for bad outcomes. The data supported this hypothesis. The correlation between MMPI-Peterson six signs and Bad Globality was nonsignificant,  $r(121) = .13$ , the partial correlation between MMPI-Peterson six signs and Bad Globality with depression controlled for was also nonsignificant,  $r(118) = .04$ , and there was no main effect for psychopathology on the analysis of variance,  $F(1,117) = .21$ , ns and no indication of a significant interaction effect with psychopathology and depression for Bad Globality,  $F(2,117) = 1.41$ , ns.

#### Bad Composite

It was hypothesized that depressed subjects have higher composite scores (i.e., combined scores of internality, stability, and globality) for bad outcomes than nondepressed subjects. The correlation between

BDI scores and Bad Composite was  $r(121) = .31, p < .01$ ; the correlation between MAACL-D scores and Bad Composite was  $r(121) = .30, p < .01$ ; and the correlation between MMPI-D scores and Bad Composite was  $r(121) = .25, p < .01$ . The partial correlation between BDI scores and Bad Composite with psychopathology partialled out was  $r(118) = .31, p < .01$ . Analysis of variance using the Bad Composite scores yielded two significant effects. There was a main effect for depression,  $F(2,117) = 7.20, p < .005$  and an interaction effect for depression by psychopathology,  $F(2,117) = 3.33, p < .05$ . Means and standard deviations of all groups are listed in Table 3. With respect to the main effect for depression, a Neuman-Keuls test on the data showed that at a .01 level of significance the Severely Depressed group had a significantly greater Bad Composite score than the Nondepressed group. In order to understand the depression by psychopathology impact, another Neuman-Keuls test was performed and showed that at a .01 level of significance both Severely Depressed-High Psychopathology and Severely Depressed-Low Psychopathology groups had higher Bad Composite scores than the Nondepressed-High Psychopathology group. Moreover, by using a .05 level of significance the Mildly to Moderately Depressed-High Psychopathology group also had a higher Bad Composite score than the Nondepressed-High Psychopathology group.

The hypothesis that Low Psychopathology and High Psychopathology groups do not differ in their composite scores for bad outcomes was supported except for the interaction effect described above. The correlation between MMPI-Peterson six signs and Bad Composite scores

Table 3

Means and Standard Deviations of Composite Scores for Bad Outcomes for Depression, Psychopathology, and Depression by Psychopathology

<u>Depression as a Main Effect</u>	<u>N</u>	<u>M</u>	<u>SD</u>	<u>Psychopathology as a Main Effect</u>	<u>N</u>	<u>M</u>	<u>SD</u>
Nondepressed	39	79.38	15.29	Low Psychopathology	65	84.85	13.81
Mildly to Moderately Depressed	45	84.91	13.84	High Psychopathology	58	85.60	17.38
Severely Depressed	39	91.36	15.68				

Depression by Psychopathology

	Nondepressed			Mildly to Moderately Depressed			Severely Depressed		
	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>
Low Psychopathology	22	83.82	13.44	26	82.46	12.89	17	89.82	15.15
High Psychopathology	17	73.65	15.99	19	88.26	14.72	22	92.55	16.33

was  $r(121) = .05$ , ns. When depression was partialled out, the correlation between MMPI-Peterson six signs and Bad Composite scores remained nonsignificant,  $r(118) = -.04$ . Also, there was no main effect for psychopathology on the analysis of variance,  $F(1,117) = .04$ , ns.

### Good Internality

The prediction that depressed individuals in comparison to non-depressed individuals have lower scores on internality for good outcomes was substantiated by the data. Findings revealed significant negative correlations at the .02 level of significance between BDI scores and Good Internality,  $r(121) = -.23$ ; between MAACL-D scores and Good Internality,  $r(121) = -.24$ ; but not between MMPI-D scores and Good Internality,  $r(121) = -.17$ , ns. Controlling for psychopathology yielded a partial correlation of  $r(118) = -.24$ ,  $p < .02$ . The analysis of variance revealed a significant main effect for depression,  $F(2, 117) = 4.32$ ,  $p < .05$ . The means (and standard deviations) for each depressed group are: Nondepressed = 28.46 (3.95); Mildly to Moderately Depressed = 26.36 (3.55); Severely Depressed = 25.87 (5.08). Results of the Neuman-Keuls test on these means showed that the significant main effect for depression was due to the Severely Depressed group having a significantly lower Good Internality score than the Non-depressed group ( $p < .05$ ).

