EXPERIMENTAL TESTS OF THE CAUSAL DIRECTION BETWEEN DEPRESSED AND ELATED MOODS AND ATTRIBUTIONAL STYLE

By

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ABSTRACT

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Reviewing the psychological theories of depression revealed a fundamental disagreement on the nature of the relationship between depressed thoughts and depression. A set of cognitive theories says depressions arise because of the interaction between depressogenic thought styles and painful events. The depressogenic thought patterns presumably precede the depression and are a central cause of the subsequent depressions. On the other hand, a second set of theories says these depressive thought styles arise out of the depression but do not precede it and are thus not a cause of depression.

To test these alternate thought-first and affect-first models of depression, two experiments were conducted. The subjects in the first experiment were 62 Michigan State University undergraduate women; in the second experiment, the subjects were 24 male and 23 female M.S.U. undergraduates.

The two experiments followed a similar design: subjects completed a set of questionnaires on their thought style, their level of depression and their mood; they were then randomly assigned to either a control condition or to an experimental condition designed to induce a mood change; afterwards, the initial questionnaires were again

completed. The two experiments differed in the experimental induction used to alter mood states and in the timing: in experiment one subjects returned for a second session, while experiment two was entirely completed at one sitting.

Experiment one used the Velten Mood Induction Procedure (or VMIP) to create neutral, depressed and elated moods. This procedure consists of having students sit alone in a room and read aloud a set of 60 mood related statements. In experiment two, mood was altered by watching the movie "Peege"; the control group saw a neutral science movie.

The following instruments were used in both experiments to measure depressed mood: the Beck Depression Inventory or BDI; the Multiple Affect Adjective Check List or MAACL; and a Personal Feelings Scale or PFS. The measure of depressive thought style was the Attribution Style Questionnaire or ASQ.

The results supported two conclusions. First both inductions successfully created depressed moods, although the procedures also induced additional emotions, most noticeably increased anxiety. Second, there was some support for the thought-first hypotheses of depression. There were significant predicted differences on one of three depression comparisons in experiment one and on two of six comparisons in experiment two. This support is stronger than it seems because sex interactions in experiment two necessitated separate analyses by sex, making the cells used for the comparison quite small in size. In addition, no comparisons were ever significant in a direction opposite to the predictions.

The affect-first hypotheses were not supported. Furthermore, no relation was found between induced elation in experiment one and either the affect-first model or the thought-first model.

The discussion suggested future avenues of research which might use naturalistic studies rather than experimental studies. The failure to find any support for the affect-first model was explained as being partly due to the experiment's failure to fully test the affect-first model, most notably because informed consent procedures made it impossible to alter mood without subjects being aware of the mood change. Finally, the discussion raised the possibility that the two models are actually alternate phases of a single feed-back loop process.

This dissertation is dedicated to the loving memory of my father
Max Ermann
who died this year before he could see me graduate.

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Dr. Messé was the methodologist on this dissertation, a capacity he had previously served on my masters thesis. On this work, as on the earlier work, most of the credit for the analysis belongs to him; any errors are naturally my own. Clinicians, I suspect, generally have a better conceptual understanding of research design than they have a strict mathematical understanding, and I am no exception. The value of the methodologist to people like me is directly proportional to both their knowledge and their patience, and in these regards, Dr. Messé is

the methodologist par excellence. I take the knowledge for granted, but the patience I know of first hand, since I sorely put it to the test whenever I started my tortured explanations of the project. Such clarity as may exist in my plan of analysis and results sections owe much to his careful reading of an early draft. In addition, Dr. Messé has been a friend and source of encouragement in all my research endeavors; I especially treasured this research encouragement as my most severe doubts early in my graduate school career centered on my ability to perform research.

Dr. Ferguson made a number of valuable suggestions which improved the basic experiment of this dissertation. Her advice here, as elsewhere during my graduate school career, was astute and sensible. She has always been an excellent teacher, a fine clinical supervisor and a friend throughout my graduate school career. One of my comprehensive questions was written for her this year, and she gave me enough leeway on the project so that it became the most exciting theoretical endeavor I attempted while in graduate school and one which I expect will engage my interest for some time to come. This is typical; looking back, I see many of the areas which are of most interest to me started in various classes of hers.

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TABLE OF CONTENTS

LIST OF TABLES

CHAPTER 1

INTRODUCTION AND REVIEW OF RELATED LITERATURE

Cognitive Theories of Depression

In the last two decades, psychological research on clinical depression has increasingly focused on thought processes. While the nature of depressed thinking is becoming clearer, the relation between such thinking and the causes of depression remains obscure. Two essential questions remain unanswered. First, is such thinking the cause of depression or is it the result of the depression? Second, what is the relationship between depressive thoughts, external events and the depressive syndrome; more specifically, must an external precipitating event remain in conscious awareness for depression to then follow? The theories which stress the role of conscious thought patterns in the etiology of depression include Beck's theory (1967), the original learned-helplessness theory (Seligman, 1975), and the latter's revised or attribution version (Abramson et al, 1978, Miller & Norman, 1979).

Although these theories have a similar emphasis on cognition, these theories are distinct. Looking at these theories one by one should reveal these differences and should describe the nature of depressive thinking as well.

Beck's (1967, 1974) theory was developed when his attention was turned to thought distortions unique to depressives as a group. Such distortions included arbitrary inference, selective abstraction,

overgeneralization, and magnification and minimization. He calls these distortions a "cognitive triad" because they fall into three categories, namely a negative view of the world, of the self, and of the future. The negative world view involves special sensitivity to failure: the depressive not only consistently reads his actual performance as evidence of failure (for example, by consistently under-estimating what he actually performed) but also consistently exaggerates minor difficulties as examples of major failure. Ambiguous or minor stimulii get blown into large experiences of ridicule or emptiness. In the negative view of self, the depressed person lays all blame for his negative life experiences solely upon himself. Any situation-specific failure is distorted into a personality flaw. Finally, the third feature of the triad is a negative view of the future: the patient sees the short-term and the long-term future as an endless continuation of the negative present experiences, including failure, deprivation and self-depreciation.

All other symptoms emerge from these depressed cognitions. Beck argues that the depressed mood, the motivational deficits, the increased suicidality, the increased dependency and many of the physical symptoms come from the depressive's cognitive triad. Consider affect and cognition, for example. Real life rejection or loss can lead to depression; so too can the thought that such rejection or loss has occurred. Depressed patients consistently misinterpret external information in a way that leads them to feel they have been rejected or suffered a loss. It is not that they immerse themselves in more no-win situations in real life, but rather that their internal cognitive processing system consistently distorts the experiences they have, so

that they always see themselves in these no-win situations. Just as the thoughts cause the depression, so too if the thoughts change, the depression then lifts. A student depressed because he thinks he has failed an exam will no longer be depressed if he learns the posted grades were in error.

The second major cognitive theory is Seligman's (1975) learned helplessness theory. Since even Seligman has now abandoned the original theory, I will only briefly describe it. This theory emerged from laboratory experiments involving dogs in a shuttle box, where an electric shock can be switched from side to side. To escape the shock, the dog jumps over a low barrier dividing the box. If a dog is initially given inescapable shocks, it appears unable to learn how to avoid the shock and instead just lies down and whines. This phenomenon was called learned helplessness: the dogs had generalized their initial helplessness to all later situations.

To create learned helplessness, there must be an objective situation in which environmental responses remain invariant no matter how one behaves and this response independence must be known to the individual. Later this information becomes an expectation of helplessness. Finally, this expectation has cognitive, emotional and behavioral consequences.

Seligman argues that since learned helplessness and depression have the same symptoms and the same cause, therefore learned helplessness is depression, and vice versa. Both are marked by passivity, by similar negative cognitive sets, by a similar lack of hostility, by similar physiological effects, including loss of weight,

sex drive, and appetite, and even by similar biochemical processes (norepinepherine depletion).

Finally, with regard to the original question of how thoughts relate to emotion, the theory is quite clear: people must be consciously aware of the essential environmental features for any depression to occur; should response independent contingencies exist but remain outside of awareness, no depression will result. Second, depressive affects come after this thought-environment interaction.

The outcome of the research generally shows the original theory is unconfirmed. Depue & Monroe (1978) note that learned helplessness ought to be a theory of reactive depression, but the physiological symptoms it describes are closer to endogenous depression. Costello (1978) argues that the experiments do not support the theory. An important discrepancy in the research is that subjects often react with anger instead of depression--something not predicted by the theory.

In the last two years, Miller & Norman (1979) and Abramson, Seligman & Teasdale (1978) have independently arrived at quite similar attribution theories of depression. These two theories combine an attribution model (Heider, 1958; Kelly, 1967) with learned helplessness in an attempt to correct for the criticisms directed at the latter. The theories assume that if a person finds himself in a helpless situation, he asks why he is helpless; the nature of the causal attribution determines whether he is depressed. The original learned helplessness theory always had such a cognitive component in it relating to expectancies about future outcomes, but the cognitive component had not been important; the new theories primarily focus on this cognitive aspect.

Three attributional dimensions determine the nature of depression: internal-external; stable-unstable; and global-specific. Depressive experiences are likely to be chronic if helplessness is given a stable attribution, and the depression will generalize over many areas of life if the helplessness is given a global attribution. (The third attribution dimension, internal-external, is discussed separately below.)

The following examples may clarify these attributions. This theory states that if someone loses his job, he must explain to himself how this happened and he therefore attributes the job loss to some set of factors. The nature of these attributions may then determine his reaction to the job loss. One man makes external attributions: he might say the entire economy is in recession (global) or he might say his company alone is floundering (specific); he might think this is a short-term recession (unstable) or that this is the beginning of national economic ruin (stable). A man making internal attributions might likewise vary on the other dimensions. He might say he was fired because of a global trait ("I am always unreliable") or because of a specific personnel problem ("My new boss and I just don't hit it off."); similarly he could say it was something stable about himself ("I am too dumb") or something unstable and changeable ("I need more education").

The two attribution theories differ on the role of the internal-external dimension. Abramson et al (1978) argue that helplessness can be experienced as personal helplessness or universal helplessness (the latter being helplessness not contingent on the person, such as helplessness during a tornado); internal attributions will lead to personal helplessness and lowered self-esteem while external

attributions are tied to universal helplessness and no lowered self-esteem. However both forms of helplessness lead to depression. Miller & Norman (1979) would say internal attributions are needed for depression. If external attributions are made, the person will just get angry.

The revised theory differs from the old learned helplessness theory in still other ways. According to the old theory, any uncontrolled event, good or bad, was supposed to lead to depression; in the new attribution theory, dysphoric depressive affect only results when the uncontrollability expectations are in regard to a "bad event" (bad event expectancies being the probability of aversive outcomes or the improbability of desired outcomes). The new model also has room for how self-esteem enters into the depressive equation, which the old model did not.

What the new theory gains in its accurate description of depression it loses in its explanatory power. The strength of the original learned helplessness theory was its very simplicity: depression was purely a function of a given kind of environment. If someone was in that environment, and knew it, they became depressed; conversely, if someone was depressed, they had to have been in such an environment. Overcoming depression merely involved changing the environment or learning that ways one thought about the environment no longer applied.

This simplicity is lost and the greater complexity of the attribution theory raises as many questions as it answers. We now have a theory in which two people can be placed in the same environment and not react in the same way, since we only know the reactions if we know the attributions, and each person may have his own style of making

attributions. But what determines their attributions? There is nothing in the theory to explain how and why different people have different kinds of attributions when in the same situation. Here we have a theory which may explain the process of depression but tells us nothing about why this process occurs in some people and not in others (Bowlby (1980) raised a similar objection to the Beck (1967) theory). We have lost the clear-cut relation between depression and environment and have not fully replaced it with an explanation of the individual and depression.

Returning again to the Beck and the learned-helplessness theories, in addition to the attribution theory; it is clear that all three cognitive theories say that depression begins with a pathogenic thought-pattern interacting with real events. Since all three theories assume that all the other symptoms in the depression syndrome[1] stem from this thought pattern, all three theories are aptly described as thought-first theories: the thinking determines the depression. In addition, all three theories say that thinking is directed at an external event. This event may be neutral or aversive, but it must remain at the forefront of the depressive's thinking. Depressogenic thought patterns can only create depression if the external precipitating event remains in consciousness.

Affect Theories of Depression

The psychological literature presents another and altogether different way of thinking about depression. This alternative view is that the conscious thought patterns in depression are mainly a result or manifestation of the depression and not its cause. What is the cause? Here the theories vary, but they generally start with the depression as an affect--an affect that then creates the typical depressive thought

patterns. In addition these theories say that depression can arise in the absence of any conscious awareness of an external precipitating event. Like the cognitive theories, these various affect-first theories do differ from each other and so they each need some further elaboration.

In psychiatry, most current research has been directed at biochemical theories of depression (Akiskal & McKinney, 1973, 1975; Baldessarini, 1975; Fawcett, 1975). Depression is directly traced to the biochemical processes which are presumed to control moods; since the other features of depression, including its thought features, are presumed to then come from the affect changes, these theories are all affect-first theories.

The second major group of theories which comprise the affect-first model are the various psychoanalytic theories. These theories all assume that unconscious processes can set off depression and thus all argue that depression can theoretically arise in the absence of any conscious awareness of a precipitating event. Therefore the ways we think about events cannot explain all the thought symptoms in all depressions.

Since these theories have been summarized elsewhere (e.g. Mendelson, 1974), this summary will focus on two issues of depression and depressed thought: first, can depression begin without conscious awareness of the precipitating event; and second, can depression begin as an affective change which then creates the other features of the depressive syndrome, including depressive thinking?