With respect to psychopathology, per se, it was not found to have a noticeable effect on internality scores for bad outcomes. The correlation between MMPI-Peterson six signs and Good Internality was  $r(121) = -.01$ , ns, and the correlation between MMPI-Peterson six signs and

Good Internality when depression was partialled out was  $\underline{r}(118) = .06$ , ns. Moreover, the analysis of variance did not produce a significant main effect for psychopathology,  $\underline{F}(1,117) = .33$ , ns, or a significant interaction effect between psychopathology and depression for Good Internality,  $\underline{F}(2,117) = .38$ , ns.

### Good Stability

It was predicted that the more depressed subjects have lower stability scores on good outcomes than the less depressed subjects. However, depression scores did not correlate significantly with the subjects' ratings for good outcomes. The correlation between BDI scores and Good Stability was  $\underline{r}(121) = -.12$ , ns; the correlation between MAACL-D scores and Good Stability was  $\underline{r}(121) = -.17$ , ns; and the correlation between MMPI-D scores and Good Stability was  $\underline{r}(121) = -.07$ , ns. Partialling out psychopathology did not improve the relationship between BDI scores and Good Stability,  $\underline{r}(118) = -.11$ , ns. The analysis of variance did not show any significant main effect for depression,  $\underline{F}(2,117) = 1.72$ , ns. The means (and standard deviations) of the depressed groups are: Nondepressed = 34.08 (4.30); Mildly to Moderately Depressed = 32.24 (4.26); Severely Depressed = 33.31 (5.05).

The hypothesis that Low Psychopathology versus High Psychopathology groups have similar Good Stability scores was upheld. The correlation between psychopathology and stability on good outcomes was not significant,  $\underline{r}(121) = -.04$ , nor was the correlation between psychopathology and Good Stability significant when depression was partialled out,  $\underline{r}(118) = -.01$ , nor was there an appreciable main effect for

psychopathology on the analysis of variance,  $F(1,117) = .03$ , ns, nor was there a significant interaction effect between psychopathology and depression for Good Stability,  $F(2,117) = .10$ , ns.

### Good Globality

As with Good Stability, the hypothesis that depressed subjects have lower globality scores for good outcomes than nondepressed subjects was not supported. The correlation between BDI scores and Good Globality scores was  $r(121) = -.06$ , ns; the correlation between MAACL-D scores and Good Globality was  $r(121) = -.005$ , ns; and the correlation between MMPI-D scores and Good Globality was  $r(121) = .02$ , ns. Even when psychopathology was controlled for, the partial correlation between BDI scores and Good Globality was nonsignificant,  $r(118) = -.06$ . There was no main effect for depression,  $F(2,117) = 1.01$ , ns, on the analysis of variance. The means (and standard deviations) of each of the depressed groups are: Nondepressed = 31.36 (5.32); Mildly to Moderately Depressed = 31.87 (4.83); and Severely Depressed = 30.26 (5.08).

As predicted, the Low Psychopathology versus High Psychopathology distinction made virtually no difference on subjects' ratings of globality for good outcomes. The correlation between MMPI-Peterson six signs and Good Globality was  $r(121) = -.03$ , ns; the correlation between MMPI-Peterson six signs and Good Globality when depression was partialled out was  $r(118) = -.01$ , ns; the main effect for



psychopathology on the analysis of variance was trivial,  $F(1,117) = .00$ , ns; and the interaction effect between psychopathology and depression on the analysis of variance was nonsignificant,  $F(2,117) = .33$ .

### Good Composite

Good Composite scores reflect the combined totals for internality, stability, and globality for good outcomes, and it was predicted that Good Composite scores are less for depressed rather than nondepressed individuals. This prediction was not supported by the data. Correlations between the depressive measures and composite scores for good outcomes did not reach significance, although they were in the predicted direction. Specifically, the correlation between BDI scores and Good Composite was  $r(121) = -.18$ , ns; the correlation between MAACL-D scores and Good Composite was  $r(121) = -.18$ , ns; and the correlation between MMPI-D scores and Good Composite was  $r(121) = -.10$ , ns. Even when psychopathology was partialled out, the correlation between BDI scores and Good Composite did not reach significance,  $r(118) = -.18$ , ns. The analysis of variance revealed no measurable difference between depressed and nondepressed groups on this attributional measure,  $F(2,117) = 2.10$ , ns. The means (and standard deviations) of the depressed groups are: Nondepressed = 93.90 (9.93); Mildly to Moderately Depressed = 90.60 (8.51); Severely Depressed = 89.44 (11.72).

As hypothesized, psychopathology was not found to have a significant impact on composite scores for good outcomes. The correlation

between MMPI-Peterson six signs and Good Composite was only  $r(121) = -.03$ , ns; the partial correlation between MMPI-Peterson six signs and Good Composite with depression controlled for was  $r(118) = .02$ , ns; the main effect for psychopathology on the analysis of variance was  $F(1,117) = .14$ , ns; and the interaction effect between psychopathology and depression on the analysis of variance was  $F(2,117) = .009$ , ns.

#### Evenhandedness (Good minus Bad Composite)

Good minus Bad composite signifies the general tendency to make internal, stable, and global attributions for good outcomes while making external, unstable, and specific attributions for bad outcomes. It was hypothesized that there is a closer association between attributions for good and bad outcomes in depressed people's responses than in nondepressed people's responses. To test this hypothesis, Evenhandedness scores were formed by calculating the absolute value of a person's total composite score for good events minus his total composite score for bad events. An analysis of variance revealed a significant main effect for depression,  $F(2,117) = 3.56$ ,  $p < .05$ , and a significant interaction effect for depression by psychopathology,  $F(2,117) = 3.95$ ,  $p < .05$ . Means and standard deviations of all groups are listed in Table 4. In terms of the main effect for depression, a Neuman-Keuls test on the data showed that at a .05 level of significance Mildly to Moderately Depressed subjects, in contrast to Nondepressed subjects, judged the causes of bad and good outcomes to be similar. Another Neuman-Keuls test looked at the interaction effect and found that at a .01 level of significance the Mildly to Moderately

Table 4

Means and Standard Deviations of Evenhandedness Scores for Depression, Psychopathology, and Depression by Psychopathology

<u>Depression as a Main Effect</u>	<u>N</u>	<u>M</u>	<u>SD</u>	<u>Psychopathology as a Main Effect</u>	<u>N</u>	<u>M</u>	<u>SD</u>
Nondepressed	39	16.00	12.05	Low Psychopathology	65	12.01	8.36
Mildly to Moderately Depressed	45	10.76	8.75	High Psychopathology	58	14.58	12.53
Severely Depressed	39	13.31	10.49				

Depression by Psychopathology

	Nondepressed			Mildly to Moderately Depressed			Severely Depressed		
	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>	<u>N</u>	<u>M</u>	<u>SD</u>
Low Psychopathology	22	11.64	9.40	26	11.62	7.77	17	13.12	8.24
High Psychopathology	17	21.65	12.98	19	9.58	10.04	22	13.45	12.14

Depressed-High Psychopathology group had more similar Evenhandedness scores than the Nondepressed-High Psychopathology group. At a .05 level of significance, all groups had a closer association between attributions for bad and good events than the Nondepressed-High Psychopathology group.

### Results Summary

The depressive attributional style proposed by Seligman and his associates leads to the prediction that depressed subjects, as compared to nondepressed subjects, have greater ratings of internality, stability, globality, and composite scores for bad outcomes and have lower ratings of internality, stability, globality, and composite scores for good outcomes. The findings, as described in the previous pages, were generally in agreement with this prediction. Specifically, stability, globality, and composite scores for bad events were significantly correlated in the predicted directions with the depressive measures. Internality scores for good outcomes correlated significantly in the predicted direction on all depression measures, except for MMPI-D scores. Internality scores for bad outcomes and composite scores for good outcomes tended to correlate with depression in the predicted directions and internality for bad outcomes reached full significance in the partial correlations when psychopathology was controlled for in subjects' ratings. Stability ratings and globality ratings for good outcomes were not found to be associated with depression scores.

Furthermore, it was predicted that the depressive attributional

style is more pronounced in extreme cases of depression, i.e., Severely Depressed groups are expected to exhibit the depressive attributional style more than Mildly to Moderately Depressed groups, although even Mildly to Moderately Depressed groups are still expected to exhibit the depressive attributional style. When depression was a main factor in differentiating subjects, results showed that major differences, indeed, occurred mostly between the Nondepressed and the Severely Depressed groups. Mildly to Moderately Depressed groups were less frequently distinguished from the Nondepressed groups and then usually so at only a less robust significance level. The only major exception to these findings was for Evenhandedness scores.