Some theories answer one or the other question; Freud's (1917) explanation of depression (an explanation still underlying most

psychoanalytic theory on depression) answers both questions. With regard to external precipitants, Freud notes that in depression, as in all mourning, loss is the central issue; however in some types of depression the loss is hidden and indeed must be inferred from the presence of the depression. In the more extreme of such cases, the individual is unaware of the loss; in the less extreme, he knows who he has lost but not what it is about the person that he has lost.

How can the link between the depression and the precipitating event be severed? It comes from the strong ambivalence in the depressive's feelings. In grief, the detachment of the libido from the lost object also begins primarily in the unconscious, but this process then easily moves on into the preconscious and then into the conscious system. Ambivalent conflict in depression, unlike grief in bereavement, does not readily leave the unconscious; it is blocked within the unconscious and this very blockage actually creates the melancholy. The libidinal cathexes do abandon the object (as in grief) but because of the ambivalence this occurs (and is dissimilar from grief) via a regression and object identification taking over part of the ego. Only after this regression has occurred does the depressive conflict become conscious, and the form it takes, as a conflict within the self between ego and the super-ego, is not the essential part of the conflict. The real original conflict, between self and external object, is hidden.[2]

The second question is whence arise these depressive thoughts? In one of his most famous comparisons, Freud says that in grief the world appears "impoverished" while in depression it is the ego which is impoverished and experienced as a devalued self. This is the now common observation that depressives suffer a loss of self-esteem. Freud

stresses that the depressed patient is not aware that this change has taken place within the self, or more accurately, does not recognize this self-devaluation as a change. Instead this self-devaluation or "delusional belittling" is extended backwards into the past, as if it always existed. Naturally, there is some truth in these harsh criticisms, but it is a part truth. The statements which are true refer to the after-effects of this process. For example, the depressive declares he is incapable of love--true, but only because depression makes him so.

In other words, Freud's paper clearly suggests that the thinking in depression neither precedes depression nor causes depression but instead is created by the depression process--a process which occurs when anger over object loss is directed within instead of being externalized.

The next major change in psycho-analytic theory comes from Bibring (1953), who also addresses the relation of depression and depressed thought. Bibring says all depressions "represent an affective state, which indicates a state of the ego in terms of helplessness and inhibitions of functions." Depression is thus the emotional state which occurs when the ego experiences helplessness or powerlessness. Such a state follows a narcissistic blow (Bibring is referring to secondary narcissism) which lowers self-esteem. Bibring notes three essential narcissistic goals: these are the wish to be loved; the wish to be great and strong; and last, the wish to be a good and loving person. These goals remain highly charged goals for the individual since they are internalized in the ego ideal, and hence retain both conscious and unconscious components; when the ego becomes aware that it is unable to

reach or maintain these narcissistic goals, extraordinary tensions result, and depression follows. Depression is thus an "emotional correlate of a partial or complete collapse of the self-esteem of the ego since it fails to live up to its aspirations," even while these internalized aspirations remain and exert a powerful pull.

Seligman (1975) quotes Bibring in support of his own learned helplessness theory, but this attempt to find support in Bibring represents a fundamental misunderstanding of Bibring which, if clarified, will also explicate the essential differences between the thought-first and the affect-first models.

There are two essential differences between Bibring's theory and Seligman's learned helplessness theory, differences which Seligman either ignores or misunderstands. The first is that Bibring says depression follows narcissistic blows, not real external blows. Seligman presents a theory in which a person faces a real (objective) situation in which real behaviors aimed at an achievement are consistently unrewarded by the outside world, leading to a mind set in which the individual gives up attempting to gain real rewards from the outside world. But Bibring repeatedly stresses that the most frequent cause of the depression is excessively high narcissistic ambitions; this need not have to do with lack of real abilities or real rewards.

Consider a talented music student who wins many awards but who fails to win the annual Tschaikovsky award in Moscow and becomes depressed. Given the earlier pattern of rewards, Seligman has no explanation of this depression. For Bibring it is simple: the narcissistic goals are excessive, failure follows despite an earlier

string of successes and, given the narcissistic blow to self-esteem, depression begins.

In a similar fashion, for Seligman's learned helplessness theory depression ends when real changes occur, namely either the reward pattern for old behaviors is now altered, or else the individual now institutes a new set of behaviors which is more successful in eliciting rewards. Bibring, on the other hand, suggests four possible ways depression could lift: either the narcissistic goals now appear more reachable; self-esteem is recovered aside from altering the goals; the goals are altered or just given up; or else the depression is defended against, as by hypomania. Notice that only the first two of these possibilities are even tangentially related to changes in behaviors or skills.

The second misunderstanding is in Seligman's interpretation of Bibring's phrase, "The ego's acute awareness of its helplessness." Seligman reads this as if it means that a person is cognitively conscious that he is indeed in an objective situation of helplessness. But the ego is an internal structure of the mind and ego states are internal states of the person. Perception of reality is only one ego function. The ego might also feel helpless vis a vis purely internal processes independent of external situations (e.g. a depression set off by a dream). Bibring cites the example of a successful professional man who became depressed whenever he did routine work. Not only was analysis needed to explain why doing routine work made him depressed, but the fact that this routine work triggered the depression was unknown to the man. Until he entered the analysis, he knew only that he occasionally became depressed, for reasons unknown.

Much recent psycho-analytic thinking about depression is even closer to the affect-first model, since these theories say many features of depression, including depressed thinking, is a reaction to the affect of depression. Edith Jacobson (1971) has written extensively on depression, and her work on moods, and especially depressive moods, is central to contrasting the affect-first depression model with the thought-first model.

According to Jacobson, moods are ego states in which all ego functioning, including thoughts, feelings, actions, etc., take on a "particular uniform coloring" for a shorter or longer time period. More specifically, Jacobson calls moods a "temporary fixation of generalized discharge modifications." For example, an affective tone of joy or anger is temporarily seen everywhere--within the self, in internalized objects, and in the external world, and hence in all one's thoughts and actions too. Other affect states are always directed at and derived from a specific object; moods lack specificity.

Moods often overtake us without our knowing whence they come. There is usually an external experience which sets off the mood, but we often lose track of the provocative event. For a mood change to result, the initial event must set off a great deal of tension which cannot be immediately relieved. In some cases, a major external change can lead to a mood change, as when someone is left by a lover. In other cases, however, it can be a rather minor event so long as the event activates considerable tension in the unconscious. At these times, the evocative event may even be forgotten.

All objects and indeed all stimulii are affected by moods. Emerging moods alter our internal world for a time, modifying all our

self-conceptions and object world. These changes run in parallel during a particular mood state, so in a joyful mood state I feel joyful and the entire world seems joyful too, while in an angry mood, I am angry and the entire world appears dark and brooding. As a result, we act in ways that confirm our mood colored vision of the world. In an angry mood we will see others as angry and will act angrily at them. Their quite naturally responding with anger only confirms that the world is indeed an angry arena.

When mood changes overtake us, Jacobson continues, our evaluative abilities are temporarily curtailed. In a normal mood state, people know this is time limited and the distortions will lift as the mood lifts. Even depression can be a normal mood, so long as people tell themselves, "I am thinking this way because of the depression, and when the depression lifts my view of myself and the world will also alter."

Pathology enters when people cannot work out of their mood. Three difficulties make moods pathological. The first is ego weakness, including flaws in basic reality testing or an inability to tolerate stress or loss. The second is a weakness of the super-ego. One function of the super-ego is to signal the presence of strivings which are not permitted; impairment of this signal function can lead to rapid mood swings. Finally, if a mood is tied to an unconscious conflict, then the repetition compulsion from the infantile conflict can block any effective use of reality testing. When reality testing is attempted, the new experience is distorted by the unconscious conflict so that it merely confirms the infantile distortion. The model Arieti & Bemporad present (later this chapter) is a variant of this model.

The key to mood pathology is thus the ability or inability to work one's way out of the mood, rather than the mood itself.

Even depression can be a normal mood, Jacobson adds. A depressed mood always involves aggressive conflict. Frustration creates a conflict which is turned against the self-image, creating a "conflict between the wishful self image and the image of the deflated falling self." This internal conflict between self image and ego ideal creates a narcissistic experience central to all depression: the experience of failure. There are two avenues to depression. Sometimes the depression is created by narcissistic conflict; at other times the super-ego is involved. In these second cases, the failure experience is matched by a moral judgement on the self as being bad.

Because the conflict in depression arises from an internal narcissistic conflict (ego ideal vs. self-representation), the reality testing must occur within the person too: in the end, it is the internal super-ego standards which must be satisfied and not the external standards of the world. Introspection about the self is difficult, but if the gap between ego-ideal and self representation is excessive, due to an unrealistically high ego-ideal, then even satisfactory encounters with reality may not alleviate the depression. If I feel depressed because I fail an exam, I can overcome this by studying harder, but if my standards remain excessive, then even success on future exams may appear inadequate.

Two key points in Jacobson's theory of moods should be reiterated. First, moods change the way we think, and not the reverse. Second, we need not be conscious of any external event which alters our mood.

Arieti & Bemporad (1978) propose a model similar to Jacobson's, except that they start with an inability to end a sad mood, where Jacobson started with an inability to end a depressed mood.

Arieti and Bemporad (1978) say depression is intimately tied to sadness. All painful emotions are designed to create actions which will cause them to disappear but sadness is unique because it slows us down first, making direct action less likely. The slowdown is adaptive because the function of sadness is to give people a chance to re-arrange their thoughts and goals, as when they remake their life plans. Sadness follows a cognitive appraisal of an event and then promotes a reparative change in our relation to that event. It is similar to the work of mourning.

Depression arises from deep thought patterns or schema which block this resolution of sadness. All people can use affecto-cognitive schema to evaluate external environments and then make the best possible adjustments to life circumstances. In the case of depression, there exist depressogenic cognitive patterns which may reside in the unconscious or but dimly in the conscious. These patterns lead to depression if they block the resolution of sadness.

Arieti & Bemporad may sound quite similar to Beck's cognitive theory, described earlier, so it is important to see how they differ. Arieti (Arieti & Bemporad, 1978) contrasts his own theory of depressogenic thought patterns with that of Beck and finds Beck wanting. Beck's patterns are too close to conscious thought, Arieti says, and the kind of thinking Beck describes often occurs after the depression has set in and is indeed caused by the depression. The patient may then use these thoughts as a way of justifying the depression to him or herself.

By contrast, Arieti says the essential depressogenic thought patterns he describes are unconscious or but dimly in conscious awareness.

I think Arieti misreads Beck. Beck introduces the notion of a "schema" as a slightly more abstract thought pattern than the actual verbalized thoughts of the patients. The schema itself may not be fully within the awareness of the patient; instead it is a basic thought pattern which distorts experience so that the patient experiences it as the distortions he talks about. This usage of schema is quite close to the depressogenic thought patterns Arieti talks about.

The difference between them (and between Beck and other analysts, though especially Jacobson) lies elsewhere. Beck says an event occurs and we then think about it and feel depressed. Sometimes this is because the event itself is depressing--for example, a death in the family. For depressives, however, the objective meaning of the event is less clear; instead they carry with them a mind set that consistently misreads external information in a way that makes them feel depressed. The misreading is always of external events. The cognitive distortion occurs after the event and before the depressed affect is created. Once the depressed affect occurs, the theory has little to say.

Arieti too would say that depressives misread events in a way that makes them more likely to feel sad. However psycho-analysts would add the following:

1. All misreading stems from unconscious dynamic issues and thus reflects a neurotic need. This need is not only manifested passively in response to external events, but actively insofar as the external events are sometimes created out of this neurotic need. Thus some people are prone to chose for relationships people who are especially likely to

leave them. The confirming external event is created from the internal neurotic need.

2. Depressed moods can arise in the absence of external events if related to unconscious issues. Hence external events are sometimes mis-identified as the cause of a depression.

3. Some amount of cognitive distortion normally occurs as a consequence of mood changes (rather than always the reverse). This affect caused distortion occurs even in healthy people. Healthy people can step back and think about these mood-caused distortions: they recognize that their distortion is temporary and mood related. Depressives are less able to work out of their moods because they fail to recognize that these distortions are fleeting and mood related. This is different from Beck's cognitive distortion because it occurs after any precipitating event and because it is related to mis-reading internal states and feelings.

4. If depression only arises from cognitive distortions, it can be eliminated. Psycho-analysis, on the other hand, says it is a normal mood so the issue is not the elimination of depression but the capacity to bear it (e.g. Zetzel, 1965).

Two other sets of theorists have focused on how clinical depression--including the thought features of clinical depression--are responses to a basic depression affect.

Joffe and Sandler (1965, 1967; Sandler & Joffe, 1965) argue that, like anxiety, depression is a basic affect, a built-in psychological and biological response. Depression is the human affect which follows being helpless when physical or psychic pain is suffered. It is thus an affect which can occur as early as infancy when an infant

is understimulated or starved; it can also occur at any later time in life. The depressive affect must be distinguished from the illness. The more complex features of depressive illness are attempts to block this painful affect either before it develops or after it has started.

Using a similar starting point, Schmale and Engel (Schmale & Engel, 1975; Schmale, 1964) assume that there is a basic biological system leading the individual to withdraw from excessively or inadequately stimulating environments. This biological state is experienced as two distinct affects of depression, depending on when it occurs. The earlier affect is helplessness. This occurs in the first year of life when the infant is totally dependent on the external environment for satisfaction. The infant is helpless to effect a change; if gratification is not forthcoming, it will withdraw into a sleep-like or depressive stupor and only later once again attempt to regain contact with the external environment. After these first two years, but especially during the Oedipal years, three to six, the affect is experienced as hopelessness. Here the failure to obtain gratification from a parent figure (especially the opposite sex parent) is explained as being due to one's own inadequacies--hence hopelessness.