Lastly, it was predicted that psychopathology, per se, does not have an appreciable effect on the subjects' attribution scores, so as to add credibility to the uniqueness of the learned helplessness model of depression. Except for internality for bad outcomes (i.e., the correlation between Bad Internality and depression was only significant when psychopathology was partialled out), psychopathology, alone, did not exert any noticeable impact on attributional ratings for either bad or good outcomes. However, there were some significant interactional influences of psychopathology and depression on Bad Internality, Bad Composite, and Evenhandedness.

## DISCUSSION

The present study examined predictions derived from the reformulated learned helplessness model of depression on a clinical population. For the most part, the findings supported a depressive attributional style. The more depressed subjects were, the more they attributed bad outcomes to stable and global causes, and tended to attribute bad outcomes to internal causes. Moreover, when psychopathology was partialled out of the analyses, the relationship between internality for bad outcomes and depression reached significance.

One plausible explanation for the weaker association between internality for bad outcomes and depression may be due, in part, to the Nondepressed-Low Psychopathology group. This control group was comprised of males and females who were in the initial stages of a weight loss program where the emphasis was on personal control of their weight. Since these individuals presumably perceived their weight in a negative manner and they were encouraged to take full responsibility for their weight, i.e., attribute their relative weight to something about themselves, it may be that they generalized this perception of attributing internal causation to other negative events in their lives, including the hypothetical events of the ASQ. In order to test this speculation that the Nondepressed-Low Psychopathology group may have lowered the association between internality for bad outcomes and depression, scores of the psychiatric sample were analyzed separately.

Results showed that the correlation between BDI scores and Bad Internality was, indeed, highly significant,  $r(99) = .27$ ,  $p < .01$  for depressed and nondepressed psychiatric patients.

A second plausible explanation for the weaker association between internality for bad outcomes and depression may be partially due to there being two types of internality. Janoff-Bulman (1979) distinguishes between blame directed at one's character (i.e., it happened to me because I'm the sort of person to whom such things happen) versus blame directed at one's behavior (i.e., it happened to me because I did something) and proposes that only characterological blame produces helplessness and depression. Peterson, Schwartz, and Seligman (1981) found that overall depressive symptoms were, in fact, positively correlated with internal characterological attributions for negative events, but negatively correlated with internal behavioral attributions for negative events. In the current study only one score for internality for bad outcomes was assessed, and it certainly is conceivable that not all subjects made similar types of internal attributions. Thus, any possible significant correlation between internality for bad outcomes and depression could have been lowered by internal behavioral attributions partially cancelling out the effects of internal characterological attributions for bad outcomes.

Mixed results partially support a depressive attributional style for good events. Externality for good events was significantly correlated with depression. In addition, composite scores for good events tended to correlate with depression in the predicted direction,

although the correlation did not reach full significance. The less robust relationship between ASQ indices for good outcomes and depression is consistent with past findings for mildly depressed undergraduates (Blaney et al., 1980; Seligman et al., 1979) and for unipolar male depressives (Raps et al., 1982).

Differences were found between depressed and nondepressed individuals in their general tendency to make internal, stable, and global attributions for good events and to make external, unstable, and specific attributions for bad events. Specifically, it appeared that there was a closer association between good and bad composite scores called Evenhandedness scores for the Mildly to Moderately Depressed group than the Nondepressed group. In addition, the relationship between Evenhandedness scores was found to be more similar for the Severely Depressed-Low Psychopathology and Severely Depressed-High Psychopathology subjects (as well as for the Mildly to Moderately-Low Psychopathology and Mildly to Moderately Depressed-High Psychopathology subjects) than for Nondepressed-High Psychopathology subjects. Rizley (1978) observed that subclinically depressed college students explain both success and failure in similar ways, whereas nondepressed college students provide a different ascription for success than for failure in a self-serving way. In a clinical population, Raps et al. (1982) observed that unipolar depressives were more evenhanded in their attributions for good and bad composites than schizophrenics or medical-surgical patients. Thus, in three studies, including this one, there is some evidence that depressed subjects make similar attributions



for good and bad events. Moreover, as noted by Raps et al. (1982), evenhandedness is not inconsistent with the reported finding that depressed people externalize the causes of their success, because this finding usually results from comparing attributions of depressed subjects with those of nondepressed subjects.