The depressive affects, helplessness and hopelessness, are like anxiety in that they can serve a signal function so as to avoid re-experiencing the original affect. For constitutional or experiential reasons, some people find even the signal depression is intolerable. Clinical depression (including depressive thinking) is a neurotic attempt to master the depression and prevent a traumatic re-experiencing of it (analagous to how phobias are attempts to master anxiety).

Comparing the Two Models of Depression

Despite their differences, the various cognitive theories of depression can be merged into a basic thought-first model. In the thought-first model, the mind is seen as an information processor, working like a "cool" computer. Drives, conflicts, motives--"hot" issues--play no crucial role. The external events are processed primarily to see if they are rewarding or unrewarding. These theories are concerned with the nature of external events--are the events rewarding or not?--and how these events are processed--are they seen as rewarding or not? The theories also consider the interaction between the real events and how they are mentally processed.

Depression begins when for some reason the computer begins to misfunction, reaching a conclusion of non-reward before all the data are in and analyzed. During a period of malfunctioning, the mind as computer begins to mis-read events, even reading success as if it were failure. In this model, the primary feature of depression is the inability of the mind to correctly analyze the environment. The mind places the burden of non-success upon itself either by being unable to read success where it is evident or else by blaming itself for a failure which might justifiably be blamed on something external to the self--such as bad luck, situational factors, etc.

By correcting the self-defeating cognitive thoughts, the depression is lifted. Often this involves placing the depressive in a situation where he experiences some success and then forcing him to take note of this success. The processing of self-success then alters how future information is processed. It is a bit like changing a computer program. The old program, which read failure everywhere, is ousted and

new program which correctly reads the environment is entered, so now the person can recognize his success, leading to a lifting of the depression.

For the depression to occur, the event must clearly be at the forefront of the person's mind. In addition, the way of thinking about the event (e.g., depressive attributional style) must be present before the event occurs and then remain stable afterwards.

In the alternative affect-first model the precipitating event is either absent altogether or else present but not at all in the forefront of the person's thoughts; there the characteristically depressive thought style does not precede the onset of depression but merely accompanies it. In various psychoanalytic theories there are two ways in which the onset of depression is not preceded by a person's full awareness of the external precipitating event. In the first, and more extreme case, the trigger for the depression can be in the unconscious and thus outside the realm of subject awareness (see Table 1). For example, unconscious conflict or unconscious loss or rejection by a loved one are sometimes thought to precipitate a depression (Freud, 1917, Bibring, 1953). In the less extreme and more common variation (see Table 1) the triggering event is within the person's awareness but is not easily tied by the person to the depression which follows. This is because the unconscious meaning of the event is far greater in impact and importance than the conscious meaning of the event. In such a case, the individual probably does not tie the depression to a precipitating event unless he or she is actively forced to look for a connection between the onset of the depression and the external event. Likewise, in some bio-chemical theories of depression, the changes in mood and

thought are caused by changes in body chemistry and are not necessarily tied to a concomitant external event.

This affect-first model stresses that the failure of self as agent in the world is a secondary feature of depression; what comes first is a set of feelings. As a result of such feelings (or as a defense against such feelings) we no longer wish to participate in the world. The world, as it were, no longer draws us into it. It is as if the overload of feelings breaks the circuits of our ties to world. We then take note of this breakage and read it as reflecting our lack of abilities in our retreat from the world. We may think we are ineffective, which lowers our self-esteem, but it is the feelings which have broken the circuit and not the thoughts.

The following example might clarify the affect-first model. About a month before a therapist's summer vacation, a patient starts to report feelings of depression. There is no clear precipitant which the patient can cite. The patient now feels so inadequate and unappreciated at work that he begins to consider changing careers. Based on derivatives in the material, the therapist suggests the patient's depression has been triggered by the therapist's upcoming vacation (i.e., "object-loss"). The patient vigorously denies this and claims his downcast view of his career is based on a correct assessment of the facts. Just prior to the therapist's vacation, the patient cries in the session. He is surprised by this, but recalls an old memory of father threatening to leave the family. Patient now ties his depression to the therapist's upcoming vacation, and some of the depression lifts.

This is a complicated example, but I will stress the following. First, the precipitating event is not recognized as such. Second, the

TABLE 1

THOUGHT-FIRST AND AFFECT-FIRST MODELS OF DEPRESSION

A. Thought-First Model

Depression	No Depression
Time 1. Depressive Attributional Thought Style (internal+global+stable)	No Depressive Attributional Thought Style (e.g., external attributions)
+	+
Time 2. Adverse Event (e.g., fired from job)	Adverse Event (e.g., fired from job)
↓	↓
Depression (including depressive affect)	No Depression

B. Affect-First Models

1. Strict Affect-First Model

Time 1. Event Outside Consciousness (e.g., biochemical change; unconscious conflict or object loss)

↓

Time 2. Depressed Affect

↓

Time 3. Depressed Thought (including depressive attributional style)

} together these = depression

2. Concommitant Affect-First Model

Time 1. Event Outside Consciousness (e.g., biochemical change; unconscious conflict or object loss)

↓

Time 2. Depression

↙ ↘

Depressed Affect

Depressed Thoughts (including thoughts attributional style)

event creates the depressed mood. Third, while in the depression, there arise typical depressive thoughts (in this case, depressive attributions about why he is a failure at his job). Fourth, confirmation about the real precipitant of the depression is later forthcoming, and it is not tied to the thoughts about the job which the patient expresses while in the depression. Last, the depressive thought style changes as the actual precipitant of the depression is recognized and the depression begins to lift.

The two models have different clinical implications. If the thought-first model were correct, it would be useful for clinicians to find people who are not depressed but who have a depressive thought pattern. Clinicians might then prevent depression before it occurred.

On the other hand, if the affect-first model were correct and there were no depressive attributional style in a non-depressed person, then there is no value in attempting to change this style if it appeared in a depressed person. The reason is simple: the depression may be causing the depressive thought style, and the style might disappear when the depression lifts. This is valuable information. It suggests that whenever someone who is in a depression begins to show depressive thoughts, a clinician might say "That is not you talking; it is the depression talking. You should not be making any major life decision (e.g., changing career) until the depression lifts." For someone who suffers from serious and cyclical depression, this may be vital information. Even on an everyday level, the experiment would provide a scientific basis for the therapeutic value in the old homily "Go to sleep: everything will look different in the morning." If moods affect

our thinking and if transient moods lift overnight, then this homily now becomes sound advice in addition to being comforting advice.

The key differences between the two models is summarized below:

1. The thought-first model says depressive thought precedes any depressive episode and the depressed affect is a consequence of this thought. The affect-first says depressed thought is caused by the depression; depression is either a primary affect, like anxiety, or an affect arising from conflict. In either case, conscious thought is not needed for its creation.

2. Both models agree that external events can trigger a depression. The thought-first model says such events must be consciously known and available to the person. The affect-first model says the triggering event can be forgotten or misidentified.

Testing the Two Models

Testing the two depression models could reduce the number of theories or at least indicate the limitations of some theories. But there are three fundamental difficulties in operationalizing a test of these alternative models. The first is the difficulty of experimentally inducing a mood change in people while keeping the nature of the procedure outside the realm of conscious awareness. The second is the difficulty in minimizing risk to subjects. The third is whether a change in mood equals depression or merely approximates it.

Sidestepping conscious awareness during a mood altering procedure may be virtually impossible if the subject is simultaneously to have informed consent about the procedure. In another era, there may have been ways of surreptitiously slipping a subject a mood altering drug; the subject would then not know the cause of his or her mood

change, and it would be easy to gauge whether the onset of depressed mood led to a change of cognitive style. Such a procedure raises serious ethical problems under any circumstances; it is clearly impossible given the informed consent necessities of current experimental work. Subjects must know, at the very least, that the procedure may alter their mood. Thus it is impossible to introduce a mood altering procedure which remains totally outside of subject awareness.

In addition to the informed consent requirements, current research must demonstrate limited risk to the subjects. With the research on depression this creates an insurmountable dilemma: any procedure which creates real depression is almost by definition a risk to the subject; yet any procedure which is risk free may be too mild to allow for even analogue studies in depression.

There is but one way to circumvent these twin problems: find an affect-first theory that begins with mood changes rather than clinical depression and find a thought-first theory that identifies which healthy people show pre-depressive thought patterns. Given this, an experiment can be run with a mild induction on a normal population.

For the affect-first theory, the choice is Jacobson (1971) and Arieti & Bemporad (1978); each describes a normal, mild mood (normal depression and normal sadness, respectively) which both change our thinking and which directly precede depression. For each of these theories, depression is an escalation or prolongation of these normal moods. Creating mood changes instead of clinical depression circumvents the ethical problems but not the informed consent problem: since subjects must know their moods are to be changed, the precipitating

event is not outside consciousness. Thus one premise of the affect first model is not met. Any support or non-support of the affect first model must therefore be considered weak evidence.

For the thought-first model, the recent attribution theory is ideal, since it predicts which normal people are pre-depressive. Of course, for the attribution theory, a sad mood or even a depressed mood is not the same as depression itself, if for no other reason than that depression is more stable than mood. However the attribution theory is experimentally derived from normal subjects rather than being clinically derived, so an experimental test using moods meets the theory on its own experimental terms. Recall that according to attribution theory, some people have a depressive attributional style prior to any depression. This style consists of a tendency to make internal, stable and global attributions about their life circumstances; when such depression-prone people then actually encounter a set of adverse life circumstances, they become depressed.

Seligman, Abramson, Semmel & von Baeyer (1979) cite indirect support for this new theory in studies which show that depressed students attribute failure to internal causes while non-depressed students make external attributions (Rizley, 1978; Klein, Fencil-Morse, & Seligman, 1976; and Kuiper, 1978). However, the only study directly designed to test this reformulated learned helplessness theory is the study by Seligman, Abramson, Semmel, & von Baeyer (1979). Students completed a newly created attributional style questionnaire and then completed two measures of depression. The results supported the theoretical predictions: the more depressed college students, when compared to non-depressed college students, tended to attribute bad

outcomes to stable, global, and internal causes and good outcomes to external, unstable and specific causes. The authors also found moderate to strong correlations between level of depression and overall level of depressive attitudinal style: on the Beck Depression Inventory (BDI) the correlation was .48, while on the Multiple Affect Adjective Check List (MAACL) the correlation was .24. At the end of the article, the authors say they plan a longitudinal follow-up study of individuals with a high depressive attributional style. If they obtain their expected results, they will provide rather strong evidence that their theory accounts for some types of depression.

Their study is a correlation study and not an experiment, and thus it is not able to specify the direction of effect. Wortman & Dintzer (1978) have speculated that people do not necessarily make an attribution about adverse circumstances and furthermore, that attributions may be caused by mood changes rather than by the causal agent of the mood swing. The strength of such a possibility is increased when the alternative affect-first model is considered. This affect-first model of depression could account for the very same results which Seligman et al (1979) obtained. In other words, Seligman et al may merely be <u>describing</u> a depression rather than pinpointing a <u>cause</u>. Instead of being a stable feature of personality which leaves people especially vulnerable to depression, attributional style may itself be merely the result of the depression.

A similar choice of models exists with regard to the relation between the mood of elation and the thoughts which are characteristic of an elated mood. Although Seligman does not explicitly deal with elation, it is possible to extend his theory to cover it. One could

postulate the existence of an elation attributional style which leaves people especially vulnerable to elation or joyous moods. Such an attributional style would consist of a tendency to make internal, stable and global attributions about good or pleasant outcome events. This could be operationalized in the following manner, using the Attributional Style Questionnaire developed by Seligman et al (1979): Elation Attributional Style= (global + internal + stable scores) on positive outcome events. Elation would thus again be explained by a thought-first model.

Psychoanalytic theorists have explicitly speculated on elation being related to, but the opposite of, depression (Freud, 1917, Jacobson, 1971). Edith Jacobson in particular develops a theory of elated mood which is close to the affect-first model. Here, as with depression, the thought change follows the mood change rather than preceding it. In other words, while in an elated mood, people think in a grandiose way, but this self-centered gradiosity is not necessarily a stable feature of personality which precedes the elated mood.

Footnotes

[1] The key symptoms of depression--and these symptoms are among the few facts associated with depression that rival schools accept--are as follows (Beck, 1967):

I. Emotional Manifestations
   1) dejected mood
   2) negative feelings toward the self
   3) reduction in gratification (from food, or libido, etc.)
   4) loss of emotional attachments
   5) crying spells
   6) loss of mirth response

II. Cognitive Manifestations
   1) low self-evaluation
   2) negative expectations
   3) self-blame and self-criticism

4) indecisiveness
5) distortion of body image

III. Motivational Manifestations
1) paralysis of will
2) avoidance, escapist and withdrawal wishes
3) suicidal wishes
4) increased dependency (not necessarily actual increased dependency, but rather a feeling of being increasingly dependent)

IV. Delusions
1) delusions of worthlessness
2) crime and punishment: belief that one has committed a crime and will be punished
3) nihilistic delusions
4) somatic delusions
5) delusions of poverty

[2]Hence the differences in the way grief ends when compared to depression. Grief ends when reality finally wins out and the object is accepted as dead. The work of depression involves denigrating or killing the object to weaken the libidinal ties to it. The unconscious work in depression ends either when the anger at the object has been exhausted or when the object is seen as worthless and thus can be easily abandoned. Either way, the ego at the end of depression feels superior to the object, and this superiority does not occur at the end of the grief process.

CHAPTER 2

HYPOTHESES

Four hypotheses are derived from the alternative thought-first and affect-first models.

These first two hypotheses are from the Abramson et al (1978) thought-first model.

Hypothesis One: Subjects with an initial high score on depressive attributional style will show higher depression following depressive mood treatment than will those with lower scores in attributional style.

Hypothesis Two: Subjects with initial high scores on elation attributional style will show higher elation following elation mood treatment than will those with lower scores in elation attributional style.

These two hypotheses are from the affect-first model:

Hypothesis Three: Subjects given a depression mood treatment will show a greater depressive attributional style than will subjects given other mood treatments.

Hypothesis Four: Subjects given an elation mood treatment will show a greater elation attributional style than will subjects given other mood treatments.

CHAPTER 3

METHODS

The two experiments created to test the alternative affect-first and thought-first models use the same basic paradigm: subjects complete a set of questionnaires on their attribution thought style, their level of depression and their mood; they are then randomly assigned to either a control condition or to an experimental condition designed to induce a mood change; afterwards, the initial questionnaires are again completed. The two experiments differ in the experimental induction used to alter mood states (reading versus film) and in the timing: in experiment one subjects must return for a second session, while experiment two is entirely completed at one sitting.

Experiment One

Subjects

The subjects in this experiment were 62 female undergraduates at Michigan State University. All but two participated to receive course credits.

Instruments

a) Mood Induction

All three experimental treatments (sad, happy, neutral moods) are based on the Velten Mood Induction Procedure or VMIP, a procedure which consists of having subjects read aloud some sixty mood related or

neutral statements. This procedure has been safely and successfully used in a large number of studies (Aderman, 1977; Coleman, 1975; Gouaux & Gauaux, 1971; Hale & Strickland, 1976; Matheny & Blue, 1977; Natale, 1977a, 1977b; Scheier & Carver, 1977; Strickland, Hale & Anderson, 1975; Velten, 1968). Two of these studies demonstrated that the procedure worked even when experimenter demand was controlled (Velten, 1967, 1968; Coleman, 1975). In most studies, the VMIP was not used to test a theory of depression but to study some feature of the mild dysphoric state it creates, features such as speech patterns (Natale, 1977a), gazing (1977b), and activity level (Strickland et al, 1975). In the current experiment too, the VMIP is used to create a mild dysphoric mood.

In a recent study, Frost, Graf & Becker (1979) observed that the VMIP depression treatment actually consisted of two kinds of statements: some self-evaluative statements which attack self-worth or self-esteem (self-devaluative statements); and other statements which only describe body sensations (somatic statements). Examples of self-devaluative statements are these: "I've doubted that I am a worthwhile person" and "I'm discouraged and unhappy about myself." Examples of somatic statements include "I'm getting tired out. I can feel my body getting exhausted and heavy" and "I feel terribly weak." Frost et al found that each subgroup of statements worked at least as well as the original VMIP, but the somatic statements were significantly better than the self-devaluative statements, when these two sub-groups were directly compared.

I use only the self-devaluation statements and not the somatic statements. This choice is based on a pilot study I ran to test the various induction procedures. I found that the somatic statements had

no effect on mood, while the self-devaluation statements created a strong consistent increase in depressed mood. This failure of the somatic statements is contrary to the findings of Professor Frost. There are two possible explanations for my pilot study's failure to replicate the Frost et al finding: first, M.S.U. undergraduates may systematically differ from Smith College undergraduates, and second, Frost et al might have had much stronger experimenter demand characteristics than they imagined.

The elation subjects read positive, self-referent statements connoting self-confidence, optimism and energy, such as "I feel cheerful and lively" and "On the whole, I have very little difficulty thinking clearly." In the neutral condition subjects read statements unrelated to moods or feelings. The original VMIP neutral condition contained only non-self-referent statements. To make it more comparable to the control and the elation conditions, I made half the neutral statements self-referent statements without an affective content--statements such as "I am an undergraduate at M.S.U." or "I am taking part in a psychology experiment." The other half are non-self-referent statements, such as "There is a large rose-growing center near Tyler, Texas." The two kinds of neutral statements are randomly mixed together.

A complete list of all VMIP statements used in this experiment is provided in Appendix 1.

b) Measures of Mood, Depression and Attributional Style

Two measures of mood are used in this study; these are the Personal Feeling Scale or PFS, which consists of ten 9-point ratings of immediate mood state (e.g., extremely optimistic-extremely pessimistic,

extremely happy-extremely sad) and the today form of the Multiple Affect Adjective Check List or MAACL (Zukerman & Lubin, 1965). These two mood questionnaires were chosen because they have been shown to be sensitive to mood induction procedures in a variety of studies (including Coleman, 1975; Hale & Strickland, 1976; Natale, 1977a; Strickland, Hale & Anderson, 1975; Velten, 1968); they are listed in Appendices 2 to 3. A depression mood subscale is derived from the total PFS. The MAACL (unlike the PFS) is a well-validated measure; insofar as the depression treatment only creates a depressed mood rather than real depression, this will be the most important measure.

In addition to depression, the Multiple Affect Adjective Check List can be scored for anxiety and hostility. Having three different scale scores allows for discriminant validation of the induction procedures.

Experiment one also employs a Counting Task to test the effectiveness of the depression treatment. Subjects count backwards from 100; since depression ought to make them count more slowly, the subjects in the depression condition ought to have reached a significantly higher number (i.e. closer to 100) after one minute than subjects in the other conditions. Since this task is started only after all the other questionnaires have been completed, or some ten to twenty minutes after the induction is over, it is best seen as a measure of of the strength of the induction. Should the other measures demonstrate a significant induction effect while this measure does not, it would demonstrate that the induction is weak or that it is not long lasting. This measure is not used to test the elation induction as it is less

clear what the relation between elation and speed of counting ought to be.

The study also uses the Beck Depression Inventory or BDI (Beck, 1974) which is frequently used in research to evaluate transient or reactive depression; in addition, it was the measure of depression used by Seligman et al (1979) in their initial study of the Abramson et al (1978) theory. The BDI is in Appendix 4. By using the BDI, the current experiment tests the attribution theory on its own terms. Matching test instruments with those used in the Seligman et al study makes this a fairer if more conservative test of the theory. In experimental research, a BDI score of nine or higher is used to indicate clinical depression. If the induction treatment subjects score lower than nine, then the induction created a depressed mood, but not real depression.

The BDI is not a good measure of elation, since it ranges from high depression to no depression (at zero) but does not cover the area from absence of depression through elation. By contrast, the PFS and the MAACL range from elation to no elation/no depression (at the mid-point) to high depression; they are thus better indicators of elation.

The final measure used in this study is the Attribution Style Questionnaire or ASQ (Seligman et al, 1979). This is the crucial instrument for measuring attributional style; it appears in Appendix 5. The ASQ consists of brief descriptions of hypothetical events; six are good outcome events (e.g. "You get a raise.") and six are bad-outcome events (e.g. "A date goes badly."). Subjects are asked to imagine that the events occur to them and then to write down one major cause for this event happening to them. Subjects then use seven-point Likert type scales to rate this cause as stable-unstable, internal-external, global-

specific, and important-unimportant. In this study, the six bad outcome events are scored separately and comprise the ASQ-B scale, while the six good outcome events form the ASQ-G scale. The scores are the sums of the internal-external, stable-unstable, and global-specific ratings, with high scores being more internal, global, and stable. A high depressive attributional style is operationally defined as a high score on the ASQ-B; likewise a high elation attributional style is operationally defined as a high score on ASQ-G. The elation and depression attribution styles are therefore independent of one another: subjects who are high on ASQ-B could be either high or low on ASQ-G.

Procedure

At Time One, all subjects sit together in a large room. They are given a general description of the experiment and are then asked to sign a standard department subject consent form. (As noted in Chapter One, informed consent means subjects must know the experiment may alter their mood; this knowledge violates an assumption of the affect-first model, making this a weak experimental test of that model). I then warn subjects about possible experimenter demand bias; in particular, I ask them to complete all mood and thought questionnaires based on what they are actually thinking and feeling at the time they complete it, rather than based on what they might think I might wish to hear them say. All subjects are then given copies of the BDI and the ASQ and asked to complete them. The order of the two questionnaires is counterbalanced. While subjects complete these questionnaires, a sign-up sheet will be passed around which lists all available time slots for the second part of the experiment, Time Two.

Subjects are assigned to their VMIP treatment condition (happy, sad, neutral) by a stratified sampling procedure. The stratified sample is high vs. low A.S.Q.-B score, using a median split of Time One A.S.Q.-B scores. Subjects do not learn which group they have been assigned to until the debriefing.

Time Two is at least one week later. At Time Two, each subject is alone in a room and a tape recorder is turned on; this entire part of the experiment is automated via the tape recording. In the room there are also a set of 4 x 6 index cards, a manilla envelope (containing questionnaires), a pencil, and two questionnaires atop the manilla envelope. The tape recording initially instructs subjects to turn off the machine, complete the two loose questionnaires, and then turn on the machine. These two questionnaires are the PFS and the MAACL. The order of these questionnaires is counterbalanced.

Tape-recorded instructions then ask subjects to read aloud and think about a series of statements, which are typed on the 4 x 6 inch index cards. At fifteen second intervals the tape recording tells the subjects to move on to a new card. Each subject reads a total of 60 VMIP statements; statements differ depending on treatment condition. Upon completion, they are instructed to open the manilla envelope, take out the questionnaires, and complete them in the order presented; they are again asked to turn off the tape recorder while they work, and turn it on upon completion. These questionnaires are a second copy of all questionnaires already once completed, namely a second BDI, PFS, MAACL and ASQ; these are in counterbalanced order. When these questionnaires are completed, subjects are instructed to find the blank paper and, on signal, to begin writing numbers in descending order from 100. I

remained available throughout the experiments to monitor any difficulties.

Subjects are then debriefed and dismissed. For the subjects in the sad mood condition, part of the debriefing consists of reading through a subset of elation statements to insure that no residual negative moods remain at the end of the experiment; this technique has been successfully used in most mood induction studies. In the unlikely event that any subject had continued to feel very sad, I planned to offer this subject a home phone number where I could be reached.

The design of Experiment One is summarized in Table 2-A.

Experiment Two

Subjects and Measures

Subjects for experiment two were 24 male and 23 female undergraduate volunteers.

This experiment uses the same questionnaires employed in Experiment One: BDI, MAACL, ASQ and PFS.

Procedure

The first part of the experiment is similar to the procedure used in Time One of Experiment One. Subjects sign consent forms after a brief explanation, subjects are warned about experimenter demand characteristics, and subjects then complete the BDI and ASQ. Two modifications are introduced: there is no sign-up sheet as there will be no second time for this experiment, and subjects also complete the PFS and the MAACL. The four questionnaires are distributed in a counterbalanced order.

TABLE 2

DESIGN OF EXPERIMENTS ONE AND TWO

A. Experiment One (N=62)

Time One (all subjects together in one room)	Time Two (each subject alone in a room; at least one week later)
1. Subjects Sign Consent Forms	1. Subject is Randomly Assigned to Happy, Sad or Neutral Condition (Prior to Arrival)
2. Subjects Warned of Experimenter Demand Characteristics	2. Subject Completes MAACL & PFS*
3. Subjects Complete BDI & ASQ*	3. Subject Reads VMIP Statements Aloud (Happy, Neutral or Sad Statements)
4. Subjects Sign Up for Time Two of Experiment	4. Subject Completes MAACL, PFS, BDI & ASQ*
	5. Subject Completes Counting Task
	6. Subject Debriefed

B. Experiment Two (N=47)

1. Subjects Sign Consent Forms
2. Subjects Warned of Possible Experimenter Demand Characteristics
3. Subjects Complete BDI, ASQ, MAACL, PFS*
4. Subjects are Randomly Divided into Two Equal Groups and Sent to Two Rooms
5. Subjects in One Room Watch the Movie "Peege" Designed to Create a Sad Mood; Subjects in the Second Room Watch a Mood Neutral Science Movie
6. After the Movie, All Subjects Complete BDI, ASQ, MAACL, & PFS*
7. Subjects Debriefed

*Order of questionnaires is counter-balanced.

ASQ = Attributional Style Questionnaire
BDI = Beck Depression Inventory
PFS = Personal Feelings Scale
MAACL = Multiple Affect Adjective Check List

Subjects are then randomly divided into two groups and sent to different rooms. In each room they see a movie. In one room, they see "Peege" a movie about a family visit to a dying grandmother in an old age home. This movie is designed to elicit sad or depressed feelings. In the second room they see "Triple Play," a neutral science movie about hydraulics. Movies have frequently been used to induce mood changes (e.g. Averill, 1969; Zuckerman, Lubin, Vogel, & Valerius, 1964). The movies used in this experiment were chosen in consultation with the Michigan State University film librarian. The last few minutes of "Peege," which were slightly optimistic in tone, are cut out in the showing. After the movie is shown, the BDI, ASQ, MAACL and PFS are redistributed in counterbalanced order and subjects again complete them. The design of Experiment Two appears in Table 2-B.

Plan of Analysis

The two experiments are related by the logic of multi-trait multi-method analysis. The VMIP induction of experiment one and the movie induction of experiment two are two quite different methods of creating mood change, so any hypotheses supported in both experiments are clearly not just method specific. Each experiment uses various measures of depression, which provide convergent validation of the induction effect (and of the hypotheses). Finally, if various moods and emotions are tested and the inductions only change the predicted mood, then discriminant validation has been achieved for the inductions.