As predicted (except for Evenhandedness scores), attributional differences between severely depressed people and nondepressed people were greater than attributional differences between mildly to moderately depressed people and nondepressed people. This finding lends support to the belief that mild to moderate depression is quantitatively different and not qualitatively different from severe depression. What is more, the relatively weaker association between mild to moderate depression and attributional style suggests that it may be more difficult to reach desired significance levels, and, in turn, find support for the reformulated model in a college population where depressed subjects are most often mildly to moderately depressed and not severely depressed.

Unexpectedly, no differences between males and females were found in depressive attributional style. This is somewhat surprising in light of Dweck and other researcher's learned helplessness studies with children (Dweck & Bush, 1976; Dweck et al., 1981; Dweck & Reppucci, 1973; Nichols, 1975), but would be consistent with the reviewed studies that found no sex difference on attributional style (Blaney et al., 1980; Costello, 1982; Feather & Davenport, 1981; Hammen & DeMayo, 1980). It may be that the relationship between cognitive variables and

depression is overall highly similar for men and women. Nevertheless, males and females may still differ in subtle ways, but the current analyses of the ASQ may be insensitive to these subtleties. Hammen and Padesky (1977) looked at BDI scores of 972 male and 1300 female college students. Although they found no overall sex difference in the degree of depression, a discriminant function analysis of the highest depression scores revealed a significant and interpretable sex difference in the patterns of symptom expression. Moreover, Strickland and Haley (1980) matched males and females on Rotter I-E scores (Rotter, 1966), yet found significant differences between males and females on eight out of twenty-three keyed items. Thus, as in the BDI and the Rotter I-E scale, the future research with the ASQ may also reveal a significant sex difference in attributional style when further refinement of the analyses are carried out.

Except for internality for bad outcomes (i.e., the correlation between Bad Internality and depression was only significant when psychopathology was partialled out), psychopathology, alone, did not significantly affect attributional ratings by subjects. Consequently, the depressive attributional style postulated and supported in this study using a clinical sample, appears to be uniquely related to depression. This finding is especially important since most of the learned helplessness research has not directly sorted out the effects of depression versus global psychopathology. Indeed, this study specifically looked at global psychopathology irrespective of degree of depression. In learned helplessness studies that used college

students, psychopathology was not measured. Moreover, in most learned helplessness studies that used clinical patients, the severity of psychopathology was implicitly controlled for by comparing depressed inpatients with nondepressed inpatients (Miller et al., 1982) or by comparing depressed outpatients with nondepressed outpatients (Gong-Guy & Hammen, 1980). However, it may be an erroneous assumption that all inpatients (or outpatients) have similar levels of severity of psychiatric illness. In addition, although Raps et al. (1982) compared depressed patients with schizophrenics, schizophrenia is only one type of psychopathology and all diagnoses were made by one clinician.

Although this study supported the reformulated learned helplessness model of depression, namely by demonstrating an association between depression and attributional style in a clinical sample and by finding this attributional style as being uniquely related to depression, this study did not address the question of causality. Indeed, this investigation was only correlational in nature and thus could not assess whether the attributional dimensions of internality, stability, and globality for bad outcomes precede, accompany, or follow a depressive episode. Future studies may well decide to focus on the issue of causality. In fact, a few studies have already begun to test for causality with respect to attributional style and depression by examining people's attributions at different points in time (Golin et al., 1981; Lewinsohn, Steinmetz, Larson, & Franklin, 1981). The evidence is contradictory and further longitudinal studies, especially with clinically depressed subjects are badly needed. Another method

that may be promising in future learned helplessness research concerned with causality is protocol analysis. As described by Pasahow (1981) protocol analysis involves analyzing subjects' ongoing attributional verbalizations rather than retrospective attributional verbalizations. Protocol analysis studies may be easily carried out in a college population of depressed and nondepressed subjects. Moreover, a modified version of protocol analysis may be possible in a therapy situation where depressed patients meet regularly with their therapist.

As important as the issue of causality is in learned helplessness research, an equally important issue involves a refined identification of the underlying distinguishing features of a learned helplessness depression. In other words, what specific type of depressed patient best fits the helplessness model and how can one easily and accurately diagnose such a person? In the current study a wide assortment of depressive types served as subjects, and a significant relationship between depression and attributional style was nevertheless found. However, it may be profitable in future learned helplessness research to more clearly identify the exact type of depressive. This does not mean simply classify subjects by DSM-III criteria. As Buchwald, Coyne, and Cole (1978) point out, although diagnoses of depression are based on explicit published criteria, the use of these criteria requires judgments. Instead it is suggested that subjects be classified by formal diagnosis, if possible, and then be further classified into groups on the basis of scores on self-report inventories like the MMPI or BDI. Specifically, the MMPI has the advantage of being able

to differentiate between several clinical types and thus one could purify a depressed subject by classifying him as either high depressed-low paranoid versus high depressed-high paranoid, etc. In terms of the BDI, Beck (1967) has identified four separate factors, namely, affective, cognitive, motivational, and physical factors. Thus, one could do separate analyses between each of these four factors of the BDI and the attributional indices of the ASQ, instead of simply using the overall BDI score as is currently done in learned helplessness studies.