If (and only if) the inductions work, then the specific hypotheses can be tested. The thought first hypotheses always turn on a simple comparison of means: in a specific treatment condition (e.g. depression) do subjects high in the relevant attributional style

(i.e. depressive attributional style) show higher mood levels than do the lows? The rationale for this comparison as applied to depression is as follows. The thought-first model assumes that certain situations lead to depression; in the two experiments, these situations are operationalized by the two inductions for depression. However the thought first model also assumes some people vary in their susceptibility to depression; the most susceptible are the people high in depressive attributional style. Thus if the thought-first model were correct, then in the depression inducing treatment, the most depression ought to occur among those people initially high in a depressive attributional style. By similar reasoning, the thought-first model of elation is proven if in the elation treatment, people high in elation attribution style show more elation than people low in this thought style.

The affect first hypotheses depend on an equally simple but quite different comparison: use attributional style as the dependent measure and see if a given treatment (e.g. depression) were causing a higher level of relevant attributional style (i.e. depressive attributional style) than the other treatments. The rationale for this analysis is as follows. The affect first model says that the relation between depression and depressive attributional style arises from depression causing a change in the way we think: thought style is not stable but changes as level of depression changes. Since we already know that the depressive treatment is causing more depression than the other treatments, then if the affect-first model were correct, subjects in this treatment should also show a significantly higher level in their depressive attributional style too. By similar reasoning, the affect

first hypothesis on elation is confirmed if there were a main effect of treatment on elation attributional style and if subjects in the elation condition show the highest elation attribution style score.

Experiment one basically uses a 2 x 3 analysis of covariance with two levels of attributional style (high, low) and three types of treatment (depression, neutral, elation). In experiment two, the basic analysis of covariance is a 2 x 2 x 2 analysis with two levels of attributional style (high, low) two treatments (depression, neutral) and sex (male, female).

CHAPTER 4

RESULTS

Discriminant Validation

Affirmative findings for any of the four hypotheses would be further strengthened if it could be demonstrated that the various inductions create only their predicted mood change and no other emotional change. Consider, for example, hypothesis three, which claims that changes in depressive attributional style are actually caused by changes in depression. If the depressive induction does create more than just depression--perhaps also creating significantly more boredom or more intensity--than one cannot be certain what caused the change in attributional style: was it change in depression, or was it the change in boredom or intensity?

Clearly one cannot test every possible additional emotion, but the MAACL can be rescored for both hostility and anxiety in addition to depression. All three emotional states are tested separately. Finding significant changes on hostility or anxiety would not vitiate earlier support of the four hypotheses, since there is no theoretical reason for these moods to change attributional style or depression, but lack of such findings would strengthen the logic of the experiment.

In experiment one, Table 3 addresses the issue of discriminant validation for the VMIP mood induction procedures. This table presents the results of three different 1 x 3 analyses of covariance (one mood x

three treatments) for three different measures of mood on the MAACL; each analysis controls for initial level of mood.

There was a significant treatment effect for MAACL depression. An inspection of the means for MAACL-depression in Table 3 indicates the depressed moods were in the predicted directions: the depressive treatment created the highest level of depression, the neutral treatment created a middle level of depression, and the lowest depression (or highest elation) was in the elation treatment. Thus both the depression treatment and the elation treatment worked as expected.

Unfortunately, Table 3 also indicates that the VMIP did more than just change levels of depression. There were also significant main effects for anxiety and hostility. In the case of anxiety, the means in Table 3 indicate that change in anxiety level came solely from the elation treatment. The depression treatments and neutral treatments showed almost equal levels of anxiety, while the elation treatment was only two-thirds as large. For anxiety to drop in the elation treatment makes intuitive sense, although it was not predicted. After all, if the elation treatment were working and the mood of subjects were improving, these subjects may have found they were growing more confident and more relaxed, and hence less anxious.

The treatment effects on hostility are less easy to explain, as the level of hostility seemed positively related to level of depression. One possible explanation is that the hostility levels reflected how enjoyable each treatment was: hostility went up most in subjects placed in the depression treatment since they realized they had been placed in an unpleasant experience; hostility went up slightly in the neutral condition since it was not very interesting to recite factual

TABLE 3

EXPERIMENT ONE: DEPRESSION, ANXIETY, HOSTILITY AS A FUNCTION OF TREATMENT CONDITIONS
(Pretreatment Scores Controlled)

Source	Depression Treatment (N=23)		Neutral Treatment (N=20)		Elation Treatment (N=20)		df	F
	M	SD	M	SD	M	SD		
MAACL-depression	17.93	1.71	15.22	1.79	10.95	1.80	2	3.93**
MAACL-anxiety	8.97	.79	8.61	.83	5.43	.83	2	5.58***
MAACL-hostility	9.37	.77	7.29	.81	6.60	.81	2	3.37**

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

statements; and hostility remained unchanged or even decreased in the elation treatment since subjects actually enjoyed reading aloud those uplifting statements. But whatever the explanation, the VMIP depression and elation conditions did more than just effect depression and elation, respectively.

Experiment two only had a neutral and a depression treatment, but because it used men and women, sex also had to be considered as a factor. Table 4 presents MAACL depression, anxiety, and hostility as a function of sex and treatment. Each mood was analysed by a 1 x 2 x 2 analysis of covariance, controlling for initial mood; the results suggest subjects cannot be pooled across sex. There was a significant main effect for treatment when depression was analyzed, and the depression adjusted means in Table 4 clearly indicate that the treatment effect was in the right direction: the depressive treatment created higher depression scores than the the neutral treatment.

Hostility levels remained the same for both men and women in the experiment two treatment, but in the case of anxiety, there were significant effects for treatment, sex, and sex-treatment interaction. The depressive treatment made subjects more anxious than the neutral treatment; this effect was not predicted, but it is sensible given the nature of the depression movie's subject matter, namely death and dying. Women were generally more anxious than men, and the interaction came because women in the depressive treatment were especially anxious. Again, women may have been more sensitive than men to this subject matter.

TABLE 4

EXPERIMENT TWO: DEPRESSION, ANXIETY AND HOSTILITY AS A FUNCTION OF SEX AND TREATMENT (Pretreatment Scores Controlled)

Measure	Depression Treatment				Neutral Treatment				Treatment		Sex		Sex X Treatment	
	Men (N=12)		Women (N=12)		Men (N=12)		Women (N=11)							
	M	SD	M	SD	M	SD	M	SD	df	F	df	F	df	F
MAACL-hostility	6.91	1.07	9.172	1.08	9.13	1.07	8.68	1.14	1	.64	1	.74	1	1.53
MAACL-anxiety	7.26	1.06	11.2	1.06	6.56	1.06	6.88	1.11	1	5.51**	1	4.07**	1	2.85*
MAACL-depression	18.13	2.56	26.60	2.60	16.23	2.57	15.95	2.67	1	5.79**	1	2.56	1	2.84*

* $p<.10$
** $p<.05$

Convergent Validation

Convergent validation was inspected on four measures of depression; the Beck Depression Inventory (BDI); the Personal Feelings Scale (PFS); the depression sub-scale of the Multiple Affect Adjective Check List (MAACL-d); and the Counting Task. The latter only appeared in experiment one. On the BDI, the PFS, and the MAACL, the dependent measure was post-induction depression with pre-induction depression controlled. All three were analysed using analysis of covariance. The Counting Task was analysed by analysis of variance. These analyses appear in Table 5-A for experiment one and Table 6-A for experiment two. If the depression and elation treatments worked, there should have been a significant main effect for treatment, along with the highest depression score in the depression treatment and the lowest in the elation treatment.

As column one of Table 5-A indicates, on experiment one three measures of depression (the BDI, the PFS, and the MAACL) showed a significant treatment effect. Inspection of the means indicates that on all three measures, subjects in the depressive VMIP treatment showed the highest post-induction levels of depression. These results provide further evidence that the depression treatment successfully increased depression more than the neutral or elation treatments.

The Counting Task alone did not show any significant treatment effects in experiment one. There are at least two possible explanations for this. The first explanation is that the VMIP depression treatment changed the way subjects felt about themselves, and hence changed various self-report measures, but was not strong enough to translate into actual behavior. The second explanation has to do with the order

TABLE 5

EXPERIMENT ONE: ANALYSES OF COVARIANCE FOR DEPRESSIVE ATTRIBUTIONAL STYLE SUBJECTS (ASQ-B)

Measures	Experimental Treatment			ASQ-B (High vs. Low)			Experimental Treatment X ASQ-B		
	df	MS	F	df	MS	F	df	MS	F
A. Depression Measures (pre-score controlled)									
Counting Task[1] (N=62)	2	7.86	.093	1	5.82	.07	2	44.89	.53
BDI (N=62)	2	53.21	2.40*	1	17.21	.78	2	26.05	1.18
PFS (N=61)	2	113.72	8.23****	1	15.43	1.12	2	10.18	.74
MAACL-Depression (N=62)	2	247.11	3.89**	1	67.86	1.07	2	72.91	1.15
B. Depressive Attributional Style Measure (pre ASQ-B Controlled)									
ASQ-B (N=62)	2	131.65	1.22	1	6.95	.07	2	38.65	.36

* p<.10
** p<.05
**** p<.001

[1] No pre-induction score, so reported means are unadjusted.

of the Counting Task within the overall experiment: the Counting Task began when all post-induction questionnaires had been completed. This was usually some ten to twenty minutes after the induction had concluded. It is thus possible that the treatment effects had worn off by the time subjects started the Counting Task. Either of these explanations suggests that the generalizability from this induction to real depression is limited, since either the induction effects were too

weak to translate into behavior or else they were too transient to last more than twenty minutes. Real depression affects behavior and lasts more than a half hour.

As Table 6-A, column one indicates, the experiment two depression treatment also created significantly higher levels of depression on all three measures of depression (Beck Depression Inventory, Personal Feelings Scale, and Multiple Affect Adjective Check List-depression subscale). The PFS also showed a significant main effect of sex; inspection of the means indicates this was because women showed more depression on the PFS than men. Finally, the MAACL-depression showed a significant interaction of sex and treatment: within the depression treatment, female depression scores were almost half again as large as the male scores. Just why women would have reacted more strongly than men to the treatment is unclear. Perhaps women were more sensitive to issues of object loss or perhaps they were more willing to admit such feelings; the difference might also be related to the sex of the protagonist in the depression movie. The movie was about an infirm and dying grandmother, so perhaps the women were more upset because they could identify more clearly with the protagonist.

The results of the elation induction are presented in column 1 of Table 7-A: there were significant treatment effects on two of the three measures (PFS and MAACL-D) in the 2 x 3 analysis of covariance, (controlling for initial mood level). In this analysis, elation was operationally defined as low scores on the depression measures. Inspection of the means shows that the mood change was in the expected direction: elation treatment lowered depression. Only the BDI failed to show a significant treatment effect on mood, and as noted in description

TABLE 6

EXPERIMENT TWO: ANALYSES OF COVARIANCE FOR DEPRESSIVE ATTRIBUTIONAL STYLE SUBJECTS (Pre-score Controlled) (N=47)

Measures	A. Depression Measures (pre-score controlled)						B. Depressive Attributional Style Measure[1]	
	BDI		PFS		MAACL-D			
	df	F	df	F	df	F	df	F
Treatment	1	3.01*	1	3.10*	1	6.11**	1	1.0
ASQ-B (high vs. low)	1	2.20	1	.78	1	2.35	1	2.05
Sex	1	1.66	1	4.79**	1	2.36	1	6.30**
Treatment x ASQ-B	1	1.29	1	1.60	1	1.35	1	.08
Treatment x Sex	1	.31	1	2.64	1	3.10*	1	.14
ASQ-B x Sex	1	2.21	1	.84	1	.04	1	.31
Treatment x ASQ-B x Sex	1	.01	1	.41	1	.84	1	1.23

[1]Pre- and post scores were not significantly related, so controls for pre-scores were not entered.

* $p<.10$

** $p<.05$

of the measures (pg. 36), this measure is not as appropriate for measuring elation as is the PFS or the MAACL.

Depression Hypotheses

The depression inductions clearly created some depression, so the two depression hypotheses (one and three) could be tested. Hypothesis three, the affect-first hypothesis on depression is considered first. This hypothesis says that higher depression created

TABLE 7

EXPERIMENT ONE: ANALYSES OF VARIANCE FOR ELATION ATTRIBUTIONAL STYLE SUBJECTS (ASQ-G)

Measures	Experimental Treatment			ASQ-G (high vs. low)			Experimental Treatment X ASQ-G		
	df	MS	F	df	MS	F	df	MS	F
A. Depression Measures (pre-score controlled)									
BDI (N=62)	2	51.58	2.26	1	10.42	.46	2	9.81	.43
PFS (N=61)	2	114.37	8.85****	1	53.40	4.13**	2	15.40	1.19
MAACL-Depression (N=62)	2	254.78	4.07**	1	66.80	1.07	2	98.2	1.57
B. Elation Attributional Style (ASQ-G) Measures (pre-ASQ-G controlled)									
ASQ-G	2	274.40	1.63	1.63 1	141.53	.84	2	25.99	.154

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

higher depression attribution style. For this hypothesis to be confirmed, subjects in the depression treatment would have to show the highest post-induction levels of depression attribution style. In experiments one and two, Tables 5-B and 6-B respectively indicate that there were no significant effects of treatment on depressive attribution style. Hypothesis three was not confirmed. There was no evidence that depressive attribution style levels were altered by levels of depression.

To test hypothesis one (the thought-first hypothesis on depression), subjects were sorted into high vs. low depressive attributional style groups. In experiment one this occurred before the

second experimental session. Experiment two subjects had only one session. They were therefore ranked according to their pre-induction depressive attributional style score (ASQ-B) and a median split was obtained. Subjects above the median were called "high depressive attributional style" subjects while subjects below the median were the low depressive attributional style subjects (low ASQ-B).