## SUMMARY

The present study tested hypotheses based upon the reformulated model of learned helplessness by Abramson, Seligman, and Teasdale (1978). One hundred and one psychiatric adult patients and 22 non-psychiatric adults served as subjects. On the basis of scores on the BDI, all subjects were classified into Nondepressed, Mildly to Moderately Depressed, and Severely Depressed groups. In addition, all subjects were divided into Low Psychopathology or High Psychopathology groups on the basis of MMPI-Peterson six signs.

Results were generally supportive of a depressive attributional style. Specifically, stability, globality, and composite scores for bad outcomes were positively correlated with depression, and internality for bad outcomes was positively correlated with depression when psychopathology was partialled out. Internality scores for good outcomes were negatively correlated with depression. Moreover, depressed subjects were found to be more evenhanded in their attributions to good and bad outcomes than nondepressed subjects. As expected, the depression attributional style was generally more pronounced in Severely Depressed as compared to Mildly to Moderately Depressed groups. No differences, however, were found between males and females in attributional style. Finally, as predicted, psychopathology, alone, did not significantly affect attributional ratings of subjects, although, as noted, there were some significant interactional influences of psychopathology by depression.

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APPENDIX A

Name \_\_\_\_\_

Code # \_\_\_\_\_

DIRECTIONS

Please try to vividly imagine yourself in the situations that follow. If such a situation happened to you, what would you feel would have caused it. While events may have many causes, we want you to pick only one--the major cause if this event happened to you. Please write this cause in the blank provided after each event. Next we want you to answer some questions about the cause and a final question about the situation. To summarize, we want you to:

- 1) Read each situation and vividly imagine it happening to you.
- 2) Decide what you feel would be the major cause of this situation if it happened to you.
- 3) Write one cause in the blank provided.
- 4) Answer three questions about the cause.
- 5) Answer one question about the situation.
- 6) Go on to the next situation.

YOU MEET A FRIEND WHO COMPLIMENTS YOU ON YOUR APPEARANCE.

- 1) Write down the one major cause \_\_\_\_\_
- 2) Is the cause of your friend's compliment due to something about you or something about the other person or circumstances? (Circle one number)
- |  |   |   |   |   |   |   |   |                      |
|--|---|---|---|---|---|---|---|----------------------|
| Totally due to<br>the other person<br>or circumstances | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Totally due<br>to me |
|--|---|---|---|---|---|---|---|----------------------|
- 3) In the future when you are with your friends, will this cause again influence what happens? (Circle one number)
- |   |   |   |   |   |   |   |   |  |
|---|---|---|---|---|---|---|---|--|
| Will never<br>again influence<br>what happens | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Will always<br>influence what<br>happens |
|---|---|---|---|---|---|---|---|--|
- 4) Is the cause something that just affects interacting with friends or does it also influence other areas of your life? (Circle one number)
- |   |   |   |   |   |   |   |   |  |
|---|---|---|---|---|---|---|---|--|
| Influences just<br>this particular<br>situation | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Influences all<br>situations in<br>my life |
|---|---|---|---|---|---|---|---|--|
- 5) How important would this situation be if it happened to you? (Circle one number)
- |                         |   |   |   |   |   |   |   |                        |
|-------------------------|---|---|---|---|---|---|---|------------------------|
| Not at all<br>important | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Extremely<br>important |
|-------------------------|---|---|---|---|---|---|---|------------------------|

YOU HAVE BEEN LOOKING FOR A JOB UNSUCCESSFULLY FOR SOME TIME.