Hypothesis one was tested in experiments one and two by inspecting the subjects in the depression treatment: if high depressive attributional style subjects showed significantly more depression than the low ASQ-B subjects, the hypothesis was confirmed. The test for significance is a simple, one-tailed comparison of the difference in means (t test). In experiment two, separate analyses had to be performed on the men and the women because there was a significant main effect of sex and a significant interaction involving sex.

The experiment one results of the test of hypothesis one are presented in Table 8; this table looks at subjects in the depression treatment and compares adjusted and unadjusted mean depression scores of high depressive attributional style subjects (high ASQ-B) with the means of low ASQ-B subjects. When the unadjusted means are considered (columns 2 and 3), then only one measure, the MAACL-D, showed that high ASQ-B had higher depression scores, as predicted. But Table 8, column 4, also indicates that pre-induction and post-induction depression scores significantly covaried, so the appropriate comparison should use the adjusted means, columns 5 and 6. With adjusted means, the PFS high ASQ-B subjects also showed the predicted higher level of depression. However this difference was not significant. The only significant difference was on the MAACL-D; since this difference was

both significant and in the predicted direction, hypothesis one was supported in experiment one.

Table 9 presents the critical test from experiment two of the thought-first hypothesis regarding depression, hypothesis one. Men and women were analyzed separately because of sex effects and interactions. Since pre-induction depression scores always covaried significantly with post-induction depression scores, the preferred comparison used adjusted means, columns 5 and 6. Hypothesis one predicts that in the depression treatment high ASQ-B women would show more depression than low women and this was true on two depression measures, the BDI and the MAACL. However none of the differences in means were significant, so hypothesis one was not supported.

For men, all three depression measures showed differences in adjusted means occurring in the predicted direction. In addition, the size of these differences was significant on the BDI and the MAACL. Thus hypothesis one was supported for men although it was not supported for the women. Why there should be a difference by sex is not fully clear, but the lack of results for women may in part arise because the N's in each cell became quite small when we divided both by sex and by ASQ-B level. Indeed the N's were so small that getting results for the men was more surprising than getting no results for the women.

Elation Hypotheses

Since the elation treatment created elation, the elation hypotheses (two and four) were tested. The affect first hypothesis on elation, hypothesis four, was considered first. This hypothesis says higher elation creates a higher elation attributional style (ASQ-G). As Table 7-B indicates, there was no main effect for treatment when elation

TABLE 8

EXPERIMENT ONE: DEPRESSION SCORES FOR HIGH VS. LOW DEPRESSIVE ATTRIBUTIONAL STYLE SUBJECTS (ASQ-B) IN DEPRESSIVE TREATMENTS

Measure	Depression		Covariance of Pre- and Post Scores	Adjusted Depression (Adjusted Standard Deviation)		Difference in Means
	High ASQ-B (N=11)	Low ASQ-B (N=11)		High ASQ-B	Low ASQ-B	
BDI	7.45	7.81	F=15.62 P=.0002	7.70 (1.42)	8.72 (1.44)	insignificant
PFS	17.45	18.09	F=101.50 P=.0000	18.27 (1.12)	17.28 (1.18)	insignificant
MAACL-D	21.00	17.18	F=45.10 P=.0000	20.87 (2.40)	14.95 (2.43)	t=1.73**

**p<.05, one-tailed test

TABLE 9

EXPERIMENT TWO: DEPRESSION SCORES FOR HIGH VS. LOW DEPRESSIVE ATTRIBUTIONAL STYLE SUBJECTS (ASQ-B) IN DEPRESSIVE TREATMENT

Measure	Depression: Hi ASQ-B (N=6)	Depression: Lo ASQ-B (N=6)	Covariance of Pre- and Post Scores	Adjusted Depression (Adjusted Standard Deviation): Hi ASQ-B	Adjusted Depression (Adjusted Standard Deviation): Lo ASQ-B	Difference in Means
I. Women						
BDI	8.50	6.0	F=43.17 P=.000	6.5 (1.38)	5.51 (1.34)	insignificant
PFS	22.17	23.00	F=18.74 P=.0001	20.89 (1.72)	22.77 (1.70)	insignificant
MAACL-depression	31.33	25.00	F=8.30 P=.007	29.25 (3.68)	24.41 (3.62)	insignificant
II. Men						
BDI	7.5	2.5		7.01 (1.35)	3.26 (1.35)	t=1.97**
PFS	20.67	14.17		17.05 (1.80)	16.10 (1.76)	insignificant
MAACL-Depression	23.33	12.00		22.28 (3.63)	13.85 (3.67)	t=1.63*

* p<.10, one-tailed test
**p<.05, one-tailed test

attributional style was the dependent measure. This meant that subjects in the elation treatment received no higher ASQ-G scores after the induction than did subjects in the other treatments. There was thus no evidence that elation thought style was changing as elation mood was boosted, and hypothesis four was not confirmed.[1]

Table 10 addresses the thought-first hypothesis on elation, hypothesis three, which predicts that subjects high in elation attribution style would show more elation following elation mood treatment than would subjects low in ASQ-G. Only subjects placed in the elation treatment in experiment one were considered in this table, and the elation moods of subjects initially high in their elation attribution style (high ASQ-G) were compared with the moods of low ASQ-G subjects. Subjects were ranked by elation attribution style level and a median split was obtained to create the high and low ASQ-G groups. High ASQ-G subjects always showed more elation than low ASQ-G subjects. The pre-induction and post-induction scores of elation always significantly co-varied, so the adjusted means were used for comparison. Even when initial levels of elation were thus controlled, the high ASQ-G subjects always responded to the elation treatment by obtaining higher elation than the low ASQ-G subjects. This pattern, predicted by hypothesis three, was consistent across the three measures of elation. However none of these differences were significant, so hypothesis three could not be confirmed. Since experiment two had no elation treatment, there was no test of the elation hypotheses in experiment two.

Summary of Results

1) The elation and the two depression treatments created their expected mood changes, but they also created additional emotional changes.

TABLE 10

EXPERIMENT ONE: DEPRESSION-ELATION SCORES FOR HIGH VS. LOW ELATION ATTRIBUTIONAL SUBJECTS (ASQ-G) IN ELATION TREATMENT

Measure	Depression/Elation: High ASQ-G (N=10)	Depression/Elation: Low ASQ-G (N=10)	Covariance of Pre- and Post Scores	Adjusted Elation/Depression (Adjusted Standard Deviation): High ASQ-G	Adjusted Elation/Depression (Adjusted Standard Deviation): Low ASQ-G	Difference in Means
BDI	6.10	5.90	F=13.98 P=.0004	5.75 (1.52)	5.71 (1.51)	insignificant
PFS	2.40	2.22	F=102.92 P=.0000	14.22 (1.14)	14.08 (1.20)	insignificant
MAACL-Depression	9.90	9.00	F=42.55 P=.0000	11.91 (2.52)	9.94 (2.51)	insignificant

2) The thought-first hypothesis on depression was significantly supported on one of three depression measures in experiment one and on two of three depression measures for the men in experiment two. Low attribution style subjects in the appropriate treatment were never significantly higher than high attribution style subjects, so whenever the difference in means was significant, it was in the predicted direction.

3) There was no support for the affect first hypothesis of depression.

4) There was no support for either the affect-first or the thought-first models of elation.

Footnote

[1]Furthermore, introducing a second covariate to control for initial depression level made no appreciable difference. The following covariates were attempted, and none made the results significant: pre BDI; z score for pre PFS + pre MAACL; z scores for pre PFS + pre MAACL + pre BDI.

CHAPTER 5

DISCUSSION OF RESULTS

This conclusion section will first discuss the elation hypotheses. It will then consider the thought-first model of depression and follow this by a discussion of the affect-first model.

Elation Models

The hypotheses of this experiment were derived from two models of human depression. The elation hypotheses were not based on empirical research about elation nor were they based on a separate theory of elation; instead they were derived by extending the logic behind the depression models to cover elation, a related but different mood. In particular, the attribution theory of depression related depression to a way of processing information about unpleasant events: people who laid the blame for these events outside of themselves escaped depression, while people who attributed these unpleasant outcomes to some feature of their own personality became depressed. It seemed logical that a similar situation might exist with regard to good events: some people might be especially apt to take personal credit for good events and feel terrific about themselves while other people are less apt to take credit for these pleasant events and thus would be less apt to reap the dividend of good or elated feelings. And just as the causal direction between depression and depressive attributions lay open to question, especially given the alternative affect-first model of depression, so

too the causal direction between elated mood and elated thought style was unclear.

The results of this experiment cannot be used to support either the thought-first of the affect-first models of elation. In part the failure of the results to distinguish between the two models may arise because the paradigms derived from depression may not be directly transferable to elation. In addition, the measures in this experiment were measures initially derived from theories of and research with depression, and these measures may not be suitable to elation. Thus the Attribution Style Questionnaire may not be measuring the essential thought processes conducive to elation. The dependent measures of depression may likewise not work as measures of elation: in this experiment, elation was operationally defined as the absence of depression, whereas in reality elation might be a singular mood, unrelated or perhaps more than the mere absence of depression. Thus any future studies specifically aimed at investigating the thought-first or affect-first models of elation should begin with measures both specifically designed to measure elation and validated with elation.

Thought-First Model of Depression

The thought-first model, as represented by the attribution theory of depression, was supported by this experiment. The critical test of this theory was whether the depressive treatment created more depression in the high depressive attributional style subjects (high ASQ-B) than in the low ASQ-B subjects. There were nine of these critical tests: three depression measures were tested in experiment one, and three each were tested for men and for women separately in

experiment two. Of these nine tests, three were significant in the predicted direction and the other six were insignificant. Thus where the difference in means did reach a level of significance, the direction was always in the predicted direction. When additional factors are considered--namely the very small N's in each box of the critical tests (N's=six per box on the six critical tests in experiment two) and that support was not method specific, occurring in two quite different treatments--then the support for the attribution theory is all the more striking.

But before considering any clinical applications of this theory, the limitations of the experiment must also be considered. The primary question is whether this is a study of an analogue of depression or a study of a real (albeit induced) depression. There are two pieces of evidence which shed light on this issue. The first is the failure in experiment one of the Counting Task even when all self-report measures showed a significant depressed mood in the treatment condition. Two explanations were offered for this failure of the Counting Task to be affected by the induction: either the induction was too weak to change behavior (as opposed to changing self-reported feelings) or else the induction had worn off in the ten to twenty minutes between the end of the induction and the completion of the questionnaires, when the Counting Task started. Either explanation suggests the induction created a depressed mood rather than real clinical depression.

The second piece of evidence on this issue is more clear-cut. On one of the three depression measures, the BDI, there exist normative data which compare clinical depression with levels in the normal population. We can thus see if the levels of depression created by the

inductions reached the levels of clinical depression. On the BDI, clinical depression for experimental purposes begins at a score of 9. In experiment one, the experimental treatment created a mean BDI score of 8.4; on this measure in experiment two, the men have a score of five and the women of six. This means that neither induction created clinical depression.

If the inductions were only creating mood changes and not clinical depression, then the direction of future research is clear. The relation found in this experiment between attribution style and depressed mood must now be repeated in a naturalistic experiment, and this relationship must also be demonstrated to exist at levels of clinical depression. These two research directions will be discussed in turn.

Experimental studies are powerful research tools but somewhat artificial. Now that we have experimentally demonstrated how depressive attribution style predicts later depression, this relationship must also be explored outside the laboratory, in the real world. It should be possible to measure depressive attributional style in a group of people and to follow them over time to see if the high depressive attributional style people were more prone to depression following bad events. Seligman et al (1979) say they are currently undertaking such a study. One possible way to conduct this study would be as follows: use the attributional style questionnaire to obtain a measure of depressive attributional style on a large group of incoming undergraduates before they arrive at college, and later see if their depressive attribution score predicts a depressive response to either leaving home or to any failure experiences when the first set of grades are returned. To

accurately test the theory, students would have to be controlled on their levels of intelligence and previous academic success. Some measure of ambition (or an ambition/ability ratio) might be obtained too, as some alternative theories of depression say that depression will arise if ambition exceeds ability (i.e. excessively high narcissistic goals), presumably independent of depressive attributional style. Finally the dependent variables measuring later depression will have to be inspected to see if results indicate only depressive mood or the more serious and higher scores of clinical depression.

A similar study could be undertaken in a clinical population. People who obtained clinical help for their depression could be tested upon discharge (from hospital or from out-patient clinic) for their level of depressive attributional style (controlled for level of depression at discharge). Follow-up studies could then be attempted to see if people with high depressive attributional styles were more likely to later suffer a recurrence of depressive episodes.

Affect-First Model of Depression

The affect-first model suggests that the thought-first features of depression such as attributional style are not the causes of depression but merely the result of depression, and likely to go up as levels of depression increase. This study provides no support for the affect-first model: in both experiments, treatments which significantly increased depression had no significant effect on changing depressive attributional style.

Does this mean the affect first model should be dismissed? And if not, what future research should be attempted to test the affect-first model?

The first question is easier to answer than the second. An accurate test of the affect-first model would change moods without subjects being aware of what had caused this change. As noted in Chapter One, current informed consent procedures make this impossible. Since the experiment only partially tests the affect-first model, the results are more suggestive than conclusive. It is too soon, pending further studies, to dismiss the affect-first model.

What these further studies could be remains a difficult question. In the last year there has been a renewal of interest in how information and especially affect can be processed outside of conscious awareness (Shevrin & Dickman, 1980; Zajonc, 1980). For example, Wilson 1979) found that during a dichotomous listening task, people given information at below threshhold levels showed an affective response to this information--a liking response--even when they could not identify the information. Such dichotomous listening experiments might eventually be used to see if affective information is unconsciously processed in a different way during depression.