- 6) Write down one major cause \_\_\_\_\_
- 7) Is the cause of your unsuccessful job search due to something about you or something about other people or circumstances? (Circle one number)
- |  |   |   |   |   |   |   |   |                      |
|--|---|---|---|---|---|---|---|----------------------|
| Totally due to<br>other people or<br>circumstances | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Totally due<br>to me |
|--|---|---|---|---|---|---|---|----------------------|
- 8) In the future when looking for a job, will this cause again influence what happens? (Circle one number)
- |   |   |   |   |   |   |   |   |  |
|---|---|---|---|---|---|---|---|--|
| Will never<br>again influence<br>what happens | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Will always<br>influence what<br>happens |
|---|---|---|---|---|---|---|---|--|

- 9) Is the cause something that just influences looking for a job or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--

- 10) How important would this situation be if it happened to you?  
(Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

YOU INVEST MONEY IN THE STOCK MARKET AND MAKE A PROFIT.

- 11) Write down one major cause \_\_\_\_\_

- 12) Is the cause of your making a profit in the stock market due to something about you or something about other people or circumstances? (Circle one number)

Totally due to other people or circumstances	1	2	3	4	5	6	7	Totally due to me
--	---	---	---	---	---	---	---	----------------------

- 13) In the future when investing in the stock market, will this cause again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	--

- 14) Is the cause something that just affects investing in stocks or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--

- 15) How important would this situation be if it happened to you?  
(Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

A FRIEND COMES TO YOU WITH A PROBLEM AND YOU DON'T TRY TO HELP THEM.

- 16) Write down the one major cause \_\_\_\_\_
- 17) Is the cause of your not helping your friend due to something about you or something about other people or circumstances? (Circle one number)
- |  |   |   |   |   |   |   |   |                   |
|--|---|---|---|---|---|---|---|-------------------|
| Totally due to other people or circumstances | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Totally due to me |
|--|---|---|---|---|---|---|---|-------------------|
- 18) In the future when a friend comes to you with a problem, will this cause again influence what happens? (Circle one number)
- |   |   |   |   |   |   |   |   |                                    |
|---|---|---|---|---|---|---|---|------------------------------------|
| Will never again influence what happens | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Will always influence what happens |
|---|---|---|---|---|---|---|---|------------------------------------|
- 19) Is the cause something that just affects what happens when a friend comes to you with a problem or does it also influence other areas of your life? (Circle one number)
- |   |   |   |   |   |   |   |   |                                      |
|---|---|---|---|---|---|---|---|--------------------------------------|
| Influences just this particular situation | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Influences all situations in my life |
|---|---|---|---|---|---|---|---|--------------------------------------|
- 20) How important would this situation be if it happened to you? (Circle one number)
- |                      |   |   |   |   |   |   |   |                     |
|----------------------|---|---|---|---|---|---|---|---------------------|
| Not at all important | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Extremely important |
|----------------------|---|---|---|---|---|---|---|---------------------|

YOU GIVE AN IMPORTANT TALK IN FRONT OF A GROUP AND THE AUDIENCE REACT NEGATIVELY.

- 21) Write down the one major cause \_\_\_\_\_
- 22) Is the cause of the audience reacting negatively due to something about you or something about other people or circumstances? (Circle one number)
- |  |   |   |   |   |   |   |   |                   |
|--|---|---|---|---|---|---|---|-------------------|
| Totally due to other people or circumstances | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Totally due to me |
|--|---|---|---|---|---|---|---|-------------------|



- 23) In the future when giving talks, will this cause again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	--

- 24) Is this cause something that just influences giving talks or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--

- 25) How important would this situation be if it happened to you? (Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

YOU DO AN IMPORTANT PROJECT WITH A GROUP AND FIND THAT THE PROJECT TURNS OUT WELL.

- 26) Write down the one major cause \_\_\_\_\_

- 27) Is the cause of the group working well together due to something about you or something about the other people or circumstances? (Circle one number)

Totally due to other people or circumstances	1	2	3	4	5	6	7	Totally due to me
--	---	---	---	---	---	---	---	----------------------

- 28) In the future when working on a group project, will this cause again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	--

- 29) Is this cause something that just affects group projects or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--



- 37) Is the cause of your not getting the work done due to something about you or something about the other people or circumstances? (Circle one number)

Totally due to other people or circumstances	1	2	3	4	5	6	7	Totally due to me
--	---	---	---	---	---	---	---	-------------------

- 38) In the future when doing the work that others expect, will this again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	------------------------------------

- 39) Is the cause something that just affects doing work that others expect you to do or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--------------------------------------

- 40) How important would this situation be if it happened to you? (Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
----------------------	---	---	---	---	---	---	---	---------------------

YOU AND YOUR SPOUSE (BOYFRIEND/GIRLFRIEND) WERE HAVING PROBLEMS GETTING ALONG BUT YOU WERE ABLE TO RESOLVE THE DIFFICULTIES.