A second, albeit indirect approach, would derive from affect-first theories those personality types thought to be prone to depression. The key to such an approach is obtaining a personality criterion not related to the processing of external information. If the criterion could be operationalized, a new set of depression-prone subjects could be identified. If they showed a wide range of attributional styles, then they could be studied in a fashion similar to the future research proposed for the thought-first model: such people could be followed over time to see if they were more depression prone than controls (with levels of depressive attributional style being

controlled). An example of this approach is provided by Blatt (1974) who has extended psychoanalytic object relations theory to depression. He has recently started to devise ways of empirically identifying various depression-prone personality types based on his theoretical extension (Blatt, S., D'Afflitti, J. P., & Quinlan, D. M., 1976).

Two Models or One?

A basic premise of this study was that there are two fundamentally different models of depression. Before these models are set aside, one last possibility must be considered in passing: these models, however different, may be alternate aspects of a single on-going process in which affect alters thought which alters affect which alters thought, etc. In reality, perhaps, the relation between depressed thought and depressed affect may be closer to a feed-back loop.

In part this feed-back possibility arises from an inadequacy of the cognitive theories noted earlier. These theories do not explain whence there arise the differences in thought-style that the cognitive theories describe so well. After all, why should one person develop a depressive attributional thought-style while another develops a less pathogenic cognitive pattern? Bowlby (1980) has suggested that the cognitive theories are incomplete because they omit any etiological explanation, and he suggests Beck's theory can be subsumed under his own theory of early attachment difficulties (most especially loss) creating depressogenic thought patterns.

However there is another body of developmental research which suggests that there exist temperamental differences from birth, and that among these temperamental differences are mood variations (Buss, A., Plomin, R., & Willerman, L., 1973; Scarr, S., 1969; Thomas, A.,

Chess, S. & Birch, H., 1968). The work of Thomas et al in particular suggests that there are some infants whose emotional temperament leaves them especially vulnerable to behavior disorders. Perhaps the origin of depressive thinking likewise arises out of the interaction between inherited mood characteristics and particular environments (e.g., early loss experiences). This line of reasoning can only be investigated by careful developmental research.

CHAPTER 6

SUMMARY AND CONCLUSIONS

Reviewing the psychological theories of depression revealed a fundamental disagreement on the nature of the relationship between depressed thoughts and depression. A set of cognitive theories says depressions arise because of the interaction between depressogenic thought styles and painful events. The depressogenic thought patterns presumably precede the depression and are a central cause of the subsequent depressions. On the other hand, a second set of theories says these depressive thought styles arise out of the depression but do not precede it and are thus not a cause of depression.

To test these alternate thought-first and affect-first models of depression, two experiments were conducted. The subjects in the first experiment were 62 Michigan State University undergraduate women; in the second experiment, the subjects were 24 male and 23 female M.S.U. undergraduates.

The two experiments followed a similar design: subjects completed a set of questionnaires on their thought style, their level of depression and their mood; they were then randomly assigned to either a control condition or to an experimental condition designed to induce a mood change; afterwards, the initial questionnaires were again completed. The two experiments differed in the experimental induction used to alter mood states and in the timing: in experiment one subjects

returned for a second session, while experiment two was entirely completed at one sitting.

Experiment one used the Velten Mood Induction Procedure (or VMIP) to create neutral, depressed and elated moods. This procedure consists of having students sit alone in a room and read aloud a set of 60 mood related statements. In experiment two, mood was altered by watching the movie "Peege"; the control group saw a neutral science movie.

The following instruments were used in both experiments to measure depressed mood: the Beck Depression Inventory or BDI; the Multiple Affect Adjective Check List or MAACL; and a Personal Feelings Scale or PFS. The measure of depressive thought style was the Attribution Style Questionnaire or ASQ.

The results supported two conclusions. First both inductions successfully created depressed moods, although the procedures also induced additional emotions, most noticeably increased anxiety. Second, there was some support for the thought-first hypotheses of depression. There were significant predicted differences on one of three depression comparisons in experiment one and on two of six comparisons in experiment two. This support is stronger than it seems because sex interactions in experiment two necessitated separate analyses by sex, making the cells used for the comparison quite small in size. In addition, no comparisons were ever significant in a direction opposite to the predictions.

The affect-first hypotheses were not supported. Furthermore, no relation was found between induced elation in experiment one and either the affect-first model or the thought-first model.

The discussion suggested future avenues of research which might use naturalistic studies rather than experimental studies. The failure to find any support for the affect-first model was explained as being partly due to the experiment's failure to fully test the affect-first model, most notably because informed consent procedures made it impossible to alter mood without subjects being aware of the mood change. Finally, the discussion raised the possibility that the two models are actually alternate phases of a single feed-back loop process.

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APPENDICES

APPENDIX 1

VELTEN MOOD INDUCTION PROCEDURE (VMIP)

VMIP: Elation Statements

Please read each of the following statements out loud. As you look at each statement, focus your observation only on that one. The tape recording will tell you when to move on to the next card.

These statements are intended to create a certain mood. Your success at coming to experience this mood will largely depend on your willingness to accept and respond to the idea in each statement and to allow each suggestion to act upon you without resistance.

Attempt to respond to the feeling suggested by each statement. Then try to think of yourself as definitely being and moving into that mood state.

If it is natural for you to do so, try to visualize a scene in which you have had such a feeling.

If you feel the urge to laugh, it will probably be because humor is a good way to counteract unwanted feelings or it might be because you feel yourself going into that mood. Try to avoid laughing.

Today is neither better nor worse than any other day.

I do feel pretty good, though.

I feel light-hearted.

This might turn out to be one of my good days.

If your attitude is good, then things are good, and my attitude is good.

I've certainly got energy and self-confidence to spare.

I feel cheerful and lively.

On the whole I have very little difficulty in thinking clearly.

My parents are pretty proud of me most of the time.

I'm glad I'm in college--it's the key to success nowadays.

For the rest of the days, I bet things will go really well.

I'm pleased that most people are so friendly to me.

My judgement about most things is good.

It's encouraging that as I get further into my major, it's going to take less study to get good grades.

I'm full of energy and ambition--I feel I could go a long time without sleep.

This is one of those days I can grind out school work with practically no effort at all.

My judgment is keen and precise today. Just let someone try to put something over on me.

When I want to, I can make things turn out fine.

I feel enthusiastic and confident now.

There should be opportunity for a lot of good times coming along.

My favorite song keeps running through my head.

Some of my friends are so lively and optimistic.

I feel talkative--I feel like talking to almost anybody.

I'm full of energy and am ready to get to like the things I am doing on campus.

I'm able to do things accurately and efficiently.

I know good and well that I can achieve the goals I set.

Now that it occurs to me, most of the things that have depressed me wouldn't have if I just had the right attitude.

I have a sense of power and vigor.

I feel so vivacious and efficient today--sitting on top of the world.

It would really take something to stop me now.

In the long run, it's obvious that things have gotten better and better during my life.

I know that in the future I won't over-emphasize so-called 'problems.'

I'm optimistic that I can get along very well with most of the people I meet.

I'm too absorbed in things to have time for worry.

I'm feeling amazingly good today!

Things look good; things look great!

I feel that many of my friendships will stick with me in the future.

I can find the good in almost anything.

I am particularly inventive and resourceful in this mood.

I feel superb! I think I can work to the best of my ability.

I feel so gay and playful today I feel like surprising someone by telling a silly joke.

I feel an exhilirating animation in all I do.

I feel highly perceptive and refreshed.

My memory is in rare form today.

In a buoyant mood like this one, I can work fast and do it right the first time.

I can concentrate hard on anything I do.

I feel industrious as heck--I want something to do!

Life is firmly in my control.

I wish somebody would play some good loud music!

This is great--I really do feel good. I <u>am</u> elated about things.

I'm really feeling sharp now.

This is just one of those days when I'm ready to go.

I feel like I'm bursting with laughter--I wish somebody would tell me a joke or give me an excuse!

I'm full of energy.

God, I feel great.

VMIP: Neutral Statements

Please read each of the following statements out loud. As you look at each statement, focus your observation only on that one. The tape recording will tell you when to move on to the next card.

These statements are intended to create a certain mood. Your success at coming to experience this mood will largely depend on your willingness to accept and respond to the idea in each statement and to allow each suggestion to act upon you without resistance.

Attempt to respond to the feeling suggested by each statement. Then try to think of yourself as definitely being and moving into that mood state.

If it is natural for you to do so, try to visualize a scene in which you have had such a feeling.

If you feel the urge to laugh, it will probably be because humor is a good way to counteract unwanted feelings or it might be because you feel yourself going into that mood. Try to avoid laughing.

I am a woman.

Japan was elected to the United Nations almost fourteen years after Pearl Harbor.

This quarter I am enrolled in a psychology course.

Oklahoma City is the largest city in the world in area, with 631.161 square miles.

Right now I am reading this statement aloud.

At the end appears a section entitled "Bibliography notes."

Michigan is where I now reside.

We have two kinds of nouns denoting physical things: individual nouns and mass nouns.

I attend Michigan State University.

There is a large rose-growing center near Tyler, Texas.

Today I have had to walk up some stairs.

I am sitting by myself in a room.

This book or any part thereof must not be reproduced in any form.

Agricultural products comprise seventy percent of the income.

While I read these statements I am also listening to a tape recording.

I attend a university located in East Lansing.

Saturn is sometimes in conjunction, beyond the sun from the earth, and is not visible.

I am a high school graduate.

Some streets were still said to be listed under their old names.

I dressed myself today.

I am participating in the second part of this experiment.

I am a female undergraduate.

The system is supervised by its Board of Regents.

When the plant bent down under its own weight, its branches began to take root.

By participating in this experiment, I am helping advance knowledge.

There isn't a scientific explanation for every U.F.O. sighting.

Completing this experiment gains me credit in my psychology course.

During high school, I took mathematics courses.

The Hope Diamond was shipped from South Africa to London through the regular mail service.

The review is concerned with the first three volumes.

I can hear my own voice while I talk.

The ship was ancient and would as soon be retired from the fleet.

I have heard a number of different college lectures at this university.

Two men dressed as repairmen will appear shortly after the van pulls up.

The wood was discolored as if it had been held in a fire.

This is not the only building I have entered.

At different times in my life I have taken exams.

A light was noticed in the dark outside and it moved eerily towards the house.

During the first part of this experiment, I sat in a room with many other people.

Painting in a few other non-European countries is treated in a separate volume.

A recent study revealed that one half of all college students were unable to find summer jobs.

The school I attend is a co-ed school.

The map would prove useless as a beginning guide.

The speaker outlined a plan whereby the deficit could be eliminated.

I am seated in a chair.

In my hand I am holding an index card.

Black and white pictures are arranged in ten sections.

The voices are only at night, and whisper words, terrible words.

The papers had been front-paging it for days.

I have already completed some questionnaires in this experiment.

The organization depended on the people for support.

I sometimes use a pencil when I write.

In 1965, Elizabeth made the first state visit by a British monarch to Germany in 56 years.

The foods I eat vary in taste.

It was their sixth consecutive best-seller.

VMIP: Depression Statements

Please read each of the following statements out loud. As you look at each statement, focus your observation only on that one. The tape recording will tell you when to move on to the next card.

These statements are intended to create a certain mood. Your success at coming to experience this mood will largely depend on your willingness to accept and respond to the idea in each statement and to allow each suggestion to act upon you without resistance.

Attempt to respond to the feeling suggested by each statement. Then try to think of yourself as definitely being and moving into that mood state.

If it is natural for you to do so, try to visualize a scene in which you have had such a feeling.

If you feel the urge to laugh, it will probably be because humor is a good way to counteract unwanted feelings or it might be because you feel yourself going into that mood. Try to avoid laughing.

Today is neither better nor worse than any other day.

However, I feel a little low today.

I'm too tired and gloomy to care about anything.

Sometimes I wonder whether school is all that worthwhile.

It has occurred to me more than once that study is basically useless, because you forget almost everything you learn anyway.

I don't concentrate anymore. I just want to forget about everything.

I've had daydreams in which my mistakes kept occurring to me--sometimes I wish I could start over again.

I just can't make up my mind; it's so hard to make simple decisions.

I just don't care about anything. Life just isn't any fun.

It takes too much effort to convince people of anything. There's no point in trying.

I couldn't remember things well right now if I had to.

It's so discouraging the way people don't really listen to me.

I've doubted that I'm a worthwhile person.

It often seems that no matter how hard I try, things still go wrong.

Things are easier and better for other people than for me. I feel like there's no use in trying again.

I've noticed that no one seems to really understand or care when I complain or feel unhappy.

I'm not very alert; I feel listless and vaguely sad.

I'm uncertain about my future.

Too often I have found myself staring into the distance, my mind a blank, when I definitely should have been studying.

I'm discouraged and unhappy about myself.

Things are worse now than when I was younger.

I've lain awake at night worrying so long that I've hated myself.

I'm not sure school is helping me very much.

I'm so tired.

Some very important decisions are almost impossible for me to make.

The way I feel now, the future looks boring and hopeless.

I fail in communicating with people about my problem.

My thoughts are so slow and downcast--I don't want to think or talk.

Things are easier and better for other people than for me. I feel like there's no use in trying again.

There have been days when everything went miserably wrong.

I just can't make up my mind; it's so hard to make simple decisions.

I feel terribly weak.

Often people make me very upset. I don't like to be around them.

I fail in communication with people about my problems.

It's so discouraging the way people don't really listen to me.

I can't get people to understand me.

I've felt so alone before that I could have cried.

Everything seems to take so much effort.

I get so discouraged because I can't solve my problems.

Sometimes I've wished I could die.

I just don't care about anything. Life just isn't any fun.