- 41) Write down the one major cause \_\_\_\_\_

- 42) Is the cause of the problems being resolved due to something about you or something about other people or circumstances? (Circle one number)

Totally due to other people or circumstances	1	2	3	4	5	6	7	Totally due to me
--	---	---	---	---	---	---	---	-------------------

- 43) In the future when trying to resolve problems, will this cause again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	------------------------------------

- 44) Is this cause something that just affects getting along with your spouse (boyfriend/girlfriend) or does it also influence other areas of your life? (Circle one number)

Influence just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
--	---	---	---	---	---	---	---	--

- 45) How important would this situation be if it happened to you?  
(Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

YOU APPLY FOR A POSITION THAT YOU WANT VERY BADLY (e.g., IMPORTANT JOB, GRADUATE SCHOOL ADMISSION, etc.) AND YOU GET IT.

- 46) Write down one major cause \_\_\_\_\_

- 47) Is the cause of your getting the position due to something about you or something about other people or circumstances? (Circle one number)

Totally due to other people or circumstances	1	2	3	4	5	6	7	Totally due to me
--	---	---	---	---	---	---	---	----------------------

- 48) In the future when applying for a position, will this cause again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	--

- 49) Is the cause something that just influences applying for a position or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--

- 50) How important would this situation be if it happened to you?  
(Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

## YOU GO OUT ON A DATE AND IT GOES BADLY.

- 51) Write down the one major cause \_\_\_\_\_
- 52) Is the cause of the date going badly due to something about you or something about other people or circumstances? (Circle one number)

Totally due to other people or circumstances	1	2	3	4	5	6	7	Totally due to me
--	---	---	---	---	---	---	---	----------------------

- 53) In the future when dating, will this cause again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	--

- 54) Is the cause something that just influences dating or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--

- 55) How important would this situation be if it happened to you? (Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

## YOU AND THE MEMBERS OF YOUR HOUSEHOLD HAVE BEEN GETTING ALONG WELL.

- 56) Write down the one major cause \_\_\_\_\_
- 57) Is the cause of your household getting along due to something about you or something about the other people or circumstances? (Circle one number)

Totally due to other people or circumstances	1	2	3	4	5	6	7	Totally due to me
--	---	---	---	---	---	---	---	----------------------

- 58) In the future in your household, will this cause again influence what happens? (Circle one number)

Will never again influence what happens	1	2	3	4	5	6	7	Will always influence what happens
---	---	---	---	---	---	---	---	--

59) Is the cause something that just affects how your household gets along or does it also influence other areas of your life? (Circle one number)

Influences just this particular situation	1	2	3	4	5	6	7	Influences all situations in my life
---	---	---	---	---	---	---	---	--

60) How important would this situation be if it happened to you?  
(Circle one number)

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

APPENDIX B

## INFORMED CONSENT STATEMENT

for

Project Title: Is there a depressive attributional style?

I agree to participate in a study about people's feelings, thoughts, beliefs, and behavior. In the following packet I will be asked to respond to true-false items, multiple choice items, short answer questions, and to check words. I am aware that filling out the questionnaires may take up to 3 hours of my time. I realize that all my answers will be held in strict confidence and in no case will my name or identity be disclosed when the findings of this study are reported.

I understand that my cooperation in this study is purely voluntary. In the event that I decide not to participate or I choose not to complete the questionnaires, there will be no effect on the quality of my medical care.

I may ask any questions about this study or about the procedures that are unclear to me.

Name: \_\_\_\_\_ Date: \_\_\_\_\_

Witness: \_\_\_\_\_ Date: \_\_\_\_\_

Person administering the  
informed consent: \_\_\_\_\_ Date: \_\_\_\_\_



APPROVAL SHEET

The dissertation submitted by Donna Munic has been read and approved by the following committee:

Dr. Thomas Petzel, Director  
Professor, Psychology, Loyola

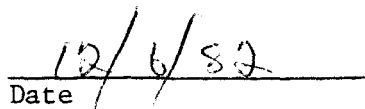
Dr. James Johnson  
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Dr. Alan DeWolfe  
Professor, Psychology, Loyola

The final copies have been examined by the director of the dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the dissertation is now given final approval by the Committee with reference to content and form.

The dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

  
Director's Signature

  
Date