Too often I have found myself staring listlessly into the distance, my mind a blank.

I just can't seem to do things right very often.

Even the simplest decisions are almost impossible for me.

I have too many bad things in my life.

I can't seem to get people to like me.

I don't concentrate or move. I just want to forget about everything.

I often feel there is something terribly wrong with me.

I feel terribly tired and indifferent to things today.

Everything seems utterly futile and empty.

My future seems bleak and hopeless.

Every now and then I feel so tired and gloomy that I'd rather just sit than do anything.

I want to go to sleep and never wake up.

All of the unhappiness of my past life is taking possession of me.

Life seems too much for me anyhow. My efforts are wasted.

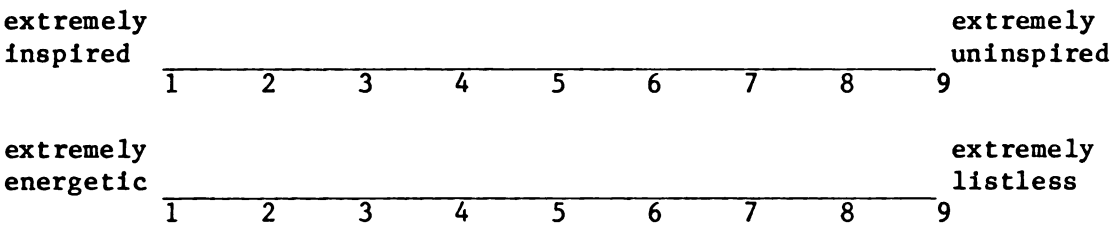
APPENDIX 2

PERSONAL FEELINGS SCALE (PFS)

(Note: PFS Depression subscale = optimistic/pessimistic + energetic/listless + reversed scores for (sad/happy + hopeless/hopeful)
Maximum score = 36

Please Indicate the Way You Are Feeling RIGHT NOW

extremely optimistic	1	2	3	4	5	6	7	8	9 extremely pessimistic
extremely sad	1	2	3	4	5	6	7	8	9 extremely happy
extremely nervous	1	2	3	4	5	6	7	8	9 extremely calm
extremely open	1	2	3	4	5	6	7	8	9 extremely closed
extremely steady	1	2	3	4	5	6	7	8	9 extremely unsteady
extremely confident	1	2	3	4	5	6	7	8	9 extremely unconfident
extremely hopeless	1	2	3	4	5	6	7	8	9 extremely hopeful
extremely angry	1	2	3	4	5	6	7	8	9 extremely serene


extremely inspired
extremely uninspired
1 2 3 4 5 6 7 8 9
extremely energetic
extremely listless
1 2 3 4 5 6 7 8 9

APPENDIX 3

MULTIPLE AFFECT ADJECTIVE CHECK LIST(MAACL)

D = depression sub-scale (Note: Maximum score = 40)
A = anxiety sub-scale (Note: Maximum score = 21)
H = hostility sub-scale (Note: Maximum score = 28)
+ = item must be checked to be counted on sub-scale
- = item must not be checked to be counted on sub-scale

Directions: On these pages you will find words which describe different kinds of moods and feelings. Mark an X beside the words which describe how you feel now - today. Some of the words may sound alike, but we want you to check all the words that describe your feelings. Work rapidly.

1. __ active(D-)	2. __ adventurous	3. __ affectionate
4. __ afraid(A+)	5. __ agitated	6. __ agreeable(H-)
7. __ aggressive	8. __ alive(D-)	9. __ alone(D+)
10. __ amiable(H-)	11. __ amused	12. __ angry(H+)
13. __ annoyed	14. __ awful(D+)	15. __ bashful
16. __ bitter(H+)	17. __ blue(D+)	18. __ bored
19. __ calm(A-)	20. __ cautious	21. __ cheerful(A-)
22. __ clean	23. __ complaining	24. __ contented(A-)
25. __ contrary	26. __ cool	27. __ cooperative

28. __ critical	29. __ cross	30. __ cruel(H+)
31. __ daring	32. __ desperate(A+)	33. __ destroyed(D+)
34. __ devoted	35. __ disagreeable(H+)	36. __ discontented(H+)
37. __ discouraged(D+)	38. __ disgusted(H+)	39. __ displeased
40. __ energetic	41. __ enraged(H+)	42. __ enthusiastic(D-)
43. __ fearful(A+)	44. __ fine(D-)	45. __ fit(D-)
46. __ forlorn(D+)	47. __ frank	48. __ free(D-)
49. __ friendly(H-)	50. __ frightened(A+)	51. __ furious(H+)
52. __ gay(D-)	53. __ gentle	54. __ glad(D-)
55. __ gloomy(D+)	56. __ good(D-)	57. __ good-natured(H-)
58. __ grim	59. __ happy(A-)	60. __ healthy(D-)
61. __ hopeless(D+)	62. __ hostile	63. __ impatient
64. __ incensed	65. __ indignant	66. __ inspired(D-)
67. __ interested(D-)	68. __ irritated(H+)	69. __ jealous
70. __ joyful(A-)	71. __ kindly(H-)	72. __ lonely(D+)
73. __ lost(D+)	74. __ loving(A-)	75. __ low(D+)
76. __ lucky(D-)	77. __ mad(H+)	78. __ mean(H+)
79. __ meek	80. __ merry(D-)	81. __ mild
82. __ miserable(D+)	83. __ nervous(A+)	84. __ obliging
85. __ offended(H+)	86. __ outraged(H+)	87. __ panicky(A+)
88. __ patient	89. __ peaceful(D-)	90. __ pleased
91. __ pleasant(A-)	92. __ polite(H-)	93. __ powerful

94. __ quiet	95. __ reckless	96. __ rejected(D+)
97. __ rough	98. __ sad(D+)	99. __ safe(D-)
100. __ satisfied	101. __ secure(A-)	102. __ shaky(A+)
103. __ shy	104. __ soothed	105. __ steady(A-)
106. __ stubborn	107. __ stormy(H+)	108. __ strong(D-)
109. __ suffering(D+)	110. __ sullen	111. __ sunk(D+)
112. __ sympathetic(H-)	113. __ tame(H-)	114. __ tender
115. __ tense(A+)	116. __ terrible(D+)	117. __ terrified(A+)
118. __ thoughtful(A-)	119. __ timid	120. __ tormented(D+)
121. __ understanding(H-)	122. __ unhappy(D+)	123. __ unsociable
124. __ upset(A+)	125. __ vexed(H+)	126. __ warm
127. __ whole(D-)	128. __ wild	129. __ willful(H-)
130. __ wilted(D+)	131. __ worrying(A+)	132. __ young(D-)

APPENDIX 4

BECK DEPRESSION INVENTORY (BDI)

(Note: Maximum score = 63)

INSTRUCTIONS: In each of the following groups of statements, circle the one statement which best describes how you feel RIGHT NOW.

1.
1) I do not feel sad
2) I feel blue or sad.
3) I am blue or sad all the time and I can't snap out of it.
4) I am so sad or unhappy that it is quite painful.
5) I am so sad or unhappy that I can't stand it.

2.
1) I am not particularly pessimistic or discouraged about the future.
2) I feel discouraged about the future.
3) I feel I have nothing to look forward to.
4) I feel that I won't ever get over my troubles.
5) I feel that the future is hopeless and that things cannot improve.

3.
1) I do not feel like a failure.
2) I feel that I have failed more than the average person.
3) I feel I have accomplished very little that is worthwhile or that means anything.
4) As I look back on my life all I can see is a lot of failures.
5) I feel I am a complete failure as a person (parent, wife, daughter, etc.)

4.
1) I am not particularly dissatisfied.
2) I feel bored most of the time.
3) I don't enjoy things the way I used to.
4) I don't get satisfaction out of anything any more.
5) I am dissatisfied with everything.

5.
1) I don't feel particularly guilty.
2) I feel bad or unworthy a good part of the time.
3) I feel quite guilty.
4) I feel bad or unworthy practically all the time now.
5) I feel as though I am very bad or worthless.

6.
1) I don't feel I am being punished.
2) I have a feeling that something bad may happen to me.
3) I feel I am being punished or will be punished.
4) I feel I deserve to be punished.
5) I want to be punished.

7.
1) I don't feel disappointed in myself.
2) I am disappointed in myself.
3) I don't like myself.
4) I am disgusted with myself.
5) I hate myself.

8.
1) I don't feel I am any worse than anybody else.
2) I am critical of myself for my weaknesses or mistakes.
3) I blame myself for my faults.
4) I blame myself for everything bad that happens.

9.
1) I don't have any thoughts of harming myself.
2) I have thoughts of harming myself but I would not carry them out.
3) I feel I would be better off dead.
4) I feel my family would be better off if I were dead.
5) I have definite plans about committing suicide.
6) I would kill myself if I could.

10.
1) I don't cry any more than usual.
2) I cry now more than I used to.
3) I cry all the time now. I can't stop it.
4) I used to be able to cry but now I can't cry at all even though I want to.

11.
1) I am no more irritated now than I ever am.
2) I get annoyed or irritated more easily than I used to.
3) I feel irritated all the time.
4) I don't get irritated at all at the things that used to irritate me.

12.
1) I have not lost interest in other people
2) I am less interested in other people now than I used to be.
3) I have lost most of my interest in other people and have little feeling for them.
4) I have lost all my interest in other people and don't care about them at all.

13.
1) I make decisions about as well as ever.
2) I try to put off making decisions.
3) I have great difficulty in making decisions.
4) I can't make any decisions at all any more.

14.
1) I don't feel I look any worse than I used to.
2) I am worried that I am looking old or unattractive.
3) I feel that there are permanent changes in my appearance and they make me look unattractive.
4) I feel that I am ugly or repulsive looking.

15.
1) I can work about as well as before.
2) It takes extra effort to get started at doing something.
3) I don't work as well as I used to.
4) I have to push myself very hard to do anything.
5) I can't do any work at all.

16.
1) I can sleep as well as usual.
2) I wake up more tired in the morning than I used to.
3) I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
4) I wake up early every day and can't get more than 5 hours sleep.

17.
1) I don't get any more tired than usual.
2) I get tired more easily than I used to.
3) I get tired from doing anything.
4) I get too tired to do anything.

18.
1) My appetite is no worse than usual.
2) My appetite is not as good as it used to be.
3) My appetite is much worse now.
4) I have no appetite at all any more.

19.
1) I haven't lost much weight, if any, lately.
2) I have lost more than 5 pounds.
3) I have lost more than 10 pounds.
4) I have lost more than 15 pounds.

20.
1) I am no more concerned about my health than usual.
2) I am concerned about aches and pains or upset stomach or constipation.
3) I am so concerned with how I feel or what I feel that it's hard to think of much else.
4) I am completely absorbed in what I feel.

21.
1) I have not noticed any recent changes in my interest in sex.
2) I am less interested in sex than I used to be.
3) I am much less interested in sex now.
4) I have lost interest in sex completely.

APPENDIX 5

ATTRIBUTION STYLE QUESTIONNAIRE (ASQ)

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 the 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 the other person or circumstances	1	2	3	4	5	6	7	Totally due to me

3) In the future when you are with your friends, will this cause again be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

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

5) 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 HAVE BEEN LOOKING FOR A JOB UNSUCCESSFULLY FOR SOME TIME.

6) Write down the 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 other people or circumstances	1	2	3	4	5	6	7	Totally due to me

8) In the future when looking for a job, will this cause again be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

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 BECOME VERY RICH.

11) Write down the one major cause_____

12) Is the cause of your becoming rich due to something about you or something about other people or circumstances?

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

13) In your financial future, will this cause again be present?

Will never again be present	1	2	3	4	5	6	7	Will always be present

14) Is the cause something that just affects obtaining money or does it also influence other areas of your life?

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?

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 be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

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 REACTS 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 be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

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 A PROJECT WHICH IS HIGHLY PRAISED.

26) Write down the one major cause_____

27) Is the cause of being praised due to something about you or something about the other people or circumstances?

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

28) In the future when doing a project, will this cause again be present?

Will never again be present	1	2	3	4	5	6	7	Will always be present

29) Is this cause something that just affects doing projects or does it also influence other areas of your life?

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

30) How important would this situation be if it happened to you?

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

YOU MEET A FRIEND WHO ACTS HOSTILELY TOWARD YOU.

31) Write down the one major cause_____

32) Is the cause of your friend acting hostile 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

33) In the future when interacting with friends, will this cause again be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

34) Is the cause something that just influences interacting with friends 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

35) 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 CAN'T GET ALL THE WORK DONE THAT OTHERS EXPECT OF YOU.

36) Write down the one major cause_____

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 cause be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

39) Is the cause something that just affects doing work that others expect of you 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

YOUR SPOUSE (BOYFRIEND/GIRLFRIEND) HAS BEEN TREATING YOU MORE LOVINGLY.

41) Write down the one major cause_____

42) Is the cause of your spouse (boyfriend/girlfriend) treating you more lovingly due to something about you or something about other people or circumstances?

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

43) In future interactions with your spouse (boyfriend/girlfriend) will this cause again be present?

Will never again be present	1	2	3	4	5	6	7	Will always be present

44) Is this cause something that just affects how your spouse (boyfriend/girlfriend) treats you or does it also influence other areas of your life?

Influences 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?

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 the 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 be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

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 be present? (Circle one number)

Will never again be present	1	2	3	4	5	6	7	Will always be present

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 GET A RAISE.

56) Write down the one major cause_____

57) Is the cause of your getting a raise due to something about you or something about other people or circumstances?

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

58) In the future on your job, will this cause again be present?

Will never again be present	1	2	3	4	5	6	7	Will always be present

59) Is this cause something that just affects getting a raise or does it also influence other areas of your life?

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?

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